

**Transactions between Family Psychosocial Stressors and Externalizing Symptoms  
from Infancy to Adolescence: Interactions with Gender and Self-regulation**

by

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## Chapter I

### Externalizing Symptoms across Infancy to Young Adulthood

Externalizing symptoms such as aggression and delinquency characterize serious behavioral problems and psychiatric disorders that disproportionately affect a wide age-range of young people (Achenbach, Howell, Quay, & Conners, 1991; American Psychiatric Association, 2000). Antisocial behavior problems, conduct problems, and disruptive behavior problems are all subsumed under the umbrella-term, *externalizing symptoms* (Achenbach et al., 1991; Yoshikawa, 1994). This broadband syndrome overlaps both conceptually and empirically with these various forms of behavioral problems such that they share many similar risk factors and effective interventions (Farrington, 2009; Schulenberg & Zarrett, 2006). While we know a great deal about the risk factors that contribute to these classifications of behavioral problems, referred to broadly as *externalizing symptoms* throughout this chapter, our understanding of their bidirectional effects with children and adolescents' externalizing symptoms is lagging far behind (Dodge & Pettit, 2003; Hinshaw, 2002). Thus, we are substantially lacking in etiological knowledge regarding how youths contribute to their adjustment problems through reciprocal exchanges with their social environments. Integrating a risk factor approach with a developmental perspective can help advance our understanding of the etiology and development of externalizing symptoms. More specifically, a transactional framework from developmental psychology provides a better means of establishing the



direction of causality between environmental risk factors and youth externalizing symptoms than typical unidirectional approaches (e.g., Sameroff & Chandler, 1975). Furthermore, integrating tests of transactions with interactions of individual characteristics such as gender can identify groups of youths who are at elevated risk of developing externalizing symptoms when facing environmental stressors.

In this chapter, I begin by discussing widespread costs of externalizing symptoms in the U.S. I then elucidate the etiology and development of externalizing symptoms by reviewing their risk factors and pathways. Next, I describe how a developmental perspective using a transactional framework can clarify understanding of bidirectional processes through which risk factors in the social environment and the individual reciprocally contribute to the development of their adjustment problems, with a focus on prevalent family psychosocial stressors and externalizing symptoms that typically peak in early phases of the lifespan. I then discuss how combining this transactional approach with tests of interactions of child gender and self-regulation may reveal individual differences in risk for developing externalizing symptoms in the presence of family psychosocial stressors. I present a conceptual model to illustrate this integrative approach (see Figure 1). Lastly, I describe three distinct longitudinal studies presented in subsequent chapters (Chapter II, III, and IV) that incorporate transactional and interactional approaches to investigate bidirectional effects between family psychosocial stressors and externalizing symptoms, and moderating effects of child gender and self-regulation. These three studies are summarized in an integrative fashion in Chapter V and discussed in regards to implications for advancing knowledge of externalizing symptoms and their prevention and intervention. As described below, steep costs associated with

externalizing symptoms across the lifespan necessitate a better understanding of their etiology and development.

### **The Societal Costs of Externalizing Symptoms**

Heightened levels of externalizing symptoms among children and adolescents that are severe enough to warrant lawful apprehension and/or clinical referral are quite prevalent in the U.S. and accrue major fiscal and health costs (Dodge & Pettit, 2003; Loeber & Farrington, 2000; Yoshikawa, 1994). According to the Office of Juvenile Justice and Delinquency Prevention, over 2.1 million youths under the age of 18 were arrested in the U.S. in 2008 (Puzzanchera, 2009). It is estimated that a typical youth who has a high risk of becoming a career criminal has about six altercations with police and 23 offenses by 18-years-of-age, and costs society between \$4.2 and \$7.2 million in his lifetime due to direct costs of illegal acts and indirect costs from loss of productivity and wages (Cohen & Piquero, 2009). These high-risk youths collectively account for approximately 50% of all U.S. crime throughout their lives. This disproportionate figure is truly significant given that the total estimated cost of crime in the U.S. exceeds \$1 trillion per year (Dodge, 2008).

Youths with externalizing symptoms also accrue a disproportionate amount of costs across many welfare agencies and health services. For example, the majority of clinical referrals for child and adolescent mental health services are for externalizing symptoms and their associated problems (Dodge & Pettit, 2003; Loeber & Farrington, 2009). Child and adolescent psychiatric illnesses such as attention-deficit/hyperactivity disorder and conduct disorder reflect core problems with externalizing symptoms (APA, 2000). Adults with a history of significant externalizing symptoms in their youth also are

at elevated risk for violence, criminality, diminished educational achievement, financial problems, work problems, unemployment, divorce, accidents and injury, drug and alcohol dependence, and other physical and mental health problems throughout their lifetimes (Loeber & Farrington, 2000; Macmillan & Hagan, 2004; Moffitt, Caspi, Harrington, & Milne, 2002; Odgers et al., 2008; Patterson, DaBaryshe, & Ramsey, 1989; Yoshikawa, 1994).

Substantial direct and indirect costs of externalizing symptoms to individuals and society, as well as research demonstrating that their origins begin early in the lifespan, have encouraged the scientific community to examine younger populations to identify early developmental precursors and processes of externalizing symptoms (Cohen & Piquero, 2009; Dodge & Pettit, 2003; Loeber & Farrington, 2000; Patterson et al., 1989; Yoshikawa, 1994). In the following, I review a broad range of risk factors associated with externalizing symptoms and explicate pathways children and adolescents follow when developing externalizing symptomatology.

### **The Etiology and Development of Externalizing Symptoms**

Researchers have identified an immense constellation of risk factors and pathways to externalizing symptoms that are too numerous to summarize in this chapter (e.g., Dodge & Pettit, 2003). To create a more manageable review, I highlight psychosocial risk factors, and proceed through Bronfenbrenner's ecological systems model (1994), beginning with the most distal risk factors found in the social-cultural milieu and then at the community-level to more proximal risk factors found in close interpersonal relationships and then those housed within the individual. I also discuss proximal influences as experts agree that externalizing symptoms mainly originate from risk

factors either mediated through or residing within the family and child (Dodge & Pettit, 2003; Farrington, 2009; Loeber & Farrington, 2000; Moffit & Caspi, 2001). Throughout this review to emphasize the usefulness of a transactional–developmental perspective to the study of externalizing symptom etiology and development, I specify relations between risk factors and their associated externalizing symptoms, their respective phases of ontogeny, and their overlap with risk factors across system levels.

**Social-cultural risk factors.** Societies contribute to children and adolescents' externalizing symptoms through violence exposure in mass media, normative beliefs, and cultural values. Exposure to violence in television, film, video games, and news outlets increases aggression and violent behavior in both the short- and long-term across the lifespan. Mass media violence exposure helps create cognitive and emotional structures in young people that affect perceptions of and attitudes toward violence that increase risk for externalizing symptoms (Huesmann & Taylor, 2006; Tremblay, 2000). Youths raised in societies that deem some acts of violence as justifiable or instrumental are more likely to assimilate attitudes toward use of violence as an acceptable problem-solving response, which can increase violent behavior throughout childhood and adolescence (Andreas & Watson, 2009; Dodge & Pettit, 2003).

Societal influences on attitudes and beliefs also account for regional differences in rates of homicide and other violent crimes in the U.S. For example, in comparison to Northerners in the U.S., Southerners have more favorable attitudes toward use of violence as an appropriate response to insult, as a means of self-protection, and as a socializing tool to discipline children (Dodge & Pettit, 2003; Nisbett, 1993). Experiments have demonstrated that White Southerners are more sensitive to provocation, become

angrier in response to insult, and are more primed for violent retaliation than White Northerners. A culture of honor, rooted in the herding livelihood of their European ancestry, is believed to be the distal cause of White Southerners' heightened proclivity to violence when threatened, and why the rural South is more violent than socioeconomically similar regions in the North (Nisbett, 1993).

Societal norms regarding appropriate uses of violence also are correlated with other social risk factors for externalizing symptoms. Adults who have positive attitudes toward violence are more likely to support gun ownership for protective purposes (Nisbett, 1993). Cultural values, such as the constitutional right to keep and bear arms, have shaped societal norms that increase young peoples' risk of violent behavior by directly influencing more proximal risk factors at the community- and neighborhood-level. For example, relatively lax gun control policies and the wide availability of firearms in the U.S. have contributed to violent offending among American youth (Dodge & Pettit, 2003). Child delinquents frequently use weapons when they commit crimes and half of all murders committed by children are with guns (Loeber & Farrington, 2000). In addition to adding to the availability of deadly weapons, cultural norms regarding appropriate uses of violence influence adults' attitudes toward use of physical discipline when children misbehave (Nisbett, 1993). The cultural normativeness of physical discipline use, in turn, moderates how negative these experiences are on children's adjustment problems (Lansford et al., 2005). Children who perceive physical discipline as a normative parenting technique are less likely to manifest elevated aggression when they experience it than children who do not view it as a common practice. Thus, social-

cultural risk factors influence children's externalizing symptoms by altering more proximal risks in the home and neighborhood (Dodge & Pettit, 2003; Farrington, 2009).

**Community and neighborhood risk factors.** Many significant community-level risk factors for externalizing symptoms are correlates of severe economic disadvantage (Yoshikawa, 1994). Risk factors for children's aggression and adolescents' chronic conduct problems include living in a crowded urban area, high residential mobility, low social cohesion, high adult unemployment rates, and poor quality housing (Chung & Steinberg, 2006; Dodge & Pettit, 2003; Farrington, 2009; Loeber & Farrington, 2000). Correlates of poverty characterize neighborhoods that are elevated in crime and disproportionately populated by residents with serious externalizing symptoms. Such pervasive effects of poverty potentiate risk factors for child externalizing symptoms such as heightened rates of violent crime (Aisenberg & Herrenkohl, 2008).

Exposure to community violence is a major risk factor for adolescents' physical aggression and violent behavior that frequently occurs in impoverished communities (Gorman-Smith, Henry, & Tolan, 2004; Gorman-Smith & Tolman, 1998; McLoyd, 1998; Ozer, 2005). Whereas adolescents often develop externalizing symptoms in response to community violence, young children tend to react with greater psychological distress and trauma (Fowler, Tompsett, Braciszewski, Jacques-Tiura, & Baltes, 2009). Violence exposure, however, predicts increases in older children's normative beliefs favoring aggression, which indirectly contribute to later aggression and violent behavior (Guerra, Huesmann, & Spindler, 2003). More often than in neighborhoods, youths frequently experience interpersonal violence in schools (Slovak, Carlson, & Helm, 2007).

Large urban schools in impoverished areas are more likely to have classrooms with high levels of peer aggression and violence (Thomas, Bierman, & The Conduct Problems Prevention Research Group, 2006). Attending a school with high levels of student aggression and delinquency elevates risk for child aggression and adolescent delinquency (Farrington, 2009; Thomas et al., 2006). Moreover, exposure to violent classmates can influence youths' own attitudes toward and use of violence (Dodge & Pettit, 2003). It is not clear, however, whether the structure and practices of schools constitute causal risk factors for externalizing symptoms or merely reflect their composition of students who have significant behavioral problems (Farrington, 2009). Hence, neighborhood-level risks also indirectly contribute to youths' delinquent behavior by elevating peer deviance and their collective offending (Chung & Steinberg, 2006).

**Risk factors in peer relationships.** Peer rejection in early childhood and deviant peer influences in childhood and adolescence are particularly salient risk factors for externalizing symptoms (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Dishion, Piehler, & Myers, 2008; Farrington, 2009). Young children who begin socializing with peers confront challenges inhibiting aggressive impulses and negative emotions (Calkins, 2009; Olson, Sameroff, Kerr, & Lunkenheimer, 2009; Rubin, Bukowski, & Parker, 2006). Failure adapting to stage-salient tasks of early childhood can lead to rejection from conventional and prosocial peers and increase levels of externalizing symptoms such as aggression (Dodge & Pettit, 2003; Patterson et al., 1989). Aggressive and disruptive children tend to be disliked by many peers, but they can be popular among aggressive and nonaggressive youths such as bullies (Dishion et al., 2008; Farrington, 2009).

Dishion and colleagues (2008) propose that early peer rejection contributes to the formation of deviant groups of similarly rejected and aggressive youths. Young children who develop chronic externalizing symptoms acquire suboptimal prosocial skills to develop and maintain positive interpersonal relationships, but they are better at engaging in deviant conversations with peers who share similar unconventional attitudes and interests. Friendships can emerge through deviancy training among unconventional youths who reinforce each other's antisocial tendencies (Dodge & Pettit, 2003). This process is more pronounced during the transition to adolescence, when peers become increasingly salient socializing agents (Rubin et al., 2006). Deviant peer influences are among the most potent social risk factors for adolescents' antisocial behavior problems (Dishion et al., 2008; Dodge, Dishion, & Lansford, 2006). Most crimes after age 17 are committed alone, but delinquent acts in adolescence are typically committed among peer groups. Peer delinquency is strongly correlated with adolescents' delinquency and violent behavior, but parenting factors such as parental supervision, reinforcement, and involvement in adolescents' lives appear to be critical influences on adolescents' externalizing symptoms (Farrington, 2009; Zimmerman, Steinman, & Rowe, 1998). Deviant peer influences, furthermore, are less likely to have an enduring influence on youths' externalizing symptoms unless exposure is stable and frequent (Bronfenbrenner & Morris, 2006; Dodge & Pettit, 2003; Thomas et al., 2006). Family members often are more consistently present in youths' lives and earlier on, which suggests that family risk factors for externalizing symptoms take precedence over deviant peer influences.

**Risk factors in family relationships.** Coercive family interactions are central to the etiology of externalizing symptoms, such as young children's disruptive behavior and



adolescents' antisocial behavior problems (Compton, Snyder, Shrepferman, Bank, & Shortt, 2003; Loeber & Farrington, 2000; Moffitt & Caspi, 2001; Patterson et al., 1989). Major family psychosocial stressors for children and adolescents' externalizing symptoms can be categorized into three areas: parenting, parental psychopathology, and family conflict. Parenting characterized as being less sensitive and responsive to youths' social-emotional needs predict increases in their externalizing symptoms (Rothbaum & Weisz, 1994). For example, consistently low maternal warmth is linked with more externalizing behavior problems in early childhood (Eisenberg, Zhou, Spinrad, Valiente, Fabes, & Liew, 2005; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005). Low levels of parental approval, guidance, supervision, involvement, and warmth and high levels of coercive and authoritarian parenting predict externalizing symptoms in young people (Deater-Deckard et al., 1998; Olson, Ceballo, & Park, 2002; Patterson et al., 1989; Rothbaum & Weisz, 1994). Unresponsive parenting that communicates parents' lack of interest in and acceptance of their children worsens externalizing behavior problems in childhood (Campbell, Shaw, & Gilliom, 2000) and antisocial behavior problems in adolescence (Loeber & Farrington, 2000; Moffitt & Caspi, 2001). The ways parents respond to children's behavioral problems can further exacerbate behavioral problems.

Parental discipline is another domain of parenting associated with children's externalizing symptoms (Gershoff, 2002; Kerr, Lopez, Olson, & Sameroff, 2004; Olson et al., 2002). Harsh and power-assertive forms of punitive discipline, such as spanking with a hand or object, increase risk of externalizing behavior problems, and sometimes constitute abuse, which also predicts later antisocial behavior problems (Deater-Deckard et al., 1998; Gershoff, 2002; Keiley, Howe, Dodge, Bates, & Pettit, 2001). In contrast,

inductive discipline provides young children with clear rules, explanation of consequences, and reasoning to elicit understanding from children about proper conduct, so they internalize rules and learn prosocial skills (Hart, DeWolf, Wozniak, & Burts, 1992; Pettit, Bates, & Dodge, 1997). Low levels of inductive discipline predict more externalizing behavior problems and less prosocial behavior in preschoolers (Hart et al., 1992) and middle school children (Krevans & Gibbs, 1996; Pettit et al., 1997). Ineffective discipline and insensitive and unresponsive parenting are especially problematic in families characterized by elevated levels of mental health problems (Berg-Nielsen, Vikan, & Dahl, 2002; Connell & Goodman, 2002).

Parental psychopathology, particularly maternal depression, is a critical contributor to parenting deficits and negative parent–child interactions (Berg-Nielsen et al., 2002). Both clinical and subclinical levels of maternal depressive symptoms are risk factors for children and adolescents’ externalizing symptoms (Cummings, Keller, & Davies, 2005; Du Rocher Schudlich & Cummings, 2007; Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007; Gartstein & Fagot, 2003; Weinfeld, Ingerski, & Moreau, 2009). Mothers who have elevated depressive symptoms demonstrate more parenting deficits and dysfunctional interactions with young children that contribute to externalizing behavior problems (Chronis et al., 2007; Goodman & Gotlib, 1999; Lovejoy, Graczyk, O’Hare, & Neuman, 2000). Younger children, such as infants and toddlers, are at greatest risk of developing externalizing symptoms when exposed to maternal depression due to their substantial dependence on caregivers and their greater likelihood of experiencing adverse parenting (Beardslee, Bemporad, Keller, and Klerman, 1983; Connell & Goodman, 2002; Cummings & Davies, 1994). Antisocial

parents and delinquent siblings also increase risk for externalizing symptoms, as well as other correlates of coercive family interactions, such as marital and family conflict (Compton et al., 2003; Cummings et al., 2005; Dodge & Pettit, 2003; Loeber & Farrington, 2000; Patterson et al., 1989).

Family and marital conflict, domestic violence, and parental divorce and separation exacerbate children and adolescents' externalizing symptoms (Dodge & Pettit, 2003; Farrington, 2009; Patterson et al., 1989). Domestic violence and parents' violent attitudes further contribute to youths' favorable attitudes toward violence and their later use of it (Slovak et al., 2007; Solomon, Bradshaw, Wright, & Cheng, 2008). Family conflict peaks in frequency during early adolescence and is characterized by more intense anger than interpersonal conflict at other ages; however, consistent high-intensity fighting among family is not normative during adolescence (Collins & Steinberg, 2006; Laursen, Coy, & Collins, 1998). Family conflict predicts increases in adolescents' violent behavior and chronic antisocial behavior problems (DuRant, Cadenhead, Pendergrast, Slavens, & Linde, 1994; Odgers et al., 2008; Paschall, Ennett, & Flewelling, 1996). Continuous instability in the family environment is associated with increases in aggression during late childhood and adolescence and elevated risks of violence and criminal behavior in adulthood (Bronfenbrennar & Morris, 2006). Further, community-related stressors such as neighborhood violence, disorganization, and poverty indirectly contribute to delinquency by exacerbating family risks (Chung & Steinberg, 2006; Yoshikawa, 1994).

Family sociodemographic risk factors at birth such as low socioeconomic status (SES) are some of the most consistent and robust predictors of externalizing symptoms throughout childhood and adolescence (Dodge & Pettit, 2003; Farrington, 2009;

Patterson et al., 1989). Family SES is often measured by household income and parents' highest level of education and occupational status (Hollingshead, 1979). Low levels of SES are associated with more intense risk factors for externalizing symptoms like harsh physical discipline (Deater-Deckard et al., 1998; Gershoff, 2002) and maternal depression (Cummings & Davies, 1994; Lovejoy et al., 2000). In addition, effects of SES on externalizing symptoms are mediated through family socializing mechanisms, such as poor child rearing, and parental psychopathology (Belsky, 1984; Campbell, Matestic, von Stauffenber, Mohan, & Kirchner, 2007; Campbell et al., 2000; Dodge & Pettit, 2003; Farrington, 2009; Yoshikawa, 1994). Thus, risk factors from distal ecological systems such as community stressors can compromise proximal interactions and relationships.

This review has to this point focused on risk factors and correlates of children and adolescents' externalizing symptoms emanating from their social ecologies. In the following, I review several individual factors that may combine with psychosocial risk factors to increase children's risk for externalizing symptoms.

**Risk factors residing within the individual.** One of the earliest identified child-centered risk factors for externalizing symptoms is an early pattern of difficult temperament often described as frequently irritable, fussy, undercontrolled, resistant, and difficult to soothe (Calkins, 2009; Compas & Reeslund, 2009; Deater-Deckard et al., 1998; Dodge & Pettit, 2003; Farrington, 2009; Loeber & Farrington, 2000).

Temperament is defined as constitutionally-based individual differences in reactivity and self-regulation (Posner & Rothbart, 2000; Rothbart & Bates, 1998). *Reactivity* refers to an individual's positive and negative emotional arousal to stress, whereas *self-regulation* is the ability to modulate that physiological arousal and inhibit emotional and behavioral

impulses. Thus, temperament influences cognitive, emotional, and behavioral responses to stress that are instrumental to children's early coping and psychosocial adjustment (Compas & Reeslund, 2009; Lengua, 2002; Rothbart & Bates, 1998). Research linking early temperament with externalizing symptoms has produced mixed results (Calkins, 2009; Yoshikawa, 1994). For example, Moffitt and Caspi (2001) found that an early undercontrolled temperament differentiated males and females who demonstrated either child-onset or adolescent-onset antisocial behavior problems. In contrast, Aguilar, Sroufe, Egeland, and Carlson (2000) found that child- and adolescent-onset groups were distinguished by psychosocial stressors in early childhood such as elevated life stress, physical abuse, and neglect, rather than temperament characteristics. Further investigation of temperament influences is needed to resolve this inconsistency.

One promising approach has been investigating links between more clearly defined components of temperament and children's adjustment problems. Specific constituents of temperament are more consistently associated with elevated risk for children's externalizing symptoms (Lengua, 2002; Lengua, 2006; Rothbart & Bates, 1998). For example, deficient levels of self-regulation are associated with more externalizing behavior problems across early childhood to adolescence (Eisenberg et al., 2005; Gartstein & Fagot, 2003; Martel & Nigg, 2006; Olson et al., 2005). Furthermore, low levels of self-regulation amplify adverse effects of environmental risk factors on children's externalizing symptoms (Lengua, Bush, Long, Kovacs, & Trancik, 2008). Another child characteristic that predicts both elevated externalizing symptoms and suboptimal self-regulation is gender (Eiden, Edwards, & Leonard, 2007; Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006).

Gender is consistently associated with variations in externalizing severity, manifestation, etiology and development. Gender differences in disruptive behavior appear after age 2 as boys become more aggressive than girls through early childhood (Loeber & Farrington, 2000; Tremblay, 2000). A greater proportion of girls than boys show little or no aggression during childhood, but moderate and high levels of aggression can be found in both genders (Campbell, Spieker, Vandergrift, Belsky, Burchinal, & The NICHD Early Child Care Research Network, 2010). The most aggressive girls, however, are not nearly as violent as the most aggressive boys. Heightened aggression in boys and girls during childhood is predicted by sociodemographic risk factors and maternal harshness in the preschool years, but low maternal sensitivity in early childhood also predicts more aggression in girls (Campbell et al., 2010). Girls show more indirect and verbal forms of aggression in early and middle childhood (Côté, Vaillancourt, Leblanc, Nagin, & Tremblay, 2007; Dodge & Pettit, 2003; Tremblay, 2000), but boys receive higher ratings of aggression and externalizing behavior problems from peers and teachers in middle childhood (Deater-Deckard et al., 1998). Although boys manifest more severe externalizing symptoms, girls demonstrate the same range of symptomatology and are affected by similar risk factors, but in varying ways that lead to modest differences in the development of externalizing symptoms (Campbell et al., 2010; Daigle, Cullen, & Wright, 2007; Gorman-Smith & Loeber, 2005).

Distinct gender differences in the course and correlates of delinquent behavior also have been found (Kroneman, Loeber, & Hipwell, 2004). Boys are much more likely than girls to demonstrate early-onset delinquency (Kroneman et al., 2004; Loeber & Farrington, 2000) and life-course-persistent antisocial behavior problems (Moffitt &

Caspi, 2001; Odgers et al., 2008), although some evidence is inconsistent (Aguilar et al., 2000; van Lier, Wanner, & Vitaro, 2007). Female delinquency, which is more likely to occur in the home, is more related to a lack of parental monitoring and heightened family conflict, whereas male delinquency is more associated with early self-regulatory difficulties and neighborhood and peer risk factors (Daigle et al., 2007; Gorman-Smith & Loeber, 2005; Kroneman et al., 2004). Again, some findings are inconsistent with one another, but the general conclusion is that there are similar etiological pathways for girls and boys' externalizing symptoms with slight deviations in their severity, course, and sensitivity to certain risks factors. It is clear that gender is a critical individual characteristic that researchers should account for when investigating externalizing symptoms. As shown below, however, the strongest child-centered predictor of externalizing symptoms is earlier behavioral risk factors.

The single best predictor of chronic aggression in adolescence and adulthood is aggressive behavior in childhood (Huesmann, Eron, & Dubow, 2003; Huesmann & Taylor, 2006). Likewise, early emergence of elevated externalizing symptoms in toddlerhood or preschool is a robust predictor of externalizing symptoms in later childhood and adolescence (Campbell et al., 2010; Campbell et al., 2000). However, most young children who demonstrate some externalizing behavior do not progress to more serious and pervasive behavioral problems (Olson et al., 2009). About half of preschool-age children who manifest early externalizing symptoms stop demonstrating them after they enter school; however, early externalizing symptoms coupled with temperament-related difficulties and elevated family stress predict more serious and chronic school-age externalizing behavior problems (Campbell et al., 2010; Campbell et al., 2000; Olson et

al., 2005; Patterson et al., 1989). The majority of children who demonstrate externalizing behavior show a gradual decrease in symptoms from ages 2 to 11 (Côté, Vaillancourt, Leblanc, Nagin, & Tremblay, 2006; Farrington, 2009; Shaw, Gilliom, Ingoldsby, & Nagin, 2003). Some transitory externalizing symptoms are normative in early childhood, but these become clinically significant problems when they begin occurring across settings and interfere with age-appropriate gains in social-emotional functioning (Campbell et al., 2000; Hinshaw, 2002). Understanding and preventing the early-onset of externalizing symptoms are especially critical because they are more difficult to treat once at a diagnosable level in older children and adolescents (Campbell et al., 2000; Dodge & Pettit, 2003; Loeber & Farrington, 2000).

Children with school-age externalizing behavior problems are at increased risk of becoming child and adolescent delinquents and developing chronic and violent antisocial behavior problems in late adolescence and adulthood (Campbell et al., 2000; Deater-Deckard et al., 1998; Loeber & Farrington, 2000; Moffitt & Caspi, 2001). Consensus among experts indicates at least two distinct trajectory groups for antisocial behavior problems: an adolescent-onset group and a child-onset or life-course-persistent group (Aguilar et al., 2000; Farrington, 2009; Moffitt et al., 2002; Odgers et al., 2008; van Lier et al., 2007). Only a small subgroup of children develop life-course-persistent antisocial behavior problems, but they commit disproportionate amounts of serious crime and violence as adolescents and adults. A trend observed in the general population, called the *age-crime curve*, illustrates a rapid increase in illegal behavior during adolescence that peaks at ages 16 and 17 and is followed by an equally rapid decrease into young adulthood (Dodge & Pettit, 2003; Tremblay, 2000). Children who have life-course-



persistent antisocial behavior problems continue offending past the vast majority of their peers, and as adults account for much more than their fair share of societal problems (Dodge, 2008; Dodge & Pettit, 2003; Loeber & Farrington, 2000; Macmillan & Hagan, 2004; Moffitt et al., 2002; Yoshikawa, 1994). Given substantial evidence of early externalizing symptoms predicting later behavioral problems, their previous manifestation constitutes a major risk factor for their continuation and escalation.

In summary, risk factors for externalizing symptoms are ubiquitous across ecological systems and ontogenetic development. Social-cultural influences through mass media and societal norms directly influence children's understanding and engagement in physical aggression and violence, as well as indirectly through parents' use of physical discipline. Cultural values favoring some uses of violence indirectly affect children's externalizing symptoms through public policies that elevate risks at the levels of the community and neighborhood. Lax gun control laws in the U.S., the wide availability of firearms, and the alarming rates of firearm-related homicides involving children illustrate this association. Further at the community-level, correlates of poverty serve as risk factors for externalizing symptoms by influencing neighborhood characteristics and residents through socioeconomic pressures, violence exposure, and rampant crime. Within these societal-contexts, peers and family members play a direct role in escalating children's externalizing symptoms; however, many of the risk factors emanating from these interpersonal relationships are exacerbated by distal stressors found at the community-level and higher. Societal pressures often diminish the quality of children's interpersonal interactions and relationships with others by contributing to peer deviancy, negative parenting, parental psychopathology, and family conflict. Finally at the

individual-level, children possess a host of risk factors that make it more difficult for them to successfully traverse many challenges found in their social ecologies. Individual characteristics such as gender and self-regulation alter negative effects of environmental risk factors leading to variations in the manifestation and course of externalizing symptoms. In some cases, externalizing symptoms are more likely to emerge at an early age due to additional risks associated with children's constitutions. Collectively, risk factors for externalizing symptoms are pervasive both in their presence in children's lives and their effects on development throughout the lifespan.

To make matters more challenging for children, risk factors tend to co-occur and accumulate over time, thereby exponentially increasing risk of serious externalizing symptoms (Deater-Deckard et al., 1998; Sameroff, 2000; Yoshikawa, 1994). Single risk factors rarely act alone. The accumulation and interaction of risks across development, particularly those centered in the family and child, create multiple pathways to externalizing symptoms (Dodge & Pettit, 2003; Farrington, 2009; Hinshaw, 2002). Risk factors also vary in their stability and salience across development; for example, the type of effects risk factors have and their intensity can change over time (Compas & Reeslund, 2009). Hence, longitudinal studies with a developmental perspective are needed to examine how different risk factors affect children's externalizing symptoms over time to clarify developmental processes and causal mechanisms underlying their continuity and change. For example, Côté and colleagues (2006) examined the development of physical aggression from toddlerhood to preadolescence with a range of risk factors. Toddlers who demonstrated infrequent physical aggression followed a declining trajectory, whereas those who used it frequently were more likely to have chronic externalizing symptoms

through childhood. Frequently aggressive children were more likely to be boys who experienced early hostile and ineffective parenting and were from low-income families. Côté and colleagues (2006) found few cases of children who became physically aggressive when older who were not already aggressive in the preschool years. This study highlighted the usefulness of a developmental perspective in capturing the etiology and course of externalizing symptoms and elucidating effects of both social contextual and individual risk factors and their various pathways.

Externalizing symptoms are not fixed entities that remain through the lifespan; some symptoms appear only during early childhood while others manifest during adolescence (APA, 2000; Hinshaw, 2002; Schulenberg & Zarrett, 2006). This heterogeneous construct requires study over multiple age periods to determine why certain externalizing symptoms and effects of risks wax and wane across development. A risk factor approach combined with a developmental perspective can elucidate dynamic processes through which externalizing symptoms first emerge and either become more chronic and severe behavioral problems or decrease to normative levels found in the general population (Campbell et al., 2000; Dodge & Pettit, 2003; Farrington, 2009; Patterson et al., 1989; Yoshikawa, 1994). As discussed below, transactional theories can serve as useful developmental frameworks for empirically investigating the etiology and development of externalizing symptoms in the context of multiple risks.

### **Transactional–Developmental Frameworks**

The transactional model of development illustrates how the continuous and dynamic interplay of the individual and social environment influences developmental process (Sameroff, 2009a; Sameroff & Mackenzie, 2003). The transactional model

focuses on the bidirectional and interdependent effects between individual-level and social contextual variables over time, while controlling for their earlier levels (Sameroff, 2009b). The transactional model therefore acknowledges the plasticity of both the environment and the individual and assigns both active roles in shaping development. Early transactional perspectives focused on mother–child dyads to elucidate the bidirectional interplay of maternal caregiving and child development (Bell, 1968; Belsky, 1984; Bronfenbrenner, 1977; Sameroff & Chandler, 1975). The transactional model of development follows a dynamic approach placing ongoing parent–child transactions at the core of socialization processes (MacKenzie & McDonough, 2009).

The transactional perspective of developmental psychopathology is a more recent adaptation of the transactional model of development that extends it to the realms of child and adolescent psychopathology (Cicchetti & Toth, 1997; Sameroff, 2000). Following this perspective, psychopathology is viewed as developing from ongoing transactional processes involving the individual and the social context rather than originating from a singular risk factor from one or both. Risk factors are not assumed to be in fixed states; their stabilities are accounted for and they are allowed to change over time. This is a more sophisticated way of viewing risk factors and the etiology of adjustment problems than traditional unidirectional approaches. Most studies focus on the main effects of environmental risk factors on children’s negative outcomes (Sameroff, 2009a). The main strength of a transactional perspective is its emphasis on bidirectional effects, which provide stronger evidence of the causal direction of associations between risk factors and adjustment problems (e.g., Sameroff & Chandler, 1975). Longitudinal studies that are informed a-priori by a transactional perspective can elucidate bidirectional effects

between risk factors in the social environment and children that contribute to the escalation of their adjustment problems. Integrating a risk factor approach with a transactional–developmental perspective can help advance our understanding of the etiology and development of externalizing symptoms.

While we know a great deal about the risk factors that contribute to externalizing symptoms, as reviewed in this chapter, understanding of their bidirectional associations with children and adolescents' externalizing symptoms is relatively limited (Dodge & Pettit, 2003; Hinshaw, 2002; Sameroff, 2009a). Gaps in the literatures on externalizing-related behavioral problems and psychiatric disorders need to be addressed with longitudinal studies that focus on how children and adolescents contribute to their own externalizing symptoms through transactional exchanges with risk factors in their social environments. Research is needed that applies a transactional–developmental approach to the study of prevalent psychosocial risk factors and stage-salient externalizing symptoms in early phases of the lifespan. Given that the best predictor of children's externalizing symptoms is an earlier problem with similar behaviors (Campbell et al., 2010; Campbell et al., 2000; Côté et al., 2006; Huesmann et al., 2003; Huesmann & Taylor, 2006; Loeber & Farrington, 2000), combining their study over time with environmental risk factors would allow researchers to disentangle the effects of psychosocial stressors from the initial levels of externalizing symptoms that potentiate or explain those effects.

**Causal direction of family psychosocial risk factors and externalizing symptoms.** When and how do externalizing symptoms start? From the review of risk factors in this chapter, it is apparent that researchers have spent the majority of their efforts identifying environmental risk factors as salient sources of stress for children. The

social ecology contributes to the etiology and development of externalizing symptoms, but primary causal risk factors have not yet been determined. Loeber and Farrington (2000) posited that initial risk factors for child delinquency originate within the individual child and then within the family. Evidence of how children contribute to the etiology of their own externalizing symptoms is rapidly emerging, demonstrating that children's behavioral problems are not fully dependent on the risk factors associated with their families or broader social ecology.

Theoretical perspectives suggest that difficult or disruptive children who frequently act out can elevate parents' stress levels and alter their parenting and mental health (Bell, 1968; Belsky, 1984; Dodge, 1990; Patterson et al., 1989; Sameroff & Chandler, 1975). Relatively recent empirical evidence indicated that children and adolescents' externalizing symptoms often lead to more stressful family interactions and disturbed psychiatric states, which in turn reinforced their externalizing symptoms (Burt, McGue, Krueger, & Iacono, 2005; Cui, Donnellan, & Conger, 2007; Gross, Shaw, Burwell, & Nagin, 2009; Jenkins, Simpson, Dunn, Rasbash, & O'Connor, 2005; Shaw, Gross, & Moilanen, 2009). For example, children's externalizing symptoms exacerbated marital and family conflict (Burt et al., 2005; Cui et al., 2007; Jenkins et al., 2005) and maternal depression (Gross et al., 2009; Shaw et al., 2009), which subsequently worsened children's adjustment problems (Cummings, Papp, & Kouros, 2009). Emerging evidence has contributed a more nuanced understanding of the etiology of externalizing symptoms; one in which children and adolescents actively elicit psychosocial experiences and modify their adverse effects over time (Cicchetti & Tucker, 1994; Sameroff, 2009).

Despite the potential for a transactional perspective to elucidate our understanding of developmental psychopathology, the majority of risk research continues to focus on the main effects of contextual stressors on children and adolescents' social-emotional outcomes (Compas & Reeslund, 2009; Connell & Goodman, 2002; Cui et al., 2007; Sameroff, 2009a; Shaw et al., 2009). This presents a major barrier to understanding the etiology of externalizing symptoms, as well as their risk pathways and developmental course. Risk factors residing within the child and family are implicated in the development of clinically significant externalizing symptoms (Campbell et al., 2000; Loeber & Farrington, 2000; Moffit & Caspi, 2001; Olson et al., 2005). Relatively severe behavior problems during toddlerhood and the preschool years are prerequisites for life-course-persistent antisocial behavior problems. Externalizing symptoms in early childhood evince moderate to strong stability over time when symptoms are relatively serious, both in frequency and pervasiveness, and when the family environment is characterized as elevated in psychosocial stress (Aguilar et al., 2000; Campbell et al., 2000; Deater-Deckard et al., 1998; Dodge & Pettit, 2003; Farrington, 2009; Hinshaw, 2002; Moffitt & Caspi, 2001; Patterson et al., 1989). More attention to how children's externalizing symptoms affect their family members' mental health and relationships is needed to clarify bidirectional processes that amplify effects of psychosocial stressors.

Conceptualizing associations between externalizing symptoms and family risk factors as transactional processes can refine understanding of the etiology of serious behavioral problems and contribute further evidence of their reciprocal causality. In addition, integrating a transactional–developmental perspective with an interactional approach can further specify causal risk factors in the etiology of externalizing symptoms

(Hinshaw, 2002). Individual characteristics such as gender profoundly alter child development and need to be considered when investigating externalizing symptoms and risk factors. As shown below, bidirectional associations between environmental and individual risk factors for externalizing symptoms may vary across groups of people who have different biological and psychological characteristics.

### **Interactional Approaches to Identifying Individual Differences in Risk**

We know of individual characteristics such as gender and a difficult temperament that contribute to externalizing symptoms, but relatively little is known about how they interact with family psychosocial stressors to worsen externalizing symptoms (Hinshaw, 2002; Loeber & Farrington, 2000). Are there group differences in developmental processes contributing to externalizing symptoms? Group differences in the magnitude of effects of risk factors and in mean levels of externalizing symptoms are not evidence of differences in etiology. Few studies have examined whether group differences in gender and other characteristics interact with multiple risk factors to explain externalizing symptoms (Deater-Deckard, 1998). Furthermore, studies that have examined gender and early temperament differences for later externalizing symptoms and their associated risk factors have produced some mixed and null findings (Calkins, 2009; Daigle et al., 2007; Gorman-Smith & Loeber, 2005; Kroneman et al., 2004; Moffitt & Caspi, 2001; Yoshikawa, 1994). Deater-Deckard and colleagues (1998) found that gender did not moderate effects of cumulative risk factors on children's externalizing symptoms, but boys had higher levels of externalizing symptoms than girls. Other researchers also found more similarities than differences across genders in the origin and course of externalizing symptoms, which suggests a similar etiology for boys and girls (Aguilar et al., 2000;



Campbell et al., 2010; Odgers et al., 2008; van Lier et al., 2007). Further replication studies are needed that examine whether gender moderates effects of multiple risk factors on children's externalizing symptoms.

Gender differences in effects of risk factors and both severity and expression of children's externalizing symptoms suggest that gender may moderate the bidirectional interplay of externalizing symptoms and family psychosocial stressors (Campbell et al., 2010; Daigle et al., 2007; Gorman-Smith & Loeber, 2005; Kroneman et al., 2004; Tremblay, 2000). Girls' externalizing symptoms are less severe and more associated with problems at home, whereas early self-regulatory and behavioral difficulties are more common among boys. Examining the interaction of gender with child-family transactions may reveal gender differences in risk for developing externalizing symptoms in the presence of family risk factors. Aside from gender, temperament may play an important role in moderating transactions leading to externalizing symptoms. However, researchers have been inconsistent in how they conceptualize a difficult temperament, leading to confusion in its meaning and measurement (Farrington, 2009; Rothbart & Bates, 1998; Yoshikawa, 1994). Studying one of its constituents, self-regulation, provides more specificity when examining interactions between risks and externalizing symptoms.

Self-regulatory deficits contribute added risk to effects of psychosocial stressors on children's externalizing symptoms (Lengua, 2002; Rothbart & Bates, 1998). Children who have low self-regulation, which are more often boys, have difficulty modulating their negative emotionality and inhibiting impulses to stress, leading to problems adjusting to situational contexts (Eiden et al., 2007; Else-Quest et al., 2006). Children with low self-regulation tend to demonstrate heightened externalizing symptoms across

early childhood to adolescence (Calkins, 2009; Eisenberg et al., 2005; Gartstein & Fagot, 2003; Hinshaw, 2002; Lengua, 2006; Olson et al., 2005). Self-regulatory abilities improve rapidly across early childhood (Bell & Deater-Deckard, 2007; Posner & Rothbart, 2000) and continue to make gradual gains to young adulthood (Keating, 2004; Steinberg, 2005; Zelazo, Craik, & Booth, 2004). Individual differences in abilities are first measurable in toddlerhood and remain moderately stable into middle childhood (Kochanska & Aksan, 2006; Posner & Rothbart, 2000). Therefore, children who have low self-regulation in toddlerhood are more likely than not to continue having low self-regulation across development. The relative stability of self-regulation makes it a suitable characteristic to test as a moderator of effects of risk factors on externalizing symptoms.

While researchers have linked low self-regulation to elevated externalizing symptoms across childhood, our understanding of its role in infancy and adolescence is relatively poor (Lengua et al., 2008; Olson et al., 2005; Rothbart & Bates, 1998). Furthermore, few studies have examined added risks of self-regulatory difficulties in the presence of family psychosocial stressors. One of the few studies to do so demonstrated that maternal risk (i.e., adolescent parent status, maternal depression, and legal or mental health problems) predicted increases in internalizing symptoms from middle childhood to early adolescence among only children who had low self-regulation (Lengua et al., 2008). Environmental risk at the neighborhood-level predicted increases in both internalizing and externalizing symptoms among children who had low self-regulation, and socioeconomic risk predicted more initial symptoms. Future studies that examine the interaction of self-regulation with family–child transactions may elucidate individual differences in vulnerability to family psychosocial stressors.

A transactional–developmental perspective with an interactional component can further specify causal risk factors for externalizing symptoms that may vary for individuals who differ in characteristics such as gender and self-regulation (Hinshaw, 2002). It is widely recognized that the family environment contributes to externalizing symptoms across infancy to adulthood (Chronis et al., 2007; Compton et al., 2003; Cummings et al., 2005; Eisenberg et al., 2005; Olson et al., 2002; Moffitt & Caspi, 2001; Patterson et al., 1989; Rothbaum & Weisz, 1994; Yoshikawa, 1994). Yet, we understand relatively little about how children worsen experiences within families and contribute to their externalizing symptoms through adverse reciprocal exchanges. Research on temperament and gender differences in externalizing symptoms frame these individual characteristics as risk factors, implying that children play an active role in contributing to their behavioral problems. Furthermore, child characteristics emerge as the most powerful risk factors for externalizing symptoms when compared to risk factors across ecological systems (Deater-Deckard et al., 1998; Loeber & Farrington, 2000). To reiterate this point, the strongest predictor of externalizing symptoms is their earlier manifestation (Côté et al., 2006; Huesmann et al., 2003; Huesmann & Taylor, 2006; Tremblay, 2000). Integrating tests of bidirectional effects between externalizing symptoms and family risk factors and interactions of gender and self-regulation can elucidate transactional processes and individual differences in the etiology and development of serious behavioral problems.

A conceptual model was created illustrating transactions between children’s externalizing symptoms and family psychosocial stressors across development with interactions of gender and self-regulation (See Figure 1). This model integrated the

transactional model of development with an interactional approach used to test for moderation (Sameroff, 2009b). The two dashed arrows represent lagged effects within children's externalizing symptoms and family psychosocial stressors that represent their stability across time. The solid arrows represent their bidirectional effects or transactions and indicate how one predicts change in the other. The dotted arrow represents the interaction or moderating effects of child gender and self-regulation. Finally, the double-sided arrows represent covariances that account for similar times of measurement. To conclude this chapter, I review three longitudinal studies, presented in Chapters II, III, and IV, that utilize this conceptual model to investigate the bidirectional interplay of children's externalizing symptoms and family psychosocial stressors and their interactions with child gender and self-regulation.

### **Developmental Research Integrating Transactions and Interactions**

Limited attention to transactions between children's externalizing symptoms and family psychosocial stressors and interactions of individual characteristics presents major challenges to clarifying the etiology of externalizing symptoms (Compas & Reeslund, 2009; Dodge & Pettit, 2003; Hinshaw, 2002; Sameroff, 2009a). Chapters II, III, and IV present three longitudinal studies that followed a transactional framework to illustrate how children and adolescents' externalizing symptoms contributed to their own behavioral problems through bidirectional effects with family stressors across distinct phases of development. In addition, each study examined interactions of children's gender and self-regulation to reveal individual differences in risk. Across the three studies, my overarching hypotheses were that family psychosocial risk factors and externalizing symptoms would intensify one another over time and to a greater degree

among children and adolescents who had low self-regulation. Young people who had high self-regulation were expected to develop fewer externalizing symptoms and to be less affected by family risk factors than young people who had low self-regulation, and hence additional risk. Since young boys tend to have more severe externalizing symptoms and self-regulatory difficulties than girls (Campbell et al., 2010; Deater-Deckard, 1998; Olson et al., 2005), I also expected boys to be affected to a greater extent by family psychosocial risk factors than girls. I did not hypothesize as to whether groups would vary in how they affected family stressors, due to a lack of supporting evidence. Across these three studies, I expected that adolescents would be less adversely affected by family risk factors than younger children, since infants and toddlers often spend the majority of their time at home with family. In addition, I expected adolescents' externalizing symptoms to exacerbate family stressors to a greater extent than younger children, because their behavioral problems inflict more harm to others and have stricter legal consequences (Tremblay, 2000). Young children's externalizing symptoms, while disruptive and problematic across settings, do not endanger children or others to the same extent as delinquency and violence.

Study 1 in Chapter II examined bidirectional effects between maternal depressive symptoms and children's emerging externalizing behavior across infancy to toddlerhood. Interactions of infants' gender and functional self-regulation of crying, feeding, and sleeping were tested to determine whether they moderated the transactional interplay of maternal depressive symptoms and externalizing behavior across toddlerhood. Infancy is a period of heightened risk to negative effects of maternal depression that are associated with elevated externalizing symptoms (Cummings & Davies, 1994; Downey & Coyne,

1990; Lovejoy et al., 2000). Children's behavioral problems have been shown to worsen mothers' depressive symptoms, but few studies test this direction of effect around infancy (Connell & Goodman, 2002; Dodge, 1990; Gross et al., 2009; Shaw et al., 2009). Further, we know little about infant characteristics that moderate their risk to maternal depression (Goodman & Gotlib, 1999). I proposed that interactions of infants' gender and functional self-regulation would distinguish between infants who were more or less at risk of developing externalizing symptoms when exposed to maternal depression.

While there is some evidence of bidirectional relations between maternal depressive symptoms and children's externalizing behavior, it is not clear whether these transactions occur more frequently in different phases of development (Shaw et al., 2009). Therefore, Study 2 (Chapter III) examined older children and transactional effects between maternal depressive symptoms and externalizing behavior across the preschool period to middle childhood. Interactions of children's gender and behavioral self-regulation, or effortful control, were examined to understand whether they moderated the bidirectional interplay of maternal depressive symptoms and externalizing behavior across childhood. Similar to Study 1, I proposed that the interaction of children's self-regulation would distinguish between children who were more or less likely to develop externalizing behavior when exposed to maternal depressive symptoms. I further expected all children's externalizing behavior to predict more maternal depressive symptoms. While maternal depression is especially problematic in early childhood (Beardslee et al., 1983; Cummings & Davies, 1994), family conflict is a more prevalent and developmentally salient psychosocial risk factors for adolescents' externalizing symptoms (Collins & Steinberg, 2006; Laursen et al., 1998).

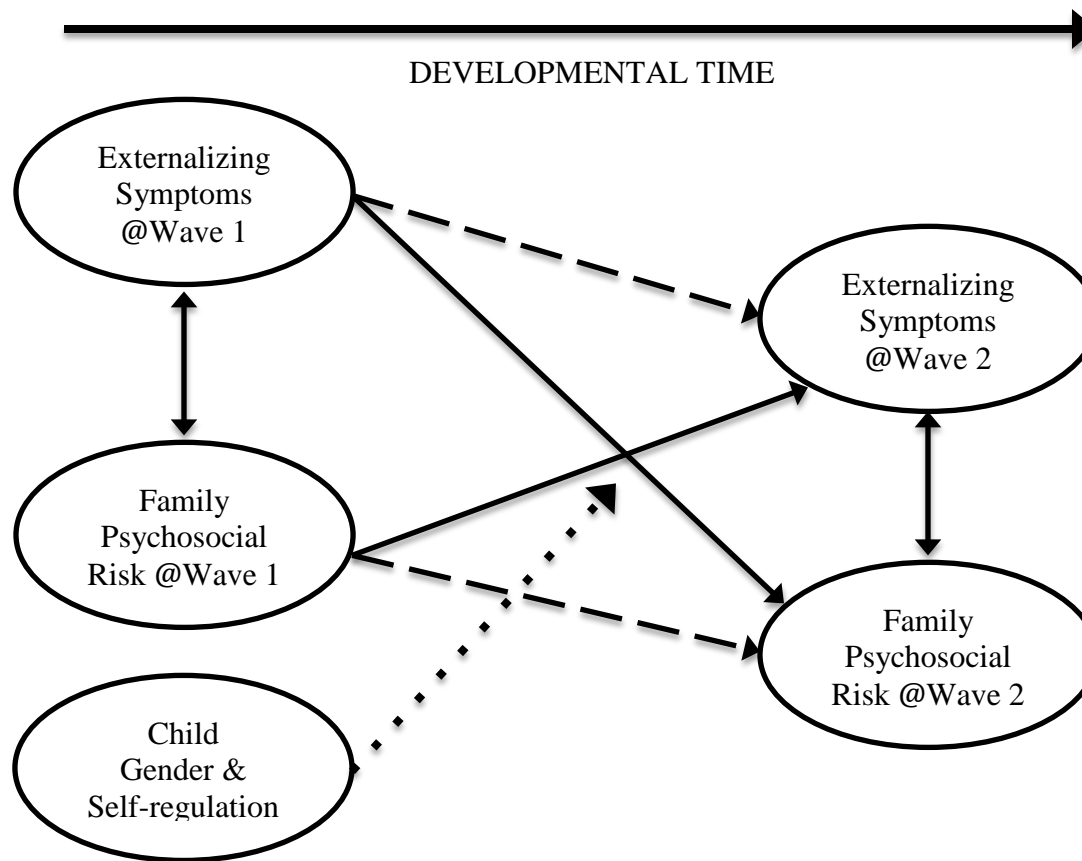
Study 3 (Chapter IV) examined bidirectional effects between family conflict and violent behavior across adolescence and their consequences for internalizing problems in young adulthood. Interactions of adolescents' gender and active coping were examined to understand whether they moderated the bidirectional interplay of family conflict and violent behavior across adolescence, as well as their effects on internalizing symptoms in young adulthood. Study 3 was unique from the previous two studies in that it included an internalizing outcome belonging to the child rather than the mother. The time from adolescence to adulthood is a transitional phase that is sensitive to internalizing problems, particularly in the presence of family conflict and violent behavior (Graber & Sontag, 2009; Laursen & Collins, 2009; Ozer, 2005; Voisin, 2007). Therefore, an internalizing outcome was included to examine whether the bidirectional interplay of adolescents' family conflict and violent behavior would directly worsen their internalizing problems. Identifying an elevated risk for adult depression was significant, because this would have placed children of these adolescents at risk for externalizing symptoms. I proposed that the interaction of elevated family conflict with adolescents' active coping, a more cognitively-sophisticated form of self-regulation than the previous two, would identify adolescents who were more likely to develop greater violent behavior and internalizing problems. By reviewing these studies in order of human development, I illustrated the progression of regulatory functioning from overseeing basic autonomic processes, such as crying and feeding, to more consciously effortful processes underlying adaptive coping responses (Compas & Reeslund, 2009).

In each study, structural equation modeling was conducted with *Mplus* (Muthén & Muthén, 2007; 2010) to test bidirectional effects between family psychosocial risk

factors and externalizing symptoms while simultaneously controlling for their continuity over time and similar times of measurement (Kline, 2005; Sameroff, 2009b). In addition, multiple-group structural equation modeling was used to test for interactions of gender and self-regulation as a means of revealing individual differences in risk.

Studying how the interface of children's gender, self-regulation, externalizing symptoms, and family psychosocial stressors elevates risk for serious behavioral problems is imperative to clarifying their etiology and development. These collective studies can contribute evidence that children actively influence their social ecologies and the etiology of their own externalizing symptoms, therefore extending the literature's focus from parental risk models, and more generally environment-to-child effects, to transactional frameworks. This may provide a clearer understanding of the onset of externalizing symptoms and whether their primary causal risk factors are located within the child, family, or their coercive exchanges. These studies also can extend the literature on early temperament by demonstrating how self-regulatory processes in infancy and adolescence alter risk for externalizing symptoms in the presence of family psychosocial risk factors. Gender differences in effects of family stressors and mean levels of externalizing symptoms can be replicated and further elucidated by investigating gender within family-child transactions and alongside the interaction of self-regulation. Taken together, the integrative approach discussed in this chapter and the three studies presented in Chapters II, III, and IV, have potential to inform the etiology and development of early externalizing symptoms by narrowing the search for causal risk factors and individual-centered mechanisms that amplify risk for more severe behavioral problems.





*Figure 1.* Conceptual model of bidirectional effects between children's externalizing symptoms and family psychosocial risk and the interaction of child gender and self-regulation. Dashed arrows are lagged effects of variables. Solid arrows are bidirectional effects of variables. The dotted arrow is the interaction of moderating variables. Double-sided arrows are covariances between variables.

## Chapter II

### **Infant Functional Self-regulation and Gender Moderate Transactions of Externalizing Behavior and Maternal Depressive Symptoms**

About 13% of all women develop postpartum depression (O'Hara & Swain, 1996), making infancy a period of particular concern for the effects of maternal depressive symptoms on child development. Researchers have consistently shown that infants who are exposed to heightened maternal depressive symptoms are at elevated risk of developing externalizing problems (Cummings & Davies, 1994; Downey & Coyne, 1990; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Children's behavioral problems have also been shown to exacerbate mothers' depressive symptoms, but relatively few studies test this direction of effect despite strong theoretical support and some empirical evidence for transactional mother-child processes (Bell, 1968; Connell & Goodman, 2002; Dodge, 1990; Sameroff & Chandler, 1975; Shaw, Gross, & Moilanen, 2009). Furthermore, we know little of infant characteristics that moderate their vulnerability to maternal depressive symptoms (Goodman & Gotlib, 1999). I propose that infants' gender and functional self-regulation of crying, feeding, and sleeping can distinguish between those who are more or less susceptible to developing behavioral problems when exposed to maternal depressive symptoms. The present study examined bidirectional effects of young children's externalizing behavior and maternal depressive symptoms in toddlerhood, and moderating effects of gender and early functional self-regulation. As

discussed below, a transactional perspective can serve as a useful framework to examine these dynamic relations.

### **A Transactional Perspective on Mother–Child Adjustment Problems**

Multiple theoretical contributions have been made that address the bidirectional nature of parent–child interactions (Bell, 1968; Belsky, 1984; Bronfenbrenner, 1977; Sameroff & Chandler, 1975). The most relevant for the present study is the *transactional perspective of developmental psychopathology*, which views the etiology of adjustment problems as resulting, in part, from ongoing exchanges between children and their social environments (Cicchetti & Toth, 1997; Sameroff, 2000). This dialectical approach emphasizes children’s active roles in shaping their development by selecting and eliciting social experiences and modifying their physiological effects through stress management (Cicchetti & Tucker, 1994; Sameroff, 2009a). Continuing parent–child transactions are considered the core process through which socialization occurs (MacKenzie & McDonough, 2009), and therefore are implicated in the development of children’s adjustment problems in the maternal depression literature (Connell & Goodman, 2002; Cummings & Davies, 1994; Dodge, 1990; Shaw et al., 2009).

Young children’s behavioral problems may be especially stressful for mothers who are vulnerable to developing depression, further exacerbating their initial symptoms. Few studies have investigated this causal direction in comparison to literature showing that maternal depressive symptoms contribute to young children’s externalizing behavior (e.g., Chronis et al., 2007; Cummings, Keller, & Davies, 2005; Gartstein & Fagot, 2003; Shaw, Gilliom, Ingoldsby, & Nagin, 2003; Weinfield, Ingerski, & Moreau, 2009). Mothers who are distressed as a result of children’s misbehavior may subsequently

respond to them in ways that worsen child adjustment problems (Belsky, 1984). Shaw and colleagues (2009) illustrated this bidirectional process when they found that maternal depressive symptoms across ages 1.5 to 6 years predicted increases in boys' conduct problems across ages 2 to 8, and boys' conduct problems at 3.5 and 5 years predicted increases in mothers' depressive symptoms at 5 and 6 years. Following a transactional–developmental perspective, I expected maternal depressive symptoms and children's externalizing behavior to predict increases in one another across toddlerhood (Cicchetti & Toth, 1997; Connell & Goodman, 2002; Dodge, 1990; Goodman & Gotlib, 1999).

Examining the transactional interplay of adjustment problems in mothers and their infants and toddlers is important for several reasons. First, normative levels of transitory externalizing behavior in toddlerhood coupled with family psychosocial stressors are more likely to progress into school-age antisocial behavior (Aguilar, Sroufe, Egeland, & Carlson, 2000; Campbell, Shaw, & Gilliom, 2000; Moffitt, Caspi, Harrington, & Milne, 2002; Shaw et al., 2003). Studies that examine the early emergence of externalizing behavior in relation to prominent family risk factors, such as maternal depression, can elucidate the etiology of more serious antisocial behavior problems. Second, the period of early infancy is believed to be especially vulnerable to the effects of maternal depression (Cummings & Davies, 1994; Goodman & Gotlib, 1999; Shaw et al., 2009). Identifying infant characteristics that attenuate their susceptibility to maternal depressive symptoms and subsequent externalizing behavior can inform preventive interventions targeting young children of mothers with depression who are at risk for chronic behavioral problems. Therefore, I was interested in examining the moderating effects of several infant characteristics in the present study, beginning with infant gender.

## **The Role of Infant Gender in Maternal Depression and Child Externalizing Behavior**

Researchers have found that child gender moderates the effects of different psychosocial risk factors on young children's externalizing behavior, with boys often demonstrating greater vulnerability to family stressors (Dodge & Pettit, 2003; Yates, Obradović, & Egeland, 2010). Boys in general tend to manifest more externalizing behavior than girls, but this difference is small until the preschool period when it begins to grow (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Eiden, Edwards, & Leonard, 2007; Fingleton, 2009). Reported gender differences in how maternal depressive symptoms contribute to children's adjustment problems have been inconsistent (Cummings & Davies, 1994; Davies & Windle, 1997; Goodman & Gotlib, 1999). Researchers have suggested, but not clearly supported, a gender-specific hypothesis that boys who are exposed to maternal depression are more prone to developing externalizing behavior, whereas girls are more likely to manifest internalizing problems (Cummings & Davies, 1994; Goodman & Gotlib, 1999). Some evidence suggests that boys are more vulnerable to maternal depressive symptoms in early childhood and girls are more vulnerable in adolescence (Davies & Windle, 1997). Given the focus on infancy in the present study, I expected maternal depressive symptoms to have a greater effect on boys' externalizing behavior than girls. No prediction was made as to whether boys and girls' externalizing behavior contributes differently to mothers' depressive symptoms, as no previous study of this potential gender difference was found. Another infant characteristic aside from gender that has been implicated in the etiology of behavioral problems and an elevated risk of maternal depression is early temperament (Calkins, 2009; Deater-Deckard et al., 1998; Dodge, 1990; Dodge & Pettit, 2003).

Temperament affects infants' initial automatic responses to stress, and both constrains and supports a range of future coping responses (Compas & Reeslund, 2009). Infants' automatic self-regulatory processing manages their arousal states before the emergence of conscious and more volitional forms of self-regulation. Self-regulation is an aspect of temperament that is associated with children's externalizing behavior (Lengua, 2006; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005; Rothbart & Bates, 1998) and maternal depressive symptoms (Choe, Olson, & Sameroff, in press; Sektnan, McClelland, Acock, & Morrison, 2009). Self-regulation also has been found to moderate young children's vulnerability to environmental risk factors (Lengua, 2002). I discuss below how self-regulation in infancy may moderate associations between their externalizing behavior and maternal depressive symptoms in toddlerhood.

### **Infants' Functional Self-regulatory Difficulties Predict Later Adjustment Problems**

The development of functional self-regulation of crying, feeding, and sleeping is essential to infants' immediate survival and future self-regulatory advances (Calkins, 2009; Porges, 1996). Infants quickly gain abilities to regulate their own autonomic processes such as breathing, feeding, and digesting, and begin to communicate their arousal state needs to caregivers through cries and other means. Infants display clear individual differences in their ability to modulate arousal states indicating early divergences in self-regulatory trajectories with important implications for development (Olson, Sameroff, Lunkenheimer, & Kerr, 2009; Rothbart & Bates, 2006). Regulatory functioning has been shown to be stable from infancy to age 5 across system levels, beginning with physiological regulation and then progressing to emotion, attention, and behavioral domains (Calkins, 2009; Feldman, 2009). Similarly, problems in managing

basic regulatory processes can cascade to deficits in more advanced forms of self-regulation in early childhood associated with externalizing problems (Bernier, Carlson, Bordeleau, & Carrier, 2010; Calkins, 2009; Olson et al., 2005). In a recent meta-analysis, infants with regulatory problems were more likely to develop later externalizing behavior than infants who did not have excessive crying and sleep problems (Hemmi, Wolke, & Schneider, 2011). Regulatory problems in infancy also predict poorer cognitive development in toddlerhood (Wolke, Schmid, Schreier, & Meyer, 2009). Early difficulties with self-regulation of crying, feeding, and sleeping in infancy lead to later self-regulatory deficits and behavioral problems.

The long-term consequences of early regulatory problems have encouraged researchers to examine how rearing environments contribute to individual differences in infant self-regulation. Infants' limited capacities to alter stressful and challenging events make them almost entirely reliant on caregivers for co-regulation of their arousal states (MacKenzie & McDonough, 2009; Olson et al., 2009). The development of functional self-regulation is supported by caregiving that provides infants with regulatory experiences that soothe their arousal and alert them to external stimuli (Sameroff, 2009a). The synchronicity of this mother–infant co-regulation determines early competency in self-regulation (Harrist & Waugh, 2002; Olson et al., 2009; Olson & Lunkenheimer 2009). Thus, problems with the quality of co-regulation lead to problems self-regulating. For example, infants of less responsive parents at 5 and 6 months are worse at regulating their stress than infants of more responsive parents (Haley & Stansbury, 2003). Poor mother–infant relationships, in general, predict more infant self-regulatory deficits (Wolke et al., 2009). Environments characterized by elevated stress challenge the optimal

development of infants' self-regulatory competence (Feldman, 2009). Infants of multiple-risk families are at increased risk of both self-regulatory and externalizing problems (Hemmi et al., 2011). Thus, a rearing environment marked by elevated maternal depressive symptoms may be a setting in which infants with functional self-regulatory difficulties are more likely to develop behavioral problems.

Mothers who are depressed tend to be less adept at meeting caregiving challenges, often resenting parenting duties and responding more negatively to children (Downey & Coyne, 1990; Goodman & Gotlib, 1999). Infants who are exposed to elevated maternal depressive symptoms often receive suboptimal support from their mothers and experience more dysregulation (Cummings & Davies, 1994; Lovejoy et al., 2000). Self-regulation has been shown to moderate effects of contextual risk on children's adjustment problems (Lengua, 2002). Infants who had low functional self-regulation were expected to be at greater risk of the negative effects of maternal depressive symptoms and to demonstrate more externalizing behavior as toddlers than infants with high self-regulation.

### **The Current Study**

The goal of the present study was to add longitudinal evidence of bidirectional relations between maternal depressive symptoms and children's externalizing behavior to the relative dearth of maternal depression research following a transactional–developmental perspective (Connell & Goodman, 2002; Dodge, 1990; Shaw et al., 2009). To further elucidate the emergence of externalizing behavior, the present study followed a community sample of families with infants across ages 7, 15, and 33 months. Because infancy is believed to be a period of particular vulnerability to maternal depression, I examined the early interplay of maternal–infant adjustment problems and tested infant



characteristics that may attenuate their susceptibility to maternal depressive symptoms (Cummings & Davies, 1994; Goodman & Gotlib, 1999; Shaw et al., 2009). I aimed to address inconsistencies in the literature and contribute evidence of how infant gender and functional self-regulation serve as moderating mechanisms of this early risk (Davies & Windle, 1997; Goodman & Gotlib, 1999).

I hypothesized that maternal depressive symptoms and children's externalizing behavior would predict increases in one another through bidirectional effects in toddlerhood (e.g., Shaw et al., 2009). Boys were expected to be at greater risk than girls to negative effects of maternal depressive symptoms resulting in their higher levels of externalizing behavior in toddlerhood (Cummings & Davies, 1994; Goodman & Gotlib, 1999). Children who had low functional self-regulation in infancy also were expected to be more adversely affected by maternal depressive symptoms and more likely to develop externalizing behavior as toddlers (Calkins, 2009; Hemmi et al., 2011; Lengua, 2002; Olson et al., 2005; Wolke et al., 2009). I did not hypothesize about whether effects of children's externalizing behavior would vary based on infant gender or functional self-regulation due to a lack of supporting evidence, making this part of the investigation exploratory. Family socioeconomic status (SES) was controlled for in the present study due to its associations with maternal depressive symptoms (Campbell, Matestic, von Stauffenber, Mohan, & Kirchner, 2007; Goodman & Gotlib, 1999; O'Hara & Swain, 1994), externalizing behavior (Deater-Deckard et al., 1998; Farrington, 2009), and self-regulatory functioning in infancy (Feldman, 2009; Hemmi et al., 2011).

## **Method**

### **Participants**

Participants in this study were among 258 families originally recruited for an investigation of environmental risk, mother–child relationships, and infant mental health (Rosenblum, McDonough, Muzik, Miller, & Sameroff, 2002). For the present study, 251 children (133 girls) were included; the remaining seven were excluded for missing data on their gender and functional self-regulation. Families were recruited from pediatric clinics in the greater area surrounding a large research university in the Midwest during their 6-month well-child visits. Mothers were asked to complete a survey about their child’s crying, feeding, and sleeping problems and whether they were able to participate in a longitudinal study of child development. Mothers predominantly identified their families as Caucasian (72%), then African American (12%), and biracial or other (14%). Seven families did not report this information. In terms of marital status, 81% of mothers were married, 10% were never married, 7% were living with the birth father, and about 2% were separated, divorced, or living with a partner who was not biologically related to the child. At recruitment, about 25% of mothers worked full time, 26% worked part time, 41% stayed at home with the baby, and the remaining 8% did not report this information. Families’ average annual household income ranged from \$2,500 to \$102,500 ( $M = \$53,179$ ,  $SD = \$28,568$ ). Twenty-three families did not report their household income.

### **Procedure**

Families were assessed when infants were 7 (T1), 15 (T2), and 33-months-old (T3). Data for the present study were collected during home visits, in which a trained graduate student research assistant interviewed mothers about demographic information and their perceptions of their children’s development and mental health. In addition, mothers completed a packet of questionnaires assessing a range of developmental and

contextual issues, such as their depressive symptoms and their children's functional self-regulation. At T2 and T3, mothers were also provided questionnaires assessing children's externalizing behavior. Only raw scores from measures were used in the study analyses.

## **Measures**

**Maternal depressive symptoms.** Mothers completed the 20-item Center For Epidemiological Studies Depression Scale at all time points (CES-D; Radloff, 1977). Items assessed a range of somatic and depressive mood symptoms (avg.  $\beta = .88$ ), such as hopelessness, poor appetite, and restless sleep. Mothers indicated the average number of days per week that they experienced each symptom using a 4-point response scale (0 indicated "less than one day" and 3 indicated "5-7 days"). The CES-D is used as a clinical screening device with a cut-off score of 16. About 19% of mothers in the sample exceeded this cut-off at T1, 18% of mothers at T2, and close to 14% of mothers at T3. These rates correspond closely with national prevalence rates of postpartum and major depression (Kessler et al., 2003; O'Hara & Swain, 1996).

**Externalizing behavior.** Mothers completed the Infant-Toddler Social Emotional Assessment at 15 and 33 months (ITSEA; Briggs-Gowan & Carter, 1998) and the Child Behavior Checklist for Ages 2–3 at 33 months (CBCL 2/3; Achenbach, 1992). The ITSEA's externalizing behavior scale (avg.  $\beta = .84$ ) consisted of 20 items assessing peer aggression, activity level, and negative emotional reactivity. Mothers responded to ITSEA items at T2 and T3 using a 3-point-response scale (0 represented "not true or rarely" and 2 represented "very true or fairly often"). The CBCL's externalizing behavior scale ( $\beta = .86$ ) consisted of 26 items assessing children's destructive and aggressive behavior. Mothers responded to CBCL items at T3 based on children's behavior during

the last two months using a 3-point-response scale (0 represented “not true” and 2 represented “very true or often true”). Mothers rated 24 children in the borderline clinical range (10%;  $T \geq 60$ ) and five children in the clinical range (2%;  $T \geq 64$ ) of the CBCL’s externalizing behavior scale at T3.

**Functional self-regulation.** Mother completed several questionnaires at T1 assessing infants’ crying, feeding, and sleeping problems during the past week. The Crying Patterns Questionnaire assessed total crying time and feeding problems in infancy (CPQ; St. James-Roberts & Halil, 1991). A 5-item crying scale ( $\beta = .81$ ) was created that assessed the total number of minutes that the child cries at various times of the day (e.g., morning, afternoon, evening). A 3-item feeding problems scale ( $\beta = .54$ ) was created that assessed the child’s appetite, picky eating habits, and difficulty to feed using a 3-point response scale (1 indicated “no problems” and 3 indicated “definite problems”). The Sleep Habits Scale assessed infants’ sleep problems (Seifer, Sameroff, Barrett, & Krafchuk, 1994). A 3-item sleep problems scale ( $\beta = .63$ ) was created that assessed whether the child sleeps too little, sleeps the right amount, and sleeps the same amount each day using a 3-point response scale (1 indicated “rarely” and 3 indicated “usually”). The two latter items were reverse-coded. Items for all scales were selected based on their specification of a regulatory problem due to the child’s behavior rather than the caregivers’ behavior. I created a total score for functional self-regulation in infancy by combining the standardized scores for these crying, feeding, and sleeping problems scales. Higher scores indicated less functional self-regulation and more regulatory problems. The total score was divided by median split (100), leaving 119 *well-regulated infants* (coded 0) and 125 *poorly-regulated infants* (coded 1).

**Family socioeconomic status.** Mothers completed a demographic questionnaire based on the Hollingshead (1979) four-factor score for family socioeconomic status assessing parents' highest level of education and occupational status at T1. Mothers reported levels of education ( $M = 5.60, SD = 1.09$ ) and occupation ( $M = 6.74, SD = 12.10$ ) indicating that most received a vocational, technical, or Bachelors' degree and worked as technicians, semiprofessionals, managers, or semiprofessionals. Fathers reported similar levels of education ( $M = 5.64, SD = 1.12$ ) and occupation ( $M = 6.33, SD = 2.27$ ) as mothers, however, 24% of fathers reported working as higher executives or major professionals. Scores ranged from 20 to 66 ( $M = 46.80, SD = 12.14$ ) representing the top four of five social strata in the Hollingshead system (1979).

### **Data Analysis Plan**

Preliminary analyses consisted of descriptive statistics and correlations, followed by tests of attrition, missing data, and gender and self-regulation group comparisons. A transactional model was then created with structural equation modeling (SEM) to test for bidirectional relations between maternal depressive symptoms and children's externalizing behavior while controlling for family SES in the full sample. As illustrated in Figure 2.1, I modeled the lagged effect stability of measures from one time point to the next (A's), the cross-lagged effect or prediction of change by MD to EXT (B's), the prediction of change by EXT to MD (C), a second-order cross-lagged effect of T1 MD to T3 EXT (D), and their within-time covariances (E's) to account for similar times of measurement. Family SES, its pathways to all constructs, and covariances between repeated measures are not shown in Figure 2.1 to simplify its graphical illustration.

Multiple-group SEM was used to compare the fit of the transactional model for boys and girls and test for moderating effects of child gender. Child gender was included as a covariate in the subsequent models. Finally, I conducted multiple-group SEM with the transactional model to compare children who had high functional self-regulation to children who had low functional self-regulation in infancy. All SEM was conducted using *Mplus* Version 6 (Muthén & Muthén, 2010) and only standardized values were reported for significant unstandardized estimates in the figures and text. All other analyses were conducted using SPSS 19.0.

## **Results**

### **Descriptive Analyses**

Table 1.1 shows the correlations, means, and standard deviations of study variables for the full sample. In addition, separate descriptive statistics were calculated for boys and girls (see Table 1.2) and for children who had low and high functional self-regulation in infancy (see Table 1.3). No problems with normality were evident based on skewness and kurtosis values. Among the full sample, higher family SES at T1 was associated with lower maternal depressive symptoms at all times points and with lower externalizing behavior at T3. More maternal depressive symptoms were associated with more externalizing behavior across all time points for the full sample (see Table 1.1). Table 1.2 shows that among boys, higher family SES at T1 was associated with lower maternal depressive symptoms at T2 and T3. Among girls, higher family SES at T1 was associated with lower externalizing behavior at T3. More maternal depressive symptoms were associated with more externalizing behavior across all time points for both genders,

except T1 maternal depressive symptoms were not related to T3 CBCL externalizing behavior for girls.

Table 1.3 shows that among children who had low functional self-regulation in infancy, higher family SES at T1 was associated with fewer maternal depressive symptoms at T2. Among children with low self-regulation, more maternal depressive symptoms were concurrently associated with more externalizing behavior at T2 and T3, but were not consistently associated across time points. Among children who had high functional self-regulation in infancy, higher family SES at T1 was associated with fewer maternal depressive symptoms at all time points and less externalizing behavior at T3 (CBCL). Among children with high self-regulation, more maternal depressive symptoms were associated with more externalizing behavior across all time points. For both self-regulation groups, child gender was not related to the other variables.

### **Attrition, Missing Data, and Gender and Regulation Group Differences**

T-tests were conducted to identify nonrandom attrition and missing data. The attrition group consisted of 43 families (17%) that had left the study by the third time point. The attrition group had lower levels of family SES at T1 ( $M = 43.29$ ,  $SD = 13.84$ ) than remaining families ( $M = 47.44$ ,  $SD = 11.66$ ),  $t(218) = 2.02$ ,  $p = .045$ . Children who were missing data on maternal depressive symptoms at T2 had lower levels of functional self-regulation in infancy ( $M = 114.10$ ,  $SD = 91.01$ ,  $n = 40$ ) than children who had these data ( $M = 154.71$ ,  $SD = 148.80$ ,  $n = 213$ ),  $t(84) = 2.30$ ,  $p = .024$ . Children who were missing data on their externalizing behavior (ITSEA) at T2 had lower levels of functional self-regulation in infancy ( $M = 113.58$ ,  $SD = 91.01$ ,  $n = 40$ ) than children who had these data ( $M = 154.81$ ,  $SD = 148.86$ ,  $n = 213$ ),  $t(83) = 2.34$ ,  $p = .022$ . Children who were

missing data at the second time point had lower functional self-regulation in infancy. There were no other differences on study variables between families.

T-tests were then conducted to compare study variables across genders and across children in the two self-regulation groups. Group means and standard deviations corresponding to these analyses are reported in Tables 1.2 and 1.3. No significant gender differences were found. Infants who had high functional self-regulation had lower externalizing behavior at T2 than infants who had low functional self-regulation,  $t(210) = -2.81, p = .005$ . These same infants had lower externalizing behavior at T3 than infants who had low self-regulation based on ratings from the CBCL,  $t(191) = -2.07, p = .039$ , and the ITSEA,  $t(190) = -2.04, p = .043$ . Similar numbers of boys and girls were in the two self-regulation groups. Infants who had high functional self-regulation at 7 months had lower levels of externalizing behavior at 15 and 33 months than infants who had low functional self-regulation.

### **Structural Equation Modeling**

As illustrated in Figure 2.2, I examined whether there were bidirectional effects of maternal depressive symptoms and children's externalizing behavior from infancy to toddlerhood while accounting for family SES. A latent factor for externalizing behavior at T3 was created that consisted of externalizing symptom scores from the CBCL ( $\beta = .83, p < .001$ ) and ITSEA ( $\beta = .73, p < .001$ ). Fit indices of the transactional model with the full sample indicated a close approximate fit of the data to the analytic model:  $\chi^2(3) = 2.12, p = .549$ . RMSEA = .00 [.00, .09]. CFI = 1.00. T1 maternal depressive symptoms predicted more T2 externalizing behavior and an increase in T3 externalizing behavior. The effect of T2 externalizing behavior on T3 maternal depressive symptoms was not



significant. All lagged effects were significant, indicating that maternal depressive symptoms were stable across 7, 15, and 33-months-of-age and children's externalizing behavior was stable from 15- to 33-months-of-age. Family SES at T1 was associated with decreases in both T2 maternal depressive symptoms and T3 externalizing behavior. The model explained 8% of the variance of T2 externalizing behavior, 30% of T2 maternal depressive symptoms, 38% of T3 externalizing behavior, and 38% of T3 maternal depressive symptoms.

### **Multiple-Group Structural Equation Modeling**

**Gender comparison.** After examining the transactional model with the full sample, I conducted multiple-group SEM to test for moderating effects of gender and determine whether maternal depressive symptoms have a greater effect on boys' externalizing behavior than girls'. I first established measurement invariance by constraining factor loadings, intercepts, and variances to be equal across groups:  $\chi^2(10) = 15.75, p = .107$ . I then compared the fit of this model to one in which these values were freely estimated:  $\chi^2(6) = 7.06, p = .315$ . A chi-square difference test ( $\Delta\chi^2$ ) indicated no difference in overall fit for the two models, establishing measurement invariance,  $\Delta\chi^2(4) = 8.69, p > .05$ . Next, an incremental approach to achieving the best fitting and most parsimonious model was followed. One structural parameter was constrained to be equal across groups and the overall model fit was compared to an unconstrained model using a chi-square difference test. If the equality constraint worsened the overall fit of the model, I eliminated the constraint in the next nested model. If the constraint did not alter the fit, it was included in the next nested model testing an additional constraint until the best fitting and most parsimonious model was identified. Following these steps, I identified a

model that was a close approximate fit to the data (see Figure 2.3):  $\chi^2(25) = 30.54, p = .205$ . RMSEA = .04 [.00, .09]. CFI = .99. All significant effects from the previous model (as presented in Figure 2.2) remained so for both genders with one major exception: The effect of T1 maternal depressive symptoms on T3 externalizing behavior was significant for boys ( $\beta = .33, p = .002, n = 115$ ), but not for girls ( $\beta = .13, p = .252, n = 133$ ),  $\Delta\chi^2(1) = 4.46, p < .05$ . Although maternal depressive symptoms at 7 months predicted more externalizing behavior at 15 months for both boys and girls, it only predicted an increase in boys' externalizing behavior at 33 months. A gender covariate was included in subsequent models to account for these differences when testing for moderating effects of functional self-regulation. Before moving on to the next multiple-group analysis, I wanted to note whether including a gender covariate would change any of the observed effects from the full sample analysis (as presented in Figure 2.2). I added gender to the transactional model and found no noticeable differences:  $\chi^2(4, N = 248) = 3.88, p = .423$ . RMSEA = .00 [.00, .10]. CFI = 1.00.

**Functional self-regulation comparison.** I tested whether functional self-regulation in infancy attenuated the interplay of maternal depressive symptoms and toddler-age children's externalizing behavior, while accounting for T1 family SES and child gender. Maternal depressive symptoms and toddler-age externalizing behavior were expected to adversely influence one another, but children who had better functional self-regulation in infancy were expected to be less vulnerable to maternal depressive symptoms and to demonstrate less externalizing behavior than children who had lower self-regulation. Again, I first tested for measurement invariance by comparing a model with fixed factor loadings and intercepts [ $\chi^2(10) = 12.99, p = .224$ ] to a model in which

they were freely estimated [ $\chi^2(8) = 12.14, p = .145$ ] and found no difference in their fit,  $\Delta\chi^2(2) = .58, p > .25$ . However, a significant difference in fit was found when I compared the former model to one in which the variances were also constrained to be equal across groups [ $\chi^2(12) = 25.32, p = .013$ ],  $\Delta\chi^2(2) = 12.32, p < .005$ . These preliminary tests established a less conservative but acceptable level of measurement invariance than the previous multiple-group model.

A similar incremental process as the gender comparison was followed to identify the best fitting and most parsimonious multiple-group model (see Figure 2.4):  $\chi^2(30) = 29.60, p = .486$ . RMSEA = .00 [.00, .07]. CFI = 1.00. Similar to previous results, T1 maternal depressive symptoms predicted more T2 externalizing behavior and an increase in T3 externalizing behavior among all children, while accounting for T1 family SES and child gender. Two effects differed across self-regulation groups: (1) The effect of T2 externalizing behavior on T3 maternal depressive symptoms was significant for *well-regulated infants* ( $\beta = .22, p = .005, n = 119$ ), but not for *poorly-regulated infants* ( $\beta = -.07, p = .484, n = 125$ ),  $\Delta\chi^2(1) = 7.50, p < .01$ ; (2) The lagged effect between T1 and T2 maternal depressive symptoms was of greater magnitude for well-regulated infants ( $\beta = .59, p < .001$ ) than for poorly-regulated infants ( $\beta = .43, p < .001$ ),  $\Delta\chi^2(1) = 6.03, p < .025$ . Although maternal depressive symptoms at 7 months predicted more externalizing behavior at 15 and 33 months, regardless of self-regulation, only the externalizing behavior of 15-month-old toddlers who had high self-regulation in infancy predicted an increase in mothers' depressive symptoms at 33 months.

All lagged effects were significant for both groups, indicating that maternal depressive symptoms were stable across 7, 15, and 33-months-of-age and children's

externalizing behavior was stable from 15- to 33-months-of-age. The continuity of maternal depressive symptoms from 7 to 15 months, however, appeared to be stronger for infants who had higher rather than lower functional self-regulation. Family SES at T1 was associated with a decrease in T2 maternal depressive symptoms. For the well-regulated and poorly-regulated groups, the model respectively explained 11% and 7% of the variance of T2 externalizing behavior, 40% and 23% of T2 maternal depressive symptoms, 35% and 46% of T3 externalizing behavior, and 51% and 30% of T3 maternal depressive symptoms.

### **Discussion**

My primary aim was to contribute evidence of dynamic transactions between maternal depressive symptoms and toddlers' emerging externalizing behavior to the growing literature on the bidirectional interplay of maternal depression and child adjustment problems (Gross et al., 2009; Nicholson, Deboeck, Farris, Boker, & Borkowski, 2011; Raposa, Hammen, & Brennan, 2011; Shaw et al., 2009). Multiple accounts have been made in the past 40 years describing transactional processes within mother-child dyads (Bell, 1968; Belsky, 1984; Cicchetti & Toth, 1997; Sameroff & Chandler, 1975). Yet, the adoption of a transactional perspective is still needed in most studies of maternal depression (Connell & Goodman, 2002; Dodge, 1990; Shaw et al., 2009). Findings initially refuted my transactional hypothesis, as I found that early exposure to maternal depressive symptoms in infancy contributed to children's externalizing behavior throughout toddlerhood. I explored the moderating effects of child gender and functional self-regulation in infancy and found both a gender interaction for maternal-effects on children's behavioral problems and a functional self-regulation

interaction for child-effects on mothers' depressive symptomatology. Moreover, I found evidence of a transactional process through which maternal depressive symptoms in infancy contributed to more externalizing behavior in toddlerhood, which in turn worsened mothers' symptoms, but only among toddlers who had high functional self-regulation at 7 months. Integrating a transactional approach with tests of interactions provided greater specificity in identifying complex bidirectional associations among a subset of mothers and children in a community sample. I discuss below the extent to which these methods confirmed my hypotheses and supported previous research.

### **The Early Interplay of Mother and Child Adjustment Problems**

I first hypothesized that there would be bidirectional effects of mothers' depressive symptoms and children's externalizing behavior in toddlerhood. Although the initial model with the full sample did not support this hypothesis and recent evidence of child effects (Nicholson et al., 2011; Raposa et al., 2011; Shaw et al., 2009), it demonstrated that exposure to maternal depressive symptoms at 7 months predicted more externalizing symptoms at 15 months and their increase at 33 months, while controlling for SES and contemporaneous depressive symptoms during toddlerhood. These findings support previous studies that found main effects of maternal depressive symptoms on young children's externalizing problems (e.g., Chronis et al., 2007; Cummings et al., 2005; Shaw et al., 2003; Weinfield et al., 2009). Although I measured maternal depressive symptoms at three time points, only the earliest ratings in infancy predicted externalizing behavior in toddlerhood. This is consistent with evidence of a sensitive period in infancy in which early exposure to maternal depression has more robust and enduring consequences for children's adjustment problems than exposure occurring later

in development (Connell & Goodman, 2002; Goodman & Gotlib, 1999; Lovejoy et al., 2000). I then turned my attention to gender differences to confirm reports that boys have more externalizing behavior and greater risk to contextual stressors than girls (Cummings & Davies, 1994; Davies & Windle, 1997; Deater-Deckard et al., 1998; Dodge & Pettit, 2003; Eiden et al., 2007; Farrington, 2009; Goodman & Gotlib, 1999; Yates, et al., 2010)

### **Gender Differences in Vulnerability to Maternal Depressive Symptoms**

I hypothesized that maternal depressive symptoms would have a greater effect on boys' externalizing behavior, which would reflect their heightened risk to early stress. Exposure at 7 months continued to predict more externalizing behavior at 15 months for both boys and girls. However, the effect of maternal depressive symptoms at 7 months on children's externalizing behavior at 33 months was moderated by gender. Exposure to maternal depressive symptoms in infancy predicted an increase in boys' behavioral problems from 15 to 33 months, but did not predict change in girls' externalizing behavior. Boys appeared to be more at risk than girls to the long-term effects of maternal symptomatology in infancy, which corroborates researchers who have suggested that boys are more sensitive to psychosocial stressors in early phases of development (Cummings & Davies, 1994; Davies & Windle, 1997; Dodge & Pettit, 2003; Goodman & Gotlib, 1999; Yates et al., 2010). This gender difference was not evident across 7 to 15 months, but emerged at 33 months, over two years after the initial assessment of depressive symptoms. Thus, mothers' symptomatology in infancy appeared to have a continuing effect on boys through toddlerhood. This was evident as children approached 3-years-old when boys typically begin displaying greater externalizing tendencies than girls (Farrington, 2009; Tremblay, 2000). No prediction was made as to whether the

effect of externalizing behavior would vary by gender, and I found no child-to-parent effects to suggest a difference. Next, I examined the moderating effects of infant regulatory competence to substantiate links among children's dysregulated functioning, externalizing problems, and maternal depression (Cummings & Davies, 1994; Cummings et al., 2005; Lengua, 2002).

### **Infant Regulatory Competence and Maternal Depressive Symptoms**

My third hypothesis was that maternal depressive symptoms would only contribute to the externalizing behavior of toddlers who had low levels of functional self-regulation in infancy. I did not find evidence supporting this. Similar to the earlier findings, maternal depressive symptoms at 7 months predicted higher levels and greater increases in externalizing behavior, respectively, at 15 and 33 months among all children, but this time while controlling for both SES and child gender. The effects of maternal depressive symptoms did not differ between infants who had high and low levels of functional self-regulation as I had expected to find. Yet consistent with previous research, infants who had more problems with crying, feeding, and sleeping had more externalizing symptoms as toddlers (Calkins, 2009; Hemmi et al., 2011; Wolke et al., 2009). Finding similar effects of maternal depressive symptoms on the behavioral problems for both groups, suggests that infants who vary in regulatory competence and later externalizing behavior do not differ in their early risk to maternal depression.

Transactional perspectives to early regulatory functioning assert that there are limits to the extent to which infant self-regulation can buffer noxious effects of poor rearing environments (Sameroff, 2009a; Sroufe, Duggal, Weinfield, & Carlson, 2000). There are caregiving situations that are so supportive that even dysregulated infants can

successfully adapt to typical developmental challenges. And conversely, there are environments marked by so many stressors that even infants with the best regulatory competence become overwhelmed, dysregulated, and develop a sequelae of adjustment problems. I found no evidence that infants' functional self-regulation contributed to differences in their vulnerability to maternal psychosocial stressors, despite research indicating moderating effects of self-regulation on stress for older children (Lengua, 2002; Lengua, Bush, Long, Kovacs, & Trancik 2008).

It is important to consider infants' self-regulation as reflecting the social context in which others provide regulation, rather than describing infant regulation as an individual characteristic (Olson & Lunkenheimer, 2009; Sameroff, 2009a; Sroufe et al., 2000). The present investigation aimed to identify child-centered moderators so I only included a measure of functional self-regulation at 7 months. While self-regulation is relatively stable over early childhood (Feldman, 2009), individual differences in regulatory competence may increase as children get older and accumulate experiences that support or hinder optimal growth in self-regulatory skills. Future endeavors that examine how self-regulatory skills develop in concert with maternal adjustment problems can help illustrate their transactional process and coordinated influence on externalizing behavior. Moreover, examining bidirectional effects of maternal and child adjustment problems can enhance understanding of child-effects on maternal depressive symptoms and mothers' vulnerability to them, as I found evidence for in the present study.

### **Infant Self-regulation Moderates Maternal Vulnerability to Child-effects**

An unexpected finding demonstrated that toddlers' externalizing behavior at 15 months predicted an *increase* in mothers' depressive symptoms at 33 months, but only



among toddlers who had high functional self-regulation in infancy. Given that these infants developed less externalizing behavior in toddlerhood than infants who had low self-regulation, it was surprising to find that their milder behavioral problems worsened mothers' depressive symptoms. This predictive effect may reflect a mismatch of high regulatory competence with elevated externalizing behavior, which mothers may perceive as more unusual and distressing than comorbid problems in regulation and behavior. Mothers of infants who had fewer crying, feeding, and sleeping problems but elevated behavioral problems later in toddlerhood may have been more likely to attribute their children's behavioral problems to their own parenting rather than to their children's negative characteristics, such as a difficult temperament. This in turn may have elevated maternal stress and depressive symptoms.

An alternative explanation for this finding, which is not mutually exclusive from the previous, is that the two groups of children differed qualitatively in the sorts of externalizing symptoms they demonstrated. Externalizing symptoms represent a heterogeneous constellation of behaviors from defiant and oppositional acts to aggressive and destructive behaviors (Hinshaw, 2002; Olson et al., 2009). Toddlers who had high regulatory competence in infancy may have displayed externalizing symptoms that are more chronically stressful for mothers than occasional acts of aggression or impulsivity. Future studies that compare the symptom profiles of young children who have varying levels of self-regulatory competence may reveal significant qualitative differences in their externalizing problems.

Children who had high functional self-regulation in infancy and their mothers were the only subgroup for whom I found evidence of a transactional association between

maternal depressive symptoms and children's externalizing behavior. By integrating tests of transactions and the interaction of a relatively stable attribute of young children, I discovered a bidirectional process that was hidden among data for the full sample. Future longitudinal research utilizing a similar method can contribute evidence of parent-child transactions that are unique to subgroups of children to the maternal depression literature.

### **Limitations**

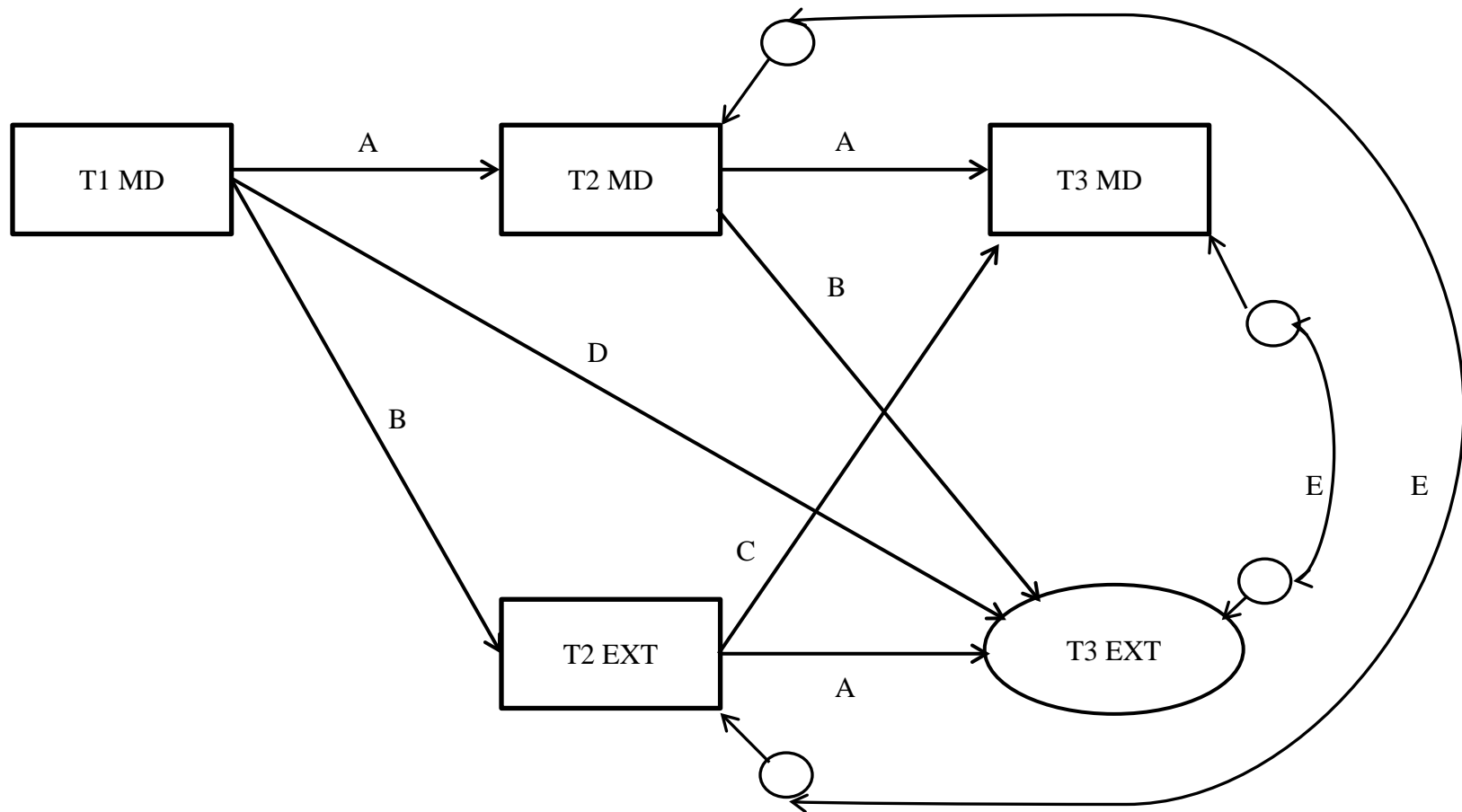
This study had several limitations that offset the generalizability of its findings. First, all data for the present study were drawn from maternal ratings, suggesting there may be problems with a lack of perspective and one method of measurement. A meta-analysis of maternal depression studies demonstrated that mother reports of child adjustment problems tended to yield larger effects than aggregated measures from multiple informants and methodologies (Connell & Goodman, 2002). Yet, stronger associations between infants' regulatory problems and externalizing behavior have been found with more rigorous measures of regulatory functioning such as interviews and expert-assessed behavioral outcomes (Hemmi et al., 2011). Future studies can build upon this investigation by using behavioral measures or experiments to assess infant regulatory problems, as well as alternative reporters for children's externalizing behavior to complement mother ratings. Additionally, associations between infants' regulation and maternal depressive symptoms are likely bidirectional and should be assessed at a moment-to-moment basis in the first few months (Goodman & Gotlib, 1999; Olson & Lunkenheimer, 2009; Sameroff, 2009a; Sroufe et al., 2000). Infants require extensive amounts of time and attention, which directly contributes to postpartum depressive symptoms (e.g., loss of sleep, extreme fatigue). This process is more pronounced when

infants have sleep regulatory problems associated with mothers' sleep difficulties, negative mood, and elevated stress (Bernier et al., 2010). Mothers are significant sources of regulation for infants and help lay a foundation for children's self-regulatory competence and vulnerability to negative stressors in toddlerhood (Bridgett et al., 2008). One of this investigation's strengths was having multiple measurement points within the first three years of children's lives, which allowed me to examine the early interface of their regulation, externalizing behavior, and maternal depressive symptoms. Relations among these constructs before 7-months-old are crucial to their later interplay and should be investigated further. Finally, this investigation included families recruited from local communities, so findings may not generalize to individuals with clinically significant symptomatology. To the best of my knowledge, neither mothers nor their young children were diagnosed with psychiatric disorders, although rates of mothers who met the CES-D's clinical cut-off were similar to national prevalence rates of depression. A recent study showed that adolescents' previous diagnoses predicted concurrent and later episodes of maternal depression with externalizing and comorbid psychopathology being particularly severe in elevating maternal stress and depression (Raposa et al., 2011). Findings demonstrated in this study may be even more pronounced when mothers have diagnosed depression. Future studies with clinical samples of mothers should be conducted to replicate and extend these findings.

## **Conclusion**

Women are at heightened risk of developing depression during their childbearing years and after giving birth, which puts young children at risk for early exposure to the behavioral and emotional sequelae of this disorder (Kessler et al., 2003; Lovejoy et al.,

2000; O'Hara & Swain, 1996). Infancy is a period of critical self-regulatory gains in autonomic processes such as feeding and sleeping (Feldman, 2009; Porges, 1996) and heightened vulnerability to negative effects of maternal depressive symptoms (Cummings & Davies, 1994; Goodman & Gotlib, 1999; Shaw et al., 2009). I replicated effects of maternal depressive symptoms on toddlers' externalizing behavior and corroborated that infancy is especially sensitive to their adverse consequences. Moreover, boys were found to be particularly susceptible to maternal depressive symptoms in infancy, leading to greater increases in their externalizing behavior as toddlers. I contributed evidence that functional self-regulatory competence interacts with behavioral problems to predict increases in maternal depressive symptoms. Difficulties with sleeping and feeding problems and excessive crying in infancy appeared to cascade to the emergence of externalizing behavior in toddlerhood. However, it was the externalizing behavior of toddlers who had fewer of these self-regulatory problems in infancy that worsened mothers' depressive symptoms. Early identification of self-regulatory deficits in infants and concurrent treatment of maternal depressive symptoms may help prevent toddler-age externalizing behavior and subsequent depressive episodes in mothers.



*Figure 2.1.* Analytic transactional model of maternal depressive symptoms (MD) across ages 7, 15, and 33 months and toddler externalizing behavior (EXT) at ages 15 and 33 months. Covariances between T1 and T3 MD and between T2 and T3 EXT are not shown to simplify the illustration. T1 = 7 months. T2 = 15 months. T3 = 33 months

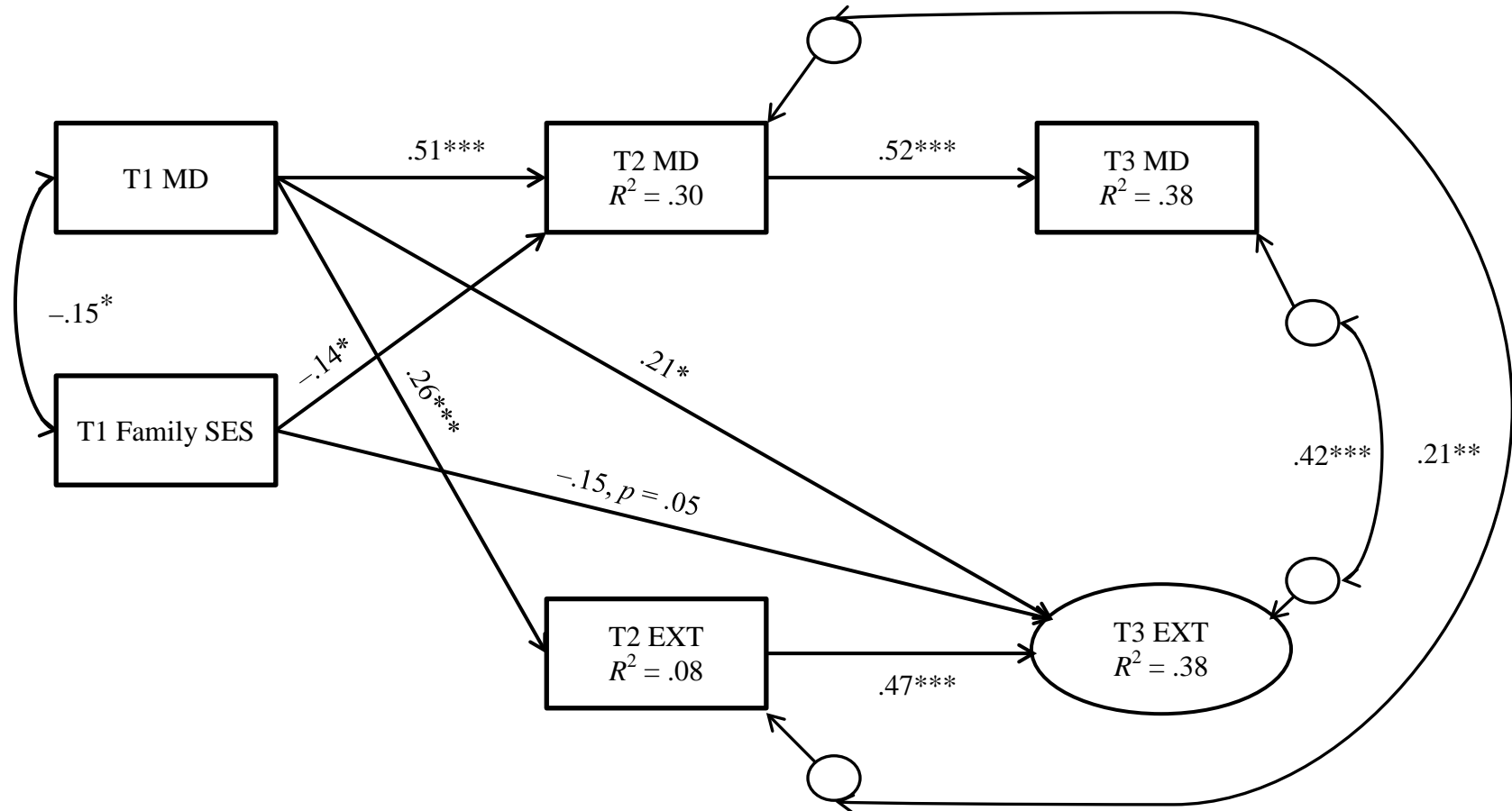


Figure 2.2. Transactional model of maternal depressive symptoms (MD) and externalizing behavior (EXT) from infancy to toddlerhood accounting for family socioeconomic status (SES).  $\chi^2(3, N = 250) = 2.12, p = .549$ . RMSEA = .00 [.00, .09]. CFI = 1.00. Nonsignificant effects are not shown. T1 = 7 months. T2 = 15 months. T3 = 33 months.  
 $*p < .05$ .  $**p < .01$ .  $***p < .001$ .

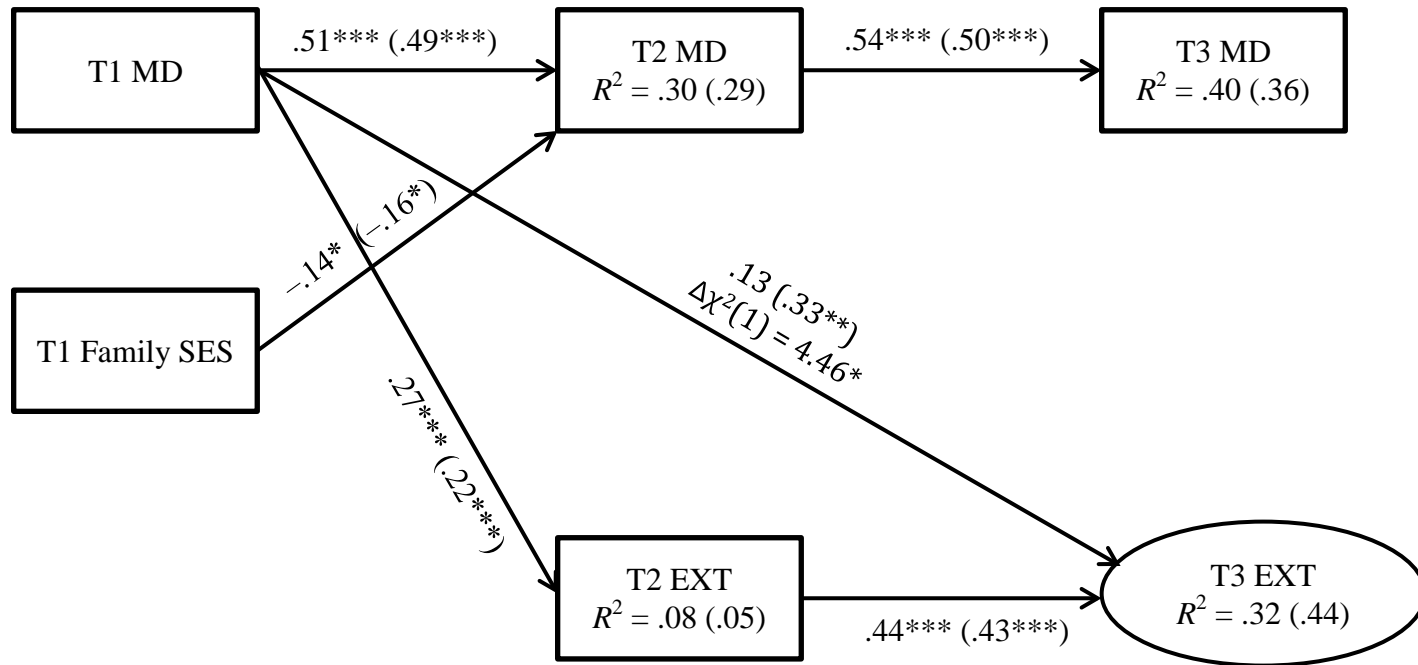


Figure 2.3. Multiple-group SEM results indicate that maternal depressive symptoms (MD) at 7 months (T1) predict more externalizing behavior (EXT) at 33 months (T3) *among only boys* (Standardized estimates in parentheses,  $n = 115$ ). Girls' estimates precede parentheses ( $n = 133$ ). Covariances, residuals, and nonsignificant paths not shown. Chi-square difference values ( $\Delta\chi^2$ ) indicate paths that differ by gender.  $\chi^2(25) = 30.54$ ,  $p = .205$ . RMSEA = .04 [.00, .09]. CFI = .99. T2 = 15 months. \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

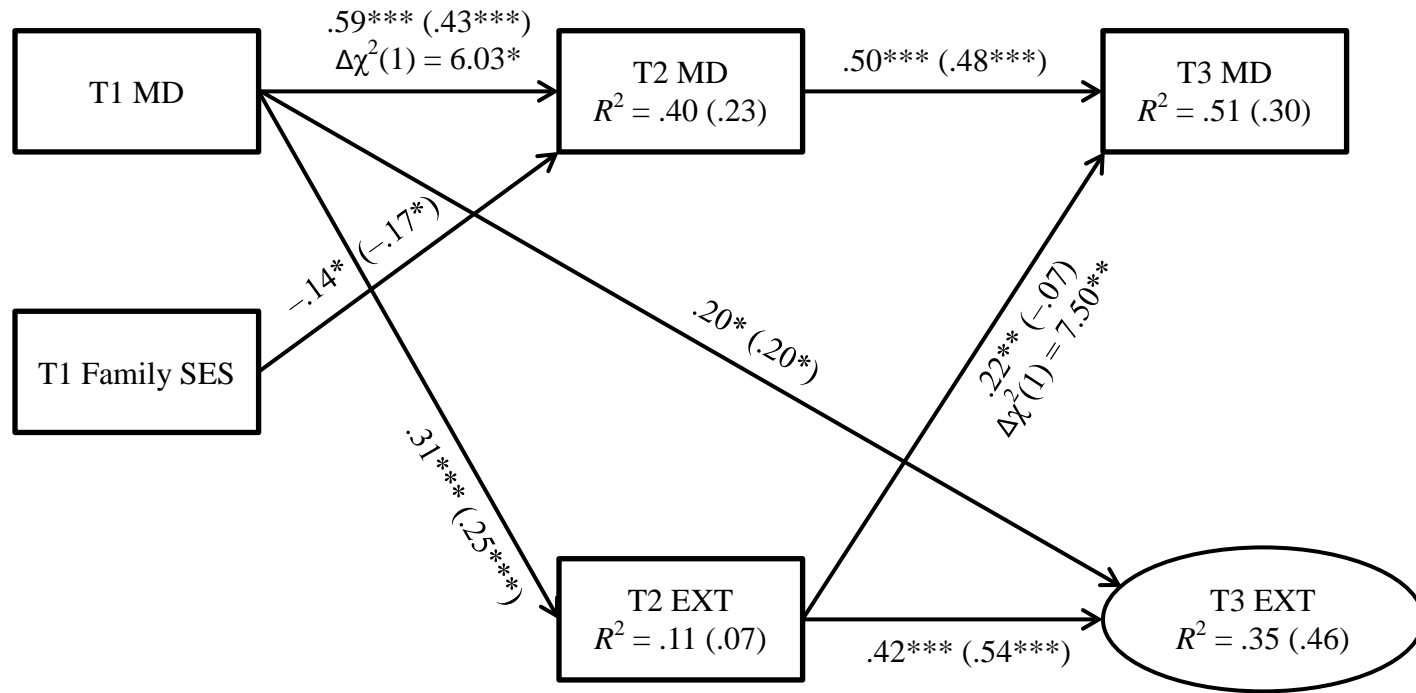


Figure 2.4. Transactional model of maternal depressive symptoms (MD) and externalizing behavior (EXT) accounting for family socioeconomic status (SES) and child gender (not shown). *Well-regulated infants'* standardized estimates precede parentheses ( $n = 119$ ), and *poorly-regulated infants'* estimates are within parentheses ( $n = 125$ ). Covariances, residuals, and nonsignificant paths not shown.  $\chi^2(30) = 29.60$ ,  $p = .486$ . RMSEA = .00 [.00, .07]. CFI = 1.00.

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



Table 1.1

*Correlations, Means, and Standard Deviations of Variables in Full Sample Structural Equation Model (N = 250)*

| Variables                          | 1      | 2      | 3      | 4      | 5      | 6      | 7     |
|------------------------------------|--------|--------|--------|--------|--------|--------|-------|
| 1. T1 Family SES                   | –      |        |        |        |        |        |       |
| 2. T1 Maternal Depressive Symptoms | –.15*  | –      |        |        |        |        |       |
| 3. T2 Maternal Depressive Symptoms | –.21** | .53*** | –      |        |        |        |       |
| 4. T2 Externalizing Behavior ITSEA | –.10   | .26*** | .32*** | –      |        |        |       |
| 5. T3 Maternal Depressive Symptoms | –.20*  | .44*** | .62*** | .28*** | –      |        |       |
| 6. T3 Externalizing Behavior ITSEA | –.19*  | .26*** | .26*** | .54*** | .39*** | –      |       |
| 7. T3 Externalizing Behavior CBCL  | –.17*  | .31*** | .26*** | .46*** | .44*** | .66*** | –     |
| <i>M</i>                           | 46.63  | 11.05  | 10.97  | .57    | 10.20  | .53    | 11.89 |
| <i>SD</i>                          | 12.19  | 8.25   | 9.12   | .27    | 8.73   | .29    | 6.26  |

*Note.* T1 = 7 months. T2 = 15 months. T3 = 33 months. SES = Socioeconomic Status. ITSEA = Infant–Toddler Social and Emotional Assessment. CBCL = Child Behavior Checklist.

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

Table 1.2

*Correlations, Means, and Standard Deviations of Variables in Multiple Group Structural Equation Model Comparing Boys (Below Diagonal, n = 115) and Girls (Above Diagonal, n = 133)*

| Variables                          | 1     | 2      | 3      | 4      | 5      | 6      | 7      | M (SD)        |
|------------------------------------|-------|--------|--------|--------|--------|--------|--------|---------------|
| 1. T1 Family SES                   | –     | –.14   | –.19   | –.10   | –.16   | –.29** | –.21*  | 45.55 (11.82) |
| 2. T1 Maternal Depressive Symptoms | –.18  | –      | .61*** | .24*   | .45*** | .24*   | .16    | 11.01 (8.55)  |
| 3. T2 Maternal Depressive Symptoms | –.27* | .41*** | –      | .27**  | .65*** | .20*   | .19*   | 10.88 (9.61)  |
| 4. T2 Externalizing Behavior ITSEA | –.13  | .30**  | .40*** | –      | .20*   | .52*** | .37*** | .55 (.25)     |
| 5. T3 Maternal Depressive Symptoms | –.24* | .43*** | .59*** | .35**  | –      | .30**  | .32**  | 10.48 (8.69)  |
| 6. T3 Externalizing Behavior ITSEA | –.12  | .31**  | .36**  | .55*** | .49*** | –      | .57*** | .50 (.27)     |
| 7. T3 Externalizing Behavior CBCL  | –.13  | .51*** | .36**  | .52*** | .57*** | .74*** | –      | 11.66 (5.86)  |
| M                                  | 48.30 | 11.11  | 11.07  | .59    | 9.84   | .56    | 12.09  |               |
| SD                                 | 12.41 | 7.92   | 8.54   | .30    | 8.81   | .31    | 6.82   |               |

*Note.* T1 = 7 months. T2 = 15 months. T3 = 33 months. SES = Socioeconomic Status. ITSEA = Infant–Toddler Social and Emotional Assessment. CBCL = Child Behavior Checklist. Variable means did not differ between boys and girls.

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

Table 1.3

*Correlations, Means, and Standard Deviations of Variables in Multiple Group Structural Equation Model Comparing Children Who had Low Functional Self-regulation in Infancy (Below Diagonal) and Children Who had High Self-regulation (Above Diagonal)*

| Variables                             | 1   | 2     | 3      | 4      | 5      | 6      | 7      | 8      | <i>M (SD)</i> |
|---------------------------------------|-----|-------|--------|--------|--------|--------|--------|--------|---------------|
| 1. Child Gender (1 = girls, 2 = boys) | –   | .11   | –.13   | –.06   | .08    | –.15   | .02    | –.05   | –             |
| 2. T1 Family SES                      | .10 | –     | –.24*  | –.21*  | –.03   | –.22*  | –.17   | –.24*  | 48.05 (11.90) |
| 3. T1 Maternal Depressive Symptoms    | .13 | –.06  | –      | .64*** | .35*** | .51*** | .35**  | .31**  | 10.29 (8.14)  |
| 4. T2 Maternal Depressive Symptoms    | .08 | –.25* | .42*** | –      | .48*** | .67*** | .39*** | .25*   | 11.03 (9.94)  |
| 5. T2 Externalizing Behavior ITSEA**  | .07 | –.14  | .19    | .24*   | –      | .52*** | .59*** | .47*** | .51 (.24)     |
| 6. T3 Maternal Depressive Symptoms    | .07 | –.17  | .39*** | .59*** | .12    | –      | .53*** | .45*** | 10.04 (8.87)  |
| 7. T3 Externalizing Behavior ITSEA*   | .16 | –.20  | .19    | .17    | .49*** | .27**  | –      | .75*** | .48 (.27)     |
| 8. T3 Externalizing Behavior CBCL*    | .10 | –.09  | .30**  | .28**  | .44*** | .42*** | .58*** | –      | 10.87 (6.19)  |
| <i>M</i>                              | –   | 45.54 | 11.77  | 10.91  | .62    | 10.35  | .57    | 12.74  |               |
| <i>SD</i>                             | –   | 12.31 | 8.32   | 8.34   | .29    | 8.63   | .30    | 6.29   |               |

*Note.* T1 = 7 months. T2 = 15 months. T3 = 33 months. SES = Socioeconomic Status. ITSEA = Infant–Toddler Social and Emotional Assessment. CBCL = Child Behavior Checklist. Asterisks on variables names indicate differences across groups. \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

### **Chapter III**

#### **Effortful Control Moderates Transactions of Externalizing Behavior and Maternal Depressive Symptoms across Childhood**

High prevalence rates of depressive symptoms among mothers and women of childbearing age elevate young children's risk for later externalizing problems (Kessler et al., 2003; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Most preschool-age children who show disruptive behavior problems gradually cease demonstrating them after school entry (Shaw, Gilliom, Ingoldsby, & Nagin, 2003). Family psychosocial stressors jeopardize this normative decrease and elevate risk for antisocial behavior in adolescence and adulthood (Aguilar, Sroufe, Egeland, & Carlson, 2000; Campbell, Shaw, & Gilliom, 2000; Moffitt, Caspi, Harrington, & Milne, 2002). For example, maternal depressive symptoms have been found to exacerbate young children's externalizing behavior and predict more serious problems in later childhood and adolescence (Chronis et al., 2007; Cummings, Keller, & Davies, 2005; Du Rocher Schudlich & Cummings, 2007; Gartstein & Fagot, 2003; Goodman & Gotlib, 1999; Weinfield, Ingerski, & Moreau, 2009). Children's behavioral problems also have been shown to worsen mothers' depressive symptoms (e.g., Shaw, Gross, & Moilanen, 2009). Few researchers have examined bidirectional effects of maternal depressive symptoms and children's externalizing behavior, despite strong theoretical support for reciprocal influences in mother-child dyads, such as the transactional model of development (Bell, 1968; Connell & Goodman, 2002; Dodge, 1990; Sameroff, 2009a). A transactional model can provide greater

explanatory power than typical unidirectional models when examining how maternal depressive symptoms and young children's early externalizing behavior contribute to the progression of externalizing problems.

In addition, identifying stable attributes of young children that moderate bidirectional effects of risk factors can clarify mechanisms that help children overcome psychosocial stressors (Compas & Reeslund, 2009). In the following longitudinal study, I proposed that children's effortful control, a set of temperament-based self-regulatory abilities associated with externalizing problems, moderates the transactional interplay of maternal depressive symptoms and child externalizing behavior (Lengua, 2002; Posner & Rothbart, 2000). I predicted that advanced levels of effortful control during the preschool years would buffer future bidirectional effects of externalizing behavior and maternal depressive symptoms, subsequently leading to fewer externalizing problems across childhood. As described below, a transactional model provides a framework for understanding these complex psychosocial exchanges occurring across development.

### **Transactional Framework**

The transactional model of development describes the continuous dynamic interplay of the individual and his or her social environment (Sameroff, 2009a). The model elucidates bidirectional, interdependent effects between child-centered and social-contextual variables that influence a developmental process (Sameroff, 2009b; Sameroff & Mackenzie, 2003). This framework places ongoing parent-child transactions at the core of socialization processes, thereby acknowledging both children's active roles in shaping their development and the plasticity of children and their social environments (MacKenzie & McDonough, 2009). The transactional perspective of developmental

psychopathology is a more recent adaptation of the former model that views adjustment problems as developing from ongoing transactions between the child and social-context, rather than from singular characteristics from either (Cicchetti & Toth, 1997; Gross, Shaw, Burwell, & Nagin, 2009). The current study incorporated this perspective when testing a transactional model of maternal depressive symptoms and children's externalizing behavior across the preschool years to middle childhood (See Figure 3.1).

Most previous researchers have focused on how mothers influence children's behavioral problems with little attention directed at how children also may exacerbate mothers' depressive symptoms (Connell & Goodman, 2002; Dodge, 1990; Gross et al., 2009; Shaw et al., 2009). For example, children exposed to high levels of maternal depressive symptoms during the preschool and early school years were found to have more externalizing problems at age 9 that increased at a faster rate than for other children (Munson, McMahon, & Spieker, 2001). These findings are informative for understanding growth of externalizing problems, but they tell us little about risk mechanisms contributing to mothers' depressive symptoms or their aggregate effect on children's later functioning. Examining young children's externalizing behavior and mothers' depressive symptoms in a transactional model can elucidate their cumulative and bidirectional effects as early risk factors for later adjustment problems in both children and mothers. In a recent study of at-risk boys, Gross and colleagues (2009) found that more disruptive behavior problems in toddlerhood were associated with a greater risk of persistent exposure to maternal depressive symptoms, which in turn predicted more antisocial behavior problems in adolescence. The current study attempted to extend these findings to both boy and girls and contribute evidence that certain constitutional differences in

young children moderate transactional associations between maternal depressive symptoms and child externalizing behavior.

Many young children who are exposed to maternal depressive symptoms and those who demonstrate early externalizing behavior do not progress to more serious and pervasive forms of adjustment problems (Berg-Nielsen, Vikan, & Dahl, 2002; Campbell et al., 2000; Olson, Sameroff, Lunkenheimer, & Kerr, 2009). Children's temperament, self-regulation in particular, can influence this process by contributing to individual differences in responses to psychosocial risk factors (Compas & Reeslund, 2009; Lengua, 2002). In addition to testing bidirectional effects in a transactional model, this study used a person-focused approach common in *resilience* research to test whether preschool-age levels of effortful control moderated risk mechanisms contributing to later externalizing problems (Luthar & Cicchetti, 2000; Masten, 2001). The potential moderating influence of children's effortful control is discussed below.

### **The Role of Children's Effortful Control in Stress and Coping**

Child effortful control (EC) is defined as a child's capacity to voluntarily inhibit a dominant response and initiate a subdominant response (Kochanska & Aksan, 2006; Posner & Rothbart, 2000; Rothbart & Bates, 1998). The unique characteristic of children's EC that distinguishes it from other constituents of temperament-based self-regulation is its role in the executive control of attention to facilitate goal-directed behavior (Bell & Deater-Deckard, 2007; Posner & Rothbart, 2000). Developmental gains in children's EC reflect an increasing flexibility and volitional control of emotional and behavioral impulses and attention across multiple settings (Kochanska & Aksan, 2006; Rothbart & Bates, 1998). Children's effortful control begins to emerge in late infancy and

develops rapidly between the ages of 2 and 6 (Bell & Deater-Deckard, 2007; Rothbart & Bates, 2006). Individual differences in EC emerge in infancy and remain moderately stable across early to middle childhood (Kochanska & Aksan, 2006; Kochanska et al., 2000; Posner & Rothbart, 2000). The stability of this construct, and its role in the selection, coordination, and storage of information, has implicated effortful control as a critical contributor to children's socialization, personality, and psychopathology (Bell & Deater-Deckard, 2007; Posner & Rothbart, 2000).

Studies have consistently shown that low levels of EC are associated with more early externalizing problems in preschool-age children and older (Eisenberg et al., 2005; Martel & Nigg, 2006; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005; Rothbart & Bates, 2006). More recently, researchers have linked children's poor EC to elevated maternal depressive symptoms (Choe, Olson, & Sameroff, in press; Lengua, Bush, Long, Kovacs, & Trancik, 2008; Sektnan, McClelland, Acock, & Morrison, 2009). High levels of EC in the preschool and the early school years have been shown to protect against risk factors by buffering their effects on externalizing outcomes (Lengua, 2002; Lengua et al., 2008). Children who have more advanced EC in early childhood may be less vulnerable to risks associated with maternal depressive symptoms and may subsequently develop fewer externalizing problems. Few researchers have found support for moderating effects of child temperament in the maternal depression literature (Connell & Goodman, 2002). This study was intended to fill this void by testing whether preschool-age EC moderated transactions of maternal depressive symptoms and child externalizing behavior.

Differences between girls and boys in the manifestation of externalizing problems and the development of self-regulation qualify tests of gender effects in the transactional



model (Eiden, Edwards, & Leonard, 2007; Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006). Girls tend to have more advanced EC and fewer externalizing problems than boys in early childhood (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Olson et al., 2005; Valiente et al., 2006). Some research suggests that effects of maternal depressive symptoms on child adjustment problems may differ by gender, although the evidence is inconclusive (Connell & Goodman, 2002; Cummings & Davies, 1994; Davies & Windle, 1997). Thus, child gender was examined in all major analyses.

### **The Current Study**

Examining transactions and interactions of child-centered and social-contextual variables can enhance explanatory models of children's externalizing problems (Hinshaw, 2002, pp. 434). To advance understanding of risk mechanisms contributing to externalizing problems, I tested whether preschool-age levels of EC moderated bidirectional effects of maternal depressive symptoms and children's externalizing behavior in a transactional model spanning across early to middle childhood (See Figure 3.1). Many previous studies of maternal depression and child development have been constrained by small samples, short-term follow-ups, retrospective reporting, lack of developmental perspective, and use of unidirectional models focusing on maternal effects on children (Campbell et al., 2007; Goodman & Gotlib, 1999; Shaw et al., 2009). This prospective longitudinal study included a relatively large community sample with data collected via multiple methods and from different informants at ages 3, 6, and 10.

The first aim of this study was to determine whether maternal depressive symptoms and child externalizing behavior showed evidence of reciprocal influence. Based on previous research (Chronis et al., 2007; Gross et al., 2009; Weinfield et al.,

2009), I hypothesized that elevated levels of maternal depressive symptoms would predict increases in children's externalizing behavior and that children's externalizing behavior would predict increases in mothers' depressive symptoms. The second aim was to determine whether preschool-age levels of EC moderated the interplay of maternal depressive symptoms and child externalizing behavior. I hypothesized that maternal depressive symptoms would predict increases in children's externalizing behavior only among children with low levels of EC in the preschool years (Lengua, 2002; Lengua et al., 2008). Children's externalizing behavior was expected to predict increases in maternal depressive symptoms among all children.

## **Method**

### **Participants**

This study included 224 children (47% girls), mothers, and subsamples of teachers involved in an ongoing longitudinal study of children considered to be at risk for school-age behavioral problems (Olson et al., 2005). Among participating children, 85% are European American, 8% are biracial, 5% are African American, and 2% represent other racial-ethnic groups. During recruitment from 1999 to 2001, 44% of mothers and 30% of fathers reported a bachelor's degree as their highest level of educational attainment and another 39% of mothers and 46% of fathers reported receiving some graduate or professional training. Hollingshead (1979) four-factor scores for family socioeconomic status (SES) were created with the occupational status and level of education attainment of each parent living in the household at recruitment. Scores for family SES ranged from 22 to 66 ( $M = 54.41$ ,  $SD = 10.98$ ) representing the top four of five social strata in the Hollingshead system. The majority of families (87%) resided in

the two highest social strata. The average annual family income was between \$60,000 and \$70,000 [\$10,000 to \$100,000+]. Two families did not report this information.

### **Procedure**

Families with preschool-age children living in communities surrounding a university in the Midwest were recruited through newspaper advertisements, fliers at childcare centers, and pediatrician referral. Advertisement targeted either hard-to-manage toddlers or typical-developing toddlers. Parents who expressed interest filled out a screening questionnaire and were briefly interviewed by telephone to determine whether families were appropriate for and could participate in a longitudinal study. Preschool-age children were recruited to represent the full range of Externalizing Problems on the Child Behavior Checklist for Ages 2–3 (CBCL 2/3; Achenbach, 1992). To meet this recruitment quota, there was an oversampling of preschoolers who were rated by their mother as demonstrating elevated externalizing behavior. Approximately 66 children (30%) were rated in the borderline clinical range for Externalizing Problems ( $T \geq 60$ ) and 27 children (12%) were rated in the clinical range ( $T \geq 64$ ). Families experiencing extreme economic hardship were not included to allow investigators to focus on parental influences rather than effects of severe environmental adversity. Children with chronic health problems, physical disabilities, or severe cognitive deficits were excluded.

A female social worker interviewed mothers in their homes to collect demographic information and administer a packet of questionnaires assessing children's behavioral adjustment and mothers' psychological distress. These in-home interviews were conducted at the first two waves of data collection. Data collection for the third wave was carried out entirely online through electronic questionnaires. In addition,

subsamples of children's teachers provided ratings of child adjustment problems at each wave. Mothers and teachers completed questionnaires at wave 1 (W1) when children were about 3-years-old ( $M = 37.74$  months,  $SD = 2.71$ ), at wave 2 (W2) when children were almost 6-years-old ( $M = 63.47$  months,  $SD = 2.69$ ), and at wave 3 (W3) when children were about 10-years-old ( $M = 10.46$  years,  $SD = .60$ ). Families were paid modestly for participating. About 80% of children's preschool teachers provided ratings of externalizing behavior at W1, 83% in the early school years at W2, and 83% in middle childhood at W3. Teachers received gift certificates for participating.

Self-regulation was directly assessed when children were about 3.5-years-old ( $M = 41.39$  months,  $SD = 2.09$ ) in 3- to 4-hour behavioral assessments administered at a local preschool on Saturday mornings. After building a rapport with children and obtaining their assent, graduate student testers administered self-regulatory tasks. Children received small gifts for participating.

## **Measures**

**Maternal depressive symptoms.** Mothers were administered the Brief Symptom Inventory (BSI) self-report measure of adult psychological distress (Derogatis, 1993; Derogatis & Fitzpatrick, 2004). Mothers rated their levels of distress in the last week to 53 items using a 5-point scale: 0 = "not at all" to 4 = "extremely". This study only includes the depression scale, which consisted of six items (avg.  $\alpha = .81$ ) assessing dysphoric mood, loneliness, feeling blue, lack of interest in things, suicidal ideation, and feelings of worthlessness and hopelessness.

Mothers reported a wide range of depression scores when compared to the BSI's subscale norms for nonpatient adult females (Derogatis, 1993). At each wave, mothers

reported an average depression score (avg.  $M = .26$ ) close to the 60<sup>th</sup> percentile for nonpatient women ( $T = 52$ ). Across the study, about 45% of mothers reported zero depressive symptoms, about 16% scored at the 50<sup>th</sup> percentile ( $T = 50$ ), about 20% scored around the 70<sup>th</sup> percentile range ( $T = 54-57$ ), and around 12% scored at or above the 84<sup>th</sup> percentile ( $T \geq 60$ ). Twelve mothers (5%) did not report this information at W2, and 42 mothers (19%) did not report this at W3.

**Externalizing behavior.** Teacher-reported raw scores for Externalizing Problems were used to measure children's externalizing behavior. Preschool teachers at W1 completed the Caregiver-Teacher Report Form for Ages 1½–5 (CTRF; Achenbach, 1997). The CTRF's Externalizing Problem score ( $\alpha = .96$ ) consisted of two highly correlated scales ( $r = .79, p < .001$ ): 17 items for Attention Problems ( $\alpha = .92$ ) and 23 items for Aggressive Behavior ( $\alpha = .94$ ). Teachers at W2 and W3 completed the Teacher's Report Form for Ages 6–18 (TRF; Achenbach & Rescorla, 2001; Achenbach, Dumenci, & Rescorla, 2002). The TRF's Externalizing Problem score (avg.  $\alpha = .94$ ) consisted of two highly correlated scales (avg.  $r = .76, p < .001$ ): 12 items for Rule-Breaking Behavior (avg.  $\alpha = .65$ ) and 20 items for Aggressive Behavior (avg.  $\alpha = .95$ ).

In addition, mothers' reported on children's externalizing behavior at W1 using the CBCL 2/3 (Achenbach, 1992) and at W2 and W3 using the CBCL 6/18 (Achenbach et al., 2002; Achenbach & Rescorla, 2001). The proportions of children rated by mothers and teachers in the borderline clinical and clinical ranges at all three waves were contrasted. Across the study, 14% of mothers rated children in the borderline clinical range for the Externalizing Problem scale ( $T \geq 60$ ) and 10% rated children in the clinical

range ( $T \geq 64$ ). In contrast, 5% of teachers rated children in the borderline clinical range for the Externalizing Problem scale and 9% rated children in the clinical range.

**Effortful control.** Child temperament, defined as individual differences in reactivity and self-regulation (Posner & Rothbart, 2000), was assessed by mother-report at W1 using an abbreviated version of the Child Behavior Questionnaire (CBQ; Ahadi, Rothbart, & Ye, 1993). CBQ scales for Inhibitory Control ( $\alpha = .72$ , 13 items) and Attentional Focusing ( $\alpha = .85$ , 14 items) represent constituents of temperament closely related to children's EC (Rothbart & Bates, 1998). Scores for Inhibitory Control and Attentional Focusing were aggregated with the total score of a toddler-age behavioral battery (Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996) assessing individual differences in toddlers' EC via six tasks ( $\alpha = .70$ ), such as the whisper, tongue, and gift delay tasks described in detail in Olson et al. (2005). All tasks were administered as games and children were reminded of the rules halfway through each. This composite score of W1 EC was dichotomized by median-split to create two groups of 112 children referred to as the *well-regulated children* and the *poorly-regulated children*.

### **Overview of Data Analysis**

Preliminary analyses focused on sample attrition and patterns of missing data. Descriptive statistics of study variables were calculated. Group differences on study variables were then examined by independent group *t*-tests to compare the *well-regulated* and *poorly-regulated children*. Next, mean changes over time in children's externalizing problems and maternal depressive symptoms were examined using repeated measure *t*-tests for the full sample and for each EC group. Subsequently, structural equation modeling (SEM) was conducted with *Mplus 6* (Muthén & Muthén, 2010) to test

bidirectional effects of maternal depressive symptoms and child externalizing behavior. As shown in Figure 3.1, a transactional model was created with repeated measures of maternal depressive symptoms and children's externalizing behavior across three waves. It consisted of first- and second-order lagged effects that accounted for the stability of constructs over time (A's and B's), first- and second-order cross-lagged effects that represented their prediction of change in one another over time (C's and D's), and three covariances that accounted for shared variance from similar times of measurement (E's). After examining the transactional model with the full sample, multiple-group SEM was conducted to test for the interaction of child gender. Boys were coded as zero and girls as one. After testing for moderating effects of child gender, it was included as a covariate when comparing the *well-regulated* and *poorly-regulated children* in a multiple-group analysis of preschool-age EC. All other analyses were conducted using SPSS 19.0.

## Results

### Attrition and Missing Data

Approximately 91% of the sample continued to participate in the study at W3, while 21 families stopped participating mainly due to family relocation. Families who stopped participating and those remaining in the study were compared with *t*-tests on all demographic variables and study measures. No group differences for study variables were found, but there were demographic differences. Fathers in the attrition group reported a lower average level of educational achievement at recruitment ( $M = 5.60$ ,  $SD = 1.23$ ; "partial college") than fathers of families who continued participating ( $M = 6.17$ ,  $SD = 1.03$ ; "bachelor's degree"),  $t(216) = 2.33$ ,  $p = .021$ . Families that left the study reported a lower average annual household income at recruitment ( $M = 7.50$ ,  $SD = 4.11$ ;

“\$40,000-\$50,000”) than remaining families ( $M = 9.46, SD = 2.85$ ; “\$60,000-\$70,000”),  $t(20) = 2.09, p = .049$ . Families in the attrition group did not differ on major study variables from the remaining families, so their data were included in final analyses.

Teachers’ ratings of externalizing behavior were missing for 44 children (20%) at W1, and 38 children (17%) at W2 and W3. Children missing teacher ratings of their externalizing behavior at W2 were more likely to have lower teacher ratings of externalizing behavior at W1 ( $M = 6.94, SD = 8.79$ ) than children who had these data ( $M = 10.78, SD = 13.13$ ),  $t(61) = -2.01, p = .049$ . Mothers who did not report their depressive symptoms at W3 ( $M = 8.22, SD = 3.67$ ) had a lower annual family income at recruitment than mothers who reported these data ( $M = 9.53, SD = 2.82$ ),  $t(51) = -2.14, p = .037$ . No other differences based on missing data were found.

### **Descriptive Statistics**

Table 2.1 shows the correlations, means, and standard deviations of study variables for the full sample. As reported in earlier studies of this sample, girls had higher levels of EC at W1 and lower levels of externalizing behavior than boys at all waves (Choe et al., in press; Olson et al., 2005). Furthermore, higher levels of EC at W1 were associated with lower levels of externalizing behavior at all waves. Scores for maternal depressive symptoms and child externalizing behavior were positively correlated with their repeated measures across time, as well as with one another to a lesser extent.

Table 2.2 shows descriptive statistics of study variables for the multiple group SEM model comparing poorly-regulated and well-regulated children. Being a girl was associated with more W1 maternal depressive symptoms in the well-regulated group and less W3 externalizing behavior in the poorly-regulated group. For the poorly-regulated



group: W1 EC was negatively related to W1 maternal depressive symptoms and W3 externalizing behavior; W1 and W2 maternal depressive symptoms were positively correlated with W3 externalizing behavior; and W1 externalizing behavior was positively correlated with W2 and W3 maternal depressive symptoms. No associations were found for the well-regulated group, except among repeated measures, which were positively correlated with one another in both groups.

### **Independent Group and Repeated Measure *t*-tests**

Figure 3.2 illustrates levels of teacher-reported Externalizing Problem raw scores for the full sample (solid black), the *poorly-regulated group* (thin dotted), and the *well-regulated group* (thick dotted). Group differences were examined with independent group *t*-tests comparing well-regulated and poorly-regulated children. Results indicated that the *well-regulated children* had fewer externalizing problems at each wave than the *poorly-regulated children*. At W1, the *poorly-regulated group* ( $M = 13.82, SD = 14.18$ ) had more than double the externalizing behavior of the *well-regulated group* ( $M = 6.65, SD = 9.69$ ),  $t(150) = 3.93, p < .001$ . At W2, a similar difference emerged between the *poorly-regulated group* ( $M = 6.30, SD = 9.72$ ) and the *well-regulated group* ( $M = 2.49, SD = 5.67$ ),  $t(148) = 3.26, p = .001$ . And again at W3, the *poorly-regulated children* ( $M = 4.85, SD = 7.36$ ) had more than double the externalizing behavior of their *well-regulated peers* ( $M = 1.83, SD = 3.75$ ),  $t(143) = 3.56, p = .001$ . As shown in Figure 3.3, no group differences in maternal depressive symptoms were found between groups.

Figure 3.2 shows a solid black line representing teacher-reported Externalizing Problem raw scores at ages 3, 6, to 10, for the full sample. For all children, externalizing behavior decreased from W1 ( $M = 10.78, SD = 13.13$ ) to W2 ( $M = 4.21, SD = 7.40$ ),

$t(148) = 6.68, p < .001$ , but did not change from W2 ( $M = 4.17, SD = 8.08$ ) to W3 ( $M = 3.45, SD = 6.23$ ),  $t(162) = 1.43, ns$ . Children's externalizing behavior was greater at W1 ( $M = 9.93, SD = 12.59$ ) than at W3 ( $M = 3.26, SD = 5.91$ ),  $t(150) = 7.25, p < .001$ .

Figure 3.2 shows a thin-dotted line and a thick-dotted line that respectively represent levels of externalizing behavior for children in the poorly-regulated and well-regulated groups. Poorly-regulated children's externalizing behavior was greater at W1 ( $M = 14.59, SD = 14.70$ ) than at both W2 ( $M = 5.97, SD = 8.41$ ),  $t(72) = 5.27, p < .001$ , and W3 ( $M = 4.60, SD = 7.08$ ),  $t(76) = 6.04, p < .001$ . Their externalizing behavior did not change from W2 to W3,  $t(84) = 1.12, ns$ . Similar changes in externalizing problems were observed among the well-regulated children. Well-regulated children's externalizing behavior was greater at W1 ( $M = 7.11, SD = 10.26$ ) than at both W2 ( $M = 2.51, SD = 5.84$ ),  $t(75) = 4.26, p < .001$ , and W3 ( $M = 1.86, SD = 3.96$ ),  $t(73) = 4.20, p < .001$ . Their externalizing behavior also did not change from W2 to W3,  $t(77) = .90, ns$ .

As shown in Figure 3.3, the well-regulated children's maternal depressive symptoms significantly decreased from W1 ( $M = .29, SD = .44$ ) to W3 ( $M = .19, SD = .32$ ),  $t(89) = 2.43, p = .017$ . No other changes in maternal depressive symptoms were found for either group of children. Both groups demonstrated a substantial decline in externalizing behavior between ages 3 and 6, followed by relatively stable externalizing behavior through age 10. Despite showing a similar trend in change over time, the well-regulated children demonstrated less than half the level of externalizing behavior of poorly-regulated children at each wave. Maternal depressive symptoms decreased from ages 3 to 10 for only the well-regulated children.

### **Structural Equation Modeling**

For all SEM results, only standardized values that were significant according to  $p$ -values from their unstandardized estimates are presented in the figures and in the text. Furthermore, all reported  $p$ -values are from unstandardized estimates, which more accurately account for standard errors than their standardized values (Kline, 2004). Figure 3.4 shows SEM results of the transactional model with the full sample:  $\chi^2(0) = .00$ ,  $p < .001$ . CFI = 1.00. RMSEA < .001 [.00, .00]. Fit indices indicated a perfect fit of the data to the fully-saturated model. Almost all lagged effects were significant, indicating that children's externalizing behavior was stable across all waves of assessment and that maternal depressive symptoms were stable from one wave to the next. None of the cross-lagged effects were significant, indicating the absence of bidirectional effects among the full sample. The model explained 22% of the variance of W2 externalizing behavior, 29% of W2 maternal depressive symptoms, 44% of W3 externalizing behavior, and 40% of W3 maternal depressive symptoms.

### **Multiple Group Structural Equation Modeling**

**Child gender effects.** After examining the transactional model with the full sample, I conducted multiple-group SEM to identify moderating effects of child gender (see Figure 3.5). An incremental approach to achieving the best fitting and most parsimonious model was followed. One structural parameter was constrained to be equal across groups and the overall model fit was compared to an unconstrained model using a chi-square difference test ( $\Delta\chi^2$ ). If the equality constraint worsened the overall fit of the model, I eliminated the constraint in the next nested model. If the constraint did not alter the fit, it was included in the next nested model testing an additional constraint until the best fitting and most parsimonious model was identified. Following these steps, I

identified the model shown in Figure 3.5:  $\chi^2(13) = 10.19, p = .68$ . CFI = 1.00. RMSEA = .00 [.00, .08]. Fit indices indicated a close approximate fit of the data to the model. As depicted in Figure 3.5, the model had two unconstrained effects: maternal depressive symptoms at W1 predicted an increase in externalizing behavior at W3 *among only boys* ( $\beta = .24, p = .001$ ),  $\Delta\chi^2(1) = 33.71, p < .001$ ; and *girls* had less stable externalizing behavior from W2 to W3 ( $\beta = .41, p < .001$ ) than *boys* ( $\beta = .61, p < .001$ ),  $\Delta\chi^2(1) = 11.37, p < .001$ . Increases in boys' externalizing behavior at W3 were predicted by mothers' depressive symptoms at W1, and their externalizing behavior was more stable from W2 to W3 than for girls. I included gender as a covariate in the next model testing for moderating effects of child EC.

**High and low effortful control.** I followed the same incremental approach to achieving the best fitting and most parsimonious model when comparing children by their preschool-age EC:  $\chi^2(18) = 11.66, p = .86$ . CFI = 1.00. RMSEA < .001 [.00, .05]. Fit indices indicated a close approximate fit of the data to the model. As depicted in Figure 3.6, the best fitting model had two unconstrained effects: maternal depressive symptoms at W1 predicted an increase in externalizing behavior at W3 *only among poorly-regulated children* ( $\beta = .21, p = .01$ ),  $\Delta\chi^2(1) = 8.69, p < .01$ ; and externalizing behavior at W1 predicted a decrease in maternal depressive symptoms at W3 *only among well-regulated children* ( $\beta = -.13, p < .05$ ),  $\Delta\chi^2(1) = 5.52, p < .05$ .

Although not shown in Figure 3.6 to simplify the visual display of the model, I found that W1 EC moderated the effect of child gender on externalizing behavior at W3; being a boy was associated with an increase in externalizing behavior at W3 *only among poorly-regulated children* ( $\beta = -.28, p < .001$ ),  $\Delta\chi^2(1) = 15.34, p < .001$ . Among children

with relatively low EC at W1, being a boy and exposure to maternal depressive symptoms at W1 predicted an increase in their externalizing behavior at W3. Children in the high EC group showed a negative relation between their externalizing behavior at W1 and maternal depressive symptoms at W3.

All observed effects were found while controlling for the stability of mothers' depressive symptoms and children's externalizing behavior across the three time points. All lagged effects were significant, except from W1 to W3 maternal depressive symptoms. For *poorly-regulated* and *well-regulated children*, respectively, this model explained 15% and 20% of the variance of W2 externalizing behavior, 29% and 28% of W2 maternal depressive symptoms, 45% and 36% of W3 externalizing behavior, and 31% and 44% of W3 maternal depressive symptoms.

## **Discussion**

My main aim was to advance understanding of risk mechanisms that contribute to the progression of children's externalizing problems following a transactional perspective of developmental psychopathology. Maternal depressive symptoms have been repeatedly shown to contribute to externalizing problems, but recent evidence indicates that this effect may be moderated by children's early self-regulation and that children's behavioral problems can worsen mothers' depressive symptoms (Compas & Reeslund, 2009; Gross et al., 2009; Lengua, 2002; Lengua et al., 2008; Shaw et al., 2009). Among predominantly White middle-class families, I found long-term bidirectional effects of child externalizing behavior and maternal depressive symptoms that were moderated by child gender and preschool levels of effortful control. These findings supported the protective effects of advanced effortful control in buffering the adverse effects of

mothers' depressive symptoms and children's externalizing behavior. Moreover, they suggest that the early interplay of these factors partly underlies the developmental progression of externalizing behavior from the preschool period to middle childhood.

### **Bidirectional Effects of Maternal Depression and Child Externalizing Behavior**

I hypothesized that maternal depressive symptoms and children's externalizing behavior would contribute to one another over time. This hypothesis was partially supported with bivariate relations in the expected direction between maternal depressive symptoms and child externalizing behavior. Consistent with previous research linking more behavioral problems in toddlerhood to elevated levels of maternal depressive symptoms (Gross et al., 2009; Shaw et al., 2009), I found that children's externalizing behavior at age 3 was positively associated with mothers' depressive symptoms at age 6. Maternal depressive symptoms at ages 3 and 6 were positively associated with children's externalizing behavior at age 10. Adjustment problems of mothers and children were positively correlated with one another across time, supporting the expected direction of association between these constructs. Despite promising bivariate results, the SEM results did not support my hypothesis of bidirectional effects when I examined the transactional model with the full sample. The model did not show effects of either maternal depressive symptom or child externalizing behavior. Prior research has consistently demonstrated adverse consequences of elevated maternal depressive symptoms for children's externalizing problems (e.g., Chronis et al., 2007; Weinfield et al., 2009). Many of these studies, however, did not include tests of bidirectional effects.

There are several explanations for why the SEM analyses did not support direct associations between maternal depressive symptoms and children's externalizing

behavior in the full sample. First, accounting for the stability of the measures across time limited the amount of variance that could be explained by other predictors. The effects of externalizing behavior and maternal depressive symptoms may not have been robust enough to predict changes in one another when controlling for their previous measurements. Second, there was a wide range of externalizing behavior among targeted children, but only 14% of the sample received borderline clinical or clinical range ratings. My inability to predict changes in children's behavioral problems may have been because the majority of the children demonstrated only mild externalizing behavior, which provided too little variance to explain with subclinical levels of maternal depressive symptoms. While the modeling results did not demonstrate bidirectional effects of maternal and child adjustment problems, correlational analyses were significant and in a direction that is consistent with my hypothesis. To circumvent problems related to the stability of the measures and their explainable variances, I separated the sample by characteristics shown to increase risk for externalizing problems to test for their moderating effects in further SEM analyses. I first examined whether child gender moderated the interplay of children's behavioral problems and maternal depressive symptoms.

### **Child Gender Differences**

Given substantial gender differences in the manifestation of externalizing behavior (e.g., Deater-Deckard et al., 1998), I compared boys and girls in the transactional model and found two differences: 1) Maternal depressive symptoms at age 3 predicted an increase in boys' externalizing behavior at age 10; and 2) Boys had more stable externalizing behavior than girls from ages 6 to 10. These findings support

evidence from studies on mostly low-income families that have focused on boys' externalizing behavior in relation to maternal depression (Gross et al., 2009; Shaw et al., 2003; Shaw et al., 2009). Preschool-age boys were more vulnerable than girls to early maternal depressive symptoms as evidenced by an increase in their behavioral problems from the early school years to middle childhood. Grouping boys and girls together in the test of the transactional model may have concealed the effects of maternal depressive symptoms on externalizing problems, especially since girls had fewer behavioral problems throughout the study duration. I included a gender covariate in the remaining models to help account for the differences mentioned above, as well as relevant bivariate relations (i.e., girls had more maternal depressive symptoms at age 3, and lower levels of externalizing behavior than boys at all ages).

### **Child Self-regulation Differences**

Findings from the next transactional model comparing children by their preschool-age effortful control (EC) supported previous literature (e.g., Lengua, 2002) and my second hypothesis: Maternal depressive symptoms at age 3 predicted an increase in externalizing behavior at age 10 among children with low levels of EC, while controlling for child gender. This is consistent with Lengua and colleagues' (2008) finding that cumulative risk, including maternal depressive symptoms, predicted subsequent adjustment problems only among children who had low self-regulation in preschool. Furthermore, these results supported meta-analytic findings that early exposure to maternal depression, such as in toddlerhood and the preschool period, is more detrimental to children's social-emotional development than later exposure (Connell & Goodman, 2002; Lovejoy et al., 2000). Exposure to maternal depressive



symptoms in infancy and toddlerhood has been linked to chronic behavioral problems, which suggests that early childhood is a period of heightened vulnerability to maternal psychosocial risk factors (Lengua et al., 2008; Shaw et al., 2009). Preschool-age boys and girls who had suboptimal self-regulation were more vulnerable to the adverse effects of early maternal depressive symptoms, as indicated by their elevated externalizing behavior in middle childhood. Moreover, these children had double the level of externalizing behavior at all measurement times compared to children who had higher levels of self-regulation during the preschool period.

Study findings refuted the prediction that children's externalizing behavior would predict increases in maternal depressive symptoms among all children, regardless of their self-regulation. Researchers found that boys' conduct problems at ages 3.5 and 5 years predicted increases in mothers' depressive symptoms at 5 and 6 years, respectively (Shaw et al., 2009). In contrast, I found that externalizing behavior at age 3 predicted a decrease in maternal depressive symptoms at age 10 only among children who had high EC in preschool. Well-regulated preschoolers' modest level of behavioral problems contributed to a decrease in mothers' depressive symptoms that was not evident until middle childhood. Mothers of poorly-regulated children demonstrated no change in their depressive symptoms and their children's externalizing behavior, despite being at elevated levels, had no effect on their mothers' depressive symptoms. I expected all observed effects of children's externalizing behavior to be negative, but evidence from the present study indicated a positive influence on maternal mental health. These findings suggest that the combination of children's advanced self-regulation and mild

externalizing behavior during the preschool years contributed to a gradual and modest improvement in mothers' depressive symptoms over roughly seven years.

It was unclear why maternal depressive symptoms and children's externalizing behavior predicted growth in one another from 3 to 10 years, rather than in a more consecutive pattern from one time point to the next (i.e., ages 3 to 6 or 6 to 10). Including second-order paths in the transactional models to account for long-term effects demonstrated bidirectional relations that would have otherwise been hidden. Upon examining bivariate relations among EC, maternal depressive symptoms, and externalizing behavior, I found that they were intercorrelated for poorly-regulated children only. These children's relatively low EC scores at 3 years were associated with more concurrent maternal depressive symptoms and more externalizing behavior at 10 years. In fact, poorly-regulated children's EC scores were only related to age 10 externalizing behavior, which was associated with more maternal depressive symptoms at ages 3 and 6. The observed relations suggest that the early influence of age 3 predictors on age 10 outcomes trumped any association involving early school-age measures at 6 years. Again, this supports the 'heightened vulnerability' argument that exposure to maternal depressive symptoms in early childhood is more likely to lead to chronic behavioral problems than experiences during the school years (Connell & Goodman, 2002; Lengua et al., 2008; Lovejoy et al., 2000).

The timing of exposure to maternal depression has been thoroughly discussed by other researchers (e.g., Cummings & Davies, 1994; Shaw et al., 2009), so I simply state that experiencing maternal depressive symptoms during infancy and toddlerhood may be worse than in later periods, because children are fully dependent on their primary

caretakers, often mothers, at a time when they require substantial support for the rapid growth of many competencies. Child regulatory deficits, for example, resulting from early psychosocial stressors may contribute to adaptive failures as children confront school, peers, and other developmental challenges of early childhood, which have enduring effects on success at later stage-salient tasks (Campbell et al., 2000; Dodge & Pettit, 2003; Olson et al., 2009). Thus, early exposure to maternal depressive symptoms may place children on a difficult trajectory where they are more likely to experience difficulties with developmental tasks and consequently manifest adjustment problems. The long-term bidirectional relations that emerged in this study may reflect the effects of accumulated exposure to mothers' depressive symptoms and children's externalizing behavior. Individual differences in self-regulation in the preschool period may distinguish between children who are more or less vulnerable to adverse mother-child exchanges and their enduring consequences. Developmental perspectives suggest that the cumulative effect of risk factors can have powerful effects on externalizing behavior, especially in parent-child transactions where relations grow exponentially over time (Dodge & Pettit, 2003; Cicchetti & Toth, 1997; Hinshaw, 2002; Sameroff, 2000). In this study of mostly middle-class families, transactions of maternal depressive symptoms and child behavioral problems and their interaction with early self-regulation may not have reached a palpable level until middle childhood. Maternal depressive symptoms and children's externalizing behavior reflect bidirectional processes of mutual influence that unfold over many years. The impact of early vulnerabilities in self-regulation may grow over time and lead to stronger relations between early stress and children's later adjustment problems.

### **The Interaction of Child Gender and Early Self-regulation**

Findings from this study suggest that preschool-age externalizing behavior is more likely to persist among children characterized by elevated maternal depressive symptoms and poor self-regulation. In addition, boys demonstrated more stable externalizing behavior from the early school years to middle childhood than girls. Thus, exposure to maternal depressive symptoms in the preschool years contributed to the progression of externalizing behavior from early to middle childhood among both boys and girls who had low levels of self-regulation. This study included both genders and extended prior research demonstrating bidirectional relations between maternal depression and boys' conduct problems (Gross et al., 2009; Shaw et al., 2009). Consistent with other studies, girls had higher levels of EC and boys had more externalizing behavior (Deater-Deckard et al., 1998; Eiden et al., 2007; Else-Quest et al., 2006; Olson et al., 2005; Valiente et al., 2006).

Findings support the critical role of preschoolers' EC in predicting later adjustment problems by increasing their associated risks with gender and maternal depressive symptoms (Eisenberg et al., 2005; Martel & Nigg, 2006; Olson et al., 2005; Rothbart & Bates, 2006). While I initially found that early maternal depressive symptoms predicted boys' elevated externalizing behavior in middle childhood, I determined that this negative effect only applied to boys and girls who had low self-regulation at age 3. I also found that low self-regulation at age 3 increased boys' risk of developing more externalizing behavior at age 10, which was separate from the effects of maternal depressive symptoms. To frame this positively: high self-regulation in the preschool years protected boys and preschoolers who were exposed to maternal depressive symptoms against continuing behavioral problems in elementary school. These findings

are consistent with other studies indicating that self-regulatory competence contributes to resilience processes by modifying children's sensitivity to environmental stressors (Buckner, Mezzacappa, & Beardslee, 2003; Lengua, 2002).

### **Limitations, Strengths, and Future Directions**

Several issues with this study warrant future investigation. The transactional models emphasized bidirectional effects between maternal depressive symptoms and children's externalizing behavior, but they did not elucidate proximal mechanisms underlying their interplay. The transactional models in this study, constructed with SEM techniques accounting for missing data and non-normality, delineated direction and magnitude of effects between mother and child adjustment problems, but they did not clarify psychosocial mechanisms underlying this dynamic process. Disturbed parenting has been related to maternal depression (e.g., Lovejoy et al., 2000), child externalizing behavior (Campbell et al., 2000; Deater-Deckard et al., 1998), and EC (Choe et al., in press; Lengua et al., 2008). Thus, it is likely that increased negative parenting or diminished positive parenting served as a proximal mediator between maternal depressive symptoms and children's externalizing problems. Mothers with elevated depressive symptoms may find it difficult meeting substantial parenting demands in early childhood, especially when children's behavioral problems challenge their competency as caretakers. In future research, suboptimal parenting should be examined in the transactional interplay between mother and child mental health outcomes.

In another investigation of this study's sample, I examined the transactional interplay of child externalizing behavior and maternal-reported inductive and physical discipline, and found that children's early externalizing problems at age 3 predicted an

increase in parental use of physical discipline at age 6 (Choe, Olson, & Sameroff, under review). Furthermore, more maternal inductive discipline use at age 3 predicted a decrease in parental physical discipline use and children's externalizing problems at age 6. Although not examined in this sample, parental discipline may be the intermediary mechanism between maternal depressive symptoms and children's externalizing behavior. Children with age-aberrant behavioral problems may evoke harsher discipline from their parents, who consequently utilize these coercive child-management techniques with greater frequency when emotionally distressed. Further study of this line of inquiry seems fruitful given the bidirectionality of parenting and child behavior.

In addition, there are other moderators that pertain to the interplay of maternal and child mental health that were not examined in this study. For example, temperament-based reactivity distinguishes children with varying levels of sensitivity to external stressors (Rothbart & Bates, 2006). Some children with low reactivity may need little self-regulation to effectively cope with contextual risk factors, whereas others who experience high arousal to psychosocial stress require substantial self-regulatory competence to manage their emotion and behavior (Sameroff, 2009a). Further research assessing temperament constructs through multiple methods, as done in this study, is needed to identify stable attributes of young children that underlie their vulnerability and protection to stress.

Finally, the external validity of my findings is limited to mainly White middle-class families. The community sample, although relatively large, was rather homogeneous in terms of ethnicity and socioeconomic status. Relatively few environmental risk factors were present and there was a wide range of early externalizing

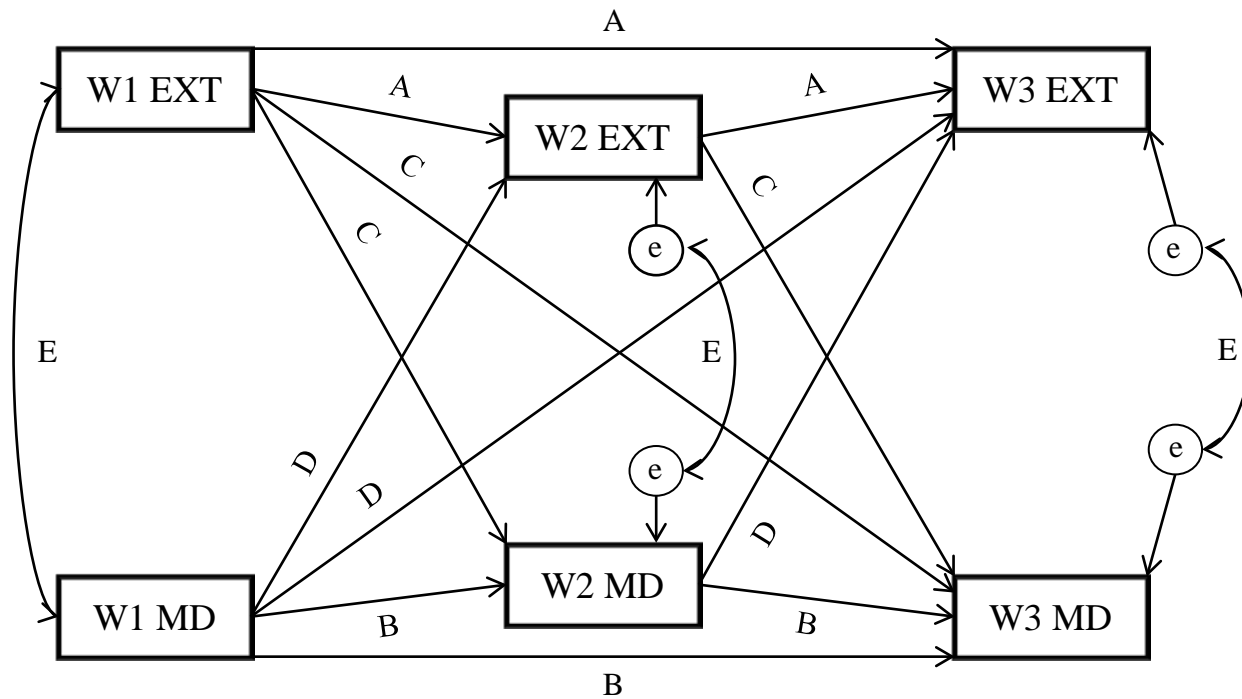
behavior among targeted children. The lack of severe ecological risks and the robust effects found in the sample suggest even subclinical levels of mothers' depressive symptoms and children's externalizing behavior can exacerbate one another. Future work can extend these findings by sampling at-risk populations such as mothers with clinical depression and/or racial-ethnic groups exposed to severe environmental risk factors that exacerbate maternal and child adjustment problems (Campbell et al., 2007; Deater-Deckard et al., 1998; Kiernan & Huerta, 2008).

### **Conclusion**

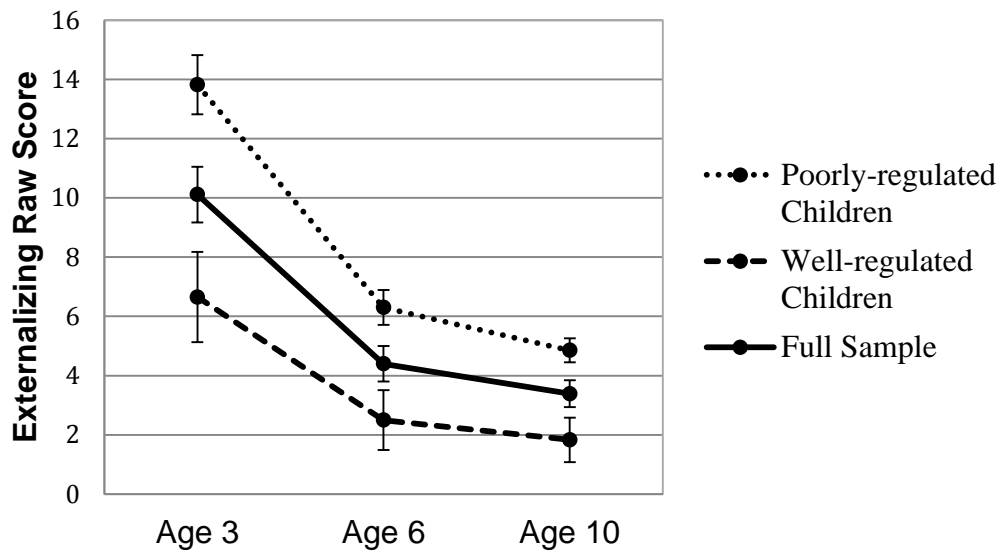
This study contributed evidence of bidirectional effects between maternal depressive symptoms and children's externalizing behavior spanning the preschool years to middle childhood, which were moderated by child characteristics. In particular, preschool-age boys and children who had low levels of self-regulation were more vulnerable to maternal depressive symptoms and were more likely to develop chronic school-age behavioral problems. The findings suggest there are benefits of advanced self-regulation in preventing children's externalizing problems, protecting against early exposure to maternal depressive symptoms, and also reducing depressive symptoms in mothers. This study is consistent with developmental psychopathology perspectives and research indicating transactional parent-child processes involving maternal depressive symptoms and child externalizing behavior (Cicchetti & Toth, 1997; Dodge, 1990; Sameroff, 2009a; Shaw et al., 2009). Teasing apart their dynamic interplay with a transactional perspective can elucidate the etiology and prevention of age-aberrant behavioral problems, as well as the intergenerational transmission of psychopathology. Interventions targeting maternal depressive symptoms and preschoolers' early

externalizing behavior and suboptimal self-regulatory skills may prevent more stable and serious adjustment problems in mothers and their children.

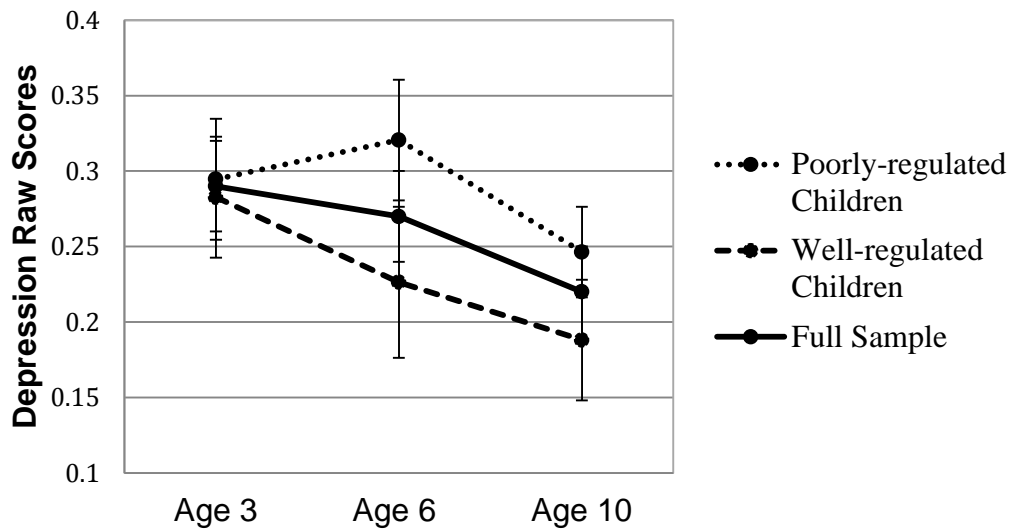




*Figure 3.1.* Our analytic structural equation model examines the continuity (A's and B's) and bidirectional effects (C's and D's) of children's externalizing behavior (EXT) and maternal depressive symptoms (MD) from the preschool years at age 3 (W1), the early school years at age 6 (W2), and middle childhood at age 10 (W3). E's represent covariances between residual terms (e's) that account for similar times of measurement.



*Figure 3.2.* Teacher-reported Externalizing Problem raw scores from age 3 in the preschool years, age 6 in the early school years, to age 10 in middle childhood by full sample and effortful control group. Standard errors are shown.



*Figure 3.3.* Mothers' Depression Scale raw scores from age 3 in the preschool years, age 6 in the early school years, to age 10 in middle childhood by full sample and effortful control group. Standard errors are shown.

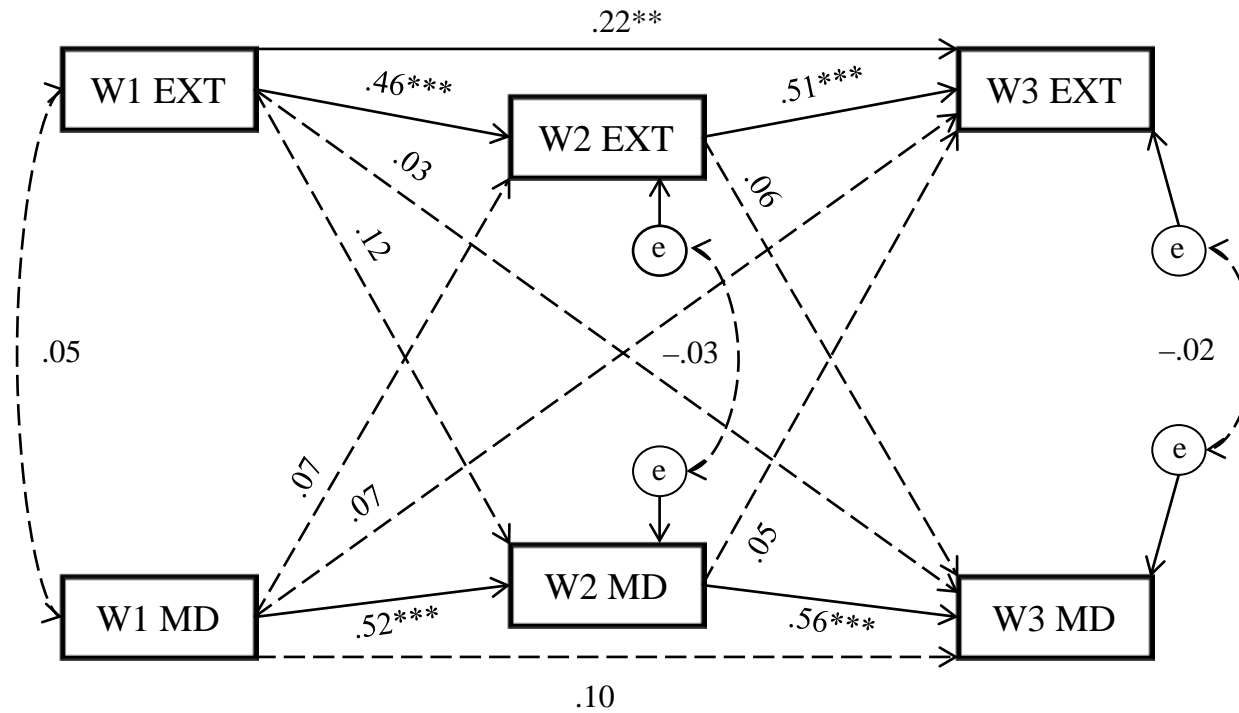


Figure 3.4. SEM results indicate stable externalizing behavior (EXT) across ages 3 (W1), 6 (W2), and 10 (W3), and stable maternal depressive symptoms (MD) from one wave to the next (MLR standardized;  $N = 224$ ). Solid arrows reflect significant effects.  $\chi^2(0) = .00, p < .001$ . CFI = 1.00. RMSEA < .001 [.00, .00]. W2 EXT  $R^2 = .22$ . W2 MD  $R^2 = .29$ . W3 EXT  $R^2 = .44$ . W3 MD  $R^2 = .40$ . \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

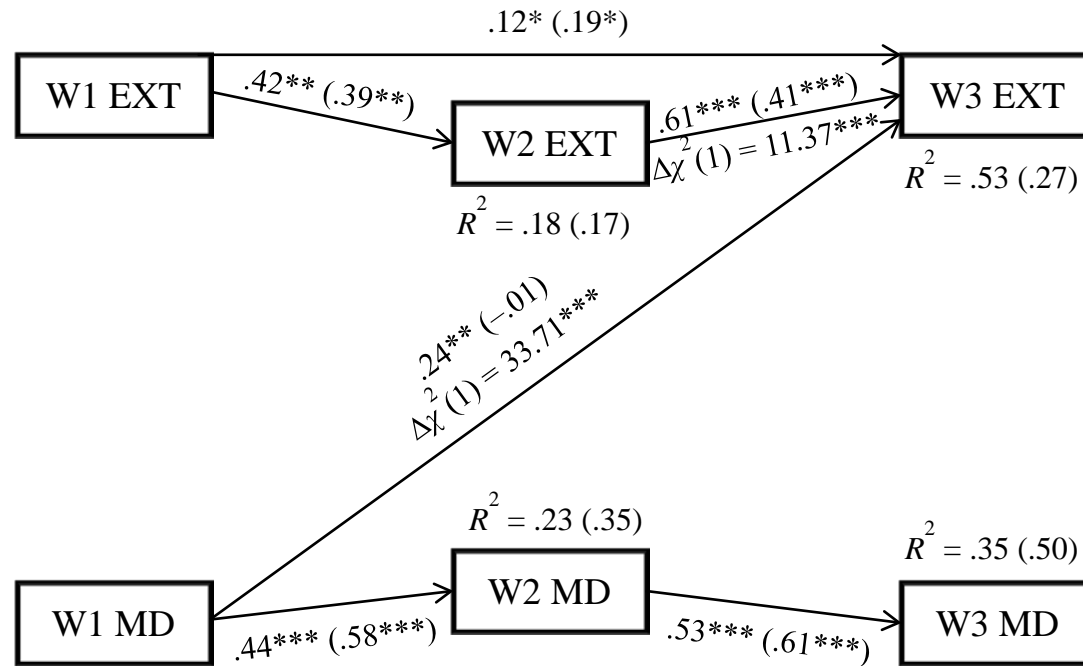


Figure 3.5. Multiple-group SEM results indicate that maternal depressive symptoms (MD) at age 3 (W1) predict increases in externalizing behavior (EXT) at age 10 (W3), *among only boys* (Standardized estimates before parentheses,  $n = 118$ ). Girls have less stable EXT from ages 6 (W2) to 10 (Estimates in parentheses,  $n = 106$ ). Covariances, residuals, and nonsignificant paths not shown. Chi-square difference values ( $\Delta\chi^2$ ) indicate paths that differ by gender.  $\chi^2(13) = 10.19$ ,  $p = .68$ . CFI = 1.00. RMSEA = .00 [.00, .08]. \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

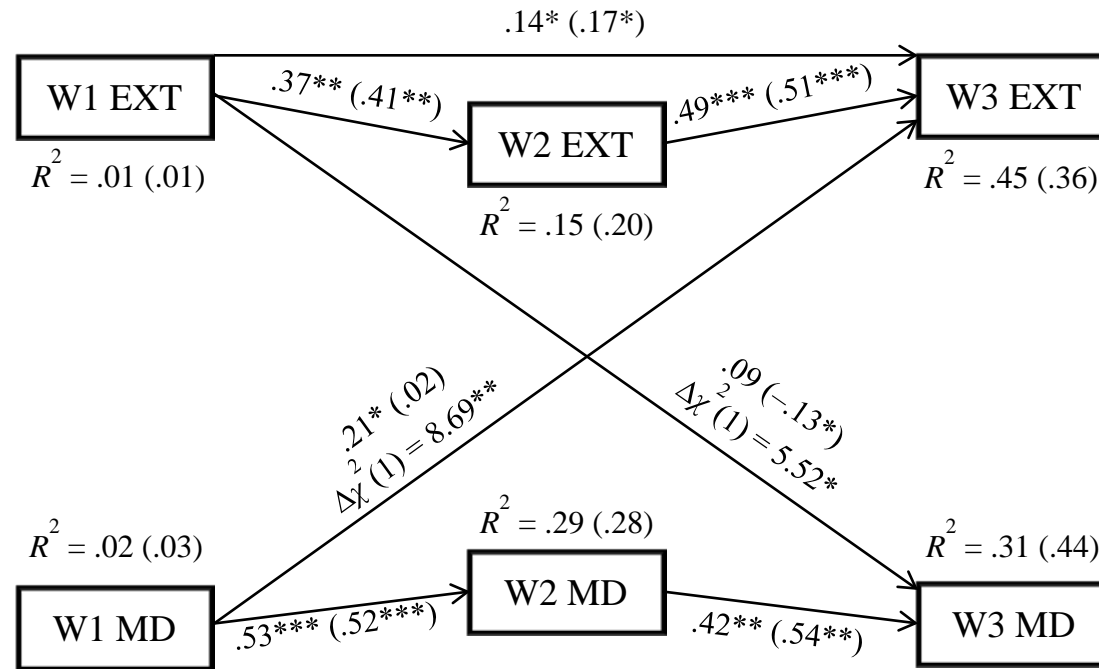


Figure 3.6. Multiple-group SEM results indicate that maternal depressive symptoms (MD) at age 3 (W1) predict increases in externalizing behavior (EXT) at age 10 (W3), among only poorly-regulated children (Standardized estimates before parentheses,  $n = 112$ ). EXT at age 3 predicts decreases in MD at age 10, among only well-regulated children (Estimates in parentheses,  $n = 112$ ). Covariances, residuals, nonsignificant effects, and gender covariate not shown. Chi-square difference values ( $\Delta\chi^2$ ) indicate paths that differ across groups.  $\chi^2(18) = 11.66, p = .86$ . CFI = 1.00. RMSEA < .001 [.00, .05]. \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

Table 2.1

*Correlations, Means, and Standard Deviations of Variables in Full Sample Structural Equation Model (N = 224)*

| Variables                            | 1       | 2       | 3      | 4      | 5      | 6      | 7   | 8    |
|--------------------------------------|---------|---------|--------|--------|--------|--------|-----|------|
| 1. Gender (0 = boys, 1 = girls)      | –       |         |        |        |        |        |     |      |
| 2. W1 Effortful Control <sup>a</sup> | .19**   | –       |        |        |        |        |     |      |
| 3. W1 Maternal Depressive Symptoms   | .15*    | –.12    | –      |        |        |        |     |      |
| 4. W1 Externalizing Behavior         | –.17*   | –.30*** | .06    | –      |        |        |     |      |
| 5. W2 Maternal Depressive Symptoms   | –.02    | –.07    | .51*** | .17*   | –      |        |     |      |
| 6. W2 Externalizing Behavior         | –.16*   | –.23**  | .08    | .43*** | .08    | –      |     |      |
| 7. W3 Maternal Depressive Symptoms   | –.02    | –.05    | .36*** | .12    | .52*** | .12    | –   |      |
| 8. W3 Externalizing Behavior         | –.26*** | –.31*** | .17*   | .44*** | .17*   | .63*** | .10 | –    |
| <i>M</i>                             | .47     | –.03    | .29    | 10.11  | .27    | 4.40   | .22 | 3.39 |
| <i>SD</i>                            | .50     | 2.24    | .42    | 12.56  | .48    | 8.16   | .38 | 6.07 |

*Note.* <sup>a</sup>W1 Effortful Control composite score of toddler-age behavioral battery total score and mother-reported child behavior questionnaire scores for Attentional Focusing and Inhibitory Control. W1 = age 3. W2 = age 6. W3 = age 10.

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

Table 2.2

*Correlations, Means, and Standard Deviations of Variables in Effortful Control Multiple Group Structural Equation Model (ns = 112)*

| Variables                            | 1      | 2      | 3      | 4      | 5      | 6      | 7      | 8      | <i>M (SD)</i> |
|--------------------------------------|--------|--------|--------|--------|--------|--------|--------|--------|---------------|
| 1. Gender (0 = boys, 1 = girls)      | –      | –.02   | .24*   | –.04   | .11    | –.11   | .02    | –.03   | .60 (.49)     |
| 2. W1 Effortful Control <sup>a</sup> | –.03   | –      | –.06   | –.12   | .02    | .02    | .09    | –.11   | 1.80 (1.21)   |
| 3. W1 Maternal Depressive Symptoms   | .08    | –.28** | –      | .03    | .50*** | .13    | .52*** | .13    | .28 (.41)     |
| 4. W1 Externalizing Behavior         | –.15   | –.13   | .09    | –      | .00    | .42*** | –.17   | .33**  | 6.65 (9.69)   |
| 5. W2 Maternal Depressive Symptoms   | –.10   | .02    | .53*** | .26*   | –      | .05    | .59*** | .03    | .23 (.46)     |
| 6. W2 Externalizing Behavior         | –.08   | –.13   | .07    | .37**  | .08    | –      | .13    | .48*** | 2.49 (5.67)   |
| 7. W3 Maternal Depressive Symptoms   | –.03   | .00    | .23*   | .24*   | .48*** | .11    | –      | .07    | .19 (.32)     |
| 8. W3 Externalizing Behavior         | –.30** | –.25*  | .22*   | .43*** | .21*   | .65*** | .08    | –      | 1.83 (3.75)   |
| <i>M</i>                             | .35    | –1.87  | .29    | 13.82  | .32    | 6.30   | .25    | 4.85   |               |
| <i>SD</i>                            | .48    | 1.33   | .43    | 14.18  | .50    | 9.72   | .42    | 7.36   |               |

*Note.* <sup>a</sup>Children with W1 Effortful Control scores at or below the median (.02) were coded as zero (*poorly-regulated group below diagonal*); participants scoring above the median were coded as 1 (*well-regulated group above diagonal*). W1 = age 3. W2 = age 6. W3 = age 10.

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



## **Chapter IV**

### **Transactions between African American Adolescents' Family Conflict and Violent Behavior: Implications for Stress and Coping**

In 2008, juveniles were apprehended in 16% of violent crime arrests in the U.S. (Puzzanchera, 2009). Although African Americans represented 16% of the juvenile population, they accounted for 52% of juvenile arrests for violent crimes. The disproportionate number of African American adolescents represented in violence statistics highlights the need for delineating the etiology and consequences of their violent behavior. Family conflict is a particularly salient risk factor for African American adolescents' violent offending (Paschall, Ennett, & Flewelling, 1996). Researchers have found that reciprocated hostility in parent–adolescent relationships exacerbates youth externalizing and internalizing problems (Laursen & Collins, 2009). Most researchers test main effects of family risk factors on adolescents' violent offending and ignore reciprocal influences in families that reinforce adjustment problems and interpersonal conflict (Spano, Vazsonyi, & Bolland, 2009). The current longitudinal study addressed this gap by examining a transactional model of African American adolescents' family conflict and violent behavior during high school and their effects on internalizing problems in young adulthood. In addition, I tested whether high active coping in adolescence buffered adverse stressors during high school and young adulthood. Identifying effective coping strategies for African American adolescents that protect them from prevalent

psychosocial stressors can clarify how youths navigate developmental challenges during the transition to young adulthood.

### **Transactional Perspective of Developmental Psychopathology**

The *transactional perspective of developmental psychopathology* is a framework emphasizing reciprocal processes of the individual and social-context contributing to the development of mental health problems (Cicchetti & Toth, 1997; Sameroff, 2009).

Psychopathology is viewed as developing from ongoing transactions between the individual and social-context. Critical to this perspective is an analytic focus on interdependent, bidirectional effects between individual and social-contextual variables. Examining bidirectional effects of violent behavior and family conflict during adolescence can elucidate their dynamic interplay as a transactional process and their unique effects on subsequent internalizing problems.

Family relationships, particularly with parents, undergo substantive transformations in early adolescence, but remain the most salient influences on social-emotional development (Collins & Steinberg, 2006; Laursen & Collins, 2009). During this time, youth gradually spend less time with family as they renegotiate familial roles and strive for autonomy, which is a quintessential task of adolescence (Eccles, Early, Fraser, Belansky, McCarthy, 1997; Steinberg, Dahl, Keating, Kupfer, Masten, & Pine, 2006). Attempts to establish autonomy often exacerbate family conflict as parents and adolescents negotiate the increasing independence youth desire (Collins & Steinberg, 2006; Laursen & Collins, 2009). Researchers have found that the frequency of parent-adolescent conflict *decreases* from early to late adolescence, but the emotional intensity of conflict *increases* from early to middle adolescence and then becomes relatively stable

(Laursen, Coy, & Collins, 1998). Contrary to popular belief, consistent high intensity fighting among family is not normative during adolescence, but it is characterized by more hostile and angry disputes than interpersonal conflict with family at other ages (Collins & Steinberg, 2006). Thus, frequency of parent–adolescent conflict generally decreases after peaking in early adolescence while its emotional intensity increases to a stable level in middle adolescence.

Frequent family conflict may be a particularly salient risk factor for African American adolescents. Elevated family conflict exacerbates internalizing and externalizing problems during adolescence and after (Gerard & Buehler, 2004; Laursen & Collins, 2009). Cross-sectional evidence indicates that more family conflict is associated with elevated violent behavior among urban African American 11- to 19-year-olds (DuRant, Cadenhead, Pendergrast, Slavens, & Linde, 1994). Moreover, 7<sup>th</sup> and 8<sup>th</sup> grade African American males report more family conflict than Whites, which is associated with their relatively higher levels of violent behavior (Paschall et al., 1996). These cross-sectional findings suggest that African American adolescents' greater violent behavior may be due in part to an increased risk for family conflict, but they do not clarify whether this association is reciprocal or whether it changes during adolescence. Longitudinal research examining the bidirectional interplay of violent behavior and family conflict during adolescence can elucidate a dynamic process that circulates stress within African American families.

Moffitt, Caspi, Harrington, and Milne (2002) examined outcomes at age 26 years among males who demonstrated life-course-persistent and adolescence-limited antisocial behavior problems. They found reciprocal influences between young men's antisocial

behavior problems and negative reactions from others, which are shown to reinforce the chronicity of problem behavior. They did not, however, examine family factors in this study. In a rare study of reciprocal family–adolescent processes, Cui, Donnellan, and Conger (2007) found that elevated marital conflict over child rearing when adolescents were 12- to 14-year-olds predicted more delinquent behavior a year later. They also found that more delinquency when adolescents were 13- to 15-years-old predicted increased marital conflict a year later. In another study, marital conflict about children exacerbated their children’s behavior problems, which consequently worsened marital conflict (Jenkins, Simpson, Dunn, Rasbash, & O’Connor, 2005). These results suggest that the interplay of adolescents’ antisocial behavior problems and family conflict creates transactional processes that further exacerbate violent behavior and family dysfunction. Yet, few researchers have examined reciprocal influences of family conflict and violent behavior during adolescence over periods longer than a year or two. Studies of the dynamic interplay between family conflict and adolescent violent behavior that explore this relationship over several adolescent years may provide useful insights into this transactional process and its consequences for adjustment problems in young adulthood.

Youth violence and family conflict are associated with internalizing problems in adolescence and young adulthood (Graber & Sontag, 2009; Laursen & Collins, 2009; Ozer, 2005; Voisin, 2007). Studies of African American adolescents that examine how reciprocal effects of risk factors influence internalizing problems during the transition to young adulthood are scarce. Researchers examining the same African American sample as this study found decreases in depressive symptoms during high school that continued to decline into young adulthood, but they did not investigate whether family conflict or

violent behavior contributed to symptoms (Repetto, Zimmerman, & Caldwell, 2008). Family stressors and serious maladaptive behavior make it more difficult for adolescents to successfully transition into healthy young adults (Steinberg et al., 2006).

The transactional interplay of African American adolescents' family conflict and violent behavior may worsen internalizing problems when transitioning into young adults for several reasons. First, during this transition, the well-being of young people tends to increase and the frequency of antisocial behavior rapidly decreases, but the incidence of psychopathology actually increases (Schulenberg & Zarrett, 2006; Steinberg et al., 2006). This transition is characterized in the U.S. by relatively limited institutional structure and support in comparison to what adolescents experience in high school. This shift to less structured and defined social-contexts during the transition to young adulthood can be stressful for many young people because their success at overcoming these challenges is dictated more by their individual characteristics than before (Schulenberg & Zarrett, 2006). African American adolescents with elevated violent behavior and family conflict at the end of high school will likely experience more challenges adapting to the demands of young adulthood during this pivotal transition than adolescents with less stressful family relationships and fewer antisocial behavior problems. As I discuss below, the added stress of transitioning to young adulthood requires active effort in dealing with psychosocial stressors to prevent subsequent adjustment problems.

### **The Role of Active Coping for African American Adolescents**

Adolescents who encounter significant adversity are at increased risk for behavioral and emotional problems in young adulthood (Steinberg et al., 2006). African American adolescents are at greater risk of exposure to violence and family adversity

than other adolescent populations (Aisenberg & Herrenkohl, 2008; Paschall et al., 1996; Voisin, 2007), so they may be more vulnerable than other groups to experiencing adverse consequences. Yet, few researchers have examined factors that help youth overcome the negative effects of this exposure. *Active coping* may help to mitigate the adverse effects of risk factors on negative outcomes (Compas & Reeslund, 2009). Active coping refers to a strong behavioral predisposition to address psychosocial environmental stressors. Evidence that active coping is associated with better adolescent adjustment supports the potential protective role of active coping for African American youth (Compas & Reeslund, 2009).

### **The Current Study**

In this longitudinal study, I examined the transactional interplay of African American adolescents' violent behavior and family conflict and their effects on internalizing problems in young adulthood. Furthermore, I tested whether active coping moderated effects of these risk factors for African Americans. Figure 4.1 depicts a transactional model of family conflict and violent behavior reflecting my hypotheses that they contribute to one another during high school through bidirectional effects and that both predict more internalizing problems in young adulthood (Andreas & Watson, 2009; Laursen & Collins, 2009; Ozer, 2005; Paschall et al., 1996; Voisin, 2007). Based on previous research (Compas & Reeslund, 2009), I expected that high active coping would buffer the bidirectional effects of family conflict and violent behavior and their effects on later internalizing problems.

## **Method**

### **Participants**

This study included 681 African American adolescents (49% male) selected from an urban city with a homicide rate over twice the national average (Michigan Department of Community Health, 2006). Students enrolled in 9<sup>th</sup> grade in four public high schools with a grade point average of 3.0 or below were eligible for participation. Initial recruitment selected 979 students (80% African American, 50% female) for a larger longitudinal study of school dropout and substance use (Zimmerman & Schmeelk-Cone, 2003). Students diagnosed by the school as emotionally impaired or developmentally disabled were not eligible. Participants were almost 15-years-old ( $M = 14.86$ ,  $SD = .65$ ) in 9<sup>th</sup> grade at wave 1 (W1).

### **Procedure**

Trained interviewers conducted structured hour-long interviews with participants during school hours. Participants were interviewed each year of high school and were followed two years later for a total of five time points. Participants reported family conflict, violent behavior, internalizing problems (i.e., symptoms of anxiety and depression), and active coping. Those who dropped out of school were interviewed in their homes or a community setting. Participants were informed that all information provided would be confidential and were paid for participating.

### **Measures**

**Violent behavior.** Eight items (avg.  $\alpha = .78$ ) recommended by the Center for Disease Control and Prevention (1995) assessed participants' fighting inside and outside of school or work, fighting in a group, hitting a teacher or supervisor, using or carrying a knife or gun, and whether they ever hurt someone badly enough to need medical aid. Participants indicated how often they engaged in each behavior during the past 12 months

using a 5-point response scale (1 = 0 times; 5 = 4+ times). Means were calculated from responses for four total scores of violent behavior during high school from W1 to W4.

**Family conflict.** Five items (avg.  $\alpha = .79$ ) from the Family Environment Scale (Moos & Moos, 1994) assessed participants' family members fighting a lot, losing their tempers, throwing things when angry, hitting, and criticizing each other. Participants selected answers best representing their family situation during the last 12 months using a 4-point response scale (1 = Hardly ever; 4 = Often). Means were calculated from responses for four total scores of family conflict from W1 to W4.

**Internalizing problems.** A latent measure of W5 internalizing problems was created with two scales from the Brief Symptom Inventory (Derogatis, 1993): depressive symptoms and anxiety symptoms. Six items ( $\alpha = .83$ ) assessed depressive symptoms: suicidal thoughts, having no interest in things, and feeling lonely, blue, worthless, and hopeless about the future. Six items ( $\alpha = .78$ ) assessed anxiety symptoms: suddenly scared for no reason, fearful, tense or keyed up, spells of terror or panic, nervousness, and restlessness. Participants indicated the frequency with which they experienced symptoms during the last 12 months using a five-point response scale (1 = Never; 5 = Very often). Total scores for each scale were calculated as means.

**Active coping.** Eight items ( $\alpha = .69$ ; James, 1994) assessed active coping. Items included: (1) "I've always felt that I could make of my life pretty much what I wanted to make of it." (2) "Once I make up my mind to do something, I stay with it until the job is completely done." (3) "I like doing things that other people think cannot be done." The items used a 5-point response scale (1 = Not true; 5 = Very true). Means were calculated from responses for five total scores of active coping from W1 to W5. A dichotomous



grouping variable derived from W1 active coping scores was created for multiple-group structural equation modeling (SEM) to compare adolescent who reported relatively high versus low active coping on hypothesized relationships. Participants with active coping scores at or above the median (4.25) were coded as 1 and labeled the “High Active Coping Group” ( $n = 368$ ). Participants who scored below the median were coded as zero and named the “Low Active Coping Group” ( $n = 311$ ).

### **Data Analysis Plan**

Preliminary analysis of sample attrition was conducted to identify systematic patterns of missing data that may have limited use of the full sample. Descriptive analyses for major study variables were conducted to examine central tendencies, distributions, and inter-correlations. SEM was used to examine a transactional model of African American adolescents’ family conflict and violent behavior during high school and internalizing problems in young adulthood (see Figure 4.1). Finally, multiple-group SEM was used to test for moderating effects of W1 active coping. Sex is a covariate in SEM models to account for the greater violent behavior of adolescent males (Côté et al., 2006; Daigle, Cullen, & Wirght, 2007; DuRant et al., 1994) and the greater family conflict reported by female adolescents (Daigle et al., 2006). In previous research of this study’s sample, females reported more depressive symptoms during high school and young adulthood than males (Repetto et al., 2008), and frequent co-occurring anxiety symptoms (Repetto, Caldwell, & Zimmerman, 2004a).

Following recommendations from Boomsma (2000), SEM models were evaluated by their chi-square ( $\chi^2$ ), comparative fit index (CFI), estimated root mean square error of approximation (RMSEA) and its 90% confidence interval. RMSEA values  $\leq .05$  indicate

close approximate fit; between .05 and .08 suggest reasonable error of approximation; and 1.0 or more suggests poor fit (Kline, 2005). CFI values greater than .90 reflect reasonably good fit. SEM was conducted using *Mplus* 5.21 and maximum likelihood with robust standard errors (MLR; Muthén & Muthén, 2007). MLR is a form of full-information maximum likelihood estimation with missing data that is robust to non-normal data (Yuan & Bentler, 2000). All other analyses were conducted with SPSS 15.0.

## Results

### Sample Attrition

Across W1 to W5, 209 participants (30.7%) were lost to attrition. Participants who left the study reported more violent behavior at W2 ( $M = 1.50, SD = .64, n = 190$ ) than those who continued ( $M = 1.39, SD = .57, n = 457$ ),  $t(645) = -2.13, p < .05$ . Participants who left also reported more violent behavior at W4 ( $M = 1.42, SD = .65, n = 162$ ) than those who stayed ( $M = 1.25, SD = .47, n = 448$ ),  $t(226) = -2.94, p < .01$ . A chi-square test indicated more males ( $n = 129$ ) than females ( $n = 80$ ) in the attrition group,  $\chi^2(1) = 18.94, p < .001$ . Despite patterns of nonrandom missing data, I included the full African American sample in analyses because measures were taken at multiple times so we could account for their stability with robust missing data estimators in SEM.

### Descriptive Analyses

Means, standard deviations, and correlations of major study variables are presented in Table 3.1. Females reported more family conflict at W1, W2, and W4 and more symptoms of depression and anxiety at W5. Males reported more violent behavior across W1 to W4. Participants who reported low active coping in 9<sup>th</sup> grade had more family conflict in 12<sup>th</sup> grade. Table 3.1 illustrates that family conflict and violent behavior

were correlated both concurrently and longitudinally and with W5 symptoms of depression and anxiety. Kurtosis values for violent behavior were relatively high at multiple waves, indicating narrow peaks in responses near the lower end of the scale (i.e., most participants reported little violent behavior).

### **Structural Equation Modeling**

**Measurement and structural model specification and fit.** An auto-regressive cross-lagged SEM model was specified to examine the continuity and transactional effects of African American adolescents' family conflict and violent behavior across four years of high school, and their effects on internalizing problems in young adulthood (see Figure 4.2). Longitudinal paths representing lagged effects (i.e., stabilities) and cross-lagged effects (transactions) extended from observed measures of family conflict and violent behavior to their later measures. Covariances connecting error terms of concurrent variables and identical constructs measured in different waves were created to account for variability in measurement error within and across time, respectively. A latent factor, W5 internalizing problems, was created from measures of W5 depressive symptoms ( $\beta = .93$ ,  $p < .001$ ) and anxiety symptoms ( $\beta = .78$ ,  $p < .001$ ). Factor loadings indicated that items adequately fit the latent factor (see Measurement Model estimates in Table 3.2). Paths were created from W4 family conflict and W4 violent behavior to the latent factor to examine direct effects of these 12<sup>th</sup> grade variables on internalizing problems in young adulthood. Sex was included as a covariate in all models, but is not shown graphically to simplify the presentation. Covariances, factor loadings, error terms, and nonsignificant effects are excluded from graphical display for the same reason.

The model in Figure 4.2 produced  $\chi^2(20, N = 681) = 49.40, p < .001$ ; CFI = .98; RMSEA < .05, 90% CI [.03, .06]. The fit indices indicated a good fit for the model, which explained about 9% of the variance of W5 internalizing problems, 30% of W4 family conflict, 39% of W4 violent behavior, 31% of W3 family conflict, 38% of W3 violent behavior, 22% of W2 family conflict, 30% of W2 violent behavior, and 1% of both family conflict and violent behavior at W1. Parameter estimates for structural paths are presented in Table 3.2 under Structural Model. Standardized parameter estimates for structural paths are presented in Figure 4.2.

As illustrated in Figure 4.2, lagged effects of family conflict (avg.  $\beta = .44, ps < .001$ ) and violent behavior were significant (avg.  $\beta = .51, ps < .001$ ). African American adolescents' family conflict and violent behavior were stable during high school.

**Bidirectional effects of family conflict and violent behavior.** I hypothesized that African American adolescents' family conflict and violent behavior would contribute to one another during high school through bidirectional effects. As shown in Figure 4.2, bidirectional effects from W2 to W3 were found; W2 family conflict predicted increased W3 violent behavior ( $\beta = .09, p < .05$ ) and W2 violent behavior predicted increased W3 family conflict ( $\beta = .10, p < .05$ ). Additionally, W1 violent behavior predicted increased W2 family conflict ( $\beta = .13, p < .01$ ). As hypothesized, African American adolescents' violent behavior in 9<sup>th</sup> and 10<sup>th</sup> grade predicted increases in family conflict in 10<sup>th</sup> and 11<sup>th</sup> grade, respectively; family conflict in 10<sup>th</sup> grade also predicted increases in violent behavior in 11<sup>th</sup> grade.

To determine whether the two significant effects of violent behavior on family conflict differed in magnitude, I constrained their parameter estimates to be equal in a

nested model that was compared to the transactional model with chi-square difference tests. The nested model did not differ in overall fit from the model in Figure 4.2:  $\chi^2(21) = 50.33, p < .001$ ; CFI = .98; RMSEA < .05, 90% CI [.03, .06];  $\Delta\chi^2(1) = .83, ns$ . The respective effects of W1 and W2 violent behavior on W2 and W3 family conflict did not appear to differ in magnitude ( $bs = .12, p < .001$ ; avg.  $\beta = .12, p < .001$ ). The effect of W2 family conflict on W3 violent behavior went through a similar test of effect size and did not appear to differ in magnitude from the previous two.

#### **Effects of family conflict and violent behavior on internalizing problems. I**

hypothesized that African American adolescents' family conflict and violent behavior in 12<sup>th</sup> grade would predict more internalizing problems in young adulthood. As hypothesized, W4 family conflict ( $\beta = .20, p < .001$ ) and violent behavior ( $\beta = .15, p < .05$ ) predicted more internalizing problems at W5 (see Figure 4.2).

**Gender effects.** A gender covariate in which boys were coded as "1" and girls were coded as "2" was included in the model to account for gender effects. Being male was associated with higher levels of violent behavior from W1 to W3. Being female was associated with higher levels of family conflict at W1, W2, and W4, and more internalizing problems at W5. Moderating effects of gender were tested with multiple-group SEM to determine whether gender interacted with relations found in the transactional model. No moderation was observed for structural paths of interest, so I continued to include gender as a covariate in subsequent models.

#### **Multiple Group Structural Equation Modeling**

After examining the model in Figure 4.2 with the full sample, I tested the transactional model with multiple-group SEM to identify moderating effects of W1 active

coping (0 = Low Active Coping Group, 1 = High Active Coping Group). An incremental approach to achieving the best fitting and most parsimonious model was followed using chi-square difference tests.

**Measurement invariance.** Measurement invariance was established by freeing estimates for factor loadings, measurement intercepts, and residual variances for indicators of W5 internalizing problems across both active coping groups, and comparing the fit of this unconstrained model to the fit of a constrained model with factor loadings, measurement intercepts, and residual variances fixed to be equal across groups. Chi-square difference tests indicated whether adding constraints in nested models worsened the overall model fit. The test of the unconstrained model produced  $\chi^2(40) = 76.78, p < .001$ ; CFI = .98; RMSEA = .05, 90% CI [.03, .07]. The test of the constrained model produced  $\chi^2(44) = 78.96, p < .001$ ; CFI = .98; RMSEA = .05, 90% CI [.03, .07]. The fit of the constrained and the unconstrained models did not differ [ $\Delta\chi^2(4) = 3.04, ns$ ] establishing measurement invariance across active coping groups.

**Structural variance.** Structural parameters were constrained to be equal across groups. Chi-square difference tests indicated equality constraints that worsened the model fit, which were then removed until the best fitting and most parsimonious model was identified,  $\chi^2(72) = 92.60, p = .052$ ; CFI = .99; RMSEA = .03, 90% CI [.00, .05] (see Figure 4.3). This model's fit was better than a fully constrained model,  $\chi^2(77) = 120.42, p = .001$ ; CFI = .97; RMSEA = .04, 90% CI [.03, .05];  $\Delta\chi^2(5) = 29.51, p < .001$ . Five effects differed across groups: the stability of violent behavior from 9<sup>th</sup> to 10<sup>th</sup> grade, the effect of 11<sup>th</sup> grade family conflict on 12<sup>th</sup> grade violent behavior, the effect of 12<sup>th</sup> grade family conflict on internalizing problems in young adulthood, and two gender effects.

Only unstandardized parameter estimates for structural paths are presented in Figure 4.3 and in the text since these more accurately reflect group differences estimated by chi-square difference tests than their standardized values.

Consistent with previous SEM results, lagged effects of family conflict (avg.  $b = .48$ ,  $ps < .001$ ) and violent behavior (avg.  $b = .52$ ,  $ps < .001$ ) were significant. A chi-square difference test indicated different parameter estimates for the lagged effect of W1 to W2 violent behavior for the High and Low Active Coping Groups. As shown in Figure 4.3, the Low Active Coping Group's violent behavior ( $b = .71$ ,  $p < .001$ ) was more stable from 9<sup>th</sup> to 10<sup>th</sup> grade relative to the violent behavior of the High Active Coping Group ( $b = .47$ ,  $p < .001$ ),  $\Delta\chi^2(1) = 4.90$ ,  $p = .027$ .

**Bidirectional effects of family conflict and violent behavior.** Contrary to my hypothesis that high active coping would buffer bidirectional effects of family conflict and violent behavior, effects between these constructs remained significant for both groups. Figure 4.3 illustrates W1 violent behavior predicted more W2 family conflict ( $b = .15$ ,  $p < .01$ ), W2 violent behavior predicted more W3 family conflict ( $b = .09$ ,  $p < .05$ ), and W2 family conflict predicted more W3 violent behavior ( $b = .08$ ,  $p < .05$ ).

A chi-square difference test demonstrated a group difference on the effect of W3 family conflict on W4 violent behavior,  $\Delta\chi^2(1) = 7.39$ ,  $p < .01$ . W3 family conflict predicted decreases in W4 violent behavior ( $b = -.08$ ,  $p < .05$ ) for the Low Active Coping Group, but was not significant for the High Active Coping Group ( $b = .06$ ,  $ns$ ). The direction of effect for the Low Active Coping Group was counter to my expectations and appeared to be an artifact of suppression, which occurs in a path model when a path coefficient of a predictor is in the opposite direction or of greater absolute magnitude than

the bivariate correlation of the predictor and outcome variables (Kline, 2005, pp. 119). This statistical paradox occurs when there are two or more independent variables predicting the same outcome and reflects their cross-correlations. The bivariate relationship of 11<sup>th</sup> grade family conflict and 12<sup>th</sup> grade violent behavior was positive for both the Low ( $r = .13, p < .05$ ) and High Active Coping Groups ( $r = .24, p < .001$ ). Therefore, the negative path coefficient observed from W3 family conflict to W4 violent behavior for the Low Active Coping Group indicated a suppression effect.

#### **Effects of family conflict and violent behavior on internalizing problems. I**

I hypothesized that high active coping would mitigate family conflict and violent behaviors' effects on internalizing problems in young adulthood. Contrary to this prediction, elevated violent behavior at W4 predicted more W5 internalizing problems for both groups ( $b = .18, p < .05$ ). A chi-square difference test provided partial support for my hypothesis with a marginal group difference for the effect of W4 family conflict on W5 internalizing problems,  $\Delta\chi^2(1) = 3.48, p = .062$ . As shown in Figure 4.3, W4 family conflict predicted more internalizing problems at W5 for adolescents with low active coping ( $b = .34, p = .001$ ), but was only marginally significant for adolescents with high active coping ( $b = .13, p = .07$ ).

#### **Discussion**

The present study's findings demonstrated a dynamic transactional process involving African American adolescents' family conflict and violent behavior. This transactional process began soon after adolescents' entry into high school when increasing levels of violent behavior contributed to more family conflict over the next school year. These findings provided evidence of bidirectional effects in which



heightened levels of family conflict and violent behavior exacerbated one another during the middle of high school. The effects of African American adolescents' violent behavior on subsequent family conflict complemented literature indicating that family conflict and stress are risk factors for violent behavior during adolescence (Aisenberg & Herrenkohl, 2008; Andreas & Watson, 2009; DuRant et al., 1994; Fergus & Zimmerman, 2005; Paschall et al., 1996). Most previous research tested only main effects of family stressors on adolescent problems, but my findings suggested that violent behavior during adolescence also serves as a risk factor for African Americans' family conflict. The dual transmission of risk in this transactional process can be further explicated by reviewing their bidirectional interplay over time.

Researchers examining reciprocal family influences have found bidirectional effects of children and adolescents' behavioral problems and marital conflict over a shorter period of time than in this study (Cui et al., 2007; Jenkins et al., 2005). These researchers proposed that marital conflict and youth behavioral problems operate as stressors on the family environment, thereby exacerbating family-wide levels of adjustment problems and conflict. In the present study, escalating violent behavior during the first two years of high school may have increasingly stressed the family system, particularly parent-adolescent relationships, and contributed to more stable conflict and violent offending throughout high school. Relatively recent research (e.g., Gross et al., 2009) and this study illustrated transactional processes of family adversity and antisocial behavior problems in adolescence, thus providing a more comprehensive account of psychosocial stressors operating on the family environment and adolescent social-emotional development. Evidence from this research supported transactional theory, the

reciprocal interplay of stressors operating on families and youth, and the importance of examining adolescent development embedded within an evolving social-context.

Findings supported my second hypothesis that heightened family conflict and violent behavior in 12<sup>th</sup> grade would predict more internalizing problems at age 20. This finding was consistent with evidence that family stressors and youth violence elevate risk for emotional problems during the transition to young adulthood (Laursen & Collins, 2009; Ozer, 2005; Steinberg et al., 2006; Voisin, 2007). Challenges encountered when facing developmental tasks during this transition, such as taking on adult roles and responsibilities, can stress young people's coping abilities and increase vulnerability to psychological disturbances (Schulenberg & Zarrett, 2006). Adolescents who experience heightened family conflict and violent behavior are less likely to successfully traverse developmental tasks and are more likely to succumb to risk factors, experience difficulties in adult roles, and develop subsequent internalizing problems than adolescents without these stressors (Masten, Burt, Roisman, Obradović, Long, & Tellegen, 2004). Elevated family conflict and violent behavior at the end of high school may indicate African Americans' increased risk for internalizing problems in young adulthood.

Previous study of this sample demonstrated that African American adolescents with consistently high levels of depressive symptoms reported more anxiety symptoms and perceived stress at the end of high school (Repetto et al., 2004a). Males who reported more depressive symptoms at age 20 used more alcohol (Repetto, Zimmerman, & Caldwell, 2004b) and marijuana (Repetto et al., 2008), suggesting that young males with internalizing problems were more likely to self-medicate as a coping response. Furthermore, researchers found reciprocal effects of alcohol use and violent behavior

from adolescence to young adulthood (Xue et al., 2009). In the general U.S. population, alcohol and marijuana use and the incidence of major depression increase after high school, but physical aggression and depressive affect decrease (Schulenberg & Zarrett, 2006). Studies of this sample demonstrated developmental trajectories of violent behavior, substance use, and depressive symptoms similar to those in the general population (Repetto et al., 2004b; Repetto et al., 2008; Xue et al., 2009). These findings collectively indicated that the transitional years following high school stress African Americans' coping responses and exacerbate adjustment problems. High levels of family conflict and violent behavior in high school undoubtedly make this transition more difficult for adolescents than for their peers with low levels of these risk factors.

Partial support was also found for my third set of hypotheses regarding the buffering effects of active coping. High levels of active coping among African American adolescents protected youth against the adverse effects of family conflict on later internalizing problems, but they did not mitigate transactions of family conflict and violent behavior. The size of the bidirectional effects did not differ in post-hoc analyses, suggesting they contributed to one another to a similar extent.

Contrary to expectation, active coping did not buffer effects of violent behavior on family conflict or internalizing problems. Violent behavior in 12<sup>th</sup> grade predicted more internalizing problems for all African American adolescents. African American adolescents reporting low active coping, however, had more stable violent behavior across the first two years of high school compared to adolescents reporting high active coping. The stability of violent behavior for the high active coping adolescents was about two-thirds the level of the low active coping adolescents in the beginning of high school,

suggesting that active coping helped adolescents desist from physical violence. These findings suggested that high active coping in adolescence buffered the noxious effects of elevated family conflict on future anxiety and depressive symptoms in young adulthood. Furthermore, violent behavior at the end of high school was predictive of internalizing problems at age 20, regardless of individual differences in active coping.

The limited protective role of high active coping supported assertions made by Gerard and Buehler (2004) that protective factors residing in the individual do little to prevent poor outcomes when the social environment is unsupportive. Youth violence and its many stressful consequences may excessively challenge adolescents' coping responses during the transition to young adulthood and strain their emotional adjustment.

Accumulated risk factors occurring across multiple contexts overwhelm young people's coping strategies and are associated with the worse outcomes (Gerard & Buehler, 2004). When exposed to many risk factors, even adolescents with effective coping skills need the support of their close family, friends, and neighbors (Ozer, 2005; Xue, Zimmerman, & Barnett, In press; Zimmerman, Steinman, & Rowe, 1998).

Active coping encompasses problem solving and efforts to alter objective stressors or one's emotional reactions to stress (Compas & Reeslund, 2009). Adolescents who respond to objectively controllable stressors or stressors they perceive to be controllable with high levels of active coping tend to be better adjusted. Active coping embodies prolonged efforts to cope with psychosocial stressors through behavioral and emotional autonomy, feelings of control, and hard work (James, 1994). Many of these same responses predict resilience among at-risk populations during the transition to adulthood (Masten et al., 2004). Although previous study of this sample reported that

active coping linearly increased during high school (Repetto et al., 2004a), I found no difference in active coping between measures taken in 9<sup>th</sup> grade and at age 20. Evidence from this study extended the work of James (1994) by demonstrating protective benefits of African American adolescents' active coping in buffering effects of family conflict on internalizing problems and reducing stability of violent behavior. Findings supported research indicating that high active coping is associated with fewer adjustment problems (Compas & Reeslund, 2009), and extended this to African American adolescents.

Although not a priority, findings from this study were consistent with literature indicating gender differences in the use of violent behavior (Côté et al., 2006; Daigle et al., 2006; DuRant et al., 1994), exposure to family conflict (Daigle et al., 2006), and the presence of internalizing problems (Nolen-Hoeksema, 2001; Repetto et al., 2008). No moderating effects of gender were found when examining reciprocal effects of family conflict and adolescent violent behavior, which is consistent with similar research (Cui et al., 2007). Furthermore, level of active coping appeared to attenuate some gender effects. Being a female was associated with greater family conflict in the first and last year of high school only among a subgroup of adolescents with low active coping. Thus, low levels of active coping for adolescent girls appeared to increase their exposure to or perception of family conflict at the beginning and end of high school.

### **Limitations, Strengths, and Future Directions**

There were several limitations of this study that warrant caution when interpreting the results. One limitation was using only self-reported data collected in structured interviews with adolescents. Adolescents and parents have discrepant views on the quality of family relationships (Laursen & Collins, 2009). Adolescents tend to make rigid

appraisals about general family relationships, whereas parents are more likely to weigh unique factors of specific relationships. Collecting reports from multiple informants can provide more comprehensive assessments of youth risk and psychopathology. Structured interviews, however, are used extensively in longitudinal research and are considered the best approach for assessing adolescent risk factors and adjustment problems (Compas & Reeslund, 2009, pp. 567). In addition, self-reports are the best method of assessing adolescent violent behavior because many aggressive acts are unknown to parents (Farrington, 2009). Future research should assess parents to complement adolescents' reports of family relationships, as well as assess both frequency and intensity of family conflict, which follow different trajectories in adolescence (Laursen et al., 1998).

In addition to measurement issues, salient structural differences between families were not statistically controlled. Adolescents who experience divorce may be more likely to develop adjustment problems, but single parents living with extended family members tend to raise better-adjusted children (Gerard & Buehler, 2004; Laursen & Collins, 2009). Extended family members lived with 18% of families in the sample, so participants' reporting of conflict may have reflected some heterogeneity in family structure. This characteristic indicated another caveat of this research, which is the limited generalizability of the findings to adolescents belonging to other racial-ethnic groups. African American youths are much more likely to be exposed to poverty and community violence, making them a high-risk population (Aisenberg & Herrenkohl, 2008; McLoyd, 1998). Thus, their levels of family conflict and violent behavior were probably higher than in the general population, indicating a limit to the external validity of the findings. Future research is warranted that examines a nationally representative sample of

adolescents and distinguishes between types of conflict within families and structural differences in the household.

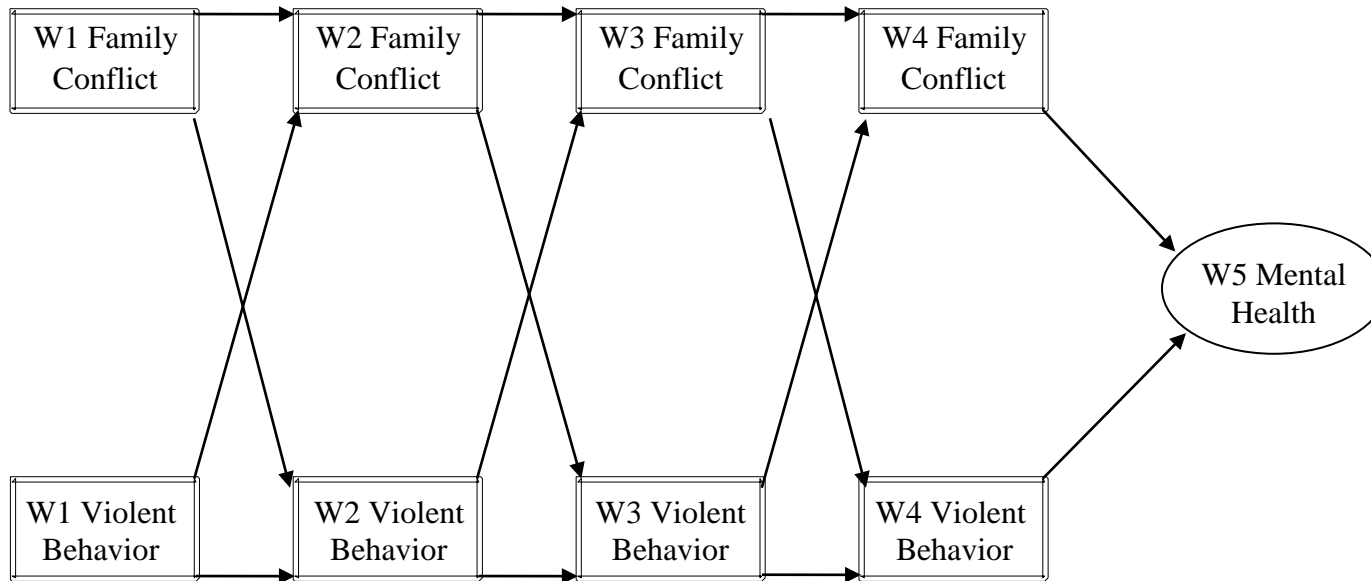
Lastly, the transactional model did not identify proximal mechanisms that operate between family conflict and violent behavior. Family conflict and other disturbances in the family can increase an adolescent's risk of violent behavior through mechanisms that were not tested in this study, such as socialization of violent attitudes, modeling of violent behavior, and coercive parenting (Aisenberg & Herrenkohl, 2008; Andreas & Watson, 2009; Dodge & Pettit, 2003; Fergus & Zimmerman, 2005; Slovak, Carlson, & Helm, 2007; Solomon, Wright, & Cheng, 2008). Likewise, adolescents' violent behavior may heighten family conflict through multiple avenues, such as weakening parental support, but further study is needed to disentangle these transactional processes. Process-oriented research can elucidate mechanisms operating within the interplay violent behavior and family conflict during adolescence (Compas & Reeslund, 2009). Identification of proximal mechanisms can inform interventions designed to buffer these transactions and prevent future adjustment problems for at-risk African American youth. Nevertheless, this study contributed evidence of a transactional process as a starting point for more focused research on mechanisms.

## **Conclusion**

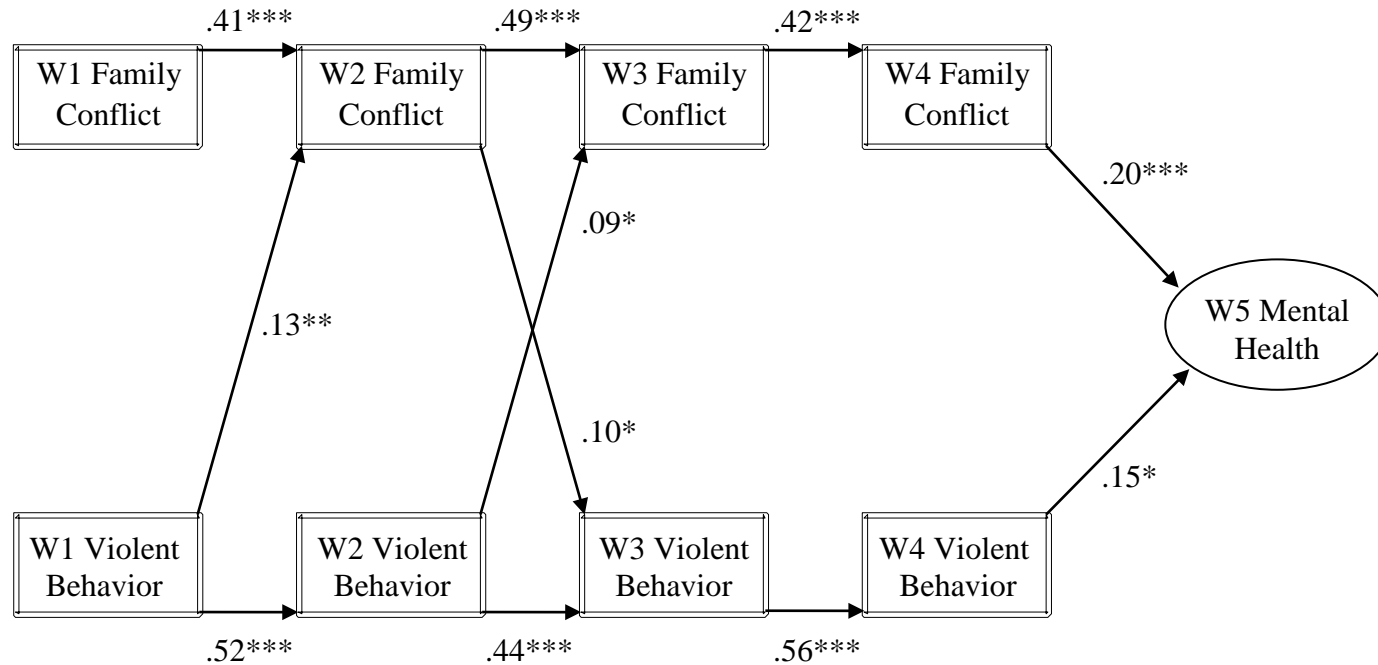
This study demonstrated transactional associations between African American adolescents' family conflict and violent behavior during high school that predicted more internalizing problems in young adulthood. Family conflict is a risk factor for adolescent engagement in youth violence and delinquency (DuRant et al., 1994; Odgers et al., 2008; Paschall et al., 1996). This study added to the dearth of literature on how adolescents'

violent behavior exacerbates family conflict. Transactional processes circulate stress within families and jeopardize adaptation across developmental transitions that represent both windows of increased vulnerability to stress and foci for preventive interventions (Schulenberg & Zarrett, 2006; Steinberg et al., 2006). Evidence from this study also suggests that active coping may help mitigate the adverse effects of family conflict in adolescence on internalizing problems in young adulthood. Further replication of these findings and an increased focus on proximal mechanisms can inform preventive interventions and reduce the burden of costs associated with internalizing problems and violent crime among young African Americans.





*Figure 4.1.* Transactional model of African American adolescents' family conflict and violent behavior and mental health in young adulthood. Time points represent 9th grade (W1), 10th grade (W2), 11th grade (W3), 12th grade (W4), and age 20 (W5). Covariances, and the gender covariate are not shown.



*Figure 4.2.* Transactional model of African American adolescents' family conflict and violent behavior and their mental health in young adulthood (Standardized solution,  $N = 681$ ).  $\chi^2(20) = 49.40, p < .001$ . CFI = .98. RMSEA < .05, 90% CI [.03, .06]. Participants were assessed in 9<sup>th</sup> grade (W1), 10<sup>th</sup> grade (W2), 11<sup>th</sup> grade (W3), 12<sup>th</sup> grade (W4), and age 20 (W5). Covariances, nonsignificant estimates, and the gender covariate are not shown.  
 \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

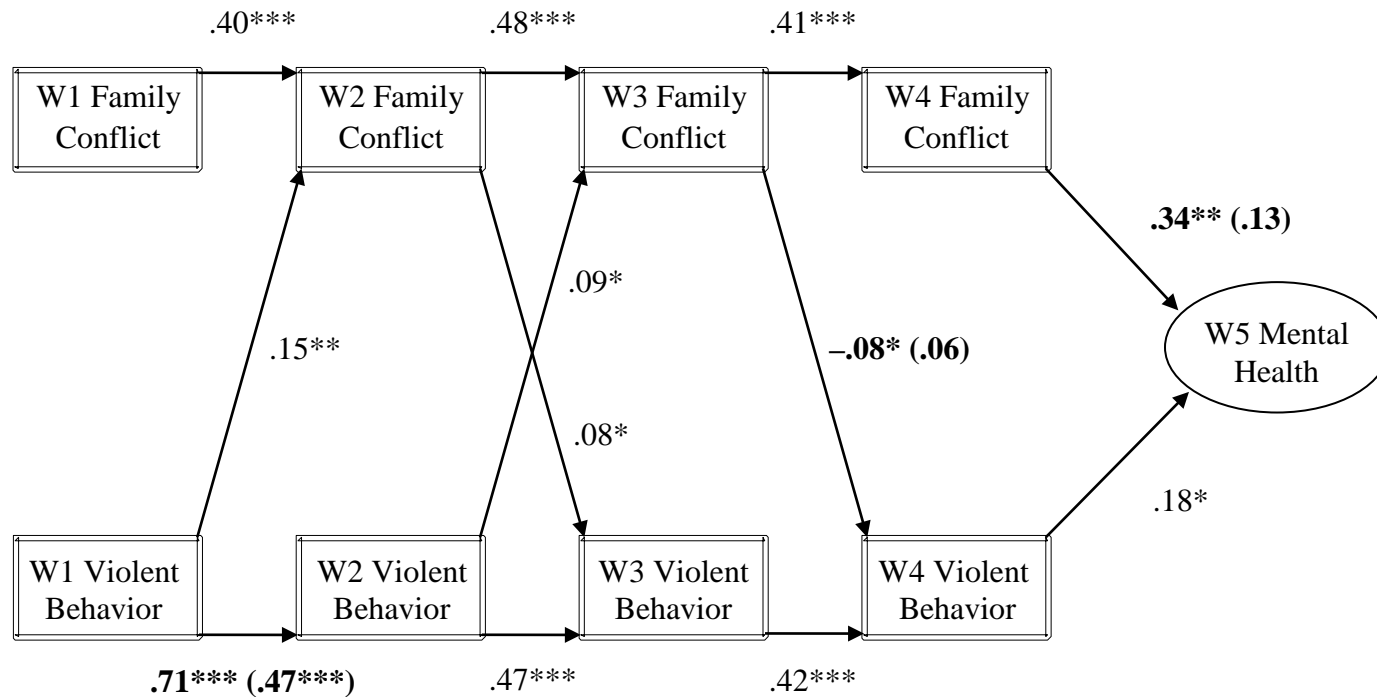


Figure 4.3. African American adolescents' active coping moderates effects of family conflict on violent behavior and mental health (Unstandardized solution).  $\chi^2(72) = 92.60, p = .052$ . CFI = .99. RMSEA = .03, 90% CI [.00, .05]. Participants with W1 active coping scores below the median were coded as zero ( $n = 311$ ), the Low Active Coping Group, and those at or above the median were coded as 1 ( $n = 368$ ), the High Active Coping Group (estimates in parentheses). Pairs of bolded estimates indicate differences between groups. Covariances, nonsignificant estimates, and the gender covariate are not shown.

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

Table 3.1

*Correlations, Means, and Standard Deviations of Major Study Variables*

| Variables                        | 1       | 2     | 3      | 4      | 5      | 6      | 7      | 8      | 9      | 10     | 11     | 12   |
|----------------------------------|---------|-------|--------|--------|--------|--------|--------|--------|--------|--------|--------|------|
| 1. Gender <sup>a</sup>           | –       |       |        |        |        |        |        |        |        |        |        |      |
| 2. W1 Active Coping <sup>b</sup> | -.04    | –     |        |        |        |        |        |        |        |        |        |      |
| 3. W1 Violent Behavior           | -.10*   | -.003 | –      |        |        |        |        |        |        |        |        |      |
| 4. W1 Family Conflict            | .09*    | -.05  | .18*** | –      |        |        |        |        |        |        |        |      |
| 5. W2 Violent Behavior           | -.15*** | -.02  | .52*** | .10*   | –      |        |        |        |        |        |        |      |
| 6. W2 Family Conflict            | .14***  | -.02  | .20*** | .45*** | .32*** | –      |        |        |        |        |        |      |
| 7. W3 Violent Behavior           | -.19*** | -.03  | .53*** | .11**  | .61*** | .25*** | –      |        |        |        |        |      |
| 8. W3 Family Conflict            | .06     | -.06  | .16*** | .33*** | .25*** | .55*** | .29*** | –      |        |        |        |      |
| 9. W4 Violent Behavior           | -.13**  | -.03  | .41*** | .07    | .51*** | .24*** | .62*** | .19*** | –      |        |        |      |
| 10. W4 Family Conflict           | .10*    | -.09* | .15*** | .26*** | .21*** | .52*** | .18*** | .54*** | .22*** | –      |        |      |
| 11. W5 Depressive Behaviors      | .13**   | -.02  | .11*   | .17*** | .13**  | .34*** | .19*** | .18*** | .15**  | .24*** | –      |      |
| 12. W5 Anxiety Symptoms          | .10*    | -.02  | .13**  | .12**  | .10*   | .28*** | .14**  | .13**  | .15**  | .19*** | .72*** | –    |
| <i>M</i>                         | 1.51    | .54   | 1.37   | 1.80   | 1.43   | 1.66   | 1.34   | 1.57   | 1.29   | 1.57   | 1.75   | 1.58 |
| <i>SD</i>                        | .50     | .50   | .55    | .45    | .60    | .63    | .55    | .60    | .53    | .60    | .71    | .61  |

Note. <sup>a</sup>(1 = male, 2 = female). <sup>b</sup>Participants with W1 active coping scores at or above the median (4.25) were coded as 1 (n = 368), indicating the High Active Coping Group, and those below the median were coded as zero (n = 311), indicating the Low Active Coping Group.

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

Table 3.2

*Unstandardized/Standardized Values for Figure 4.2 (Standard Errors in Parentheses)*

| <i>Parameter Estimate</i>                | <i>Unstandard.</i> | <i>p</i> | <i>Standardized</i> | <i>p</i> |
|--|--------------------|----------|---------------------|----------|
| <u>Measurement Model</u>                 |                    |          |                     |          |
| W5 Mental Health → W5 Depression         | 1.00               | Na       | .93                 | .00      |
| W5 Mental Health → W5 Anxiety            | .72 (.12)          | .00      | .78                 | .00      |
| <u>Structural Model</u>                  |                    |          |                     |          |
| W4 Family Conflict → W5 Mental Health    | .22 (.06)          | .00      | .20                 | .00      |
| W4 Violent Behavior → W5 Mental Health   | .19 (.08)          | .01      | .15                 | .02      |
| Gender <sup>a</sup> → W5 Mental Health   | .18 (.06)          | .01      | .13                 | .00      |
| W3 Violent Behavior → W4 Family Conflict | .05 (.05)          | .33      | .04                 | .34      |
| W3 Family Conflict → W4 Family Conflict  | .42 (.06)          | .00      | .42                 | .00      |
| Gender → W4 Family Conflict              | .10 (.04)          | .01      | .08                 | .01      |
| W3 Family Conflict → W4 Violent Behavior | -.001 (.03)        | .98      | -.001               | .98      |
| W3 Violent Behavior → W4 Violent Beh.    | .54 (.08)          | .00      | .56                 | .00      |
| Gender → W4 Violent Behavior             | -.02 (.03)         | .50      | -.02                | .50      |
| W2 Violent Behavior → W3 Family Conflict | .10 (.04)          | .03      | .10                 | .03      |
| W2 Family Conflict → W3 Family Conflict  | .47 (.05)          | .00      | .49                 | .00      |
| Gender → W3 Family Conflict              | .01 (.04)          | .80      | .01                 | .80      |
| W2 Family Conflict → W3 Violent Behavior | .08 (.04)          | .02      | .09                 | .02      |
| W2 Violent Behavior → W3 Violent Beh.    | .40 (.06)          | .00      | .44                 | .00      |
| Gender → W3 Violent Behavior             | -.14 (.04)         | .00      | -.13                | .00      |
| W1 Violent Behavior → W2 Family Conflict | .15 (.04)          | .00      | .13                 | .00      |
| W1 Family Conflict → W2 Family Conflict  | .57 (.06)          | .00      | .41                 | .00      |
| Gender → W2 Family Conflict              | .14 (.04)          | .00      | .11                 | .00      |
| W1 Family Conflict → W2 Violent Behavior | .02 (.05)          | .65      | .02                 | .65      |
| W1 Violent Behavior → W2 Violent Beh.    | .58 (.06)          | .00      | .52                 | .00      |
| Gender → W2 Violent Behavior             | -.15 (.04)         | .00      | -.13                | .00      |
| Gender → W1 Violent Behavior             | -.10 (.04)         | .01      | -.09                | .01      |
| Gender → W1 Family Conflict              | .08 (.03)          | .02      | .09                 | .02      |
| W1 Family Conflict ↔ W1 Violent Behavior | .04 (.01)          | .00      | .17                 | .00      |
| W2 Family Conflict ↔ W2 Violent Behavior | .08 (.01)          | .00      | .28                 | .00      |
| W3 Family Conflict ↔ W3 Violent Behavior | .03 (.01)          | .00      | .15                 | .00      |
| W4 Family Conflict ↔ W4 Violent Behavior | .02 (.01)          | .05      | .11                 | .05      |
| W1 Family Conflict ↔ W4 Family Conflict  | .02 (.01)          | .08      | .10                 | .07      |
| W1 Family Conflict ↔ W3 Family Conflict  | .02 (.01)          | .08      | .11                 | .07      |
| W2 Family Conflict ↔ W4 Family Conflict  | .07 (.02)          | .00      | .25                 | .00      |
| W1 Violent Behavior ↔ W4 Violent Beh.    | .03 (.01)          | .02      | .12                 | .01      |
| W1 Violent Behavior ↔ W3 Violent Beh.    | .08 (.02)          | .00      | .35                 | .00      |
| W2 Violent Behavior ↔ W4 Violent Beh.    | .03 (.02)          | .10      | .13                 | .10      |

*Note.*  $\chi^2(20, N = 681) = 49.40, p < .001$ . CFI = .98. RMSEA < .05, 90% CI [.03, .06].

<sup>a</sup>(1 = male, 2 = female).

## Chapter V

### **Integrating Transactions and Interactions for Understanding the Etiology, Development, and Prevention of Externalizing Symptoms**

The preponderance of research on externalizing symptoms and their associated disorders has approached study of their etiology with unidirectional models focusing on environmental influences (Sameroff, 2009a). A limited number of studies employ interactional or transactional approaches to examining risk factors and the progression of children's externalizing symptoms (Compas & Reeslund, 2009; Hinshaw, 2002; Sameroff, 2009b). Family psychosocial stressors, children's individual characteristics that confer additional risk, and their coercive exchanges have been implicated in the etiology and development of externalizing symptoms, but their synthesis in research until recently has been too infrequent (Dodge & Pettit, 2003; Farrington, 2009). This final chapter summarizes three longitudinal studies presented in Chapters II, III, and IV that followed a transactional framework to illustrate bidirectional effects between externalizing symptoms and family psychosocial stressors across distinct phases of development and interactions of child gender and self-regulation. The aims were to demonstrate that children also contributed to their behavioral problems in the presence of family risk factors and that individual differences in risk were attributable to their personal characteristics. I begin this chapter by briefly summarizing the three studies' similarities, differences, and collective findings. I then weigh each study's findings to their respective literatures. Next, I evaluate support for my overarching hypotheses and

expectations across the three studies, while discussing their implications for understanding the development and etiology of externalizing symptoms. Then I briefly discuss their implications for prevention and intervention. I then discuss some limitations across the three studies. I conclude this chapter by discussing how future research can extend their respective findings to advance knowledge of the etiology, development, and prevention of externalizing symptoms.

### **Examples of Developmental Research Integrating Transactions and Interactions**

The preceding chapters presented three complementary longitudinal studies of the early lifespan investigating bidirectional effects between family psychosocial risk factors and externalizing symptoms and interactions of child gender and self-regulation. These studies were methodologically similar; using advanced structural equation modeling to elucidate the transactional and interactional interplay of risk factors in the family and within the individual. They also were conceptually distinct, examining consecutively older samples of young people spanning across the developmental range of infancy to young adulthood and their age-salient family risk factors, externalizing symptoms, and self-regulatory abilities. Each investigation uncovered evidence of transactional associations between family psychosocial stressors and young peoples' externalizing symptoms. Young children and adolescents affected psychosocial stressors within their family system, either altering mothers' depressive symptoms or worsening family conflict. These effects were specific to subsets of young children and adolescents based on their gender and/or self-regulatory competence. Overall, these studies contributed unique evidence of bidirectional and moderating effects that contributed to increases in externalizing symptoms and family risk factors, as well as replicated findings of elevated

levels of externalizing symptoms among boys and young children who had low functional and behavioral self-regulation. These findings were demonstrated with White and Black families across different developmental phases of the early lifespan, providing support for the usefulness and wide applicability of transactional and interactional approaches in developmental research on externalizing symptoms.

### **Infant Functional Self-regulation and Gender Moderate Transactions of Externalizing Behavior and Maternal Depressive Symptoms**

Study 1 in Chapter II examined transactions of maternal depressive symptoms and externalizing behavior across infancy to toddlerhood and interactions of infant gender and functional self-regulation of crying, feeding, and sleeping. This study of mostly middle class White families addressed the paucity of research investigating effects of toddlers' emerging behavioral problems on maternal depression (Connell & Goodman, 2002; Shaw, Gross, & Moilanen, 2009) and infant characteristics that attenuate risk to maternal depression (Cummings & Davies, 1994; Goodman & Gotlib, 1999).

As expected from previous research, maternal depressive symptoms at 7 months predicted higher levels of externalizing behavior at 15 months and their increase at 33 months, while controlling for socioeconomic status (SES) and concurrent depressive symptoms in toddlerhood (Chronis et al., 2007; Cummings, Keller, & Davies, 2005; Weinfield, Ingerski, & Moreau, 2009). Mostly consistent with previous studies, maternal depressive symptoms in infancy only predicted an increase in boys' externalizing behavior at 33 months, although their levels of externalizing were similar to girls (Davies & Windle, 1997; Deater-Deckard, Dodge, Bates, & Pettit, 1998; Yates, Obradović, & Egeland, 2010). While initial tests refuted a transactional hypothesis, the interaction of functional self-regulation uncovered a child-to-mother effect in which externalizing



behavior at 15 months worsened mothers' depressive symptoms at 33 months only among toddlers who had high functional self-regulation in infancy. This unexpected effect, which to my knowledge had not been reported before, reflected a mismatch of good functional self-regulatory with externalizing symptoms that mothers appeared to find more distressing than those of toddlers who had low self-regulation as infants. Findings demonstrated a transactional process through which maternal depressive symptoms in infancy contributed to elevated externalizing behavior across toddlerhood, particularly for boys, and in turn heightened depressive symptoms in mothers of toddlers who had high functional self-regulation in infancy.

### **Effortful Control Moderates Transactions of Externalizing Behavior and Maternal Depressive Symptoms across Childhood**

Study 2 in Chapter III followed slightly older at-risk children across a longer developmental range than Study 1 to investigate whether transactions between maternal depressive symptoms and children's externalizing behavior occurred more prominently later in development. This study examined mother-child transactions across the preschool period to middle childhood and interactions of child gender and effortful control, a set of self-regulatory abilities associated with modulation of emotionality and inhibition of impulses that are more advanced than Study 1's functional regulatory processes (Calkins, 2009; Rothbart & Bates, 1998). Aside from extending Study 1 to an older age group, Study 2's aims were to elucidate progression of externalizing behavior across school entry and childhood by examining their transactional interplay with maternal depressive symptoms.

Initial findings with all children refuted the hypothesis of their bidirectional effects. Consistent with prior research and Study 1, maternal depressive symptoms at age

3 predicted increases in externalizing behavior at age 10 among boys and children with low effortful control (Gross, Shaw, Burwell, & Nagin, 2009; Lengua, 2002). Contrary to other studies, the externalizing behavior of well-regulated children at age 3 predicted a gradual and modest improvement in mothers' depressive symptoms at age 10 (Shaw et al., 2009). Findings illustrated that low effortful control at age 3 increased boys and girls' risk of long-term effects of maternal depressive symptoms on their externalizing problems almost seven years later. Thus, exposure to maternal depressive symptoms in the preschool years contributed to continuing externalizing behavior from early to middle childhood among both boys and girls who had low levels of self-regulation. Importantly, boys who had low self-regulation in the preschool years were at increased risk of continuing behavioral problems through elementary school.

### **Transactions between African American Adolescents' Family Conflict and Violent Behavior: Implications for Stress and Coping**

Study 3 in Chapter IV extended Studies 1 and 2 by assessing at-risk African American adolescents across four years of high school and at age 20, permitting more tests of bidirectional effects than previous studies. Family conflict is a salient risk factor during adolescence (Gerard & Buehler, 2004; Laursen & Collins, 2009) and violent behavior typically peaks around this time (Dodge & Pettit, 2003; Tremblay, 2000). This study examined their transactional interplay across ages 15 to 18 and their effects on internalizing problems at age 20. Interactions of adolescents' gender and active coping also were examined. Whereas Studies 1 and 2 examined basic functional and behavioral self-regulatory abilities, Study 3 included active coping since it represented more sophisticated forms of emotion and behavioral regulation, and adaptive self-regulatory beliefs (e.g., high self-efficacy and goal importance) associated with positive adjustment

outcomes in adolescence (Compas & Reeslund, 2009; James, 1994; Rudolph, Lambert, Clark, & Kurlakowsky, 2001). To the best of my knowledge, no other studies have examined these constructs together with transactional or interactional approaches.

Consistent with hypotheses, there were bidirectional effects between African American adolescents' violent behavior and family conflict during high school and their elevated levels predicted more internalizing problems in young adulthood. This replicated and extended the role of family conflict as a risk factor for violent behavior (DuRant, Cadenhead, Pendergrast, Slavens, & Linder; 1994; Paschall & Hubbard; 1998) and recent evidence of transactions between marital conflict and youths' antisocial behavior (Cui, Donnellan, & Conger, 2007; Jenkins, Simpson, Dunn, Rasbash, & O'Connor, 2005). Although no a priori predictions of gender differences were made, I consistently found that males reported more violent behavior and girls indicated greater family conflict, but gender did not moderate these associations. Further supporting hypotheses, family conflict in 12<sup>th</sup> grade contributed to more internalizing problems at age 20 among adolescents who reported low active coping; however, active coping did not alter relations between family conflict and violent behavior or the effect of violent behavior on internalizing problems. The transition from adolescence to young adulthood is marked by increases in internalizing symptoms, particularly in the presence of family conflict and violent behavior (Graber & Sontag, 2009; Ozer, 2005; Voisin, 2007). African American adolescents' use of active coping appeared to reduce risk associated with family conflict.

### **Advancing Knowledge of Externalizing Symptom Etiology and Development**

I hypothesized that family psychosocial risk factors and externalizing symptoms would intensify each other over time, and especially among young people who had low

self-regulation. In other words, children and adolescents who had high self-regulation were expected to develop fewer externalizing symptoms and to be less affected by family risk factors than those who had low self-regulation. I also expected boys to be more adversely affected by family risk factors than girls, but I did not hypothesize as to whether gender or self-regulation groups would vary in how they affected family stressors due to a lack of supporting evidence. Across these three studies, I expected that adolescents would be less adversely affected by family risk factors than younger children who often spend the majority of their time at home, and thus are often exposed to more concurrent family stressors. I also expected adolescents' externalizing symptoms to exacerbate family stressors to a greater extent than younger children, because their violent behavior inflicts more harm to others and has stricter legal consequences than young children's externalizing behavior (Tremblay, 2000). I discuss these topics by order of transactions, interactions of self-regulation, interactions of gender, and age differences.

**Evidence of transactional processes.** Family psychosocial risk factors and externalizing symptoms were hypothesized to worsen each other over time among all children, but this was only supported with adolescents in Study 3. African American adolescents' violent behavior and family conflict predicted increases in each other from 10<sup>th</sup> to 11<sup>th</sup> grade. Studies 1 and 2, in contrast, only demonstrated bidirectional effects between externalizing behavior and maternal depressive symptoms among subsets of young children. Adolescence may be a time when transactional processes of reciprocated stress within families occur more frequently and noticeably than earlier in development. This may be due to adolescents' violent behavior being more severe than children's externalizing symptoms. Children typically begin displaying disruptive non-delinquent

behavior in their homes and with their families, which can progressively spread to school and community settings among peers and other relatives (Loeber & Farrington, 2000). Over time, children's behavioral problems may escalate to violent and delinquent offenses directed at strangers with greater frequency than earlier in development. The increasing pervasiveness and seriousness of youths' externalizing symptoms are reflected by the *age-crime curve*'s peak in violent offending at ages 16 and 17 (Dodge & Pettit, 2003; Tremblay, 2000). In Study 3, African American adolescents' highest levels of violent behavior were in 9<sup>th</sup> and 10<sup>th</sup> grade, respectively ages 15 and 16, which contributed to subsequent increases in family conflict.

Transactions between family conflict and adolescents' violent behavior appeared to be relatively stable. Interactions of gender and active coping did not moderate their bidirectional relations during adolescence, whereas transactions between maternal depressive symptoms and young children's externalizing behavior only emerged among subsets of children who differed in self-regulation. Infants in Study 1 were all negatively affected by maternal depressive symptoms at 7 months, but only toddlers who had high functional self-regulation in infancy consequently worsened mothers' depressive symptoms across 15 and 33 months. Study 2 did not demonstrate transactions between maternal depressive symptoms and externalizing behavior within any single group. Adverse effects of early maternal depressive symptoms only contributed to later externalizing behavior in boys and children who had low effortful control at age 3. Conversely, preschoolers who had high effortful control at age 3 were the only children whose externalizing behavior contributed to decreases in mothers' depressive symptoms. Thus, specificity was needed in early and middle childhood, as transactions only emerged

among subsets of children, whereas in adolescence they were present for everyone. I further delineate the effects children and adolescents had on their families in the following discussions of interactions of self-regulation and gender and age differences.

**Interactions of self-regulation.** I hypothesized that family psychosocial stressors and externalizing symptoms would worsen each other to a greater extent among children and adolescents who had low self-regulation. This was not supported. Only Study 3 demonstrated bidirectional effects between externalizing symptoms and family psychosocial stressors among its entire sample, but active coping did not moderate them. African American adolescents' active coping only modestly contributed to individual differences in risk of developing internalizing problems. Considering the severity of adolescents' violent behavior and their contributions to family conflict, use of active coping may not have been sufficient to elevate or diminish the adverse transactions operating between adolescents and their families. To reiterate points made in Chapter IV, individual characteristics that alter effects of risk factors, such as self-regulatory abilities, do little to prevent or exacerbate externalizing outcomes when the developing person's social-context is unsupportive and full of risk factors (Compas & Reeslund, 2009; Gerard & Buehler, 2004; Ozer, 2005; Zimmerman, Steinman, & Rowe, 1998). Active coping was not associated with levels of violent behavior, so it is logical that it also did not attenuate effects of violent behavior on family conflict.

Children and adolescents who had high self-regulation were expected to develop fewer externalizing symptoms and to be less affected by family risk factors than youths who had low self-regulation. All three studies were partially consistent with these expectations. Study 1 replicated findings that good functional self-regulation of basic

processes such as feeding in infancy were associated with fewer externalizing symptoms in toddlerhood (Calkins, 2009; Hemmi, Wolke, & Schneider, 2011; Wolke, Schmid, Schreier, & Meyer, 2009). All toddlers, however, were adversely affected by their mothers' depressive symptoms. Infants' self-regulatory skills reflect the quality of the regulation provided by caregivers and are as much a characteristic of their social environment as they are of individual infants (Olson & Lunkenheimer, 2009; Sameroff, 2009a; Sroufe, Duggal, Weinfield, & Carlson, 2000). Although infants who had more regulatory problems also had more externalizing behavior as toddlers, this did not appear to be due to maternal depressive symptoms.

Study 2 demonstrated that maternal depressive symptoms at age 3 predicted an increase in externalizing behavior at age 10 among children with low effortful control. These same children had almost double the levels of externalizing behavior as their well-regulated peers. This longitudinal evidence of self-regulation moderating effects of maternal risk extended similar cross-sectional findings in middle childhood with cumulative risk factors (Lengua, 2002). It also extended findings that maternal risk predicted increases in internalizing problems, but not externalizing, across middle childhood and early adolescence for children with low effortful control (Lengua, Bush, Long, Kovacs, & Trancik, 2008). Study 2 demonstrated that preschoolers' effortful control could identify 3-year-olds who were at elevated risk of early maternal depressive symptoms and later externalizing behavior. Because this study ended in middle childhood, relations may have been stronger for externalizing behavior than internalizing symptoms, which typically increase through early adolescence (Graber & Sontag, 2009).

Relatedly, Study 3 demonstrated that African American adolescents' use of active

coping reduced adverse effects of family conflict in 12<sup>th</sup> grade on their internalizing problems at age 20. Family conflict only worsened the internalizing problems of adolescents who had low levels of active coping. Again, active coping was not associated with violent behavior, although violent behavior was more stable from 9<sup>th</sup> to 10<sup>th</sup> grade among adolescents who used less active coping. This difference in stability of violent behavior, however, did not lead to differences in levels of severity or their effects.

The interactions of effortful control in Study 2 and active coping in Study 3 were consistent with evidence that self-regulation modifies vulnerability to environmental stressors (Buckner, Mezzacappa, & Beardslee, 2003; Lengua, 2002). Self-regulation in response to stressors can either increase vulnerability to their adverse effects and consequently exacerbate problematic outcomes, or protect against effects of stress and reduce likelihood of negative outcomes (Compas & Reeslund, 2009; Lengua et al., 2008). Interpreting the interactions of effortful control and active coping within a resilience framework can illustrate the role of self-regulation in attenuating vulnerability.

In Study 2, maternal depressive symptoms were not related to externalizing behavior among the full sample. The interaction of effortful control revealed that exposure to maternal depressive symptoms in the preschool years contributed to increases in externalizing behavior at age 10, only among children who had low effortful control at age 3. Thus, low self-regulation in the preschool years increased children's vulnerability to the negative effects of maternal depressive symptoms on their externalizing behavior. Children who had high effortful control were not affected.

In Study 3, family conflict in 12<sup>th</sup> grade contributed to more internalizing problems at age 20 among the entire sample. The interaction of active coping



demonstrated a buffering effect in which family conflict no longer predicted internalizing problems in young adulthood, only among adolescents who reported high active coping in 9<sup>th</sup> grade. Therefore, high active coping in high school protected adolescents from the negative effects of family conflict on their internalizing problems. Family conflict continued to contribute to the internalizing problems of low active coping adolescents.

Studies 2 and 3 provided complementary evidence of children and adolescents' self-regulation that altered vulnerability to family psychosocial stressors in different ways. Self-regulatory processes operate in the context of stress. Too much stress can lead to over arousal, regulatory problems, and eventually disruptive behavior (Calkins, 2009; Cummings & Davies, 1994). The goal of self-regulatory functioning is to manage an individual's arousal to stress and to inhibit socially inappropriate emotional and behavioral impulses, thus maintaining homeostatic levels of arousal that facilitate adaptation to situational demands (Olson, Sameroff, Kerr, & Lunkenheimer, 2009; Posner & Rothbart, 2000). Self-regulatory processes therefore constitute coping responses that embody effortful regulation initiated in the presence of stress. Coping responses are best understood as constituents of self-regulation and are one of the primary processes through which resilience is achieved (Compas & Reeslund, 2009).

Studies 2 and 3 extended research on early temperament to coping literature by demonstrating how self-regulatory processes in early childhood and adolescence altered risk for externalizing and internalizing problems, and contributed to individual differences in vulnerability to family stressors. Study 3 showed that benefits of active coping extended to African American adolescents. In this chapter, I considered active coping as an advanced set of self-regulatory abilities and adaptive beliefs, such as high

self-efficacy, associated with positive behavioral and emotional outcomes in adolescence (Compas & Reeslund, 2009; Rudolph et al., 2001). Temperament characteristics underlie cognitive, emotional, and behavioral responses to stress that are instrumental to coping and behavioral adjustment (Lengua, 2002; Rothbart & Bates, 1998). But evidence linking early temperament difficulties with long-term externalizing outcomes has been mixed (e.g., Aguilar et al., 2000; Moffitt & Caspi, 2001). Patterns of self-regulatory difficulties more recently have been associated with youths' elevated externalizing symptoms (Eisenberg, Zhou, Spinrad, Valiente, Fabes, & Liew, 2005; Gartstein & Fagot, 2003; Lengua, 2006; Martel & Nigg, 2006; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005) and increased vulnerability to family, community, and socioeconomic risk factors (Buckner et al., 2009; Lengua, 2002; Lengua et al., 2008). Study 2 extended these findings by demonstrating effortful control's role in attenuating bidirectional associations between maternal depressive symptoms and children's externalizing symptoms. Early temperament characteristics such as self-regulation are critical to coping responses to stressors in childhood and adolescence.

The development of self-regulation increases the amount of adaptive coping responses young people have at their disposal and their flexibility in using them, therefore contributing to individual difference in vulnerability to risk factors (Compas & Reeslund, 2009). Self-regulatory abilities are critical to supporting resilience processes (Buckner et al., 2009; Masten, 2001). Resilience is a process whereby an individual overcomes significant adversity to achieve positive adaptation and healthy adjustment (Fergus & Zimmerman, 2005; Luthar, Cicchetti, & Becker, 2000; Masten, 2007a). Resilience researchers use person-centered models to identify protective and vulnerability

factors that alter adverse effects of risk factors (Luthar & Cicchetti, 2000; Masten, 2001). Protective factors are individual or environmental characteristics that alter effects of risk factors in a positive direction and are related to resilient outcomes (Compas & Reeslund, 2009; Luthar & Cicchetti, 2000). Vulnerability factors are characteristics that exacerbate the negative effects of risk factors, such as in a diathesis–stress model (Fergus & Zimmerman, 2005). Protective and vulnerability factors modify negative effects of stress in opposite ways. Both are identified with tests of interactions where a characteristic moderates the effect of a risk factor on a negative outcome in either a positive or negative direction. Studies of resilience, however, tend to ignore transactions between the individual and social-context and patterns of developmental change, presenting major challenges to understanding processes of risk and resilience (Compas & Reeslund, 2009; Masten, 2001). As illustrated in Studies 2 and 3, integrating tests of transactions and interactions provided evidence that self-regulatory processes modified youths' vulnerability to environmental risk factors and likelihood of adjustment problems. Continued efforts examining self-regulation in the presence of stress with these combined approaches can clarify vulnerability and protective factors related to externalizing symptoms, as well as resilience when confronted with their associated risk factors.

**Interactions of gender.** Evidence of gender differences in risk factors and both severity and expression of children's externalizing symptoms led me to expect that boys would be more adversely affected by family risk factors than girls (Gorman-Smith & Loeber, 2005; Kroneman, Loeber, & Hipwell, 2004; Tremblay, 2000). Consistent with this expectation, Study 1 demonstrated that maternal depressive symptoms at 7 months predicted an increase in boys' externalizing behavior at 33 months, but not in girls. In

addition, Study 2 revealed that maternal depressive symptoms at 3 years predicted an increase in boys' externalizing behavior at 10 years, and again this was not significant for girls. Boys also demonstrated more stable and higher levels of externalizing behavior across Study 2. Study 3 only demonstrated gender differences in levels of violent behavior and family conflict, with males faring worse for the former but not latter.

Gender differences across studies were consistent with research suggesting that boys are more susceptible to contextual risk factors in early childhood (Cummings & Davies, 1994; Yates et al., 2010), and that boys are more likely to manifest externalizing symptoms as adjustment problems than girls (Davies & Windle, 1997). Findings from Study 2 conflicted with previous research that showed gender did not moderate cumulative effects of early risk factors on externalizing behavior problems in middle childhood (Deater-Deckard et al., 1998). Boys' early exposure to maternal depressive symptoms predicted an increase in their externalizing behavior in middle childhood, and although this effect also was significant for both boys and girls who had low self-regulation, just being a boy with low self-regulation was enough to predict an increase in their school-age externalizing behavior. Gender interactions across Studies 1 and 2 demonstrated that boys were more vulnerable than girls to negative effects of maternal depressive symptoms in infancy and preschool years and were more likely to develop elevated externalizing symptoms in early school years and middle childhood.

I did not hypothesize whether gender or self-regulation groups would vary in how they affected family stressors due to a lack of previous research on this topic. Given that deficits in self-regulation (e.g., Olson et al., 2005) and being a boy (Deater-Deckard et al., 1998; Tremblay, 2000) are associated with elevated externalizing symptoms, one

could assume that young people who have these characteristics would be a greater burden to their parents and other relatives. Yet, no gender differences in child-to-mother effects were found in Studies 1 or 2 and no group differences in family-child effects were found in Study 3. Both Studies 1 and 2, however, demonstrated that only children who had high self-regulation affected their mothers' depressive symptom. Moreover, these effects were different signs across studies. In Study 1, the externalizing behavior of toddlers who had good functional self-regulation in infancy predicted an increase in mothers' depressive symptoms, and in Study 2, the externalizing behavior of preschool-age children who had good effortful control predicted a decrease in mothers' depressive symptoms.

Although Shaw and colleagues (2009) did not examine the role of gender or self-regulation, they found that boys' conduct problems at ages 3.5 and 5 years predicted increases in mothers' depressive symptoms at 5 and 6 years, respectively. Gross and colleagues (2009) also found that boys' noncompliance at 18 months predicted mothers' more persistent and higher levels of depressive symptoms over the next 8 years. Study 1 extended these findings to girls and boys, demonstrating that some toddlers' externalizing behavior at 15 months predicted increases in maternal depressive symptoms at 33 months, which to the best of my knowledge is earlier in development than any other study in the maternal depression literature.

Study 2 also revealed a unique child-to-parent effect in which some preschoolers' externalizing behavior predicted a decrease in mothers' depressive symptoms almost seven years later. No other study to the best of my knowledge has shown a similar effect as this. These children, who had high effortful control as preschoolers, had milder and more normative levels of externalizing behavior across childhood, which may explain

why their mothers' depressive symptoms decreased over this time span. In both examples of child-to-mother effects in Studies 1 and 2, only children who had high self-regulatory competence and modest externalizing behavior affected mothers' depressive symptoms. Further understanding the qualitative nature of their externalizing symptoms may reveal whether manifestations among children who have high self-regulation differ from those of children who have both self-regulatory difficulties and externalizing problems.

**Age differences.** Across studies, I expected that adolescents would be less adversely affected by family risk factors than younger children. Partially supporting this, I found that maternal depressive symptoms in infancy exacerbated all children's externalizing behavior in toddlerhood. In Study 2, only boys and children who had low self-regulation in the preschool-years were adversely affected by maternal depressive symptoms. All adolescents in Study 3 were affected by family conflict in the middle of high school but this occurred after their initial violent behavior in 9<sup>th</sup> grade worsened family conflict in 10<sup>th</sup> grade. Although Studies 1 and 3 provided modest support for the hypothesis that older youths are less affected by family stressors than younger children, Study 2 indicated that children's early experiences of family psychosocial stress had a lasting impact on their externalizing symptoms almost 8 years later. Similarly, studies have found that exposure to heightened levels of maternal distress and harsh physical discipline before age 5 predicted elevated externalizing problems many years later (Choe, Olson, & Sameroff, in press; Keiley, Howe, Dodge, Bates, & Pettit, 2001). These findings are consistent with notions of a sensitive period to stress in early childhood (Connell & Goodman, 2002; Lovejoy et al., 2000) and the accumulation of stress over time that collectively produces the greatest risk of escalating externalizing symptoms

(Deater-Deckard et al., 1998; Sameroff, 2000; 2010; Yoshikawa, 1994). Thus, adolescents may not be as intensely influenced by immediate family conflict as young children are with maternal depression, but all youths internalize a history of stressful family experiences that continues to influence their adjustment. The accumulation and interaction of risks across the wide age range of 3 to 10 years in Study 2 demonstrated the strength of integrating these approaches in elucidating these long-term pathways to externalizing symptoms (Dodge & Pettit, 2003; Farrington, 2009; Hinshaw, 2002).

I also expected adolescents' violent behavior to exacerbate family stressors to a greater extent than younger children's externalizing behavior, because they inflict more harm and have stricter legal consequences. As expected, adolescents' violent behavior predicted increases in family conflict at 10<sup>th</sup> and 11<sup>th</sup> grade, whereas family conflict predicted an increase in violent behavior at only 11<sup>th</sup> grade. This was the only example from the three studies in which young people affected the family system more times than they were affected by it. As explicated earlier, child-to-mother effects in Studies 1 and 2 were specific to children who had high self-regulation. Only half of the children in these two studies contributed to higher levels of maternal depressive symptoms, in contrast to all adolescents who contributed to family conflict. Granted samples differed in family risk level and racial composition, the findings from Study 3 suggested that violent behavior is a particularly stressful form of externalizing symptoms for the family system. The greater severity of externalizing symptoms during adolescence than in early childhood is a major concern for parents and other family members of adolescents with violent tendencies (Dodge & Pettit, 2003; Loeber & Farrington, 2000; Tremblay, 2000).

In sum, three studies presented in Chapters II, III, and IV, illustrated the bidirectional nature of stressors within families and individual differences in risk attributable to children's gender and self-regulation. Externalizing symptoms affected family psychosocial stressors in three successive phases of development, and in some cases, these effects were only evident after testing for interactions of individual characteristics. These unique and occasionally unexpected findings were exemplary in demonstrating the utility of a transactional framework with an interactional component in uncovering child effects. The synthesis of these methods with a developmental perspective and risk-focus contributed evidence indicating that transactional processes involving youths' externalizing symptoms and family risk factors were present throughout the early lifespan. Specificity was required in determining when in development and among which groups of individuals these bidirectional effects would be found. Individual characteristics of gender and self-regulation attenuated children's vulnerability to family psychosocial stressors and risk of developing externalizing symptoms. This research demonstrated the successive progression of regulatory functioning from overseeing basic autonomic processes, such as crying and feeding, to more consciously effortful processes underlying adaptive coping responses. Whereas in infancy these regulatory skills only appeared to attenuate children's contributions to mothers' depressive symptoms, from early to middle childhood self-regulation moderated bidirectional effects between children and mothers' adjustment problems. Further in adolescence, high active coping served as a protective factor in buffering negative effects of family conflict on adolescents' internalizing problems.



Children with deficient levels of self-regulation who reside in stressful family environments can be identified as being at elevated vulnerability to psychosocial stressors and externalizing symptoms. Self-regulatory abilities can be reliably measured early in development and targeted for intervention to promote children's positive adaptation to future developmental tasks. Identifying other individual-level vulnerabilities and protective factors in youths can inform intervention and prevention of externalizing symptoms and other adjustment problems. Next, I discuss the translational relevance of this research and a transactional and interactional approach.

### **Implications for Prevention and Intervention of Externalizing Symptoms**

Over half of all crimes in the U.S. are committed by 7% of the population, who individually cost society between \$1.6 and \$2.3 million (Dodge, 2008; Dodge & Pettit, 2003). Disproportionate costs of crime attributable to a minority of the U.S. population encourage researchers to focus on the prevention of antisocial behavior in younger populations. Implementing widespread prevention efforts targeting risk factors early in children's development can prevent the onset of stable patterns of externalizing symptoms and be more cost effective than attempting to reduce maladjustment after its emergence (Loeber & Farrington, 2000; Luthar & Cicchetti, 2000). Effective prevention programs tend to be multisystemic in targeting multiple risk and protective factors, employ behavioral interventions within real social settings, are specific to individual children, and include supports to ensure fidelity (Campbell, Shaw & Gilliom, 2000; Henggeler & Schoenwald, 2011; Olsson, Bond, Burns, Vella-Brodrick, & Sawyer, 2003; Yoshikawa, 1994). Importantly, prevention and intervention efforts tend to be more effective with children younger than 5-years-old or during the preschool period when

foundations for prosocial and antisocial behavior are established, so they should follow a developmental perspective with a strong theoretical basis for targeted youth (Dodge & Pettit, 2003; Loeber & Farrington, 2000; Luthar & Cicchetti, 2000).

Psychosocial risk factors associated with early coercive parent–child exchanges are key to the etiology of serious externalizing symptoms and are important targets of intervention (Campbell et al., 2000; Loeber & Farrington, 2000; Patterson, DaBaryshe, & Ramsey, 1989). Programs that improve children’s early caregiving environments can prevent the formation of coercive family processes and the emergence of clinically-significant externalizing symptoms (Luthar & Brown, 2007; Olsson et al., 2003; Yoshikawa, 1994). For example, evidence from studies presented in the previous chapters support targeting maternal depression prior to or during the preschool period to help avert children’s age-aberrant behavioral problems. Yet the direction of causality has been especially difficult to establish in studies of family influences and children’s development (Cowan & Cowan, 2003), and evidence from the presented studies indicated that some children’s early externalizing symptoms exacerbated mothers’ depressive symptoms.

Transactions between family risk factors and child externalizing symptoms can be better understood with prevention studies. Prevention of chronic externalizing symptoms provides opportunities to empirically test putative causal risk factors in developmental models while searching for ways to help at-risk children (Tremblay, 2000). The field of *prevention science* addresses the study of etiology and development, the design of controlled intervention trials determining the causal and meditational role of variables, the design of field trials to clarify process, and the implementation and evaluation of community prevention programs (Coie, Miller-Johnson, & Bagwell, 2000; Dodge &

Pettit, 2003). One method of determining whether maternal depression is a cause of children's externalizing symptoms, rather than a consequence, is to conduct controlled experiments with random assignment of participants to intervention and control conditions (Cowan & Cowan, 2003). The intervention should target maternal depression, and analyses should establish the intervention had a positive influence on children's behavioral outcomes associated with *improvement* in mothers' depressive symptoms.

For example, a randomized intervention study of mothers with postpartum depression demonstrated that treating mothers with interpersonal psychotherapy alleviated their depressive symptoms and parenting stress when compared to mothers with depression who were placed on a waiting list and nondepressed control mothers. However, there was no effect of recovery with treatment on mothers' responsiveness to their children, mother-child relationships, or children's subsequent behavioral problems (Forman, O'Hara, Stuart, Gorman, Larsen, & Coy, 2007). The researchers concluded that successfully treating a mother's depression does not improve mother-child exchanges or prevent children's externalizing symptoms and that a parenting component driving cognitive and behavioral change is needed to ensure mother-child interactions promote children's optimal social-emotional development (Forman et al., 2007). Thus, successful interventions should focus on proximal mechanisms that promote positive transactional exchanges between mothers and children rather than just their mental health problems.

A randomized prevention study demonstrated that a brief family intervention promoted mothers' use of positive parenting techniques in toddlerhood, which in turn prevented behavioral problems in high-risk children and coercive parent-child exchanges (Dishion, Shaw, Connell, Gardner, Weaver, & Wilson, 2008). A follow up study, further

indicated that improvements in both positive parenting and maternal depression mediated effects of this brief intervention on children's behavioral problems (Shaw, Connell, Dishion, Wilson, & Gardner, 2009). This family intervention was specifically tailored to families' needs and mostly focused on parenting and only a subgroup of families received treatment for maternal depression. To attain stronger support for the causal roles of parenting quality and maternal depression, experimental prevention trials are needed that randomly assign mothers with depression to conditions focusing on either parenting training or treatment of depression. Comparison of change in children's externalizing symptoms across these conditions can provide a clearer understanding of which foci of intervention plays a larger role in the development of children's behavioral problems.

Prevention programs that target proximal processes can delineate their influence on children's externalizing symptoms, but child-effects also should be experimentally tested to determine their relative contribution to coercive mother-child transactions. Evidence from the studies presented in the previous chapters indicated that early self-regulation skills may be fruitful targets of intervention that can help elucidate the causal role children play in their contextual risks and externalizing symptoms. Interventions that target preschool-age children's self-regulation can help prevent their later adjustment problems and difficulties adapting to salient developmental tasks such as adjusting to school and peer settings (Blair & Diamond, 2008). For example, Rueda and colleagues (2003) were able to improve preschool-age children's self-regulatory skills using computerized attention exercises in 5-day training programs. This brief training enhanced executive attention scores for 4- and 6-year-old children and led to changes in brain activity comparable to older children and adults. Contrasting change in externalizing

behavior over time between children who receive self-regulatory training and children whose mothers receive either parenting training or treatment for their depressive symptoms can clarify their causal roles in the development of age-aberrant behavioral problems. Importantly, changes in maternal depressive symptoms and parenting quality can be contrasted between child-training and control conditions to provide support for the causal role of children's behavioral problems for these family psychosocial influences.

Following the integrative approaches elucidated in these chapters, multiple treatment conditions targeting parental and child risk factors can be contrasted to various control groups to delineate causal influences on children's emerging externalizing symptoms and associated family stressors. Refinement of developmental models for externalizing symptoms can inform the design and planning of field trials, and lead to larger community prevention programs targeting at-risk children and families.

### **Limitations**

Several issues across the three studies reviewed in this chapter require discussion. Different measures and informants of externalizing symptoms contributed to some inconsistency across studies. Some measures of externalizing behavior administered in Studies 1 and 2 were part of the Achenbach System of Empirically Based Assessment instruments (ASEBA; Achenbach, 1992; 1997), considered gold standard measures in developmental research. Study 1 also included mother ratings with the Infant–Toddler Social and Emotional Assessment, which categorized externalizing symptoms as peer aggression, activity level, and negative emotional reactivity (ITSEA; Briggs-Gowan & Carter, 1998). Study 2 included teacher ratings on two forms of ASEBA measures, one for young children that conceptualized externalizing symptoms as destructive or

aggressive behaviors, and the other for older children that categorized externalizing symptoms as rule-breaking or aggressive behaviors (Achenbach, 1992; 1997; Achenbach, Dumenci, & Rescorla, 2002). Teachers indicated that more children on average in Study 2 met clinical-levels of externalizing problems (9%) than mothers in Study 1 who rated relatively few children as having clinical-level problems at 33 months (2%). In contrast, Study 3 used structured interviews to assess adolescents' reports of their violent behavior with an 8-item scale recommended by the Center for Disease Control and Prevention (CDC, 1995). No norms were available to compare adolescents' violent behavior, which made it difficult to contrast the clinical significance of their problems with children in the other studies. Furthermore, varying measures across studies suggest they examined distinct forms of externalizing; however, externalizing symptoms are a heterogeneous collection of behaviors that often co-occur and progress to more severe manifestations over time (Hinshaw, 2002; Loeber & Farrington, 2000; Tremblay, 2000). Assessing externalizing behavior in early childhood and violent behavior in adolescence captured symptoms that were most salient during those developmental phases. Similarly, informants were selected to provide the most reliable assessments of externalizing symptoms at their respective phase in development. Mothers, often the primary caretakers of infants and toddlers, were assessed in Study 1. Teachers provided another perspective to children's problems during school-age years and were assessed in Study 2 to avoid common method variance with mothers' ratings of their depressive symptoms. Adolescents in Study 3 were assessed with reliable structured interviews since they can provide the most accurate reports of their violent behavior (Compas & Reeslund, 2009; Farrington, 2008). Despite some inconsistency of informants and measures across the

three studies, all the administered assessments had good psychometrics and are used frequently in research, indicating each study had sound methods for their investigations.

Another caveat that is similar to the previously discussed was the different measures and types of family psychosocial stressors across studies. Studies 1 and 2 examined maternal depressive symptoms because they are consistently associated with externalizing symptoms across toddlerhood to middle childhood (Connell & Goodman, 2002; Cummings & Davies, 1994). Whereas Study 1 used a 20-item clinical screening instrument for depression, the Center For Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), Study 2 administered an 8-item depression symptom scale from the Brief Symptom Inventory (BSI; Derogatis, 1993; Derogatis & Fitzpatrick, 2004). No information regarding these instruments' convergent validity with each other was found, but both have excellent criterion validity (Derogatis & Fitzpatrick, 2004; Radloff, 1977). Throughout Study 1, 14% to 19% of mothers exceeded the clinical cut-off for depression, whereas in Study 2, about 32% of mothers scored at or above the 70th percentile range for nonpatient adult females. It is difficult to ascertain how comparable these scores are, but both instruments provided a wide range of scores, suggesting that they were sensitive to individual differences. Study 3 examined family conflict, which is a more frequent family stressor in adolescence (Collins & Steinberg, 2006; Gerard & Buehler, 2004; Laursen, Coy, & Collins, 1998). Comparing adolescents' experiences of family conflict to young children's exposure to maternal depressive symptoms hindered more definitive conclusion from being drawn across the three studies. However, selection of these family psychosocial stressors was sensitive to their developmental salience and thus provided findings that can more easily be compared to and integrated within their respective

literatures. All three longitudinal studies contributed evidence of bidirectional effects between their respective forms of externalizing symptoms and family psychosocial stressors, suggesting that transactional processes occur across a range of risk factors emanating from within the individual and his or her family system.

Lastly, sociodemographic characteristics of participants in each of the three studies limit generalizability of their respective findings and warrant prudent interpretation of conclusions drawn in this chapter. Participants from Studies 1 and 2 consisted of mostly White middle class children from two parent families recruited from the greater area of a large university in the Midwest. Many parents attained levels of education and occupational prestige that are higher than national averages. Single parent families living below the poverty line were not recruited to reduce confounding effects of severe socioeconomic disadvantage. In contrast, Study 3 included African American adolescents from a medium sized urban community in the Midwest characterized by poverty, high adult unemployment, community violence, crime, and other correlates of socioeconomic disadvantage associated with family risk factors and externalizing symptoms (Aisenberg & Herrenkohl, 2008; Campbell, Matestic, von Stauffenber, Mohan, & Kirchner, 2007; Chung & Steinberg, 2006; Dodge & Pettit, 2003; Farrington, 2009; Gershoff, 2002; Keiley et al., 2001). In many ways the recruitment criteria for Study 3 mirrored exclusionary criteria for Studies 1 and 2, making it difficult to form general conclusions across their respective findings. Yet, each study provided evidence of bidirectional relations between family risk factors and externalizing symptoms, suggesting that transactional processes were universal across racial-ethnic and socioeconomic groups, as well as across development from infancy to adulthood. Further



evidence in each sample of self-regulatory abilities reducing risk associated with family stressors substantiated the integral role of self-regulation in coping and adjustment.

### **Future Directions and Conclusion**

Studying the interface of children's gender, self-regulation, externalizing symptoms, and family psychosocial stressors produced some unique and unexpected findings that helped clarify the etiology and development of externalizing symptoms. Three distinct studies contributed evidence that children actively influence their social ecologies and the progression of their own behavioral problems, therefore supporting the generalizability of transactional processes across White and Black families and the developmental range from infancy to young adulthood. Interactions of children's gender and self-regulatory processes demonstrated individual differences in risk for developing externalizing symptoms and vulnerability to family psychosocial stressors. Young children and adolescents affected psychosocial stressors within their family system, either altering mothers' depressive symptoms or worsening family conflict, but some of these effects were specific to subsets of young children. Future research examining transactions between the same children and families across the early lifespan would improve on these studies with large-scale nationally representative samples and more compatible measures of externalizing symptoms and risk factors. More robust findings with better external validity would be produced, leading to a better understanding of the transactional interplay of youths' externalizing symptoms and risk factors in their social ecologies. Further studies examining parenting and child adjustment with a transactional perspective can help elucidate the bidirectional nature of parent-child exchanges, particularly those focusing on parenting behaviors that are elicited by and reinforce children's behavioral

problems. In accord with Grant and colleagues (2003), future studies can extend this research by examining mediators that explain or account for the relationship between stressors and psychopathology, as well as specificity in relations among stressors, moderators, mediators, and psychopathology. Prevention studies also can provide a better means of delineating causality among family and child factors related to externalizing symptoms than nonexperimental investigations. This may provide a clearer understanding of the onset of externalizing symptoms and whether their primary causal risk factors are located within the child, family, or their coercive exchanges. Taken together, the integrative approach discussed throughout these chapters has the potential to inform prevention of externalizing symptoms by identifying causal risk factors and individual characteristics that amplify risk for more severe behavioral problems. More knowledge of causal risk factors and their complex interplay across system levels and development can improve the early identification and treatment of young children who are at risk for chronic externalizing symptoms.

## References

- Achenbach, T. M. (1992). *Manual for the Child Behavior Checklist/2-3 and 1992 profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M. (1997). *Teacher–Caregiver Report Form for Ages 2-5*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2002). Ten-year comparisons of problems and competencies for national samples of youth: Self, parent, and teacher reports. *Journal of Emotional and Behavioral Disorders, 10*, 194–203.
- Achenbach, T. M., Howell, C. T., Quay, H.C., Conners, C. K., & Bates, J. E. (1991). National survey of problems and competencies among four- to sixteen-year-olds: Parents' reports for normative and clinical sample. *Monographs for the Society for Research in Child Development, 56*, 1–130.
- Aguilar, B., Sroufe, L. A., Egeland, B., & Carlson, E. (2000). Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: From birth to 16 years. *Development and Psychopathology, 12*, 109–132.
- Ahadi, S. A., Rothbart, M. K., & Ye, R. (1993). Children's temperament in the U.S. and China: Similarities and differences. *European Journal of Personality, 7*, 359–378.
- Aisenberg, E., & Herrenkohl, T. (2008). Community violence in context: Risk and resilience in children and families. *Journal of Interpersonal Violence, 23*, 296–315.
- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders (4<sup>th</sup> ed.—Text Revision)*. Washington, DC.
- Andreas, J. B., & Watson, M. W. (2009). Moderating effects of family environment on the association between children's aggressive beliefs and their aggression trajectories from childhood to adolescence. *Development and Psychopathology, 21*, 189–205.
- Beardslee, W. R., Bemporad, J., Keller, M. B., & Klerman, G. L. (1983). Children of parents with major affective disorder: A review. *American Journal of Psychiatry, 140*, 825–832.
- Bell, R. Q. (1968). A reinterpretation of the direction of effects in studies of socialization. *Psychological Review, 75*, 81–95.
- Belsky, J. (1984). The determinants of parenting: A process model. *Child Development, 55*, 83–96.
- Berg-Nielsen, T. S., Vikan, A., & Dahl, A. A. (2002). Parenting related to child and parental psychopathology: A descriptive review of the literature. *Clinical Child Psychology and Psychiatry, 7*, 529–552.
- Bernier, A., Carlson, S. M., Bordeleau, S., & Carrier, J. (2010). Relations between physiological and cognitive regulatory systems: Infant sleep regulation and subsequent executive functioning. *Child Development, 81*, 1739–1752.
- Blair, C., & Diamond, A. (2008). Biological processes in prevention and intervention: The promotion of self-regulation as a means of preventing school failure. *Development and Psychopathology, 20*, 899–911.
- Boomsma, A. (2000). Reporting analyses of covariance structures. *Structural Equation Modeling, 7*, 461–483.
- Bridgett, D. J., Gartstein, M. A., Putnam, S. P., McKay, T., Iddins, E., Robertson, C. et

- al., (2009). Maternal and contextual influences and the effect of temperament development during infancy on parenting in toddlerhood. *Infant Behavior and Development*, 32, 103–116.
- Briggs-Gowan, M. J., & Carter, A. S. (1998). Preliminary acceptability and psychometrics of the infant–toddler social and emotional assessment (ITSEA): A new adult-report questionnaire. *Infant Mental Health Journal*, 19, 422–445.
- Bronfenbrenner, U. (1994). Ecological models of development. In *International Encyclopedia of Education*, Vol. 3, 2<sup>nd</sup> Ed. Oxford: Elsevier
- Bronfenbrenner, U., & Morris, P. A. (2006). The bioecological model of human development. In W. Damon (Series Ed.) & R.M. Lerner (Volume Ed.) *Handbook of Child Psychology Vol. 1: Theoretical Models of Human Development 6th Ed.* (pp. 793–828). NY: Wiley.
- 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.
- Burt, S.A., McGue, M., Krueger, R.F., & Iacono, W.G. (2005). How are parent-child conflict and childhood externalizing symptoms related over time? Results from a genetically informative cross-lagged study. *Development and Psychopathology*, 17, 145–165.
- Calkins, S. D. (2009). Regulatory competence and early disruptive behavior problems: The role of physiological regulation. In S. L. Olson and A. J. Sameroff (Eds.), *Biopsychosocial Regulatory Processes in the Development of Childhood Behavioral Problems* (pp. 86–115).
- Campbell, S. B., Shaw, D. S., & Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology*, 12, 467–488.
- Centers for Disease Control and Prevention. (1995). Youth risk behavior surveillance—United States. *Morbidity and Mortality Weekly Review*, 45 (September 27), No. SS–4.
- Choe, D. E., Olson, S. L., & Sameroff, A. J. (in press). Effects of early maternal distress and parenting on the development of children’s self-regulation and externalizing behavior. *Development and Psychopathology*.
- Choe, D. E., Olson, S. L., & Sameroff, A. J. (under review). The interplay of externalizing problems and physical and inductive discipline during childhood
- Chronis, A. M., Lahey, B. B., Pelham, W. E., Williams, S. H., Baumann, B. L., Kipp, H., Jones, H. A., & Rathouz, P. J. (2007). Maternal depression and early positive parenting predict future conduct problems in young children with attention-deficit/hyperactivity disorder. Collins, W. A., & Steinberg, L. (2006). Adolescent development in interpersonal context. In W. Damon & R. M. Lerner (Series Eds.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Social, emotional, and personality development* (6<sup>th</sup> Ed., Vol. 3, pp. 1003–1067). Hoboken, NJ: Wiley.
- Cicchetti, D., & Toth, S. L. (1997). Transactional ecological systems in developmental psychopathology. In S. S. Luthar, J. Burack, D. Cicchetti & J. Weisz (Eds.), *Developmental psychopathology: Perspectives on adjustment, risk, and disorder* (pp. 317–349). New York: Cambridge University Press.
- Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the

- mind. *Development and Psychopathology*, 6, 533–549.
- Cohen, M. A., & Piquero, A. R. (2009). New evidence on the monetary value of saving a high risk youth. *Journal of Quantitative Criminology*, 25, 25–49.
- Coie, J. D., Miller-Johnson, S., & Bagwell, C. (2000). Prevention science. In A. J. Sameroff, M. Lewis, & S. M. Miller (Eds.), *Handbook of developmental psychopathology* (2<sup>nd</sup> Ed., pp. 93–112). New York: Kluwer Academic/Plenum Publishers.
- Collins, W. A., & Steinberg, L. (2006). Adolescent development in interpersonal context. In W. Damon & R. M. Lerner (Series Eds.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Social, emotional, and personality development* (6<sup>th</sup> Ed., Vol. 3, pp. 1003–1067). Hoboken, NJ: Wiley.
- Compas, B., & Reeslund, K. L. (2009). Processes of risk and resilience during adolescence. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology: Vol. 1. Individual bases of adolescent development* (3rd Edition, pp. 561–588). Hoboken, NJ: John Wiley & Sons Inc.
- Compton, K., Snyder, J., Shrepferman, L., Bank, L., & Shortt, J. W. (2003). The contribution of parents and siblings to antisocial and depressive behavior in adolescents: A double jeopardy coercion model. *Development and Psychopathology*, 15, 163–182.
- Connell, A. M., & Goodman, S. H. (2002). The association between psychopathology in fathers versus mothers and children's internalizing and externalizing behavior problems: A meta-analysis. *Psychological Bulletin*, 128, 746–773.
- Côté, S. M., Vaillancourt, T., Barker, E. D., Nagin, D., & Tremblay, R. E. (2007). The joint development of physical and indirect aggression: Predictors of continuity and change during childhood. *Development and Psychopathology*, 19, 37–55.
- Côté, S. M., Vaillancourt, T., Leblanc, J. C., Nagin, D. S., & Tremblay, R. E. (2006). The development of physical aggression from toddlerhood to pre-adolescence: A nation wide longitudinal study of Canadian children. *Journal of Abnormal Child Psychology*, 34, 71–85.
- Cowan, P. A., & Cowan, C. P. (2002). Interventions as tests of family systems theories: Marital and family relationships in children's development and psychopathology. *Development and Psychopathology*, 14, 731–759.
- Cui, M., Donnellan, M. B., & Conger, R. D. (2007). Reciprocal influences between parents' marital problems and adolescent internalizing and externalizing behavior. *Developmental Psychology*, 43, 1544–1552.
- Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *Journal of Child Psychology and Psychiatry*, 35, 73–112.
- Cummings, E. M., Keller, P. S., & Davies, P. T. (2005). Towards a family process model of maternal and paternal depression: Exploring multiple relations with child and family functioning. *Journal of Child Psychology and Psychiatry*, 46, 479–489.
- Cummings, E. M., Papp, L. M., & Kouros, C. D. (2009). Regulatory processes in children's coping with exposure to marital conflict. In S. L. Olson and A. J. Sameroff (Eds.), *Biopsychosocial regulatory processes in the development of childhood behavioral problems* (pp. 212–237). New York: Cambridge University Press.
- Davies, P. T., & Windle, M. (1997). Gender-specific pathways between maternal

- depressive symptoms, family discord, and adolescent adjustment. *Developmental Psychology*, 33, 657–668.
- Deater-Deckard, K., Dodge, K. A., Bates, J. E., & Pettit, G. S. (1998). Multiple risk factors in the development of externalizing behavior problems: Group and individual differences. *Development and Psychopathology*, 10, 469–493.
- Derogatis, L. R., & Fitzpatrick, M. (2004). *The use of psychological testing for treatment planning and outcomes assessment: Instruments for adults* (3<sup>rd</sup> ed., Vol. 3). Mahwah, NJ: Lawrence Erlbaum Associates, Inc., Publishers.
- Dishion, T. J., McCord, J., & Poulin, F. (1999). When interventions harm: Peer groups and problem behavior. *American Psychologist*, 54, 755–764.
- Dishion, T. J., Piehler, T. F., & Myers, M. W. (2008). Dynamics and ecology of adolescent peer influence. In M. J. Prinstein & K. A. Dodge (Eds.), *Understanding Peer Influence in Children and Adolescents* (72–93). New York: Guilford Press.
- Dishion, T. J., Shaw, D., Connell, A., Gardner, F., Weaver, C., & Wilson, M. (2008). The family check-up with high-risk indigent families: Preventing problem behavior by increasing parents' positive behavior support in early childhood. *Child Development*, 79, 1395–1414.
- Dodge, K. A. (1990). Developmental psychopathology in children of depressed mothers. *Developmental Psychology*, 26, 3–6.
- Dodge, K. A. (2008). Framing public policy and prevention of chronic violence in American youths. *American Psychologist*, 63, 573–590.
- Dodge, K. A., Dishion, T. J., & Lansford, J. E. (2006). Deviant peer influences in intervention and policy of youth. *Social Policy Report*, 20, 3–19.
- Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychopathology*, 39, 349–371.
- DuRant, R. H., Cadenhead, C., Pendergrast, R. A., Slavens, G., & Linder, C. W. (1994). Factors associated with the use of violence among urban Black adolescents. *American Journal of Public Health*, 84, 612–617.
- Eccles, J. S., Early, D., Fraser, K., Belansky, E., & McCarthy, K. (1997). The relation of connection, regulation, and support for autonomy to adolescents' functioning. *Journal of Adolescence Research*, 12, 263–286.
- Eisenberg, N., Zhou, Q., Spinrad, T. L., Valiente, C., Fabes, R. A., & Liew, J. (2005). Relations among positive parenting, children's effortful control, and externalizing problems: A three-wave longitudinal study. *Child Development*, 76, 1055–1071.
- Farrington, D. P. (2009). Conduct disorder, aggression, and delinquency. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology: Individual bases of adolescent development* (3rd Ed., pp. 683–722). Hoboken, NJ: John Wiley & Sons Inc.
- Feldman, R. (2009). The development of regulatory functions from birth to 5 years: Insights from premature infants. *Child Development*, 80, 544–561.
- Fergus, S., & Zimmerman, M.A. (2005). Adolescent resilience: A framework for understanding healthy development in the face of risk. *Annual Review of Public Health*, 26, 399–419.

- Finkelhor, D., Ormrod, R., Turner, H., & Hamby, S.L. (2005). The victimization of children and youth: A comprehensive, national survey. *Child Maltreatment, 10*, 5–25.
- Fowler, P. J., Tompsett, C. J., Braciszewski, J. M., Jacques-Tiura, A. J., & Baltes, B. B. (2009). Community violence: A meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. *Development and Psychopathology, 21*, 227–259.
- Gerard, J. M., & Buehler, C. (2004). Cumulative environmental risk and youth maladjustment: The role of youth attributes. *Child Development, 75*, 1832–1849.
- Gershoff, E. T. (2002). Corporal punishment by parents and associated child behaviors and experiences: A meta-analytic and theoretical review. *Psychological Bulletin, 128*, 539–579.
- Goldsmith, H. H. (1996). Studying temperament via construction of the toddler behavior assessment questionnaire. *Child Development, 67*, 218–235.
- Goodman, S. H. & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed parents: A developmental approach to the understanding of mechanisms. *Psychological Review, 106*, 458-490.
- Gorman-Smith, D., & Loeber, R. (2005). Are developmental pathways in disruptive behaviors the same for girls and boys? *Journal of Child and Family Studies, 14*, 15–27.
- Graber, J. A., & Sontag, L. M. (2009). Internalizing problems during adolescence. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology: Individual bases of adolescent development* (3<sup>rd</sup> Ed., Vol. 1, pp. 642–682). Hoboken, NJ: John Wiley & Sons Inc.
- Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin, 129*, 447–466.
- Gross, H. E., Shaw, D. S., Burwell, R. A., & Nagin, D. S. (2009). Transactional processes in child disruptive behavior and maternal depression: A longitudinal study from early childhood to adolescence. *Development and Psychopathology, 21*, 139–156.
- Guerra, N. G., Huesmann, L. R., & Spindler, A. J. (2003). Community violence exposure, social cognition, and aggression among urban elementary-school children. *Child Development, 74*, 1561–1576.
- Haley, D. W., & Stansbury, K. (2003). Infant stress and parent responsiveness: Regulation of physiology and behavior during still-face and reunion. *Child Development, 74*, 1534–1546.
- Hart, C. H., DeWolf, D. M., Wozniak, P., & Burts, D. C. (1992). Maternal and paternal disciplinary styles: Relations with preschoolers' playground behavioral orientations and peer status. *Child Development, 63*, 879–892.
- Hemmi, M. H., Wolke, D., & Schneider, S. (2011). Associations between problems with crying, sleeping and/or feeding in infancy and long-term behavioural outcomes in childhood: A meta-analysis. *Archive of Diseases in Childhood, 96*, 622–629. doi: 10.1136/adc.2010.191312
- Henggeler, S. W., & Schoenwald, S. K. (2011). Evidence-based interventions for juvenile offenders and juvenile justice policies that support them. *Social Policy Report, 25*,

1–28.

- Hinshaw, S. P. (2002). Process, mechanism, and explanation related to externalizing behavior in developmental psychopathology. *Journal of Abnormal Child Psychology*, *30*, 431–446.
- Hollingshead, A. B. (1979). *The Hollingshead four factor index of socioeconomic status*. Unpublished paper, Yale University, New Haven, CT.
- Huesmann, L. R., Eron, L. D., & Dubow, E. F. (2003). Childhood predictors of adult criminality: Are all risk factors reflected in childhood aggressiveness? *Criminal Behavior and Mental Health*, *12*, 185–208.
- Huesmann, L. R., & Taylor, L. D. (2006). The role of media violence in violent behavior. *Annual Review in Public Health*, *27*, 393–415.
- James, S. A. (1994). John Henryism and the health of African-Americans. *Culture, Medicine, and Psychiatry*, *18*, 163–182.
- Jenkins, J., Simpson, A., Dunn, J., Rasbash, J., & O'Connor, T. G. (2005). Mutual influence of marital conflict and children's behavior problems: Shared and nonshared family risks. *Child Development*, *76*, 24–39.
- Keating, D. P. (2004). Cognitive and brain development. In R. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology*. New York: Wiley & Sons.
- Keiley, M. K., Howe, T. R., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2001). The timing of child physical maltreatment: A cross-domain growth analysis of impact on adolescent externalizing and internalizing problems. *Development and Psychopathology*, *13*, 891–912.
- Kerr, D. C. R., Lunkenheimer, E. S., & Olson, S. L. (2007). Assessment of child problem behaviors by multiple informants: A longitudinal study from preschool to school entry. *Journal of Child Psychology and Psychiatry*, *48*, 967–975.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., et al. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replications (NCS-R). *Journal of American Medical Association*, *289*, 3095–3105.
- Kiernan, K. E., & Huerta, M. C. (2008). Economic deprivation, maternal depression, parenting and children's cognitive and emotional development in early childhood. *The British Journal of Sociology*, *59*, 783–806.
- Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2<sup>nd</sup> ed.). New York: Guilford Press.
- Kochanska, G., Murray, K., Jacques, T. Y., Koenig, A. L., & Vandegeest, K. A. (1996). Inhibitory control in young children and its role in emerging internalization. *Child Development*, *67*, 490–507.
- Kroneman, L., Loeber, R., & Hipwell, A. E. (2004). Is neighborhood context differently related to externalizing problems and delinquency for girls compared with boys? *Clinical Child and Family Psychology Review*, *7*, 109–122.
- Lansford, J. E., Chang, L., Dodge, K. A., Malone, P. S., Oburu, P., Palmérus, K. et al. (2005). Physical discipline and children's adjustment: Cultural normativeness as a moderator. *Child Development*, *76*, 1234–1246.
- Laursen, B., & Collins, W. A. (2009). Parent-child relationships during adolescence. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology*:



- Contextual influences on adolescent development* (3<sup>rd</sup> Ed., Vol. 2, pp. 3–42). New York: Wiley.
- Laursen, B., Coy, K. C., & Collins, W. A. (1998). Reconsidering changes in parent–child conflict across adolescence: A meta-analysis. *Child Development, 69*, 817–832.
- Lengua, L. J. (2002). The contribution of emotionality and self-regulation to the understanding of children’s response to multiple risk. *Child Development, 73*, 144–161.
- Lengua, L. J., Bush, N. R., Long, A. C., Kovacs, E. A., & Trancik, A. M. (2008). Effortful control as a moderator of the relation between contextual risk factors and growth in adjustment problems. *Development and Psychopathology, 20*, 509–528.
- Loeber, R., & Farrington, D. P. (2000). Young children who commit crime. Epidemiology, developmental origins, risk factors, early interventions, and policy implications. *Development and Psychopathology, 12*, 737–762.
- Lovejoy, M. C., Graczyk, P. A., O’Hare, E., & Neuman, G. (2000). Maternal depression and parenting: A meta-analytic review. *Clinical Psychology Review, 20*, 561–592.
- Luthar, S. S., & Brown, P. J. (2007). Maximizing resilience through diverse levels of inquiry: Prevailing paradigms, possibilities, and priorities for the future. *Development and Psychopathology, 19*, 931–955.
- Luthar, S.S., & Cicchetti, D. (2000). The construct of resilience: Implications for intervention and social policies. *Development and Psychopathology, 12*, 857–885.
- Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development, 71*, 543–562.
- Lynch, M., & Cicchetti, D. (2002). Links between community violence and the family system: Evidence from children’s feeling of relatedness and perceptions of parent behavior. *Family Processes, 41*, 519–532.
- Mackenzie, M. J., & McDonnough, S. C. (2009). Transactions between perception and reality: Maternal beliefs and infant regulatory behavior. In A. J. Sameroff (Ed.), *The transactional model of human development: How children and contexts shape each other* (pp. 35–54). Washington, DC: APA.
- Macmillan, R., & Hagan, J. (2004). Violence in the transition to adulthood: Adolescent victimization, education, and socioeconomic attainment in later life. *Journal of Research on Adolescence, 14*, 127–158.
- Masten, A. S. (2001). Ordinary magic: Resilience processes in development. *The American Psychologist, 56*, 227–238.
- Masten, A. S. (2007a). Competence, resilience, and development in adolescence: Clues for Prevention Science. In D. Romer & E.F. Walker (Eds.), *Adolescent psychopathology and the developing brain: Integrating brain and prevention science*. New York: Oxford.
- Masten, A. S. (2007b). Resilience in developing systems: Progress and promise as the fourth wave arises. *Development and Psychopathology, 19*, 921–930.
- Masten, A. S., Burt, K. B., Roisman, G. I., Obradović, J., Long, J. D., & Tellegen, A. (2004). Resources and resilience in the transition to adulthood: Continuity and change. *Development and Psychopathology, 16*, 1071–1094.
- Mathiesen, K. S., Tambs, K. (1999). The EAS Temperament Questionnaire—Factor structure, age trends, reliability, and stability in a Norwegian sample. *Journal of Child Psychology and Psychiatry, 40*, 431–439.

- Michigan Department of Community Health (2006). *1989-2004 Michigan resident death files*. Lansing, MI: Vital Records and Health Data Development Section, MDCH.
- Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology, 13*, 355–375.
- Moffitt, T. E., Caspi, A., Harrington, H., & Milne, B. J. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years. *Development and Psychopathology, 14*, 179-207.
- Moos, R. J., & Moos, B. S. (1994). *Family environment scale manual: Development, applications, research, 3<sup>rd</sup> Edition*. Palo Alto: Consulting Psychologists Press, Inc., 1994.
- Muthén, L. K., & Muthén, B. O. (2007). Mplus version 5.2 user's guide. Los Angeles, CA: Muthén & Muthén.
- Muthén, L. K., & Muthén, B. O. (2010). Mplus version 6 user's guide. Los Angeles, CA: Muthén & Muthén.
- Nicholson, J. S., Deboeck, P. R., Farris, J. R., Boker, S. M., & Borkowski, J. G. (2011). Maternal depressive symptomatology and child behavior: Transactional relationship with continuous bidirectional coupling. *Developmental Psychology, 47*, 1312–1323.
- Nisbett, R. E. (1993). Violence and the U.S. regional culture. *American Psychologist, 48*, 441–449.
- Nolen-Hoeksema, S. (2001). Gender differences in Depression. *Current Directions in Psychological Science, 10*, 173–176.
- O'Hara, M. W., & Swain, A. M. (1996). Rates and risk of post-partum depression: A meta-analysis. *International Review of Psychiatry, 8*, 37–54.
- Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., Poulton, R., Sears, M. R., Thomson, W. M., & Caspi, A. (2008). Female and male antisocial trajectories: From childhood origins to adult outcomes. *Development and Psychopathology, 20*, 673–716.
- Olson, S. L., Ceballo, R., & Park, C. (2002). Early problem behavior among children from low-income mother-headed families: A multiple risk perspective. *Journal of Clinical Child and Adolescent Psychology, 31*, 419–430.
- Olson, S. L., & Lunkenheimer, E. S. (2009). Expanding concepts of self-regulation to social relationships: Transactional processes in the development of early behavioral adjustment. In A. J. Sameroff (Ed.), *The transactional model of human development: How children and contexts shape each other* (pp. 55–76). Washington, DC: APA.
- Olson, S. L., Sameroff, A. J., Kerr, D. C. R., Lopez, N. L., & Wellman, H. M. (2005). Developmental foundations of externalizing problems in young children: The role of effortful control. *Development and Psychopathology, 17*, 25–45.
- Olson, S. L., Sameroff, A. J., Kerr, D. C. R., & Lunkenheimer, E. S. (2009). Self-regulatory processes in the development of early disruptive behavior: The preschool to school transition. In S.L. Olson & A.J. Sameroff (Eds.). *Biopsychosocial Regulatory processes in the development of childhood behavior problems*. NY: Cambridge University Press.

- Olsson, C. A., Bond, L., Burns, J. M., Vella-Brodrick, D. A., & Sawyer, S. M. (2003). Adolescent resilience: A concept analysis. *Journal of Adolescence*, *26*, 1–11.
- Ozer, E. J. (2005). The impact of violence on urban adolescents: Longitudinal effects of perceived school connection and family support. *Journal of Adolescent Research*, *20*, 167–192.
- Paschall, M. J., Ennett, S. T., & Flewelling, R. L. (1996). Relationships among family characteristics and violent behavior by Black and White male adolescents. *Journal of Youth and Adolescence*, *25*, 177–197.
- Paschall, M. J., & Hubbard, M. L. (1998). Effects of neighborhood and family stressors on African American male adolescents' self-worth and propensity for violent behavior. *Journal of Consulting and Clinical Psychology*, *66*, 825–831.
- Patterson, G. R., DeBaryshe, B. D., & Ramsey, R. (1989). A developmental perspective to antisocial behavior. *American Psychologist*, *44*, 329–335.
- Pettit, G. S., Bates, J. E., & Dodge, K. A. (1997). Supportive parenting, ecological context, and children's adjustment: A seven-year longitudinal study. *Child Development*, *68*, 908–923.
- Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, *12*, 427–441.
- Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*, 385–401.
- Raposa, E. B., Hammen, C. L., & Brennan, P. A. (2011). Effects of child psychopathology on maternal depression: The mediating role of child-related acute and chronic stressors. *Journal of Abnormal Psychology*, *39*, 1177–1186.
- Repetto, P. B., Caldwell, C. H., & Zimmerman, M. A. (2004a). Trajectories of depressive symptoms among high-risk African American adolescents. *Journal of Adolescent Health*, *35*, 468–477.
- Repetto, P. B., Zimmerman, M. A., & Caldwell, C. H., (2004b). A longitudinal study of the relationship between depressive symptoms and alcohol use in a sample of inner-city Black youth. *Journal of Studies on Alcohol*, *65*, 169–178.
- Repetto, P. B., Zimmerman, M. A., & Caldwell, C. H. (2008). A longitudinal study of depressive symptoms and marijuana use in a sample of inner-city African Americans. *Journal of Research on Adolescence*, *18*, 421–447.
- Rosenblum, K. L., McDonough, S., Muzik, M., Miller, A., & Sameroff, A. (2002). Maternal representations of the infant: Associations with infant response to the still face. *Child Development*, *73*, 999–1015.
- Rothbart, M. K., & Bates, J. E. (1998). Temperament. In W. Damon (Series Ed.) and N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, personality development* (pp. 105–176). New York: Wiley.
- Rothbart, M. K., & Bates, J. E. (2006). Temperament. In W. Damon & R. Lerner (Series Eds.), & N. Eisenberg (Vol. Ed.), *Handbook of child psychology, Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). New York: Wiley.
- Rothbaum, F., & Weisz, J. R. (1994). Parental caregiving and child externalizing behavior in nonclinical samples: A meta-analysis. *Psychological Bulletin*, *116*, 55–74.
- Rubin, K. H., Bukowski, W., & Parker, J. D. (2006). Peer interactions, relationships and groups. In W. Damon, R. Lerner (Series Ed.) & N. Eisenberg (Volume Ed.)

- Handbook of Child Psychology: Vol. 3: Social, Emotional, and Personality Development 6<sup>th</sup> Ed.* (pp. 571–645). NY: Wiley
- Rudolph, K. D. et al., (2001). Negotiating the transition to middle school: The role of self-regulatory processes. *Child Development, 72*, 929–946.
- Rueda, M. R., Rothbart, M. K., McCandliss, B. D., Saccomanno, L., & Posner, M. I. (2003). Training, maturation, and genetic influences on the development of executive attention. *Proceedings of the National Academy of Sciences, 102*, 14931–14936.
- St. James-Roberts, I., & Halil, T. (1991). Infant crying patterns in the first year: Normal community and clinical findings. *The Journal of Child Psychology and Psychiatry, 32*, 951–968.
- Sameroff, A. J. (2000). Developmental systems and psychopathology. *Development and Psychopathology, 12*, 297–312.
- Sameroff, A. J. (2009a). The transactional model. In A. J. Sameroff (Ed.), *The transactional model of human development: How children and contexts shape each other* (pp. 3–21). Washington, DC: APA.
- Sameroff, A. J. (2009b). Designs for transactional research. In A. J. Sameroff (Ed.), *The transactional model of human development: How children and contexts shape each other* (pp. 23–32). Washington, DC: APA.
- Sameroff, A. J., & Mackenzie, M. J. (2003). Research strategies for capturing transactional models of development: The limits of the possible. *Development and Psychopathology, 15*, 613–640.
- Seifer, R., Sameroff, A. J., Barrett, L. C., & Krafchuk, E. (1994). Infant temperament measured by multiple observations and mother report. *Child Development, 65*, 1478–1490.
- Sektnan, M., McClelland, M. M., Acock, A., & Morrison, F. J. (2010). Relations between early family risk, children's behavioral regulation, and academic achievement. *Early Childhood Research Quarterly, 25*, 464–479.
- Shaw, D., Connell, A., Dishion, T. J., Wilson, M., & Gardner, F. (2009). Improvements in maternal depression as a mediator of intervention effects on early childhood problem behavior. *Development and Psychopathology, 21*, 417–439.
- Shaw, D. S., Gilliom, M., Ingoldsby, E. M., & Nagin, D. S. (2003). Trajectories leading to school-age conduct problems. *Developmental Psychology, 39*, 189–200.
- Shaw, D. S., Gross, H. E., & Moilanen, K. L. (2009). Developmental transactions between boys' conduct problems and mothers' depressive symptoms. In A. J. Sameroff (Ed.), *The transactional model of human development: How children and contexts shape each other* (pp. 77–96). Washington, DC: APA.
- Sheehan, M. J., & Watson, M. W. (2008). Reciprocal influences between maternal discipline techniques and aggression in children adolescent. *Aggressive Behavior, 34*, 245–255.
- Slovak, K., Carlson, K., & Helm, L. (2007). The influence of family violence on youth attitudes. *Child & Adolescent Social Work Journal, 24*, 77–99.
- Snyder, J., Cramer, A., Afrank, J., & Patterson, G. R. (2005). The contributions of ineffective discipline and parental hostile attributions of child misbehavior to the development of conduct problems at home and school. *Developmental Psychology, 41*, 30–41.

- Solomon, B. S., Bradshaw, C. P., Wright, J., & Cheng, T. L. (2008). Youth and parental attitudes toward fighting. *Journal of Interpersonal Violence, 23*, 544–560.
- Spano, R., Vazsonyi, A. T., & Bolland, J. (2009). Does parenting mediate the effects of exposure to violence on violent behavior? An ecological–transactional model of community violence. *Journal of Adolescence, 32*, 1321–1341.
- Sroufe, A. L., Duggal, S., Weinfield, N., & Carlson, E. (2000). Relationships, development, and psychopathology. In A. J. Sameroff, M. Lewis, & S. M. Miller (Eds.), *Handbook of developmental psychopathology* (2<sup>nd</sup> Ed., pp. 75–91). New York: Plenum Publishers.
- Steinberg, L. (2005). Cognitive and affective development in adolescence. *TRENDS in Cognitive Sciences, 9*, 69–74.
- Steinberg, L. (2008). Juvenile justice: A future of children event. The Brookings Institution.
- Steinberg, L., Dahl, R., Keating, D., Kupfer, D.J., Masten, A., & Pine, D.S. (2006). The study of developmental psychopathology in adolescence: Integrating affective neuroscience with the study of context. D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Developmental neuroscience* (2<sup>nd</sup> Ed., Vol. 2, pp. 710–741). Hoboken, NJ: John Wiley & Sons Inc.
- Thomas, D. E., Bierman, K. L. & the Conduct Problems Prevention Research Group (2006). The impact of classroom aggression on the development of aggressive behavior problems in children. *Development and Psychopathology, 18*, 471–487.
- Tremblay, R.E. (2000). The development of aggressive behaviour during childhood: What have we learned in the past century? *International Journal of Behavioral Development, 24*, 129–141.
- van Lier, P. A. C., Wanner, B., & Vitaro, F. (2007). Onset of antisocial behavior, affiliation with deviant friends, and childhood maladjustment: A test of the childhood- and adolescent-onset models. *Development and Psychopathology, 19*, 167–185.
- Voisin, D. R. (2007). The effects of family and community violence exposure among youth: Recommendations for practice and policy. *Journal of Social Work Education, 43*, 51–66.
- Weinfield, N. S., Ingerski, L., & Moreau, S. C. (2009). Maternal and paternal depressive symptoms as predictors of toddler adjustment. *Journal of Family Studies, 18*, 39–47.
- Xue, Zimmerman, M. A., & Barnett, (In press). Parental support and the longitudinal relationships between exposure to community violence and violent behavior among African American adolescents. *Journal of Research on Adolescence*.
- Xue, Zimmerman, M. A., & Cunningham, (2009). Longitudinal study of the relationship between alcohol use and violent behavior among urban African-American youths from adolescence to emerging adulthood. *American Journal of Public Health, 99*, 2041–2048.
- Yates, T. M., Obradović, J., & Egeland, B. (2010). Transactional relations across contextual strain, parenting quality, and early childhood regulation and adaptation in a high-risk sample. *Development and Psychopathology, 22*, 539–555.
- Yoshikawa, H. (1994). Prevention as cumulative protection: Effects of early family support and education on chronic delinquency and its risks. *Psychological*

- Bulletin*, 115, 28–54.
- Yuan, K. H. & Bentler, P. M. (2000). Three likelihood-based methods for mean and covariance structure analysis with nonnormal missing data. *Sociological Methodology*, 30, 165–200.
- Zelazo, P. D., Craik, F. I. M., & Booth, L. (2004). Executive function across the life span. *Acta Psychologica*, 115, 167–183.
- Zimmerman, M. A., & Schmeelk-Cone, K. H. (2003). A longitudinal analysis of adolescent substance use and school motivation in African American youth. *Journal of Research on Adolescence*, 13, 185-210.
- Zimmerman, M. A., Steinman, K. J., & Rowe, K. (1998). Violence among urban African-American adolescents: The protective effects of parental support. In S. Oskamp, & X. B. Arriaga (Eds.), *Addressing community problems: Research and intervention* (pp. 78–103). Newbury Park, CA: Sage.