Examining the Reciprocal Relationship between Parental Negativity and Negative Emotionality during Adolescence: A Biometric Cross-Lagged Model

by Tova A. Jacobs

B.A. in Psychology, May 2005, The George Washington University
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Dissertation directed by

Jody M. Ganiban
Professor of Psychology

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 - 1346

The Columbian College of Arts and Sciences of The George Washington University certifies that Tova Anne Jacobs has passed the Final Examination for the degree of Doctor of Philosophy as of March 7, 2014. This is the final and approved form of the dissertation.

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Tova Anne Jacobs

Dissertation Research Committee:

Jody M. Ganiban, Professor of Psychology, Dissertation Director

Rolf A. Peterson, Professor Emeritus of Psychology and of Psychiatry and Behavioral Sciences, Committee Member

Sharon F. Lambert, Associate Professor of Psychology, Committee Member

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Abstract of Dissertation

Examining the Reciprocal Relationship between Parental Negativity and Negative Emotionality during Adolescence: A Biometric Cross-Lagged Model

Adolescence represents a key developmental period when the interplay between temperament and parenting may be most apparent and has implications for adolescents' adjustment. The current study utilized a cross-lagged, biometric model to explore the reciprocal relationship between Adolescent Negative Emotionality and Parental Negativity across adolescence. Analyses examined stability and change in Adolescents' Negative Emotionality and Parental Negativity over a three year period, as well as the impact of each construct on each other over time. To gain further understanding of the mechanisms that underlie links between temperament and parenting, genetic and environmental contributions to stability and change in each construct were also examined.

This study focused on a subset of the families within the Nonshared Environment in Adolescent Development (NEAD) project (N=395) who were assessed twice, 3 years apart. This sample included 5 sibling pair types that resided in nondivorced or stepfamilies: Monozygotic (N=63) and Dizygotic (N=75) twin pairs, and Full Sibling (N=153), Half Sibling (N=60 pairs), and Unrelated Sibling (N=44) pairs.

Overall findings for mothers and fathers indicated that: (1) there is moderate stability in Negative Emotionality and Parental Negativity over time, (2) Negative Emotionality and Parental Negativity influence each other over time; and (3) genetic and environmental factors account for variance in Negative Emotionality and Parental Negativity within each age examined, and contribute to stability and change. These

findings support the presence of bidirectional effects between Parental Negativity and Adolescent Negative Emotionality, and underscore the need for intervention programs targeting the parent-child relationship during adolescence.

Table of Contents

List of Figures

List of Tables

Chapter 1: Introduction

When considering the developmental outcomes of children, both parenting (e.g., Maccoby & Martin, 1983; Mcleod, Weisz, & Wood, 2007; Loeber and Stouthamer-Loeber, 1986) and temperament (e.g., Rothbart & Bates, 1998; Sanson, Hemphill, & Smart, 2004) have been independently established as important predictors of children's later adjustment. However, less is known about how these factors influence each other. Further insight into this issue would enhance understanding of parents' and children's roles in child development. When examining the relationship *between* parenting and temperament, researchers have generally proposed two models: (1), parenting influences the development of children's temperament (parent effects models) and (2), children's temperament influences parenting (child effects models). More recently, researchers have put forth models that emphasize the mutual influence of temperament and parenting on each other—or in transaction with one another—in predicting children's developmental trajectories (e.g., Lengua, 2006; Patterson, 1982; Putnam, Sanson, & Rothbart, 2002; Scarr & McCartney, 1983; Van der Bruggen, Stams, Bogels, & Paulussen-Hoogeboom, 2010).

While previous researchers have examined the interplay between temperament and parenting during infancy and early childhood, few studies have focused on these factors during adolescence. Adolescence is a period of developmental transformation in cognition, emotional functioning, and family relationships. Moreover, it represents a period in which temperament may be expressed more strongly (Collins & Steinberg, 2006; Larson & Lampman-Petraitis, 1989) and the emotional overture of the parent-child relationship is in transition (Feinberg, Howe, Reiss, & Hetherington, 2000). Indeed,

adolescence is a period in which conflict in the parent-child relationship increases and closeness decreases (Eisenberg, et al., 2008; Laursen, Coy, & Collins, 1998; McGue, Elkins, Walden, & Iacono, 2005; Steinberg & Silk, 2002) as the adolescent struggles with identity formation, hormonal changes and a heightened desire for autonomy (Erikson, 1968; Schwartz, Cote, & Arnett, 2005). As such, this developmental period represents a time when the interplay between temperament and parenting may be most apparent.

Despite the developmental importance of adolescence, research on the relationship between parenting and temperament has traditionally focused on documenting these effects in infancy and early toddlerhood (e.g., Maccoby & Martin, 1983; Paulussen-Hoogeboom, Stams, Hermanns, Peetsma, & van den Wittenboer, 2008). Moreover, most research focuses either on the effects of parenting on temperament *or* the effects of temperament on parenting (e.g., Braungart-Rieker, Hill-Soderlund, & Karrass, 2010; Crockenberg & Leerkes, 2006; Fish, Stifter, & Belsky, 1991; Ganiban et al., 2011; O'Connor, Hetherington, Reiss, & Plomin, 1995; Paulussen-Hoogeboom et al., 2008); transactional models have been tested infrequently (e.g., Eisenberg et al., 1999; Eisenberg et al., 2008; Lengua, 2006; Lengua & Kovacs, 2005).

The proposed study examines the interplay of temperament and parenting during adolescence. Specifically, the proposed study uses a longitudinal, cross-lagged model to examine the influences of parental negativity on negative emotionality, as well as the influences of negative emotionality on parental negativity, across adolescence. Furthermore, a behavioral genetic research design is employed to enhance understanding of the mechanisms through which temperament and parenting affect each other during this period. This approach distinguishes child-based genetic influences from

environmentally-based influences on parenting and temperament. Using behavioral genetic methods, the current study can foster a better understanding of the relationship between temperament and parenting across adolescence.

The next section provides an overview of the constructs of temperament and parenting. This section will be followed by an in-depth discussion of the potential mechanisms that underlie the effects of each upon the other from the perspective of parent effects, child effects, and bidirectional effects models.

Temperament

Temperament is understood to be an important predictor of children's developmental outcomes (e.g., Rothbart & Bates, 1998; Rothbart & Bates, 2006; Sanson, Hemphill & Smart, 2004; Sanson et al., 2009; Saudino, 2005) and can be broadly defined as constitutionally-based individual differences in emotional, motor and attentional reactivity and self-regulation (Rothbart & Bates, 1998). One temperament trait, negative emotionality, has been consistently linked with psychological outcomes, including the development of internalizing disorders (e.g., Klump, McGue, & Iacono, 2002; Kagan & Snidman, 1999; Lonigan, Phillips, & Hooe, 2003; McClowry, 1994; Rende, 1993) and externalizing disorders (e.g., Hagekull, 1994; Singh & Waldman, 2010). Negative emotionality relates to one's tendency to react to stressors with high degrees of negative affect, including anger, irritability, fear, or sadness (Rothbart, Ahadi, & Hershey, 1994), and is synonymous with negative reactivity (Sanson et al., 2009) and negative affectivity (Stice & Gonzales, 1998).

Researchers specifically note that adolescence is a time of heightened stress (Larson & Lampman-Petraitis, 1989; Collins & Steinberg, 2006), when negative

emotionality can be exacerbated or reinforced (Ge & Conger, 1999). With stability estimates ranging from .31 to .75 from middle childhood to later adulthood, negative emotionality (or neuroticism in adult personality research) becomes increasingly more stable throughout the lifespan (Caspi, Roberts, & Shiner, 2005; Roberts & DelVecchio, 2000) particularly during late adolescence to early adulthood (Shiner, 2005). It is for these reasons that this study focuses specifically on the development of negative emotionality from early to later adolescence.

Maternal Negativity

In regard to parenting, there is general consensus that parental negativity influences child outcomes (e.g., Maccoby, 1992; Putnam, Sanson, & Rothbart, 2002). Parental negativity is defined as the frequency and intensity of disputes in a relationship as well as feelings of anger. *Maternal* negativity has consistently been found to be predictive of negative developmental outcomes (e.g., Rothbaum & Weisz, 1994; Rubin, Burgess, Dwyer, & Hastings, 2003), and inversely associated with positive developmental outcomes (Pettit, Bates, & Dodge, 1997) in childhood as well as in adolescence (e.g., Collins & Steinberg, 2006; Steinberg, & Silk, 2002).

Of note, the majority of studies that investigate parental negativity only include the construct of *maternal* negativity (e.g., Bates, Pettit, Dodge, & Ridge, 1998; Combs-Ronto, Olson, Lunkenheimer, & Sameroff, 2009; Morris, Silk, Steinberg, Sessa, Avenevoli, & Essex, 2004; Rubin, Burgess, Dwyer & Hastings, 2003). Historically, child development research has focused on mothering, as fathers were thought to play less of an active role in child-rearing (Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000). Most research conducted with fathers has concentrated on the positive benefits of

paternal involvement, particularly when the relationship is supportive (Amato & Rezac, 1994).

Paternal Negativity

Cabrera and colleagues (2000), however, note the growing importance of investigating the effects of fathering – both positive and negative aspects – on children's developmental trajectories as mothers in the workforce are sharing more parenting responsibilities. Researchers have begun to document that fathering shapes child development and emotional regulation, but acknowledge that the relationship may differ from that with mothering (e.g., Collins & Russell, 1991; Ge, Conger, Lorenz, Shanahan, & Elder, 1995; Gottman, Katz, & Hooven, 1997). However, limited research has investigated the association between paternal negativity and negative emotionality.

Research on the relationship between *maternal* negativity and temperament – in particular, the trait of negative emotionality – suggests that parenting and child temperament influence each other (Rothbart & Bates, 1998; Sanson, Hemphill, & Smart, 2004). However, some researchers suggest that a child's temperament may have less of an impact on fathering than mothering (Belsky, Jaffee, Sligo, Woodward, & Silva, 2005), as fathers tend to be less reactive to their child's negative behaviors than mothers (Woodworth, Belsky, & Crnic, 1996). Indeed, fathers have been found to disengage from adolescents who are more difficult, ultimately monitoring them less (Reiss, Neiderhiser, Hetherington, & Plomin, 2000). Other researchers posit that fathers engage in more unsupportive responses when they have limited parental resources and children with high negative emotionality (Wong, McElwain, & Halberstadt, 2009). Yet, during adolescence, fathers may also have decreased sensitivity than in earlier years (Reiss et al., 2000).

The relationship between paternal negativity and negative emotionality throughout childhood and adolescence is poorly understood. Therefore, this study examines both maternal and paternal negativity, which is defined as the frequency and intensity of disputes in a relationship as well as feelings of anger. In subsequent sections of this review, unidirectional models of parents' influences on child temperament and child's influences on parenting are presented. Additionally, a transactional model that focuses on bidirectional influences of parenting and temperament is presented.

Parent-Effects Model: Parents' Influence on Child Temperament

Parenting in early childhood. Researchers have consistently documented the role of mother's parenting on the development of negative emotionality throughout the lifespan. When focusing on infancy and early childhood, parental sensitivity has been found to predict changes in negative reactivity over time. When evaluating factors that contributed to the continuity of negative emotionality, Fish, Stifter, and Belsky (1991) found that infants with more sensitive mothers remained lower in negative emotionality from birth to 5 months than did infants with less sensitive mothers. Crockenberg (1987) showed that irritable infants, when coupled with punitive mothers, were angrier and more disagreeable than irritable infants without punitive mothers. Similarly, another study found that infants whose mothers were more sensitive showed slower increases in fear reactivity over the period from 4 to 16 months (Braungart-Rieker, Hill-Soderlund, & Karrass, 2010). Crockenberg and Leerkes (2006) found similar results when evaluating change in negative emotionality from 6 months to 2.5 years old; children were more likely to show a stable association in their levels of negative emotionality if their mothers were rated as less sensitive. Conversely, mothers who show sensitivity towards their

infants' distress, aid their infants to maintain optimal levels of emotional arousal, and help to induce positive emotions in times of stress, have infants who show higher levels of positive emotionality and may learn appropriate emotion regulation strategies (Eisenberg, Cumberland, & Spinrad, 1998).

Parenting in adolescence. The majority of studies examining the influence of parental negativity on temperament focus mainly on infancy and early childhood; therefore, it is unclear whether parental negativity continues to directly affect or exacerbate negative emotionality during *adolescence*. One study found that parental punitive or distress reactions at ages 8-10 predicted children's negative emotionality at ages 10-12 (Eisenberg et al., 1999). Lengua (2006) also found that parental rejection from ages 8-12 increased children's fear and irritability one year later. Nonetheless, both researchers note the dearth of studies examining the influence of parental negativity on temperament during adolescence.

While numerous studies document the effects of parenting on behavioral outcomes in adolescence (Putnam, Sanson & Rothbart, 2002; Rothbart & Bates, 1998), fewer investigate the influence of parenting on *temperamental* changes. This may be in part due to the common conception of temperament as constitutionally based and stable, i.e., influenced by genes, and less by the environment. Notably, several studies show that negative parenting leads to greater occurrences of internalizing problems such as depression and anxiety — outcomes traditionally associated with negative emotionality (Klump, McGue, & Iacono, 2002; Kagan & Snidman, 1999; Lonigan, Phillips, & Hooe, 2003; McClowry, 1994; Rende, 1993; Rothbart & Bates, 1998); however, we are unable to make conclusions about whether parenting shapes *temperament* in these studies, or

whether parenting is mitigating or exacerbating the associations between negative emotionality and negative psychological outcomes.

The parent-child relationship continues to be important through adolescence; concurrently, temperament is still developing. Adolescence is an important time to examine temperament development as researchers have suggested that the stability of temperament from infancy through adolescence has been correlated to be as low as 0.2 (Putnam, Sanson, & Rothbart, 2002). Further, adolescence may be a transitional time when negative emotionality may become more salient (Eisenberg et al., 2008). Parenting may be one of the key factors that contributes to changes in negative emotionality during this time.

Attachment theory. Attachment security is one mechanism through which negative parenting, including harshness, hostility, and insensitivity, is thought to influence the development of children's negative emotionality (Shaver & Mikulincer, 2007). According to attachment theory, children have an innate attachment behavioral system that is activated during times of threat or distress and which motivates them to seek protection and closeness from an attachment figure ("AF") as a way to restore their internal security (Bowlby, 1969; 1973). When the attachment figure responds appropriately and consistently to the child's needs, internal security is restored and the child feels comfortable and safe. However, anxiety may be evoked if the parent is unresponsive to the child's needs. Children may also feel and use anger to alert attachment figures that their needs have not been met or have been rejected. Therefore, according to attachment theory, children experience and learn to regulate the emotions of love, anxiety, and anger within the context of their first attachment relationship (Bowlby,

1969). The degree to which children experience, express, and regulate negative emotions could reflect the extent to which the primary AF has responded consistently and sensitively to their needs.

Attachment styles have commonly been characterized as secure or insecure, each of which affects the development of effective or ineffective emotion regulation. Early emotional self-regulation is preceded by emotional co-regulation with their parent (Shaver & Mikulincer, 2007). Parental warmth and responsiveness promotes the development of a secure attachment, as the parent acts as a secure base and provides the child with a safe haven in times of distress. Subsequently, the securely attached child develops a 'felt security' because his or her needs are being met. For example, infants of mothers who are more sensitive may feel more protected because they have a secure base with which to develop more confidence and the ability to self-soothe (Ainsworth et al., 1978; Posada, Carbonell, Alzate, & Plata, 2004). Thus, children with a history of secure attachment tend to be better equipped to regulate their emotions because they have more assurance that their caregivers will be available to help, and because they may have more confidence in their own ability to regulate emotions (Mikulincer & Shaver, 2012).

Children with insecure attachments, on the other hand, have two predominant responses for emotion regulation when their needs are not being met by their caregiver: avoidance or anxiety (Mikulincer & Shaver, 2005). An avoidant strategy is characterized by deactivating strategies, or a desire for emotional distance and independence due to perceptions of the attachment figure as rejecting and unresponsive (Bowlby, 1969). According to attachment theory, when parents ignore or reject the child's anger, the world is perceived as a hostile place, in which needs are not met, but frustrated. As a

result, the child may learn to suppress anger or develop hostile attributional biases, the tendency to interpret others' behavior as hostile when it is not (Steinberg & Dodge, 1983) which can perpetuate their anger or aggression (Crick & Dodge, 1996). Even if a child's anger is not overtly expressed to AFs, avoidant children nevertheless exude hostility. Thus, misperceived hostility *or* suppressing anger can both contribute to heightened negative emotionality, and contribute to temper tantrums and outbursts (Kobak, Cole, Ferenz-Guillis, & Fleming, 1993).

Children who perceive their AFs as inconsistently responsive or withholding of love and affection may consequently become preoccupied with the potential loss or unavailability of their attachment figures, leading to higher baseline levels of anxiety and anger. These children tend to display an anxious strategy characterized by hyper-activating strategies, e.g., becoming hyperaware of perceived threat, leading to increased negative emotionality, particularly when separated from their caregiver (Cassidy & Kobak, 1988; Mikulincer & Shaver, 2005). To gain the attention of the AFs, these children may amplify their bids for attention through clinginess and expressing their anxiety to their AFs more intensely than others (Mikulincer, 1998; Mikulincer, Shaver & Pereg, 2003). Furthermore, individuals with hyper-activating strategies are also expected to experience higher levels of anger if they perceive their AFs as unresponsive or withholding of affection or support.

Attachment in adolescence. Several studies provide empirical support for the impact of the parent-child relationship on the development of emotional reactivity through the mechanism of attachment. Mikulincer (1998) investigated the relationship between attachment style, physiological signs of anger, and attribution of hostile intent in

adolescents and adults. Participants, categorized by secure and insecure attachment, were asked to rate their anger in response to emotionally evocative hypothetical scenarios, while their physiological response (heart rate) was monitored. As expected, securely attached individuals' anger played a functional role (e.g., repair a relationship, problem solve) and was expressed in situations that warranted such a response. In contrast, anxiously attached individuals expressed anger in unwarranted situations, and felt a lack of control over their anger expressions (e.g., excessive rumination, flooded by their angry thoughts). Avoidant individuals were found to show physiological signs of emotional arousal, yet their anger was not overtly reported, suggesting a tendency to experience hostility without it being consciously expressed or acknowledged. Notably, the emotional expression of anger varied based on the attachment status of the participant. Similar findings have been found with the expression of fear, anger, and sadness—the components of negative emotionality—at a young age. Children as young as 14 months of age showed varying emotional expression trajectories as a function of their attachment histories (Kochanska, 2001). Taken together, this body of research suggests that parenting contributes to how one expresses emotions such as anger, anxiety, and fear in adolescence and adulthood via the mechanism of attachment styles.

In summary, parents influence children's expression of negative emotions during childhood and shape propensities to experience and regulate anxiety and anger that persist throughout the lifespan. Hazan and Shaver (2003) provide empirical support for this notion by demonstrating the parallels between attachment behaviors during infancy and attachment behaviors during adolescence and adulthood. As in early childhood, adolescents display similar emotional responses during times of perceived threat or stress.

Moreover, Kobak, Cole Ferenz-Gillies, and Fleming (1993) discovered that adolescents who viewed their mothers as currently being attuned and supportive (characteristics of a secure attachment) experienced less angry outbursts toward their parent and vice versa. However, it remains unclear whether exposure to high levels of parental negativity during adolescence exacerbates traits of negative emotionality because adolescents continue to feel rejected, scared, or even angry with their parents. Attachment theory proposes that children with insecure attachments tend to use avoidant (deactivating) or anxious (hyper-activating) strategies and are more likely to experience anxiety, distress, and anger, than those with a secure attachment (Mikulincer, 1998). Thus, attachment theory describes the mechanism through which parenting influences the development of negative emotionality.

Child-Effects Model: Childs' Influence on Parenting

There has been an increasing awareness of the active role a child plays in shaping his or her development (Rothbart & Bates, 1998; Thomas, Chess, Birch, Hertzig, & Korn, 1963). Children's traits and behaviors are thought to influence and shape their own social and emotional development (Bell, 1968); indeed, individual differences in characteristics can elicit or evoke varied responses from others, thereby adjusting one's experience. Some of these characteristics (e.g., temperamental traits) may be genetically-influenced (Plomin, Pederson, McClearn, Nesselroade & Bergeman, 1988). Children's genetically-influenced characteristics are thought to influence their behavior (Rothbart & Bates, 1998; Sanson & Prior, 1999); their perception of their environment (Rothbart, Ahadi & Hershey, 1994); and the reactions of others around them (Paulesson-Hoogeboom, Stams, Hermanns, & Peetsma, 2008; Akker, Dekovic, Prinzie & Asscher,

2010). Previous research has found that certain temperamental traits can have a significant influence on the parent-child dynamic (Bell, 1968; Bates, Pettit, & Dodge, 1995; Braungart-Rieker, Garwood, & Stifter, 1997; Campbell, 1979) and can elicit or evoke certain parenting behaviors (Damon & Eisenberg, 1998; Lengua, 2006; Lengua & Kovacs, 2005; Plomin, Defries, & Loehlin, 1977; Scarr & McCartney, 1983). The process by which a child's genetically influenced characteristics evoke distinctive responses from their caregiver is referred to as an evocative gene-environment correlation (Scarr & McCartney, 1983).

Several studies have found temperamental characteristics to evoke or elicit negative parenting behaviors (O'Connor, Hetherington, Reiss, & Plomin, 1995; Sanson & Rothbart, 1995). One temperamental characteristic that has been especially associated with evocative child-based effects is negative emotionality. Negative emotionality has been associated with less supportive, harsher and more restrictive parenting (Paulesson-Hoogeboom, Stams, Hermanns, & Peetsma, 2007; Sanson, Hemphill & Smart, 2004; Rothbart & Bates, 1998). Using a sample of 3 year-old children, Paulesson-Hoogeboom and colleagues (2008) found that authoritative parenting mediated the relationship between children's negative emotionality and externalizing and internalizing problems. Although this study could not interpret the causality or directionality of the findings, this study nonetheless posits that a child's genetically-influenced characteristics evoke certain parenting behaviors which in turn influence behavior problems in children. For example, a child who is more affable may elicit more warmth and responsiveness from a parent, whereas a child with high reactivity and poor self-regulation may elicit more hostile or punitive parenting. Ganiban et al. (2011) used biometric analyses to assess the degree to

which child-based genetic effects account for genetic and environmental contributions to parents' negativity and warmth. Their findings supported an evocative gene-environment model: child-based genetic factors explained significant variance in negative parenting, and these effects were stronger when children demonstrated higher levels of negative emotionality. Temperament characteristics that were not as provocative (e.g., shyness) did not show this pattern. However, this study was unable to verify directionality or causality of the variables because it was cross-sectional in design.

Adoption studies have also provided evidence of evocative child effects on parenting. Within the adoption design, one can examine the extent to which children's genetically influenced characteristics predict parenting. This design eliminates the possibility that genetic factors shared by parents and children account for associations between child characteristics and parenting (i.e., passive gene-environment correlation). In a seminal study, Ge et al., (1996) demonstrated compelling evocative effects utilizing a longitudinal adoptive design in order to dispel a passive gene-environment correlation. Adopted children exhibiting hostile traits were found to receive harsher parenting over time than those children without the same traits. These results suggest that genetically-influenced characteristics (hostile behaviors) accounted for the variance in the parenting received. In an attempt to replicate these findings, O'Connor, Deater-Deckard, Fulker, Rutter, and Plomin (1998) found similar patterns: children genetically-predisposed to antisocial behaviors were more likely to receive negative parenting from their adoptive parents. Lipscomb et al. (2011) found that adoptive mothers were attuned to their adopted child's negative emotionality (as measured by birth parent negative emotionality) which predicted decreases in adoptive parent efficacy over time for those children with the

predisposition.

Several researchers have found an evocative link between negative emotionality and negative parenting throughout development (Eisenberg, 1999; Ge & Conger, 1999; Lee & Bates, 1985; Lengua, 2006; Lengua & Kovacs, 2005; Paulussen-Hoogeboom, Stams, Hermanns, Peetsma, & van den Wittenboer, 2008; van den Akker, Dekovic, Prinzie, & Asscher, 2010; van der Bruggen, Stams, Bogels, & Paulussen-Hoogeboom, 2010); however, their findings are in the context of a bidirectional model, such that both temperament and parenting mutually influence each other over time. In this model, also referred to as a transactional model, parents and children's genetically influenced characteristics and behaviors dynamically interact throughout a child's development (Sameroff, 2000). "Transactions" occur between negative emotionality and negative parenting, in that, changes in one may increase or decrease the response of the other or may elicit or initiate a new response (Sameroff, 2009).

Bidirectional Model

Parenting and temperament may influence each other, promoting more positive qualities and exacerbating more negative ones (Lengua, 2006; Lengua & Kovacs, 2005). Children's negative emotionality has been a commonly studied trait within transactional models because it has consistently been found to powerfully evoke negative parenting (Ganiban et al., 2010; Paulussen-Hoogeboom, Stams, Hermanns, Peetsma, & van den Wittenboer, 2008). A transactional model suggests that child characteristics elicit certain parenting behaviors, which in turn, may sustain, reinforce, exacerbate, or evoke child behaviors, further shaping the initial expressed characteristic (Sanson & Rothbart, 1995; Rothbart & Bates, 1998). For example, a child high in negative emotionality may evoke

specific parental responses such as anger, impatience, hostility, or inconsistent discipline. This parental response might then reinforce the child's negative emotionality (e.g., angrier outbursts, high emotional reactiveness, etc.). If these aversive interactions and reactions are consistent in the child's development, over time, the child's negative emotionality and the parent's behaviors are mutually shaping and reinforcing each other. This reciprocal process between negative parenting and negative emotionality can become a coercive family process that gradually intensifies over time without intervention (Patterson, 2002; Scaramella & Leve, 2004).

Several studies have supported the notion of a bidirectional relationship between parenting and temperament; however, the majority of these studies focus on infancy and early childhood (e.g., Hemphill & Sanson, 2000; Maccoby & Martin, 1983; Maccoby, Snow, & Jacklin, 1984; Paulussen-Hoogeboom, Stams, Hermanns, Peetsma, & Wittenboer, 1998; Van den Boom & Hoeksma, 1994). Few studies have explored a transactional model during adolescence. Lengua and Kovacs (2005) found bidirectional effects between negative emotionality and parenting in a sample of students in 3rd through 5th grade. Child irritability predicted parental frustration with efforts to discipline or enforce limits. In turn, inconsistent or unpredictable rules and consequences amplified children's levels of frustration and irritability. Of note, the opposite effect was also observed; positive emotionality predicted greater maternal acceptance across 1 year. Similarly, in a sample of pre-adolescent children, child negative emotionality and negative parenting at Time 1 predicted how parents and children dealt with conflict 4 years later during adolescence (Time 2). Parents of children with high negative emotionality during the initial assessment (based on parent and teacher reports), exhibited

higher levels of negativity and less warmth during conflicts at Time 2. The results suggest that a child's genetically influenced characteristics accounted for some of the continuity in the quality of parent-child relationship, and that both parents and children can continue to influence one another and exacerbate a negative dynamic over time (Eisenberg et al., 2008).

Using an adolescent sample, Eisenberg et al. (1999) examined the mutual influences of parents' negative reactions and children's negative emotionality. A transactional model was supported: children's externalizing negative emotions expressed towards parents at 6-8 years of age predicted greater parental distress and more punitive discipline 2 years later (ages 8-10), and parents' punitive reactions predicted more anger, irritability, and hostility at ages 10-12. Analyses controlled for the stability in parenting and negative emotionality and their prior associations, suggesting a progressive cycle in which parenting and children's negative emotionality continued to influence each other over time. Lengua (2006) followed a group of 8-12 year olds each year over a 3-year period from middle childhood to adolescence. Baseline levels of temperament and parenting predicted changes in each other at the 2nd and 3rd follow-up visits. Specifically, irritability predicted parental frustration and inconsistent parenting, and over time, maternal rejection predicted increased levels of irritability. Very recent research has begun to not only look at the bidirectional relationships between parenting and temperament, but also the underlying mechanisms that may explain their association (Burt, McGue, Krueger, & Iacono, 2005; Moberg, Lichtenstein, Forsman, & Larsson, 2010).

Underlying Mechanisms of the Child and Parent Effects Models

The most recent trend in the literature on the influences of parenting on temperament and vice versa provides evidence for a bidirectional relationship. Further studies have demonstrated that the mutual influences of parenting and temperament continue to develop over time; however, only a handful of studies have examined the etiology (e.g., genetic or environmental) of the bidirectional association (Burt, McGue, Krueger, & Iacono, 2005). Save a few studies, prior research can only speak to the phenotypic relationship between temperament and parenting and make assumptions about the etiology based on the directionality of findings. Indeed, researchers have assumed that parent-driven effects on temperamental change reflect an environmental mechanism and that an evocative effect is accounted for by child-based genetically driven characteristics. While these assumptions have some basis theoretically, few studies have explicitly demonstrated their veracity. The current study sets out to test these assumptions utilizing a biometrically informed cross-lagged design.

A behavioral genetic design allows researchers to identify the underlying mechanism through which negative emotionality is related to parental negativity by disentangling genetic from environmental contributions to understand the magnitude of their influence (Waldman, 2007). This can be done by including pairs of siblings with a varying degree of genetic relatedness, and based on the degree of similarity between these sibling groups, making inferences about genetic and environmental contributions. Behavioral genetic studies can provide broad estimates of the impact of children's genetic makeup upon their negative emotionality as well as the parenting they receive. Behavioral genetic designs can also estimate the relative importance of environmental

influences on negative emotionality and parenting. Environmental contributions are commonly separated into *shared* and *nonshared* influences. Shared environmental influences are common experiences that make two siblings similar, whereas nonshared environmental influences are unique experiences that make siblings different. Incorporating a behavioral genetic design within a cross-lagged structural equation model allows us not only to examine the bidirectional relationship between negative emotionality and parental negativity and their mutual influence over time, controlling for their pre-existing relationship, but also informs us whether genetic (a), shared (c), or nonshared (e) environmental influences account for associations between these constructs over time.

Neiderhiser and colleagues (1999) conducted one of the first studies to examine the genetic and environmental contributions to the cross-lagged associations between parenting and child outcomes using a multivariate genetic analysis. Over a 3-year period, they found that child-based genetic factors primarily accounted for the bidirectional influence of maternal negativity on adolescent depressive symptoms and antisocial behaviors and vice versa; however, they examined the cross-lagged associations in two separate models which did not allow them to constrain for the pre-existing relationship of maternal negativity and temperament at Time 1 nor speak to the underlying mechanism that explains the stability and change in their associations over time. Burt, McGue, Krueger, and Iacono (2005) developed a cross-lagged model that could account for the stability and mutual influences in adolescent externalizing symptoms and parent-child conflict, controlling for a pre-existing relationship, and decompose the underlying mechanism into genetic, shared, or nonshared components. Their results also supported a

bidirectional model; externalizing symptoms and parent-child conflict independently influenced each other over time, even after controlling for their prior relationship. Further, Burt and colleagues (2005) also found that child-based genetic factors and environmental mechanisms accounted for parenting effects on externalizing symptoms and child-based genetic influences primarily accounted for the influence of externalizing behaviors on parent-child conflict, supporting an evocative model.

A handful of other researchers have used longitudinal genetically informed designs to better understand the direction and underlying mechanism of related variables. Larsson, Viding, Rijsdijk, and Plomin (2008) found a bidirectional relationship between parental negativity and antisocial behavior in early childhood. Genetic, shared and nonshared environment accounted for the cross-lag from parenting to antisocial behaviors, whereas genetic factors primarily explained the cross-lag from antisocial behaviors to parenting, with some nonshared environmental influences, as well. Using the same model, Moberg, Lichtenstein, Forsman, and Larsson (2010) found that child-based genetic effects accounted for the majority of the relationship between parenting and internalizing symptoms, supporting a true evocative model. Of note, these findings held true for daughters and not for sons, indicating potential sex differences in how the expression of internalizing symptoms uniquely influences parenting received.

The few studies that have used a cross-lagged biometric model have focused on parenting and behavioral outcomes (e.g., internalizing and externalizing symptoms). Previous studies have not examined negative emotionality and parental negativity utilizing a cross-lagged biometric model. Additionally, few studies have focused on temperament change in the adolescent period, as a result of parenting behaviors. This gap

in the research literature may reflect the common belief that temperament is constitutionally-based and stable over time (Rothbart & Bates, 1998; Sanson, Hemphill, & Smart, 2004). However, genes do not entirely determine adolescent temperament; indeed, environmental influences may minimize or magnify genetic predispositions (Rutter, 1989) and shape personality formation. Furthermore, previous research has suggested that adolescence is an important time when temperament development continues to reorganize and solidify (Ge & Conger, 1999). Ganiban, Saudino, Ulbricht, Neiderhiser and Reiss (2008) found that during adolescence, temperament stability was primarily explained by genetic factors, but changes in temperament were primarily explained by environmental factors. Environmental influences on temperament change in adolescence was also illustrated in a 6-year longitudinal study by Ge and Conger (1999), which examined the effect of psychological distress experienced in adolescence on personality formation. They discovered that psychological distress experienced from 7th to 10th grade influenced the development of negative and positive emotionality in 12th grade. Further, many researchers specifically note that adolescence is a time of heightened negative emotionality (Collins & Steinberg, 2006; Larson & Lampman-Petraitis, 1989) which can impact the parent-child relationship (Larson, Coy & Collins, 1998), in particular with mothers (Steinberg & Silk, 2002). These findings not only indicate the importance of looking at parental influences on temperamental change (and vice versa), but also the need to focus on adolescence as an important period of transition. Therefore, the current study set out to do both using a cross-lagged biometric model.

Current Study

The current study used a cross-lagged biometric model to examine the direction and etiology of Parental Negativity and adolescents' Negative Emotionality, and their mutual influence over time. By using a behavioral genetic approach, the researcher was able to better understand the underlying mechanisms of the constructs' mutual influence.

The current study investigated the following research questions:

1. Do Maternal and Paternal Negativity during early adolescence contribute to changes in Negative Emotionality later in adolescence?
    a. Is this cross-lagged effect explained by genetic, shared or nonshared environmental influences?
2. Does Negative Emotionality during adolescence contribute to changes in mothers' and fathers' Negativity later in adolescence?
    a. Is this cross-lagged effect explained by genetic, shared or nonshared environmental influences?

In accordance with prior research the following hypotheses were made:

1. Based on previous research, it was hypothesized that Maternal and Paternal Negativity at Time 1 would contribute to changes in Negative Emotionality at Time 2, after controlling for their association at Time 1 and each variable's stability over time.
    a. It was hypothesized that a significant cross-lagged effect between Parental Negativity and adolescent Negative Emotionality would be explained

primarily by environmental mechanisms, consistent with a parent effects model.

2. Based on previous research, it was hypothesized that adolescent Negative Emotionality at Time 1 would contribute to changes in Maternal and Paternal Negativity during Time 2, after controlling for their association at Time 1 and each variable's stability over time.

    a. It was hypothesized that a significant cross-lagged effect between Parental Negativity and adolescent Negative Emotionality would be explained primarily by child-based genetic factors consistent with a child effects model.

Chapter 2: Methods

Participants

The Nonshared Environment and Adolescent Development (NEAD) project is a nationwide sibling study that included nondivorced families and stepfamilies from across the country (Reiss et al., 2000). To be included in the study, participating families were required to have two adolescent siblings of the same gender within 4 years of age from one another (M=1.61 years apart $\pm$ 1.29 years) and be married or remarried for at least 5 years prior to the first wave of data collection (M=8.9 $\pm$ 3.7 years of marriage). Families were assessed two times, 3 years apart. In total, 720 families were recruited for the first wave; however, 12 families were excluded because the sibling pairs' genetic relatedness was not provided. The remaining families (N=708) were grouped into six sibling categories. Within nondivorced families, sibling groups included monozygotic (MZ) twin

pairs (N=93), dizygotic (DZ) twin pairs (N=99), and full sibling (FI) pairs (N=95). Within stepfamilies, sibling groups included full sibling (FS) pairs (N=182), half siblings (HS) pairs (N=109), and genetically unrelated sibling (US) pairs (N=130). The sample included a total of 1416 adolescents. The average age for Sibling 1 was 13.5 $\pm$ 2.0 years and for Sibling 2 was 12.1 $\pm$ 1.3 years. Three hundred ninety five families were retained for the 2nd wave of data collection [N of sibling pairs = (MZ=63; DZ=75; FI=58; FS=95; HS=60; US=44)]. At Time 2, the mean age of Sibling 1 was 16.11 years (S.D. = 2.10) and Sibling 2 was 14.67 years (S.D. =1.86). The lower number of participants at Time 2 reflected the eligibility requirements: both siblings had to still be residing at home with their parents in order to be eligible at Time 2.

Two commercial survey companies compiled a list of 675,000 households, from which participants were recruited through random digit dialing and national market panels. The participating families were primarily Caucasian (mothers = 94%; fathers = 93%), middle class (mean incomes ranging from $25,000 to $35,000), and educated (on average, mothers and fathers completed 13.6 and 14 years of school, respectively).

Twin Zygosity

Interviewer, parent, and self-report ratings of the twins' physical similarity (e.g., eye and hair color) were assessed using a questionnaire developed by Nichols and Bilbro (1966) in order to determine zygosity classifications. Prior research has demonstrated that this method of categorizing zygosity is at least 90% accurate when compared to DNA testing (Nichols & Bilbro, 1966; Spitz et al., 1996). When a twin pair was rated as being physically different from one and other, allowing others to make a distinction between the two, the pair was classified as dizygotic. Twin pairs, who were rated as not having

any physical differences, were classified as monozygotic. The zygosity of 7% of the twins could not be identified using this method because of conflicting cross-rater reports. Because biometric analyses used varying degrees of genetic relatedness to understand phenotypic variation, twins without reliable zygosity classifications were excluded from the analyses.

Procedures

Two three-hour home visits were completed two weeks apart at each Wave of data collection. Home visits included interviews of all family members and observations of their interactions. Each visit was videotaped.

Questionnaires, some of which were mailed ahead of time, were completed by both parents and each adolescent sibling. The questionnaires covered topics including the adolescent's temperament characteristics, received parenting, and psychological adjustment.

Measures

Adolescent temperament. Mothers and fathers completed the EAS Temperament Survey-Parent Form (Buss & Plomin, 1984) for each adolescent sibling involved in the study. The EAS consists of twenty descriptive statements of the adolescent's behavior during the past two weeks. Using a 5-point Likert scale, parents were asked to rate the degree to which each statement described their adolescents, with ratings of "1" indicating that the statement was not at all characteristic of the adolescent to "5" indicating that the statement was very much characteristic of the adolescent. The current study focused on a subset of items (N=5, α's = .81 - .86 for mothers and fathers) that comprised the negative emotionality subscale. Items in this scale measure an adolescent's tendency to express

negative affect; specific examples include crying easily, fussing, getting upset easily, and reacting intensely when upset. Given that the mothers' and fathers' reports of sibling 1's and 2's negative emotionality were significantly correlated at each age (*r's* ranged from .42 to .48), their ratings were combined to form a multi-rater composite of adolescent negative emotionality. The utility of multiple informants has been widely advocated to reduce single rater bias (e.g., Natsuaki et al., 2010).

Parental Negativity. In addition to an observational measure, three measures were used to assess mothers' and fathers' behavior toward each sibling.

Parent-Child Relationship (PCR; Hetherington & Clingempeel, 1992). Mothers, fathers, and their children were asked to rate positive and negative aspects of their relationship using 5-point Likert scales (from *not at all* to *extremely*). The PCR comprised 36 items for the parent and 31 items for the child. Both parent and child reported on two scales: closeness ("How much does this person understand you?") and conflict ("How often does this person get into disagreements or fights with you?"). Internal reliabilities for closeness and conflict averaged .87 and .75 for parents and .91 and .71 for child ratings, respectively. Two-month test-retest correlations ranged from .87 to .91 in previous studies (Hetherington & Clingempeel, 1992). On the PCR measure, the items were summed and higher scores indicated more disagreements.

Parent Child Disagreements (PCD; Hetherington & Clingempeel, 1992). This questionnaire assessed parent-child disagreements over a variety of issues. Mothers, fathers, and children completed this questionnaire. Each item first uses a dichotomous scale to indicate whether the child and parent have disagreed about an issue in the past month. If they have disagreed, the parent and child must indicate how often they

disagreed using a 6-point Likert scale ranging from 1 (more than once a day) to 6 (not at all), and how bad the disagreement was using a 5-point Likert scale ranging from 1 (extremely bad) to 5 (not bad at all). The items measured parent-child disagreements on areas including household routines (e.g., chores), behavior to other family members (e.g., behavior toward brothers and sisters), adolescent issues (e.g., dating), and deviant behavior (e.g., use of alcohol). The sum mean score across all items was used as an index of total disagreements. This measure showed adequate internal reliability for parents (alpha=.87) and children (alpha = .86).

Parent Discipline Behavior (PDB; Hetherington & Clingempeel, 1992): Parents and children used this scale to report on parents' use of various disciplinary strategies. This scale included 43 items, and three subscales: nattering, punitive, and yielding to coercion behaviors. The latter two subscales (punitive, and yielding to coercion) were used in this study. *Punitive discipline* corresponds to parents' use of hostile, coercive behaviors (e.g., "Yelled at you about something you did wrong"). Yielding to coercion related to parents' tendency to give into the child's demands (e.g., "Let you get away with bad behavior"). For both subscales, the items were summed and higher scores indicated more punitive or yielding behaviors. Both subscales demonstrated adequate internal reliabilities for parents (alphas = .78 - .85) and children (alphas = .74 - .88).

Conflict Tactics Scale (CTS; Straus, 1979). Mothers, fathers, and their children completed this 18-item measure which assessed how parents act during a conflict with each sibling. Items were rated on a 5-point Likert scale ranging from *not at all common or typical* to *very common or typical*, yielding three scales: reasoning (e.g., discussed the issue calmly), symbolic aggression (e.g., insulted or swore), and violence (e.g., hit or

tried to hit with something). The CTS items were summed and higher scores indicated more aggression. For parental reports, internal reliabilities for the reasoning and symbolic aggression scales averaged .41 and .79, respectively; for child reports, internal reliabilities averaged .43 and .80, respectively. Only the symbolic aggression scale was used in the current study.

Observational Measure (O'Conner, Hetherington, Reiss, & Plomin, 1995). Dyadic interactions between parents and children were given global ratings by trained coders. The coders rated three components of familial negativity: *Anger/Hostility* (e.g., extreme negative, angry, or contemptuous remark made by the parent or child; or nonverbal behaviors such as tone of voice, poor eye contact, and ignoring the other person); *Coercion* (e.g., the degree to which the parent or child expressed his or her needs or opinions in a negative, controlling or stubborn manner); and *Transactional Conflict* (e.g., the frequency and intensity of reciprocated anger/hostility in the dyad). The global ratings assessed the frequency and intensity of observed behaviors.

Composite scores. In the interest of data reduction, principal component factor analysis with a varimax rotation was used to create multi-measure composites of parenting (see Plomin, Reiss, Hetherington, & Howe, 1994). These analyses used mothers' and fathers' self-report, child reports, and observational data. Factor analyses yielded three factors that were similar for mothers and fathers: negativity, warmth, and monitoring. The parental negativity factor domain included: Parent Discipline Behavior (PDB: punitive scale loading = .79; and yielding to coercion scale loading = .50); Parent-Child Relationship (PCR: conflict scale loading = .68); Conflict Tactics Scale (CTS: symbolic aggression scale loading = .59). Given the focus on negativity in the current

study, only these measures, in addition to the observational data, were used in the subsequent analyses.

Analyses

The relative contributions of genetic, shared and nonshared environmental factors to phenotypes can be estimated by examining intraclass phenotypic correlations across sibling pairs who vary in genetic relatedness (i.e., MZ, DZ, FI, FS, HS, and US pairs). MZ twins have the same genotype and therefore share 100% of their genes. DZ twins and full siblings share 50% of their segregating genes, on average. Half siblings share approximately 25% of their segregating genes. Lastly, unrelated siblings, from different biological parents do not share any genetic relatedness. Therefore, if MZ correlations > DZ and FS/FI correlations > HS correlations > US correlations, it suggests that phenotypic associations are explained by genetic factors. If correlations amongst sibling groups are similar, it suggests that phenotypic associations are explained by the siblings' shared environment. If there is no systematic pattern across sibling types, it suggests that phenotypic associations may be explained by unique experiences or measurement error (Plomin & Daniels, 2008).

Biometric model fitting allows for more precise estimates of genetic, shared, and nonshared environmental contributions to each behavior and their association than intraclass correlations. Biometric models estimate the magnitude of additive genetic, and shared and nonshared environmental contributions to phenotypes. The additive genetic component (A) accounts for the total contributions of genetic factors to Adolescent Negative Emotionality or Parental Negativity. The shared environmental component (C) accounts for environmental influences that make siblings similar for each construct. This

latent factor is a source of between-family differences. The nonshared environmental component (E) accounts for individual-specific experiences that make siblings different in regard to parenting or temperament. (E) also includes measurement error. This latent factor is a source of within-family effects.

A biometric cross-lagged model was used to examine the associations between Adolescent Negative Emotionality and Parental Negativity across a 3-year period (see Fig. 1). This model constrains all cross-age associations to function as partial regression coefficients (b_{11}, b_{22}, b_{12} and b_{21}). The two cross-age parameters estimate the stability of Adolescent Negative Emotionality (b_{11}) and Parental Negativity (b_{22}) over a 3-year period, controlling for the pre-existing association of the phenotypes at Time 1. The cross-lagged paths estimate the independent contribution of Adolescent Negative Emotionality at Time 1 on Parental Negativity at Time 2(b_{12}) and the independent contribution of Parental Negativity at Time 1 on Adolescent Negative Emotionality at Time 2(b_{21}), controlling for the stability of both phenotypes and the association between these phenotypes at Time 1. The model also partitions variance for each phenotype into genetic, and shared and nonshared environmental components at each Time. For example, at Time 1, paths a_1, c_1, e_1 indicate the degree to which latent genetic (A1), shared environmental (C1), and nonshared environmental (E1) factors account for variance in variable 1 (Adolescent Negative Emotionality), and paths a_2, c_2, e_2 provide estimates of the contributions of latent genetic (A2), shared environmental (C2), and nonshared environmental (E2) factors to variable 2 (Parental Negativity). However, paths a_3, c_3, e_3, a_4, c_4, and e_4 correspond to genetic (A3, A4), shared (C3, C4) and nonshared (E3, E4) environmental contributions to Adolescent Negative Emotionality and Parental

Negativity that are unique to Time 2. The model also estimates genetic, shared and nonshared correlations between Adolescent Negative Emotionality and Parental Negativity at Time 1 (r_{a12}, r_{c12}, r_{e12}) and Time 2 (r_{a34}, r_{c34}, r_{e34}). The Time 2 correlations control for phenotypic associations at Time 1.

The parameter estimates from the cross-lagged model can be used to estimate four types of effects: Stability effects, cross-lagged effects, common effects at Time 1 and residual effects at Time 2 (Larsson, Viding, Rijsdijk, & Plomin, 2008; Moberg, Lichtenstein, Forsman, & Larsson, 2011). All pathways are illustrated in Figure 1:

(1) *Stability effects* relate to rank order stability in Negative Emotionality and Parental Negativity from Time 1 to Time 2. These effects are partitioned into genetic, shared environmental, and nonshared environmental components.

   a. Genetic:
      i. Genetic variance of Negative Emotionality at Time 2 that is accounted for by genetic effects at Time 1 ($a_1^2 * b_{11}^2$).
      ii. Genetic variance of Parental Negativity at Time 2 that is accounted for by genetic effects at Time 1 ($a_2^2 * b_{22}^2$).
   b. Shared environment:
      i. Shared environmental variance of Negative Emotionality at Time 2 that is accounted for by genetic effects at Time 1 ($c_1^2 * b_{11}^2$).
      ii. Shared environmental variance of Parental Negativity at Time 2 that is accounted for by shared environmental effects at Time 1 ($c_2^2 * b_{22}^2$).
   c. Nonshared environment:
      i. Nonshared environmental variance of Negative Emotionality at Time 2 that

is accounted for by nonshared environmental effects at Time 1 ($e_1^2 * b_{11}^2$).

ii. Nonshared environmental variance of Parental Negativity at Time 2 that is accounted for by nonshared environmental effects at Time 1 ($e_2^2 * b_{22}^2$).

(1) *Cross-lagged effects* relate to the extent to which Parental Negativity and Negative Emotionality account for change in each other over time. These effects can also be partitioned into genetic, shared environmental, and nonshared environmental components:

a. Genetic:

i. Genetic variance of Negative Emotionality at Time 2 that is explained by genetic influences of Parental Negativity at Time 1, independent of any prior genetic association between the two variables at Time 1 ($a_2^2 * b_{21}^2$).

ii. Genetic variance of Parental Negativity at Time 2 that is explained by genetic influences of Negative Emotionality at Time 1, independent of any prior genetic association between the two variables at Time 1 ($a_1^2 * b_{12}^2$).

b. Shared environment:

i. Shared environmental variance of Negative Emotionality at Time 2 that is explained by shared environmental influences of Parental Negativity at Time 1, independent of any prior shared environmental association between the two variables at Time 1 ($c_2^2 * b_{21}^2$).

ii. Shared environmental variance of Parental Negativity at Time 2 that is explained by shared environmental influences of Negative Emotionality at Time 1, independent of any prior shared environmental association between the two variables at Time 1 ($c_1^2 * b_{21}^2$).

c. Nonshared environment:

i. Nonshared environmental influences of Negative Emotionality at Time 2 that is explained by nonshared environmental influences of Parental Negativity at Time 1, independent of any prior nonshared environmental association between the two variables at Time 1 ($e_2^2 * b_{21}^2$).

ii. Nonshared environmental influences of Parental Negativity at Time 2 that is explained by nonshared environmental influences of Negative Emotionality at Time 1, independent of any prior nonshared environmental association between the two variables at Time 1 ($e_1^2 * b_{21}^2$).

(1) *Common effects* describe the degree to which covariance between Negative Emotionality and Parental Negativity at Time 1 account for variance in Negative Emotionality and Parental Negativity at Time 2. These effects are further broken down into genetic, shared, and nonshared environmental components for each construct.

a. Genetic:

i. Genetic influences that are shared by Negative Emotionality and Parental Negativity at Time 1 [$2* (b_{21} * a_2 * r_{a12} * a_1 * b_{11})$].

b. Shared environment:

i. Shared environmental influences that are shared by Negative Emotionality and Parental Negativity at Time 1 [$2* (b_{21} * c_2 * r_{c12} * c_1 * b_{11})$].

c. Nonshared environment:

i. Nonshared environmental influences that are shared by Negative Emotionality and Parental Negativity at Time 1 influences [$2* (b_{21} * e_2 *$

$r_{e12} * e_1 * b_{11})]$.

(1) *Residual effects* relate to variance in Negative Emotionality and Parental Negativity at Time 2 that is independent of Time 1.

a. Genetic:

i. Genetic influences of Negative Emotionality at Time 2 that is independent of genetic influences of Negative Emotionality at Time 1 (a_3^2).

ii. Genetic influences of Parental Negativity at Time 2 that is independent of genetic influences of Parental Negativity at Time 1 (a_4^2).

b. Shared environment:

i. Shared environmental influences of Negative Emotionality at Time 2 that is independent of shared environmental influences of Negative Emotionality at Time 1 (c_3^2).

ii. Shared environmental influences of Parental Negativity at Time 2 that is independent of shared environmental influences of Parental Negativity at Time 1 (c_4^2).

c. Nonshared environment:

i. Nonshared environmental influences of Negative Emotionality at Time 2 that is independent of nonshared environmental influences of Negative Emotionality at Time 1 (e_3^2).

ii. Nonshared environmental influences of Parental Negativity at Time 2 that is independent of nonshared environmental influences of Negative Emotionality at Time 1 (e_4^2),

The Mx Statistical package was used to conduct biometric analyses. Mx was explicitly designed to estimate genetic ("A"), shared ("C") and nonshared ("E") environmental variance of behaviors, or the "ACE" model, by using sibling pairs that vary in genetic relatedness (Neale, Boker, Xie, & Maes, 2003). Maximum Likelihood estimation based on raw data was used to estimate all paths and to obtain model fitting statistics. The fit of the full ACE model was determined based on the -2LL (minus twice the log-likelihood). Several nested models were also fit. These models tested the significance of the cross-lagged paths through systematically setting each path to "0", and determining if this constraint lead to a worse model fit.

The Likelihood Ratio Test (LRT) was used to compare the fit of the nested models to the full ACE model. The difference in fit between models can be assessed by the difference in -2LL values (Δ-2LL) relative to the differences in the degrees of freedom (Δdf) between the models. Within the context of the current study's hypotheses, we tested each cross-lagged path separately. To do so, we set one path to "0" at a time. A Δ2LL difference score of 3.84 or higher with a Δdf of "1" is indicative of a significant difference in model fit. If the model fit is worse, than the path set to "0" is significant. If the Δ2LL score is not significant, then the more parsimonious model is selected, because this indicates model fit does not deteriorate with elimination of the cross-lagged path. With regard to the current study's research questions, the following pathways were estimated:

1. Does Maternal and Paternal Negativity during early adolescence contribute to changes in Negative Emotionality in later adolescence?
    a. Is this cross-lagged effect explained by genetic, shared or nonshared

environmental influences ($a_1^2 * b_{12}^2$); ($c_1^2 * b_{12}^2$); ($e_1^2 * b_{12}^2$)?

2. Does Negative Emotionality during adolescence contribute to changes in mothers' and fathers' negativity in later adolescence?

    a. Is this cross-lagged effect explained by genetic, shared or nonshared environmental influences ($a_2^2 * b_{21}^2$); ($c_2^2 * b_{21}^2$); ($e_2^2 * b_{21}^2$)?

Chapter 3: Results

All analyses controlled for gender and age differences between siblings. All variables were standardized to a mean of 0 and a standard deviation of 1.

Phenotypic Correlations

The pattern of phenotypic correlations between measures was similar for both parents (presented in Table 1). There were moderate relationships between Parental Negativity and Adolescent Negative Emotionality at Time 1 (paternal, r=0.32; maternal, r=0.32). There was moderate stability in both Parental Negativity (paternal, r=0.62; maternal, r=0.65) and Negative Emotionality from early to later adolescence (r=0.65). The cross-lagged correlations between Adolescent Negative Emotionality at Time 1 and Parental Negativity at Time 2 were moderate (paternal, r=0.29; maternal, r=0.32), as were the cross-lagged correlations between Parental Negativity at Time 1 and Adolescent Negative Emotionality at Time 2 (paternal, r=0.23; maternal, r=0.31).

Cross-Sibling Intraclass Correlations

Table 2 contains the intraclass correlations for Paternal Negativity, Maternal Negativity, and Negative Emotionality at Time 1 and 2 by sibling zygosity. The intraclass correlations can provide a first indication of the genetic and environmental influences on

individual differences in Parental Negativity and Adolescent Negative Emotionality. In all cases, the intraclass correlations were greater for MZ twins than for DZ twins, suggesting genetic influences for all phenotypes. For parenting, the DZ correlations are larger than half the MZ correlations, suggesting shared environmental influences. Finally, the correlations for Paternal Negativity and Maternal Negativity are roughly similar for siblings of high genetic relatedness. However, for siblings of less genetic relatedness, the correlations for Maternal Negativity are higher than for Paternal Negativity in half-siblings, and lower than Paternal Negativity for unrelated siblings.

Model Fitting

The full cross-lagged ACE model was fit first for Paternal Negativity and Negative Emotionality (*-2LL* = 10804.878, *Adjusted BIC* = -1792.785) and for Maternal Negativity and Negative Emotionality (*-2LL* = 10785.048, *Adjusted BIC* = -1822.236). All paths that were estimated as not significantly different from zero were dropped to create a more parsimonious model. For the model including Paternal Negativity, the following pathways were dropped: paths related to shared environmental contributions to each variable and their association at Time 1 (r_{c12}, r_{c34}, c_1, c_3); paths related to genetic influences on Paternal Negativity at Time 2 and to the association between the two variables at Time 2 ($r_{a34,}$ a_4^2); and paths related to residual nonshared environmental factors accounting for the association between the two variables at Time 1 (r_{e12}). Elimination of these paths did not significantly affect the fit of the model (Paternal Negativity model fit: *-2LL* = 10796.528, *Adjusted BIC* = -1800.466). For the cross-lagged model including Maternal Negativity, pathways related to shared and nonshared environmental associations between negative parenting and Adolescent Negative

Emotionality at Time 1 and 2 (r_{c12}, r_{c34}, r_{e12}, r_{e34}) were dropped as well as unique shared environmental pathways for Negative Emotionality at Time 1 and 2 (c_1 and c_3); elimination of these paths did not significantly affect the fit of the model (Maternal Negativity model fit: *-2LL* = 10793.532, *Adjusted BIC* = -1828.155). Model fit statistics and parameter estimates from the full and nested models are summarized in Table 3. The most parsimonious models, with parameter estimates, are illustrated in Figures 2 and 3.

How Much of the Variance in Paternal Negativity and Adolescent Negative Emotionality is Accounted for by Genetic, Shared and Nonshared Environmental Factors?

Time 1. To estimate genetic and environmental contributions to the variance of each variable, paths linking the latent variables (A, C and E) to each variable were estimated and squared (paths a_1^2, c_1^2, e_1^2, a_2^2, c_2^2, and e_2^2 found in Figure 2). These values are included in Figure 2. Negative Emotionality was primarily accounted for by genetic factors (62%) at Time 1 and partially accounted for by nonshared environmental factors (38%) at Time 1; shared environmental factors did not contribute (0%). Genetic and environmental factors both accounted for variance in Paternal Negativity (A = 46%; C = 32%; E = 22%) at Time 1.

Time 2. As summarized in Table 4, at Time 2, genetic and environmental factors explained variance in Paternal Negativity and Negative Emotionality. These variance estimates reflect the sum of stability, cross-lagged, joint, and time specific effects for each variable. Each of these effects is included in the right side of Table 4, and is described in detail below.

Genetic factors accounted for 53% of variance in Negative Emotionality, and 18%

of variance in Paternal Negativity. Nonshared environmental factors also explained significant variance in Negative Emotionality (47%) and Paternal Negativity (50%). Shared environmental effects were also moderate for Paternal Negativity (31%), but trivial for Negative Emotionality (0.4%). Overall, we noted decreasing genetic contributions, increasing nonshared environmental contributions, and relatively stable shared environmental contributions for each construct from Time 1 to Time 2.

Stability effects. Paternal Negativity and Adolescent Negative Emotionality were moderately stable over time: Time 1 Negative Emotionality accounted for 37% variance in Time 2 Negative Emotionality (i.e., $b_{11}^2 = 0.37$), while Time 1 Paternal Negativity explained 31% of the variance in Time 2 Paternal Negativity (i.e., $b_{22}^2 = 0.31$). The biometric analyses indicated that stability in Negative Emotionality was mainly accounted for by genetic factors ($a_1^2 * b_{11}^2 = 0.23$). Nonshared environmental factors also explained 14% of variance in Time 2 Negative Emotionality ($e_1^2 * b_{11}^2 = 0.14$). Stability in Paternal Negativity was explained by genetic ($a_2^2 * b_{22}^2 = 0.14$) and environmental factors (Shared: $c_2^2 * b_{22}^2 = 0.10$; Nonshared: $e_2^2 * b_{22}^2 = 0.07$). Therefore, genes (14%) and environmental (17%) factors almost equally contributed to stability in Paternal Negativity.

***Cross-lagged effects*.** Paternal Negativity and Negative Emotionality at Time 1 accounted for changes in each other over time. Paternal Negativity at Time 1 independently accounted for 1.3% of variance in Negative Emotionality at Time 2 after controlling for their relationship at Time 1, i.e., $b_{21}^2 = .013$. This cross-lagged effect was almost equally explained by genetic and environmental factors. Specifically, genetic factors related to Time 1 Paternal Negativity accounted for 0.6% variance in Time 2

Negative Emotionality, i.e. $a_2^2 * b_{21}^2 b_{21} = 0.006$. Shared and nonshared environmental factors related to Time 1 Paternal Negativity jointly explained 0.7% of variance in Time 2 Negative Emotionality, i.e., $c_2^2 * b_{21}^2 = 0.004$ and $e_1^2 * b_{21}^2 b_{21} = 0.003$, respectively. Negative Emotionality independently accounted for 0.8% of Paternal Negativity at Time 2, after controlling for their relationship at Time 1, i.e., $b_{22}^2 = 0.008$. Most of this cross-lagged effect was explained by genetic influences on Negative Emotionality at Time 1 ($a_1^2 * b_{12}^2 = 0.005$). Nonshared environmental factors also contributed to the cross-lagged effect ($e_2^2 * b_{12}^2 = 0.003$). In summary, Negative Emotionality and Negative Parenting at Time 1 predicted modest changes in each other over time. Moreover, these cross-lagged effects were due to both genetic and environmental factors.

Joint effects. Negative Emotionality and Paternal Negativity were moderately correlated at Time 1. Genetic factors accounted for this association (r_{a12}=.55). Furthermore, the covariance between Negative Emotionality and Paternal Negativity at Time 1 explained 4% of variance in Time 2 Paternal Negativity, i.e., $2 * (b_{21} * a_2 * r_{a12} * a_1 * b_{11}) = 2 * (0.11 * 0.68 * 0.55 * 0.79 * 0.61 = 0.04)$. Similarly, covarying genetic influences on both variables at Time 1 explained 3% of the variance in Paternal Negativity at Time 2 ($2 * (b_{12} * a_1 * r_{a12} * a_2 * b_{22} = 2 * (0.09 * 0.79 * 0.55 * 0.68 * 0.56 = 0.03$).

***Time specific effects*.** Although Time 1 variables accounted for moderate variance in the Time 2 variables, there were also genetic and environmental influences unique to Time 2. Specifically, after controlling for Time 1 influences, nonshared environmental factors accounted for 33% of variance in Negative Emotionality, and genetic factors accounted for 25%. After controlling for Time 1 influences, nonshared environmental

factors primarily accounted for residual variance in Paternal negativity (43%), and shared environmental factors explained twenty-one percent of the variance.

How Much of the Variance in Maternal Negativity and Adolescent Negative Emotionality is Accounted for by Genetic, Shared and Nonshared Environmental Factors?

Time 1. To determine genetic and environmental contributions to the variance of each variable, parameter estimates associated with the latent variables A, C and E were squared (i.e., paths a_1^2, c_1^2, e_1^2, a_2^2, c_2^2, and e_2^2 found in Figure 3). Negative Emotionality was primarily accounted for by genetic factors (64%) at Time 1 and partially accounted for by nonshared environmental factors (36%) at Time 1; nonshared environmental factors did not contribute (0%). Genetic and environmental factors both accounted for the variance in Maternal Negativity (A = 55%; C = 24%; E = 20%) at Time 1.

Time 2. As summarized in Table 5, at Time 2, genetic and environmental factors explained variance in Maternal Negativity and Negative Emotionality. Specifically, genetic factors accounted for 52% of variance in Negative Emotionality, and 32% of variance in Maternal Negativity. Nonshared environmental factors also explained significant variance in Negative Emotionality (49%) and Maternal Negativity (42%). Shared environmental effects were also moderate for Maternal Negativity (25%), but trivial for Negativity Emotionality (0.2%). These variance estimates reflect the sum of stability, cross-lagged, joint, and time specific effects for each variable. Each of these effects is described in detail below, and included in the right side of Table 5. With regard to general patterns of change for variance in Maternal Negativity and Adolescent Negative Emotionality from Time 1 to Time 2, we found decreasing genetic

contributions, increasing nonshared environmental contributions, and relatively stable shared environmental contributions were apparent.

Stability effects. Maternal Negativity and Adolescent Negative Emotionality were moderately stable over time: Time 1 Negative Emotionality accounted for 37% variance in Time 2 Negative Emotionality (i.e., $b_{11}^2 = 0.37$), while Time 1 Maternal Negativity explained 36% of the variance in Time 2 Maternal Negativity (i.e., $b_{22}^2 = 0.36$). The biometric analyses indicated that stability in Negative Emotionality was mainly accounted for by genetic factors, i.e., $a_1^2 * b_{11}^2 = 0.24$. Nonshared environmental factors, however, explained 14% of variance in Time 2 Negative Emotionality, i.e. $e_1^2 * b_{11}^2 = 0.14$. Stability in Maternal Negativity over time was explained by genetic ($a_2^2 * b_{22}^2 = 0.20$) and environmental factors (Shared: $c_2^2 * b_{22}^2 b_{22} = 0.09$; Nonshared: $e_2^2 * b_{22}^2 b_{22} = 0.07$). Therefore, genes (20%) and environmental (16%) factors almost equally contributed to stability in Maternal Negativity.

Cross-lagged effects. Maternal Negativity and Negative Emotionality at Time 1 accounted for changes in each other over time. Maternal Negativity at Time 1 independently accounted for 0.8% of Negative Emotionality at Time 2 after controlling for their relationship at Time 1, i.e., $b_{21}^2 = 0.008$. This cross-lagged effect was equally explained by genetic and environmental factors. Specifically, genetic factors related to Time 1 Maternal Negativity accounted for 0.4% of the variance in Time 2 Negative Emotionality, i.e. $a_2^2 * b_{21}^2 b_{21} = 0.004$. Shared and nonshared environmental factors related to Time 1 Maternal Negativity jointly explained 0.4% of variance in Time 2 Negative Emotionality, i.e., $c_2^2 * b_{21}^2 = 0.002$ and $e_1^2 * b_{21}^2 b_{21} = 0.002$, respectively.

Negative Emotionality independently accounted for 0.6% of Maternal Negativity at

Time 2, after controlling for their relationship at Time 1, i.e., $b_{22}^2 = 0.006$. Most of this cross-lagged effect was explained by genetic influences of Negative Emotionality at Time 1 ($a_1^2 * b_{12}^2 = 0.004$). Nonshared environmental factors also contributed to the cross-lagged effect ($e_2^2 * b_{12}^2 = 0.002$). Shared environmental factors did not contribute to the cross-lagged effects. In summary, Negative Emotionality and Negative Parenting at Time 1 predicted modest changes in each other over time. Moreover, these cross-lagged effects were due to both genetic and environmental factors.

Joint effects. Negative Emotionality and Maternal Negativity were moderated correlated at Time 1. As illustrated in Figure 3, genetic factors underlie this association at Time 1 (r_{a12}=.53). Shared and nonshared environmental factors did not contribute to this association. The covariance between Negative Emotionality and Maternal Negativity at Time 1 explained 3% of variance in Time 2 Maternal Negativity, i.e., $2 * (b_{21} * a_2 * r_{a12} * a_1 * b_{11}) = 2 * (0.09 * 0.74 * 0.55 * 0.80 * 0.61 = 0.03)$. Similarly, shared genetic influences on both variables at Time 1 explained 3% of variance in Maternal Negativity at Time 2 ($2 * (b_{12} * a_1 * r_{a12} * a_2 * b_{22} = 2 * (0.08 * 0.8 * 0.53 * 0.74 * 0.60 = 0.03)$.

Time specific effects. Although Time 1 variables accounted for moderate variance in the Time 2 variables, there were also Time 2 specific genetic and environmental influences. Nonshared environmental factors and genetic factors unique to Time 2 respectively accounted for 35% and 24% of variance in Negative Emotionality. No Time 2 specific shared environmental contributions were found (0%). Nonshared environmental factors unique to Time 2 primarily accounted for the variance in Maternal Negativity (35%). Shared environmental factors unique to Time 2 explained an additional 16% of variance in Maternal Negativity. Genetic factors were trivial (0.9%).

Chapter 4: Discussion

Adolescence represents a key developmental period when the interplay between temperament and parenting may be most apparent and has implications for adolescents' adjustment. A cross-lagged biometric model was used to better understand associations between temperament and parenting across adolescence. Further, analyses explored the underlying child-based environmental and genetic factors accounting for these associations. Finally, the degree to which associations between negative emotionality and parenting differed for mothers and fathers was also explored.

Overall findings for mothers and fathers indicated that: (1) there is moderate stability in Negative Emotionality and Parental Negativity over time; (2) Negative Emotionality and Parental Negativity were associated with each other at each age and over time; and (3) genetic and environmental factors both contribute to variance in Negative Emotionality and Parental Negativity within each age examined, as well as to stability and change, and cross-lagged associations. Collectively, these findings underscore the importance of child-based genetic and environmental contributions to both adolescent temperament and parenting. Furthermore, these findings are indicative of bidirectional effects between Parental Negativity and Adolescent Negative Emotionality.

Although the author anticipated that environmental mechanisms would primarily account for the relationship between Parental Negativity at Time 1 and Adolescent Negative Emotionality at Time 2, only partial support for this hypothesis was found. The study's second hypothesis, that a significant cross-lagged effect between Parental Negativity and Adolescent Negative Emotionality would be explained primarily by child-based genetic factors, was fully supported. These findings will be discussed in more

detail.

How Much of the Variance in Parental Negativity and Negative Emotionality is Due to Genetic and Environmental Factors from Early to Later Adolescence?

Adolescent Negative Emotionality at Time 1 was primarily accounted for by genetic factors, consistent with prior temperament research based on twin samples (e.g., Goldsmith, Buss & Lemery, 1997; Plomin & Rowe, 1977; Rothbart & Bates, 1998) and as reported in previous publications with this sample that used single reporters of adolescents' temperament (e.g., Ganiban et al., 2008; Saudino, McGuire, Hetherington, Reiss, & Plomin, 1995). Specifically, genetic factors accounted for about two-thirds of the variance in Negative Emotionality, while nonshared environmental factors accounted for the remaining variance. Approximately three years later, at Time 2, there nonshared environmental influences on Adolescent Negative Emotionality appeared to increase, while genetic contributions decreased. By Time 2, genetic and nonshared environmental factors each accounted for approximately half of the variance in Adolescent Negative Emotionality. Parental Negativity showed a similar pattern. At Time 1 genetic and environmental factors accounted for nearly equal variance in Maternal and Paternal Negativity. However, genetic influences declined over time, while nonshared environmental influences increased. By Time 2, most variance in Parental Negativity was explained by environmental factors.

Patterns for both Adolescent Negative Emotionality and Parental Negativity suggest that adolescents' temperament and parents' negativity may be influenced more by the adolescents' unique experiences as they grow older. These findings are consistent with phenotypic studies that indicate increasing influences of unique academic and social

pressures on adolescents' emotional functioning and relationships with their parents (Harris, 1995) as they assume more responsibility in their life (Maccoby, 1992). Increases in nonshared environmental contributions to negative emotionality and parenting may also be influenced by changes in the extent to which an adolescent chooses to engage in the parent-adolescent relationship (Elkins, McGue, & Iacono, 1997). Adolescence is a developmental time period in which individuation occurs, and self-identity starts to coalesce (Erikson, 1968). Therefore, adolescents may assert their autonomy more strongly, and build and develop relationships outside of the family (Harris, 1995). In turn, this change may create more conflict and negative affect within the parent-child relationship. Further, an adolescent may express more negative affect in response to some common challenges during adolescence, including developing intimate relationships, pressure to engage in drug or alcohol use and hormonal changes (Harris, 1995).

How Do Parental Negativity and Adolescent Negative Emotionality at Time 1 Contribute to Parental Negativity and Adolescent Negative Emotionality at Time 2?

Maternal and Paternal Negativity were found to be moderately stable, in line with previous research focusing on early childhood (Larsson et al., 2008) and late adolescent/young adult twin samples (Moberg et al., 2010). Specifically, Maternal Negativity at Time 1 accounted for 36% of variance in Maternal Negativity at Time 2, and Paternal Negativity at Time 1 explained 31% of variance in Paternal Negativity at Time 2.

Stability in Maternal and Paternal Negativity was jointly accounted for by genetic and environmental factors, consistent with previous research with the NEAD sample (Reiss et al., 2000) and with other samples (Larsson et al., 2008; Moberg et al., 2010).

These findings suggest that genetic and environmental factors that are influencing a parent's tendency to react to their teenagers with anger and hostility at Time 1 may continue to do so over time. For example, environmental factors such as interparental conflict affect parenting at Time 1, and these effects may persist through Time 2. Similarly, genetic factors that affect parenting at Time 1 continue to do so at Time 2. Within the current study, stability estimates for Parental Negativity controlled for the impact of Adolescent Negative Emotionality. Therefore, observed stability in parental negativity could not be explained by stable child negative emotionality. However, other genetically influenced characteristics such as antisocial behavior or poor self-control may have contributed to stability in Parental Negativity over time (Moffitt, 2005). For example, an adolescent who displays antisocial behaviors may evoke Parental Negativity in early and later adolescence. Previous research has found that antisocial behaviors are stable across adolescence and may elicit negative parenting (Larsson et al., 2008; O'Connor et al., 1998).

Changes in Maternal and Paternal Negativity from Time 1 to Time 2 were partially explained by adolescents' Negative Emotionality at Time 1. Higher adolescent Negative Emotionality at Time 1 was associated with more Maternal and Paternal Negativity at Time 2. This effect was primarily driven by child-based genetic factors associated with Negative Emotionality at Time 1. This finding is consistent with child-based evocative effects on parenting. A genetically influenced characteristic, Adolescent Negative Emotionality, predicted more Parental Negativity over time. This pattern of results may mean that a child who has difficulty regulating his or her emotions may elicit a more hostile response from his or her parent.

Similar to Parental Negativity, adolescents' Negative Emotionality was also moderately stable over time. However, this stability was primarily explained by genetic factors. This pattern of findings is consistent with previous research suggesting that Negative Emotionality has a genetic basis, and is relatively stable over time (Bouchard & Loehlin, 2001; Caspi, Roberts, & Shiner, 2005; Ganiban, Saudino, Ulbricht, Neiderhiser & Reiss, 2008; Gillespie et al., 2004; McGue, Bacon, & Lykken, 1993; Roberts & DelVecchio, 2000; Viken et al., 1994). There were also changes in Negative Emotionality over time, and Parental Negativity at Time 1 contributed to these changes, even after controlling for their prior relationship at Time 1. Both environmental and child-based genetic factors associated with Parental Negativity at Time 1 were related to Time 2 Negative Emotionality. It is possible that environmental factors present at or prior to Time 1 such as the attachment history between parent and child may have influenced the adolescent's negative emotionality. Consistent with attachment theory, harshness, hostility, and insensitivity can negatively affect a child's development of healthy emotional regulation (Shaver & Mikulincer, 2007). Therefore, adolescents with an insecure attachment history may be more likely to experience and express dysregulated anxiety, distress, and anger (Mikulincer, 1998). The impact of genetic factors being mediated by parenting may suggest that parenting is reinforcing the expression of Negative Emotionality. On the other hand, a child's genetic characteristics may have elicited more negative parenting at Time 1, which in turn, may sustain, reinforce, exacerbate, or evoke child behaviors, further shaping the initial expressed characteristic (Rothbart & Bates, 1998; Sanson & Rothbart, 1995).

Overall, this pattern of findings for both cross-lags supports a reciprocal model between parent and child. Although it was anticipated that environmental mechanisms would primarily account for the relationship between Parental Negativity at Time 1 and Adolescent Negative Emotionality at Time 2, this effect was jointly explained by genetic and environmental factors. Consistent with child evocative effects model, it was also hypothesized that a significant cross-lagged effect between Parental Negativity and Adolescent Negative Emotionality would be explained primarily by child-based genetic factors related to Time 1 Negative emotionality. Even though most of this effect was explained by genetic factors, environmental factors also played a role. Therefore, when considered together, both cross-lagged effects indicate that adolescent negative emotionality and parent negativity influence each other over time and their effects are driven by the adolescents' genetic makeup and by a combination of family wide factors (shared environment) and the adolescents' unique experiences.

How Do Genetic and Environmental Factors Influence the Age-Specific Covariance Between Parental Negativity and Adolescent Negative Emotionality?

For mothers and fathers, genetic factors solely accounted for the correlation between Parental Negativity and Adolescent Negative Emotionality at Time 1 (r_{a12}= .53 - .55). These findings suggest that the same set of genes that affect Negative Emotionality also account for variance in Parental Negativity. As described previously, children's genetically influenced tendency to express negative affect may have elicited more negative parental responses (Eisenberg, 1999; Ganiban et al., 2011). At Time 2, Adolescent Negative Emotionality and Parental Negativity were also correlated for both mothers and fathers. For mothers, common genetic effects continued to account for the

entire correlation between Maternal Negativity and Adolescent Negative Emotionality. In contrast, for fathers, covariance at Time 2 was solely explained by nonshared environmental factors. This suggests that in later adolescence, the adolescent-father relationship may not be as child-driven as during early adolescence. Rather, a common set of environmental factors appeared to affect children's negative emotionality and their fathers' negative parenting.

These findings are consistent with prior research suggesting that contextual stressors such as marital stress may have greater influence on fathers' parenting than mothers' parenting (Reiss, Neiderhiser, Hetherington, & Plomin, 2000). For example, Ulbricht and colleagues (2013), using the same sample as the current study, found that when there is high marital conflict, mothers become more responsive to their child's genetically influenced characteristics. In contrast, environmental influences on parenting increased for fathers. In the current study, environmental factors such as marital conflict might have had a simultaneous effect on fathers' and adolescents' negativity. Alternatively, adolescents' new relationships outside of the family system could also have had an impact on the adolescents' behavior and fathers' behaviors. For example, as teenagers start to individuate from their families, they may seek new peer groups and express more negative affect and conflict with their parents. Fathers may not approve of their teenagers attempts to become more autonomous, or appreciate their selection of friends, and act more negatively to their children.

Of note, mothers were generally the primary caregivers and may have had more direct contact with the children. A more involved mother may have more opportunities to be influenced by their child's behavior, and this may partially explain a mother's

tendency to be more in tune with and influenced by her child's genetically influenced characteristics across adolescence. However, other studies, using more contemporary samples, have yielded similar patterns of genetic and environmental contributions to parenting (Narusyte et al., 2008), and suggest that mothers are more responsive to children's genetically influenced characteristics than fathers (e.g., Narusyte et al., 2011).

Is There Evidence for Differences in the Maternal and Paternal Model?

The results from our model fitting analyses revealed similar findings for Maternal and Paternal Negativity. Notably, the cross-age stability, cross lags, and unique and joint genetic and environmental contributions at Time 1 followed qualitatively similar patterns for mothers and fathers. In contrast, at Time 2, genetic and environmental contributions to negative parenting varied for mothers and fathers: children's genetic makeup accounted for more variance in mothers' parenting than fathers' parenting. This pattern is consistent with previous studies that suggest that father's parenting is more influenced by environmental factors than mothers' parenting (Ulbricht et al., 2013). However, the current analyses suggest that this effect may emerge in later adolescence.

Environmental factors that explain the differences in the maternal and paternal models at Time 2 may not be as apparent at Time 1 and may explain the initial similarities. For example, Time 1 analyses may have captured the parent-child dynamic before outside influences, such as peer group, became more important. When the child was younger, mothers and fathers parenting was more responsive to the child's genetically-influenced characteristics. In later adolescence, influences outside of the home, and the emergence of individuation and self-exploration, may begin to affect mothers and fathers parenting differently.

Clinical Implications

The findings highlight the importance of both the parents' and child's role in their relationship during adolescence, and the bidirectional effects of Negative Parenting and Adolescent Negative Emotionality. This reciprocal process between Negative Parenting and Negative Emotionality may reflect a coercive family process that has gradually emerged over time (Patterson, 2002; Scaramella & Leve, 2004). Prior research suggests that parenting styles, and the goodness-of fit between parent and child, can influence the development of temperament characteristics (Rettew, Stanger, McKee, Doyle & Hudziak, 2006; van der Bruggen, Stams, Bogels & Paulussen-Hoogeboom, 2010), consistent with these findings. Conversely, findings also indicate that children are not passive receivers of environmental influences – they can affect their environments, including the parenting that they receive. Thus, clinical interventions should target both the parent and the adolescent.

For parent interventions, two types of clinical education may be beneficial. First, it would be important to provide parents with validation and an enhanced understanding of the possible evocative effects of their children's behaviors. Second, parents should also be aware of how their own behaviors may further reinforce their children's negative emotionality. For example, though an evocative model suggests that adolescents' temperament is evoking parenting behaviors, some parents may be more reactive than others. Providing education to parents about their own reactivity may provide an opportunity for parents to modulate their own natural responses to negative emotionality. Additionally, if parents are educated about their adolescent's predispositions, they may be able to better modulate their own parenting styles to better support their child's

strengths, and to foster effective regulation of negative emotions. Indeed, mothers who are able to show sensitivity toward their child during distress may help him or her to maintain more optimal levels of positive emotionality and to learn more appropriate emotion regulation strategies (Eisenberg, Cumberland, & Spinrad, 1998; Rothbart & Bates, 1998).

Further, family interventions targeting the parent-adolescent relationship should be considered. Given that environmental factors partially explain the relationship between Negative Parenting and Negative Emotionality, the quality of the child-parent relationship may be an important consideration and the attachment history may be an important target in a therapeutic setting. During adolescence, the attachment relationship between the parent and child could continue to impact and potentially exacerbate negative emotionality. Similarly, the behaviors of the adolescent may continue to impact and potentially elicit parental negativity. Treatments targeting the parent-child relationship are more commonly found for younger children (e.g., Parent-Child Interaction Therapy (PCIT); Zisser & Eyberg, 2010; Incredible Years; Webster-Stratton & Reid, 2002) or target more externalizing, antisocial behaviors (e.g., Parent-Management Training (PMT); Forgatch & Patterson, 2005). These interventions operate under the principle that a solid foundation between the parent and child, created through parent praise, positive reinforcement, and parent-child collaborative time, is essential in order to develop healthy interactions and decrease conflict. Perhaps then, in adolescence, these same principles can be applied in order to alter a family dynamic characterized by conflict.

The findings further indicate some nuanced differences in the father-child and mother-child relationship. Clinicians can make efforts to help fathers be aware of possible environmental and contextual stresses that may be contributing to their own parenting and their responses to their adolescent. Some research suggests that when adolescents are reactive, fathers have a tendency to disengage from the relationship (Stoneman, Gavidla–Payne, & Floyd, 2006; Woodworth, Belsky, & Crnic, 1996). Coupled with the current findings, this implies that in later adolescence, fathers should be encouraged to be aware of any patterns of increasing distance in their relationship with their child.

The current study focused on negative emotionality due to its link to adolescent adjustment and depression. Given the stability in negative emotionality, one may assume that intervening early to help children cope with fear, anger, and reactivity would be especially important. Given that emotional dysregulation is an inherent component of depression (Kochanska, 1993; Rothbart, Ahadi, & Hershey, 1994), strengthening a child's self-regulatory skills could be important to modulate his or her temperamental reactivity (Derryberry & Reed, 1998; Derryberry & Rothbart, 1997; Rothbart, Ahadi & Hershey, 1994; Rothbart, Ellis, Rueda & Posner, 2003). Thus, successful early interventions should focus on developing emotion regulation skills and coping strategies. Specifically, targeting effortful control may prove to be an effective intervention, as it has been shown to moderate the known association between negative emotionality and depressive symptoms (Bernier, Carlson, & Whipple, 2010; Eisenberg, et al., 2003; Eisenberg, et al., 2009; Fonagy & Target, 2002; Rothbart, Ellis, Rueda & Posner, 2003). Consideration of possible moderators that influence the relationship between negative

emotionality and depression could be critically important, as measuring negative emotionality independent of other characteristics is insufficient. As an example, consider the adolescent with high negative emotionality coupled with low self-regulatory skills; an adolescent with this combination of temperamental characteristics is likely at much greater risk for the development of psychopathology when contrasted to an adolescent with equivalent levels of negative emotionality, yet with high self-regulatory skills (Rothbart & Bates, 1998).

The relative proportion of negative emotionality and effortful control, and how they interact with one another, will be an important avenue to continue to explore to better inform the prevention of adolescent depression. Given that adolescence can be an important period – one characterized by heightened emotionality (Collins & Steinberg, 2006; Larson & Lampman-Petraitis, 1989) and an increase in depressive symptoms (Krueger, Caspi, & Moffitt, 2000; Newman, et al., 1996) – underscores the importance of continued research to identify youth at risk for depression and the implementation of early interventions prior to adolescence.

Limitations and Future Directions

The participants in our sample were not diverse (e.g., middle to high SES, mostly Caucasian, two parent families) and relatively mentally healthy which limits generalizability. Due to low diversity, our findings may not extend to other cultures. For example, in cultures in which adolescence is not a period of individuation, one might conjecture that nonshared environmental influences may not increase as significantly in later adolescence. Further, the results may not generalize to families that are newly divorced, remarried, or those with single parents. In these family structures, or in

environments where there are generally more stressors (e.g., low SES, marital conflict), environmental influences may be more apparent.

The NEAD sample included a higher rate of remarried families compared to the general population. Remarried families with step- or half-siblings likely encompass even more diverse family dynamics. Future studies could compare the relationship between adolescent negative emotionality and parenting in other samples since parents may differentially react to biological and non-biological children.

Additionally, the current study may not capture the dynamic nature of the family system. The present model focused on the mother- and father-child relationship separately, when in fact, both relationships play a role contemporaneously (Minuchin, 1985). More intricate family systems may be playing a role as well (e.g., sibling relationships or parent-sibling relationships) and could influence the child (Minuchin, 1985). Future research on the development of adolescent temperament should consider incorporating a family system perspective in which more complex family dynamics are explored.

While the current study found significant bidirectional cross-lagged effects between Parental Negativity and Adolescent Negative Emotionality, the effects were quite modest (i.e., .002-.005). The significant within age correlations between Parental Negativity and Adolescent Negative Emotionality at Time 1 speak to the probability that their association likely developed earlier in the parent-child relationship. Collecting data from earlier time points would allow the study to understand several additional inquiries. First, it would allow researchers to more accurately detect the important period during which the relationship between Parental Negativity and Adolescent Negative

Emotionality is first noticed, and provide further evidence for early intervention. Second, although in the current model we do not have three time points to fully illustrate a coercive model, the partial underlying genetic factors of the parent-driven model indicate that the child's genotype has already elicited the Parental Negativity. Assessing additional time points would allow researchers to properly document this coercive cycle, as has been described in previous literature (Patterson, 2002; Scaramella & Leve, 2004).

Despite the need for further research, the current study begins to document the mutual influences of parenting and temperament over time, and extends the literature by directly examining the etiology of this bidirectional association. The current study allows for clarity in the genetic and environmental underpinnings of the complex relationship between parenting and the development of temperament that phenotypic studies are limited in addressing. The findings speak to the importance of the parent-child relationship during adolescence, and the need to help the family navigate this important period. Moreover, the findings hint at a coercive cycle between parenting and negative emotionality that may be instigated prior to adolescence, implicating the need for earlier intervention. Indeed, early interventions should be created to enrich and enhance both child and parent protective factors. Given the documented associations between parent-child conflict, negative emotionality and psychopathology, early intervention is critical to help redirect developmental trajectories.

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Table 1

Phenotypic Correlations Among Study Variables

	1	2	3	4	5	6
Time 1						
1. Negative Emotionality	_					
2. Maternal Negativity	.32	_				
3. Paternal Negativity	.32	.62	_			
Time 2						
4. Negative Emotionality	.65	.31	.23	_		
5. Maternal Negativity	.32	.65	.45	.37	_	
6. Paternal Negativity	.29	.46	.62	.33	.60	_

Note. All correlations significant for $p < .0001$. All variables were standardized to have a mean of 0 and a standard deviation of 1.0

Table 2

Cross-Sibling Intraclass Correlations Among Study Variables

	MZ	DZ	FI	FS	HS	US
Time 1						
Maternal Negativity	0.78	0.51	0.47	0.50	0.54	0.14
Paternal Negativity	0.74	0.64	0.43	0.41	0.40	0.44
Negative Emotionality	0.66	0.29	0.29	0.06	0.04	- 0.16
Time 2						
Maternal Negativity	0.67	0.61	0.47	0.39	0.37	0.23
Paternal Negativity	0.66	0.67	0.26	0.43	0.25	0.37
Negative Emotionality	0.62	0.02	0.14	0.09	0.06	- 0.12

Note. MZ = Monozygotic Twins; DZ = Dizygotic Twins; FI = Full Siblings from non-divorced families; FS = Full Siblings from divorced families; HS = Half-Siblings; and US = Unrelated Siblings.

Table 3

Model Fit for Adolescent Negative Emotionality and Paternal and Maternal Negativity

Model Fitting Paternal Negativity	DF	ΔDF	-2LL	Δ-2LL	Adjusted BIC
ACE Full	4253	--	10804.88	--	-1792.79
Trimmed Model: Set paths to 0: r_{c12}, r_{c34}, c_1^2, c_3^2, r_{e12}, r_{a34}, a_4^2	4264	7	10796.53	8.35	-1800.47

Model Fitting Maternal Negativity	DF	ΔDF	-2LL	Δ-2LL	Adjusted BIC
ACE Full	4260	--	10785.05	--	-1822.24
Trimmed Model: Set paths to 0: r_{c12}, r_{c34}, r_{e12}, r_{e34}	4264	4	10793.53	8.48	-1824.77

Table 4

Genetic and Environmental Variance at Time 1 and Time 2: Adolescent Negative Emotionality and Paternal Negativity

	Time 1		**Time 2**		**Time 2 Specific Effects**							
	NE	**PN**	**NE**	**PN**	**Negative Emotionality (NE)**				**Paternal Negativity (PN)**			
Variance Components	Total Variance	Total Variance	Total Variance	Total Variance	Stability Variance	Unique Variance	Cross-Lag Variance	Common Variance	Stability Variance	Unique Variance	Cross-Lag Variance	Common Variance
A	.62	.46	.53	.18	.23	.25	.006	.04	.14	0	.005	.03
C	0	.32	.004	.31	0	0	.004	0	.10	.21	0	0
E	.38	.22	.47	.50	.14	.33	.003	0	.07	.43	.003	0

Note. NE = Negative Emotionality; PN = Paternal Negativity; A = Additive Genetic, C = Shared Environment and E = Non-shared Environment. Paths correspond to those depicted in Figure 2.

Table 5

Genetic and Environmental Variance at Time 1 and Time 2: Adolescent Negative Emotionality and Maternal Negativity

	Time 1		**Time 2**		**Time 2 Specific Effects**							
	NE	**PN**	**NE**	**PN**	**Negative Emotionality (NE)**				**Paternal Negativity (PN)**			
Variance Components	Total Variance	Total Variance	Total Variance	Total Variance	Stability Variance	Unique Variance	Cross-Lag Variance	Common Variance	Stability Variance	Unique Variance	Cross-Lag Variance	Common Variance
A	.64	.55	.52	.32	.24	.24	.004	.03	.20	.09	.004	.03
C	0	.24	.002	.25	0	0	.002	0	.09	.16	0	0
E	.36	.20	.49	.42	.14	.35	.002	0	.07	.35	.002	0

Note. NE = Negative Emotionality; MN = Maternal Negativity; A = Additive Genetic, C = Shared Environment and E = Non-shared Environment. Paths correspond to those depicted in Figure 2.

Figure 1

Biometric Cross-Lagged Model

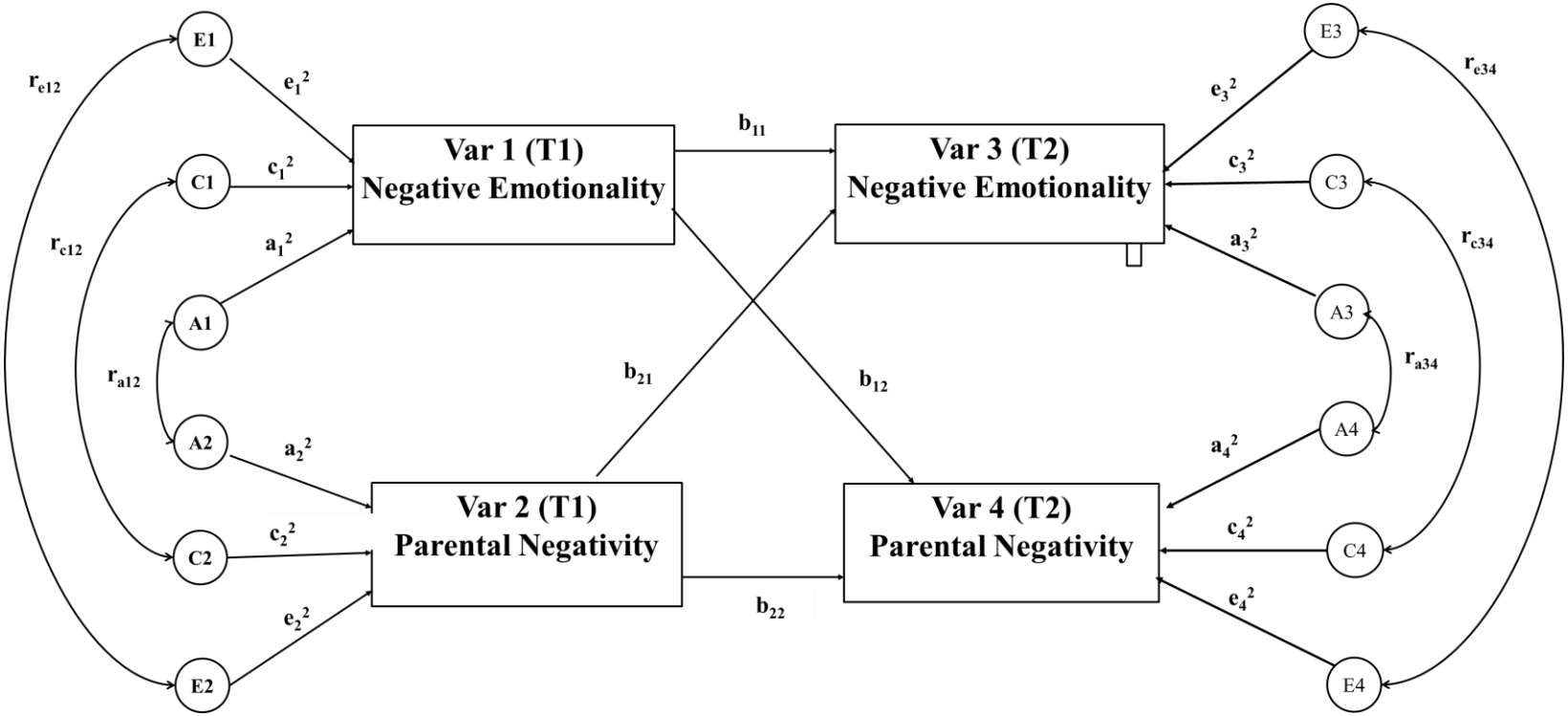

Figure 1. Path diagram of a biometric cross-lagged model. Parental negativity and negative emotionality are the measured variables, while circular shapes represent A (additive genetic), C (shared environment) and E (non-shared environment). Included in the model are also standardized path estimates for A, C and E ($a_1, c_1, e_1, a_2, c_2, e_2, a_3, c_3, e_3, a_4, c_4, e_4$), their correlations ($r_{a12}, r_{c12}, r_{e12}, r_{a34}, r_{c34}, r_{e34}$), two cross-age parameters (b_{11}, b_{22}), and two cross-lagged parameters (b_{12}, b_{21}).

Figure 2

Biometric Cross-Lagged Model: Adolescent Negative Emotionality and Paternal Negativity

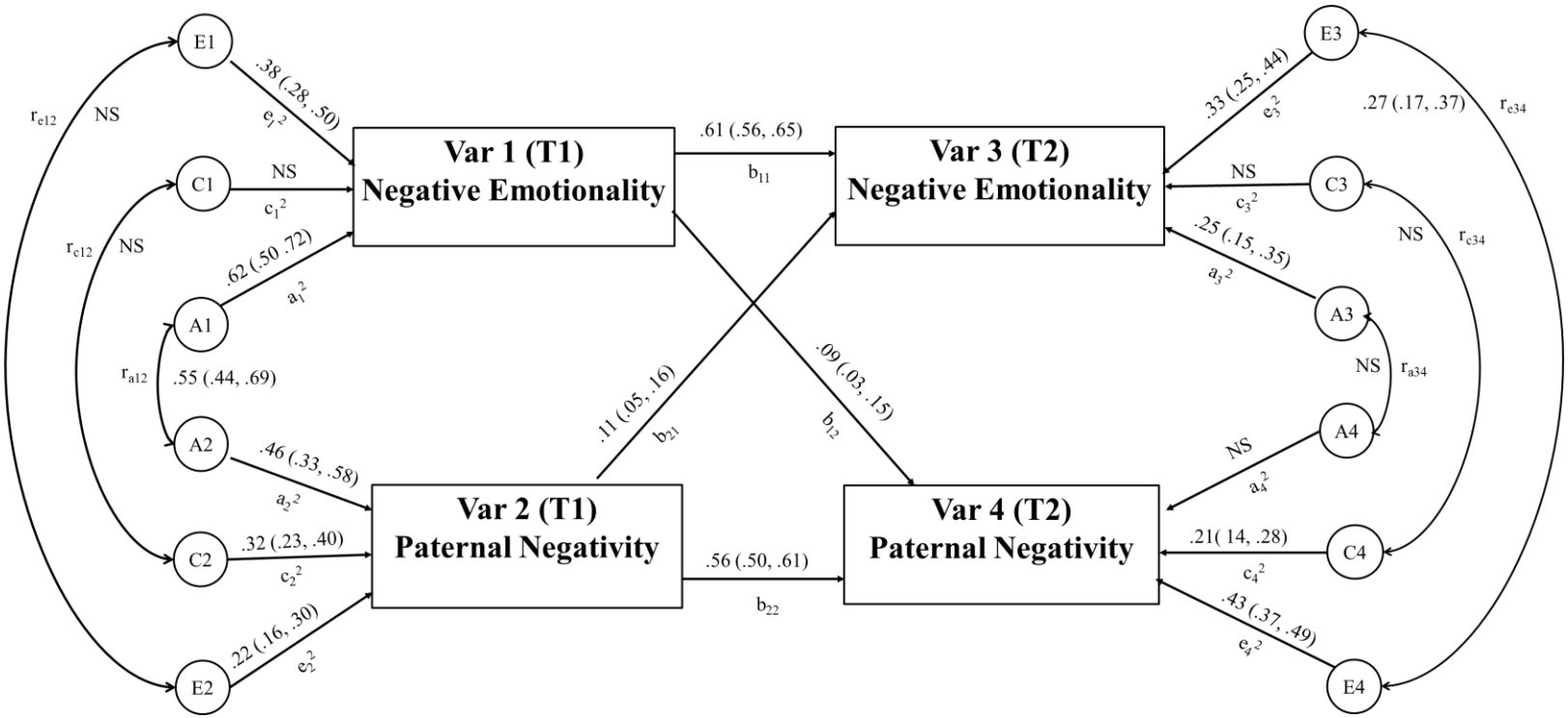

Figure 2. Path diagram of a biometric cross-lagged model. Paternal negativity and Adolescent Negative Emotionality are the measured variables, while circular shapes represent A (additive genetic), C (shared environment) and E (non-shared environment). Included in the model are also standardized path estimates for A, C and E (a_1, c_1, e_1, a_2, c_2, e_2, a_3, c_3, e_3, a_4, c_4, e_4), their correlations (r_{a12}, r_{c12}, r_{e12}, r_{a34}, r_{c34}, r_{e34}), two cross-age parameters (b_{11}, b_{22}), and two cross-lagged parameters (b_{12}, b_{21}). Unless noted as "NS", all paths are significant at the $p<.05$ level.

Figure 3

Biometric Cross-Lagged Model: Adolescent Negative Emotionality and Maternal Negativity

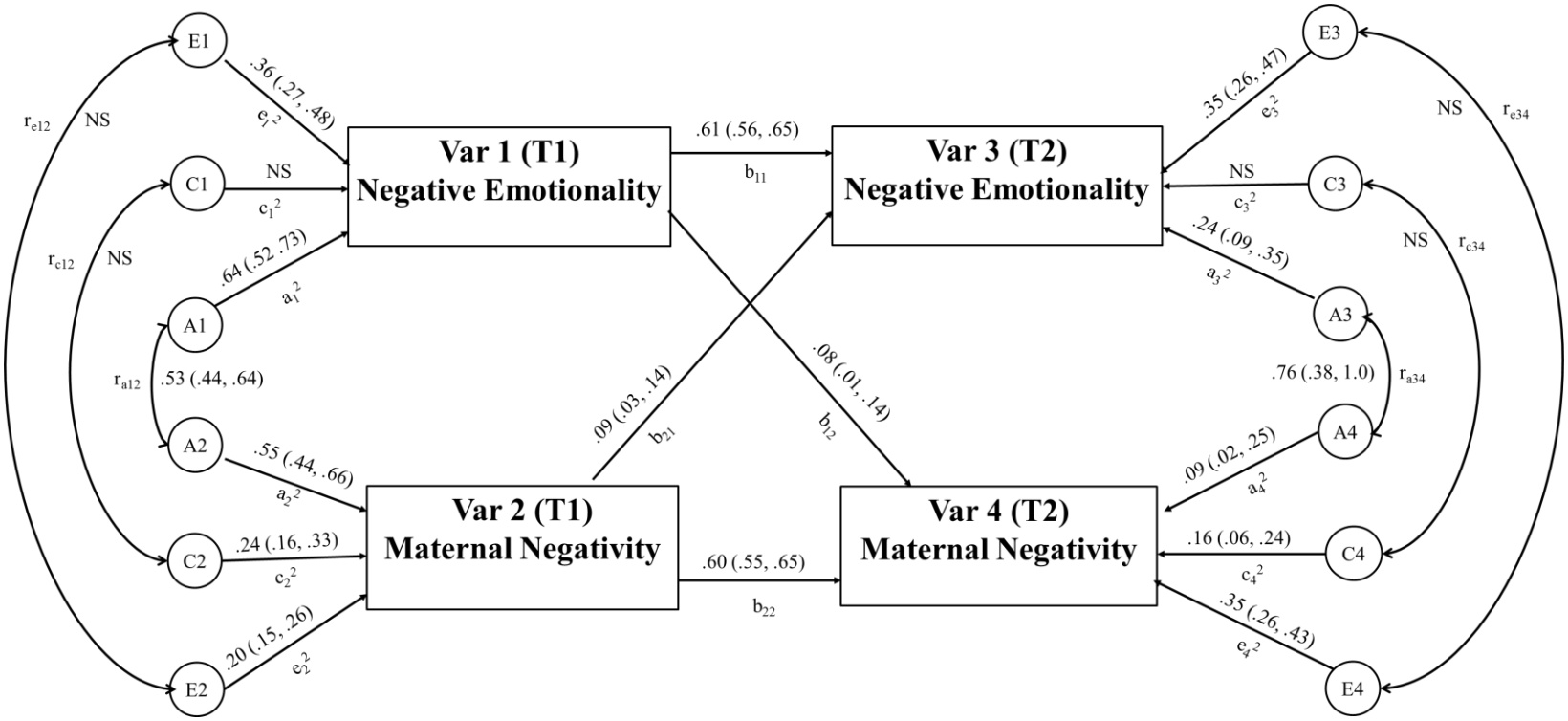

Figure 3. Path diagram of a biometric cross-lagged model. Maternal negativity and Adolescent Negative Emotionality are the measured variables, while circular shapes represent A (additive genetic), C (shared environment) and E (non-shared environment). Included in the model are also standardized path estimates for A, C and E (a_1, c_1, e_1, a_2, c_2, e_2, a_3, c_3, e_3, a_4, c_4, e_4), their correlations (r_{a12}, r_{c12}, r_{e12}, r_{a34}, r_{c34}, r_{e34}), two cross-age parameters (b_{11}, b_{22}), and two cross-lagged parameters (b_{12}, b_{21}). Unless noted as "NS", all paths are significant at the $p<.05$ level.