Understanding Behavior Problems and Competencies across Childhood through the Contributions of Parental Warmth and Rejection and Dopamine, Vasopressin, and Neuropeptide-Y Genes

by

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ABSTRACT

Externalizing behaviors are pervasive, widespread, and disruptive across a multitude of settings and developmental contexts. While the conventional diathesis-stress model typically measures the disordered end of the spectrum, studies that span the range of behavior, from externalizing to competence behaviors, are necessary to see the full picture. To that end, this study examined the additive and nonadditive relations of a dimension of parenting (ranging from warm to rejecting), and variants in dopamine, vasopressin, and neuropeptide-y receptor genes on externalizing/competence in a large sample of predominantly Caucasian twin children in toddlerhood, middle childhood, and early adolescence. Variants within each gene were hypothesized to increase biological susceptibility to both negative and positive environments.

Consistent with prediction, warmth related to lower externalizing/higher competence at all ages. Earlier levels of externalizing/competence washed out the effect of parental warmth on future externalizing/competence with the exception of father warmth in toddlerhood marginally predicting change in externalizing/competence from toddlerhood to middle childhood. Warmth was a significant moderator of the heritability of behavior in middle childhood and early adolescence such that behavior was less heritable (mother report) and more heritable (father report) in low warmth environments. Interactions with warmth and the dopamine and vasopressin genes in middle childhood and early adolescence emphasize the moderational role gene variants play in relations between the rearing environment and child behavior. For dopamine, the long variant related to increased sensitivity to parent warmth such that

the children displayed more externalizing behaviors when exposed to rejection but they also displayed more competence behaviors when exposed to high warmth. Vasopressin moderation was only present under conditions of parental warmth, not rejection. Interactions with neuropeptide-y and warmth were not significant. The picture that emerges is one of gene-environment interplay, wherein the influence of both parenting and child genotype each depend on the level of the other. As genetic research moves forward, gene variants previously implicated as conferring risk for disorder should be reexamined in conjunction with salient aspects of the environment on the full range of the behavioral outcome of interest.

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"Nobody trips over mountains. It is the small pebble that causes you to stumble. Pass all the pebbles in your path and you will find you have crossed the mountain." – Author Unknown

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Introduction

Children face many challenges to successful maturation. Children's behavioral outcomes are one arena in which development is measured. A considerable percentage of mothers of toddlers (5-13%) report moderate to severe externalizing behaviors in their children (Campbell, Shaw & Gilliom, 2000), with the umbrella of externalizing behaviors encompassing aggressive, defiant, and delinquent behavior. As many as 10-15% of preschoolers exhibit clinical or subclinical behavior problems (Briggs-Gowan, Carter, Skuban, & Horwitz, 2001; Campbell, 1995), and over 21% of children age 9 to 17 contend with some form of mental disorder (Surgeon General Report, Satcher, 1999). Additionally, in a large longitudinal community sample, 13.3% of children had a diagnosed mental disorder at any one measurement point across middle childhood, while almost 40% of children experienced at least one or more disorders over the eight year duration of the study (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003).

The purpose of the current study was to examine the etiology of externalizing behaviors and behavioral competence in a twin study that spans across three time points, from toddlerhood to middle childhood to middle adolescence. Parental warmth (and conversely, parental rejection) is examined both as a predictor and as a moderator of the heritability of behavioral outcomes. Additionally, hypothesized plasticity gene variants are considered as moderators of the phenotypic relations between parental warmth/rejection and behavioral outcomes.

Although externalizing, internalizing, and Attention Deficit Hyperactivity Disorder (ADHD) are comorbid to some degree (Lilienfeld, 2003), their genetic etiology is partially independent and they show different patterns of prediction (Krueger, 1999). The focus of this paper is on externalizing behaviors and, on the other end of the spectrum, behavioral competence (i.e., adaptive social skills and prosocial behaviors). Externalizing behaviors are particularly disruptive across multiple settings and early-onset externalizing behaviors are particularly concerning, as such behaviors have been shown to strongly predict later antisocial outcomes (Moffit, 1993). If allowed to remain unchecked, externalizing behaviors have lasting effects, including early onset substance use (King, Iacono, & McGue, 2004) and impaired social functioning in adulthood (Bongers, Koot, van der Ende, & Verhulst, 2008). Given the societal implications of life-course deviant behavior, the study of the developmental progression of externalizing behaviors, and factors that can alleviate or moderate negative outcomes is of particular importance.

Historically, more attention has been paid to behavioral deficits and disordered outcomes than to behavioral competence. Indeed, across the board, psychological research often focuses on the negative while failing to measure the positive (Belsky & Pluess, 2009). Positive psychology and resilience research have successfully demonstrated the added benefits of studying positive adaptation (Masten & Obradovic, 2006; Moffitt, 2005; Vanderbilt-Adriance & Shaw, 2008). There is considerable stability in the successful handling of developmental tasks across ages (Masten et al., 2004), and research has shown three types of competence in late childhood (ages 8-12):

academic, conduct (both at home and in school), and social (Masten et al., 1995) that can serve as a contrast to externalizing behaviors.

The need for a continuum of behavioral outcomes (ranging from positive to negative) and parenting (ranging from warm to rejecting) is informed by the differential susceptibility hypothesis (Belsky, 1997; Belsky & Pluess, 2009). The differential susceptibility model has shown utility as an alternative to the diathesis-stress model, which presupposes that certain individuals are inherently more susceptible to adversity, and, when exposed to a stressful situation, are then more likely to experience negative outcomes. This diathesis-stress framework initially informed genetic research, resulting in the search for risk gene variants. Belsky's differential susceptibility framework, on the other hand, emphasizes that characteristics internal to the individual result in increased susceptibility to environmental effects, both beneficially and detrimentally. The differential susceptibility hypothesis is supported when individuals with one genotype (or characteristic) are both more at-risk for negative outcomes when exposed to a negative experience and more competent when exposed to a positive experience.

Evolutionary theory supports the differential susceptibility hypothesis (Belsky, 1997; Belsky & Pluess, 2009). Natural selection (the process through which biological traits become more or less common by way of their influence on reproductive fitness) should act to preserve genetic variation in a population to ensure the survival of some across dynamic environments (Orr, 2009). Beyond maintaining basic variation in behavior, evolution is hypothesized to maintain variation in susceptibility to environmental

influences (Belsky, 1997). In advantaged environments, susceptibility offers an advantage to fitness. In disadvantaged environments, less susceptibility may be an advantage, although one can argue that susceptibility may still provide an advantage (Belsky, 1997). If a negative or disadvantaged environment leads susceptible individuals towards externalizing behaviors, this may better prepare them to fight for what they need in order to survive. For example, among the Masai tribe of East Africa, infants with a difficult temperament (indexed by higher levels of fussiness and crying) were more likely to survive during a famine (DeVries, 1984). Infants with difficult temperaments were thought to survive at better rates because their behaviors increased the likelihood of being fed. To test the differential susceptibility hypothesis, constructs must be measured in a way that spans the full range of experiences and outcomes.

Human adaptational systems (which regulate responses to environments) are influenced by genetic variation in individuals. In fact, genes underlie all behavioral development (Lykken, Bouchard, McGue & Tellegen, 1993; Plomin & Rutter, 1998; Turkheimer & Gottsman, 1996). According to Pepper's (1961) world view framework, behavior genetic theory falls under the mechanist world view alongside other popular theories including learning theory, social cognitive theory, and information processing theory. Mechanist theories recognize two main causes of behavior; efficient causes that are external to the individual and material causes that are internal components of the individual. Contemporary research also takes into account what Pepper

called formal causes – those which address the interaction of parts of the system.

The current wave of genetically-informed research accepts the importance of both genes and the environment for behavior and development, embraces measured gene studies, and focuses on multifactorial origins for behaviors (Deater-Deckard et al., 2005; Rutter et al., 2006). Individual differences across development are generally attributed to a combination of gene-gene, environment-environment, or gene-environment interplay, with variability in both genes and environments potentially having additional independent main effects (Shanahan & Hofer, 2005).

A gene-environment interaction is a measure of how genetic factors moderate the impact of an individual's environment. Certain genetic factors can predispose one to be more or less susceptible to environments that lead to externalizing problems. In addition to the gene-environment interactions and main effects examined in this study, it is important to note the potential for epigenetic influences (experiences that alter the expression of an individual's genes), and gene-environment correlation (Deater-Deckard et al., 2005). With gene-environment correlation, genetic factors influence the likelihood that one experiences an environment expected to contribute to externalizing problems. Different study designs can explicitly test these various types of gene-environment interplay.

Quantitative behavior genetic methodology compares groups of individuals that share genes to different extents (e.g., identical and fraternal twins, full and half siblings) which disentangles genetic and environmental

influences on outcomes (Moffitt, 2005). Heritability estimates represent the proportion of variance in the behavior, or phenotype that can be attributed to genetic variance in the population (Neale & Cardon, 1992). The current study employs the most common behavior genetic model, which includes monozygotic (identical; MZ) and dizygotic (fraternal; DZ) twins who are genetically related 100% and roughly 50%, respectively. In the classic twin study, a quick estimate of heritability is twice the difference between MZ and DZ twins ($h^2 = 2[r_{mz} - r_{dz}]$; Falconer & MacKay, 1996).

More sophisticated behavior genetic models build upon the ACE model (otherwise known as a univariate Cholesky model), which parses phenotypic variance into additive genetic (A), shared environment (C) and nonshared/unique environment (E) components. A, C and E are latent, unmeasured constructs. Additive genetic influences are genetic contributions that additively influence a given trait or behavior. Shared environment influences are aspects of the environment that increase similarity between individuals. Nonshared environment influences are aspects of the environment that decrease similarity between individuals, whether by differential treatment (actual or perceived) or different experiences (e.g., different classrooms, friends). Nonshared environment influences also encompass measurement error in quantitative genetic models (Nagoshi, 1994).

Non-genetically informed studies can lead researchers to conflate sources of variance and reach incorrect suppositions with regards to direction and magnitude of effect. Variance in commonly-used measures of the environment

(such as parenting) is partially attributable to genetic factors. Beyond establishing heritability estimates, the behavior genetic framework allows for the testing of process models which further our understanding of the ways in which genes and environments cause stability and change over time and interact to influence behavior.

There is also an important distinction to be made between the C component in a standard variance decomposition model and the C component in a model that includes a predictor or moderator with both environmental and genetic components. Parental warmth, considered in this study as a moderator of the heritability of externalizing and competence, conflates influences of the child's environment with genes shared between the parent and child (Deater-Deckard, Fulker, & Plomin, 1999). As a result, the C component in an ACE model with parenting included is no longer a clean partition of the influences of the environment (South, Krueger, Johnson, & Iacono, 2008). For the sake of specificity, when referring to ACE models including parental warmth, I will use the term *experience* rather than *environment*.

Behavior problems are an ideal outcome for a genetically-informed investigation because of the extensive research into their etiology (Lemery & Doelger, 2005; Miles & Carey, 1997). Externalizing problems have fairly high rank-order stability across childhood (Campbell, Pierce, Moore, Marakovitz, & Newby, 1996), although mean levels of externalizing decrease over middle childhood (Campbell, 1995). The predictive nature of externalizing behaviors is evidenced very early on, with externalizing behaviors in five year olds

predicting mother and teacher report of externalizing behaviors at age seven and ten (Denham et al., 2000). Early externalizing is one of the strongest predictors of later antisocial behavior (White, Moffitt, Earls, Robins, & Silva, 1990).

Heritability estimates of externalizing have a broad range. For example, heritability in middle childhood has been estimated at 0.35 (Burt, McGue, Krueger, & Iacono, 2005) and at .60 in adolescence (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). Genetic influences tend to increase with child age (Lemery & Doelger, 2005; McGue, Bouchard, Iacono, & Lykken, 1993). Nonshared environmental influences are significant, but shared environmental influences tend to be weak, especially among older children (McGue et al., 1993). A common vulnerability factor with a heritability of 0.80 has been found to underlie externalizing disorders (e.g., conduct disorder, alcohol dependence, and drug dependence) in adolescence (Hicks et al., 2004).

In addition to shifting with age of the child, heritability estimates may differ for mother and father reports of externalizing behaviors. Mothers often have more frequent interactions with children, and so may be reporting on more heritable behavior that is averaged across many contexts, while fathers may see more situationally-driven behavior. Mother report of externalizing does yield higher heritability estimates than that of other reporters (Eaves et al., 1997). A study of parent ratings of child externalizing concluded that although parents observe and report with considerable similarity, there is an additional component that is unique to each parent contributing 4-14% of the genetic variance (van der Valk, van den Oord, Verhulst, & Boomsma, 2003).

This study attributed the most variance to the genetic component for boys and girls with both mother and father report, but also included a modest shared environmental component at seven years of age.

Last, research on externalizing must account for aspects of the environment or individual that are conflated with externalizing and can, if left unchecked, bias conclusions. Socioeconomic status (SES) is related to behavior problems such that children from lower SES families exhibit higher levels of problem behavior (Kupersmidt, Bryant, & Willoughby, 2000; Qi & Kaiser, 2003), especially physical aggression. Child sex is also related to behavior problems, with boys exhibiting more externalizing behaviors than girls (American Psychiatric Association, 1994). In early childhood behavior problems may not differ by sex (Campbell, 1998). A review by Qi and Kaiser (2003) found that in only five of 16 studies did researchers find a significant sex difference in preschool-aged children. Due to these potential relations, SES and sex are included as covariates in analyses.

Genetic research on positive development, compared to behavior problems, has been less common (Knafo et al., 2008). Competence, defined in toddlerhood as behaviors such as sustained attention, prosociality, imitative play, and compliance with parents, also has a strong genetic component (Van Hulle, Lemery-Chalfant, & Goldsmith, 2007), with girls displaying higher mean levels of competence than boys in early childhood. For commonly observed behavior (the common factor of mother and father report), competence was heritable for both boys and girls (A = 0.67 and 0.61, respectively) with a modest shared environment component (C = 0.22 and

0.26, respectively) and the remainder of variance accounted for by the nonshared environment. General competence has been found to be more heritable in boys than girls in toddlerhood (Saudino, Carter, Purper-Quakil, & Gorwood, 2008).

Prosocial behaviors (e.g., empathy, sympathy, and altruism) can be distinguished from cognitive and affective aspects of competence (Hoffman, 2000; Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008). Research conducted on prosocial behaviors, defined as behaviors enacted voluntarily and with the intent to help others (Eisenberg & Fabes, 1998), generally results in modest to moderate heritability estimates with a declining shared environment factor across developmental periods (Rushton, Fulker, Neale, Nias, & Eysenck, 1986; Zahn-Waxler, Robinson, & Emde, 1992). There is, however, disagreement among studies. Using a stepfamily design, problem behaviors were found to be strongly heritable (A = 0.76) in early childhood with no measurable influence of the shared environment, but prosocial behaviors (e.g., sharing and kindness) were largely influenced by the nonshared environment and only slightly heritable (A = 0.15; Deater-Deckard, Dunn, O'Connor, Davies, & Golding, 2003).

Parallel to externalizing, most studies find that competence seems to become more heritable with age. In toddlers, researchers have reported significant shared environment components (Knafo & Plomin, 2006a; Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008; Van Hulle et al., 2007). In middle childhood the significant shared environment component remains (Edelbrock, Rende, Plomin, & Thompson, 1995) with a low to moderate

genetic factor (A = 0.08 to 0.48). In contrast, by adolescence, competence (scholastic, social, physical, and athletic) appears to be more strongly heritable without a significant contribution from the shared environment (Gregory, Light-Hausermann, Rijsdijk, & Eley, 2009; Hur & Rushton, 2007; Knafo & Israel, 2008; Knafo & Plomin, 2006b; McGuire, Neiderhiser, Reiss, Hetherington, & Plomin, 1994; 1999). A sample of Taiwanese adolescents also yielded moderate heritabilities for adolescent competence (0.43 to 0.51; Kuo, Lin, Yang, Soon, & Chen, 2004). A full review of the genetic and environmental contributions to prosocial behavior is available (see Knafo & Israel, 2008).

In contrast to the finding of Saudino and colleagues (2008) that competence is more heritable in boys in toddlerhood, others have found that the heritability of competence is stronger in adolescent girls than in boys, particularly in the activity domain (Kuo et al., 2004). In addition to considerations of child sex, mother report of prosocial behavior may be less genetically-influenced than observer report (Hur & Rushton, 2007; Zahn-Waxler, Schiro, Robinson, Emde, & Schmitz, 2001), at least among young children.

In general, externalizing and behavioral competence show similar patterns of genetic and environmental influence across development, although mixed findings in both areas warrant further study. Externalizing and behavioral competence are strongly negatively correlated, even when comparing observer reports (Laible, Carlo, Torquati, & Ontai, 2004). Additional support for combining behavior problems and competence into one

continuous dimension appears in the resilience literature (Buckner, Mezzacappa, & Beardslee, 2003), where a behavioral continuum from depression and anxiety to successful emotional and behavioral functioning yielded similar (but stronger) results than an approach that kept depression/anxiety and successful functioning as separate indicators of resilience.

Measured aspects of the environment can be incorporated into classic behavior genetic models to strengthen their design (Rutter, Pickles, Murray & Eaves, 2001). Bronfenbrenner's (1999) bioecological model defines development as a function of forces arising from multiple settings and from the relations among these different settings. Therefore, an approach that integrates multiple levels is useful for elucidating development (Cook, 2003; Kagan, 2000; Magnusson, 2000). Development is then modeled as a product of the additive or interactive relations between the person and his or her experiences. Relevant experiential predictors of externalizing and competence typically include harsh parental control strategies, inconsistent family routines, uninvolved or highly controlling parenting styles, and disorganized classroom environments (Wachs, 2005).

A particularly salient experience that functions to increase positive outcomes across developmental periods is caring relationships with others (Baumrind, 1971). This caring relationship with the caregiver or other figure offers the child support, positive regard, and trust. In fact, the effects of parenting remain a significant predictor of child behavior over time even after controlling for child characteristics (Collins, Maccoby, Steinberg,

Hetherington, & Bornstein, 2000). This caring relationship is one dimension of authoritative parenting, in which the caregiver is caring and involved while remaining firm and consistent with discipline and guidelines (Steinberg, 2000). For children in risk environments, a positive relationship with an adult (particularly a parent) can help diminish or prevent child behavior problems (Masten & Coatsworth, 1998). Additionally, behavior competence is longitudinally predicted by parenting, with warm parenting predicting increases in child prosocial behaviors and strict, punitive parenting predicting decreases in child prosocial behaviors (Eisenberg & Fabes, 1998).

Parenting is operationalized in many different ways (e.g., attitudes about parenting, fixed constellations of behaviors, or specific behaviors). The preponderance of research to date on positive parenting has utilized dimensions of parenting, typically guided by Baumrind's (1966; 1967) typology of authoritarian (low warmth, high control), authoritative (high warmth, high control), permissive (high warmth, low control), and neglectful (low warmth, low control) parenting styles. Studies that classify individuals along multiple dimensions of parenting are useful, but also of importance is research that focuses on the individual contributions of different dimensions. By combining the effects of warmth and control, researchers may lose sight of the distinct predictive patterns of each. For example, parental rejection (low warmth), but not control, predicts depressed mood in girls (Hipwell et al., 2008).

Warmth has been distinguished from other parenting behaviors, such as responsiveness to distress (Davidov & Grusec, 2006), by having unique contributions to child outcomes like prosociality and empathy. Important to this study, more harsh, rejecting parenting has been found to relate to the development of externalizing problems (Deater-Deckard, Dodge, Bates, & Pettit, 1998) while higher levels of warmth and acceptance related to positive child outcomes, such as prosociality and cooperativeness (Maccoby & Martin, 1983; Ruchkin, Eisemann, Hagglof, & Cloninger 1998). In a study that will be revisited later in this review, warm and responsive parenting predicted lower externalizing in toddlerhood, moderated by ethnicity and DRD4 genotype (Propper, Willoughby, Halpern, Carbone, & Cox, 2007). In adolescence and adulthood, parental rejection related to higher hostility (Meesters, Muris, & Esselink, 1995), and positive, warm parenting in early childhood predicted decreased adolescent externalizing, moderated by the child's effortful control (Eisenberg, Zhou, Spinrad, Valiente, Fabes, & Liew, 2005).

Eisenberg and colleagues (1999) posit that parenting causes externalizing behaviors when the parenting leaves the child emotionally stimulated and unresolved. Parental warmth, more so than rejection, should be more effective in terms of returning children to baseline after an upsetting event. Punitive or minimizing parental reactions were associated with children's low socio-emotional competence (Jones, Eisenberg, Fabes, & MacKinnon, 2002), and a general lack of parental warmth related to delinquency and aggression (East, 1991). Parental rejection should then increase externalizing behaviors and decrease behavioral competence. In a large study of middle childhood in

the Netherlands, child perception of high levels of parental rejection and low levels of parental warmth related to higher levels of psychopathology (Buschgens et al., 2010).

Why is parental warmth particularly important in the development of externalizing and behavioral competencies? Parent-child interactions characterized by parental warmth model positive feelings and enable the child to experience the intrinsic rewards implicit in this type of interaction (MacDonald, 1992). According to MacDonald, paternal warmth may be particularly relevant for child outcomes. Paternal warmth indicates a capacity for high-investment parenting that is evolutionarily adaptive. Humans epitomize ecology's K strategists (MacArthur & Wilson, 1967). K strategists are organisms that, through evolution, have chosen to focus on quality of offspring rather than quantity in the hopes of reproducing offspring that can compete successfully for limited resources. The trade-off (a prolonged period of immaturity) means that human children require a long period of resources and support. Children with invested fathers experience greater levels of both, making offspring of involved fathers more competitive. To the extent that paternal warmth indexes parental investment in the child, it may show a different pattern of relations than maternal warmth to behavioral outcomes in the child.

Modeling of positive feelings by parents may have an indirect effect on child externalizing and competencies through the child displaying more positive social behavior. Parental positive affect has been shown to relate indirectly to children's social competence, mediated by the child's expressed

positive affect (Isley, O'Neil, Clatfelter, & Parke, 1999). In addition to potential mediated effects, parental warmth has been found to have direct links to children's competence in kindergarten and first grade (Isley et al., 1999). Interestingly, excessive parental warmth (over and above parent-child positive affect that was shared and mutual) in this study was associated with diminished child social competence. Noncontingent or inappropriate warmth from parents may indicate a lack of parental sensitivity.

Beyond phenotypic studies, genetically-informed research has linked parent positivity and negativity to heritable aspects of child prosocial behaviors (Knafo & Plomin, 2006a). Specifically, from age two to age seven, parent positivity predicted increased child prosocial behaviors at later time points and parent negativity predicted decreased child prosocial behaviors at later time points (though less strongly than did parent positivity). Genetic influences on prosocial behavior increased from 0.32 at age two to 0.61 at age seven (averaged across child sex and parent and teacher report) while shared environmental influences declined from 0.47 to 0.03 across the same ages.

Some studies find evidence to support reciprocal relations, with two separate types of genetic influence examined. A child's heritable trait can elicit the type of parenting they receive (i.e., gene-environment correlation). For example, mothers who demonstrate more positive expressiveness toward their children had children who, in turn, exhibited more positive expressiveness to the mother (Cassidy, Parke, Butkovsky, & Braungart, 1992). This may be, in part, because in twin studies parental warmth and negativity have been shown to have genetic components that could be a result

of gene-environment correlation (Deater-Deckard, Fulker, & Plomin, 1999). Parents' genes influence parenting behaviors like warmth and rejection (Losoya, Callor, Rowe, & Goldsmith, 1997), and in a study of adult identical twins, 25% of the variance in parenting was attributable to gene-environment correlation (Plomin, McClearn, Pederson, Nesselroade, & Bergeman, 1989), further illustrating the complex interplay between genes and experiences. Clearly, genes do appear to play some role in the elicitation of parental behaviors, however this is the case for many measures of experience.

Most genetically-informed studies of parental warmth have focused on gene-experience correlation. Thus far, only one genetically-informed study has tested the hypothesis that parental warmth can moderate the expression of genetic influences on externalizing in adolescents. Researchers found evidence of significant moderation of the heritability of aggression by family level warmth (Rowe, Almeida, & Jacobson, 1999). The authors clustered individuals within school and reported that individuals in schools with higher levels of family warmth had lower levels of aggression. Furthermore, in schools with higher family warmth, sibling correlations increased with the degree of relatedness providing evidence for a strong heritability for aggression (A = 0.65) and no measureable contribution of shared experiences. In schools with lower family warmth, there were weaker estimates of heritability (A = 0.13) and a conversely higher importance of shared experiences. Negative experiential factors are hypothesized to be more strongly related to development, in essence washing out some of the potential genetic effect on individual differences (Bronfenbrenner & Cici, 1994). To the

extent that parental rejection creates a negative environment, children in a negative environment will display a phenotype that is less genetically-influenced than it would have been under better environmental circumstances. For example, children with greater genetic propensity for prosocial behavior will not display as much prosocial behavior in a disadvantaged environment as they would in an advantaged environment. Conversely, children with greater genetic propensity for externalizing behavior will display these behaviors in a disadvantaged environment, but the cause of the behavior will be conflated between additive genetic influences and the negative experiential factor of the parental rejection. In more advantaged environments however, in this case those in which the child experiences more parental warmth and less rejection, there are more opportunities for genetic effects, allowing the full range of heritable influences. Bronfenbrenner and Cici's (1994) theoretical argument lends credence to Rowe and colleagues' (1999) results. Given the family-level nature of the research question however, there is a need to replicate their findings without the potentially confounding school-level clustering.

The study by Rowe and colleagues (1999) considered warmth as a moderator of the heritability of aggression only in adolescence. Parental warmth/rejection has not previously been considered as a moderator of the heritability of behavior competence in children. The relations of parental warmth to child behavioral outcomes may well shift with child age. A meta-analysis of research on the relation between caregiving behaviors (approval, guidance, motivational strategies, synchrony, coercive control, and

restrictiveness) and externalizing in nonclinical samples tested two hypotheses pertaining to the age of the child. One hypothesis was that parent behaviors have stronger effects with younger children, because young children lack exposure to alternative influences (e.g., peer groups, other adults) and spend more time with parents. The alternative hypothesis was that parent-child interactions over the years could yield cumulative effects, resulting in stronger relations between parenting behavior and outcomes in older children (Rothbaum & Weisz, 1994). Using observational and interview methods to ascertain the influence of parental behaviors on externalizing behaviors, the meta-analysis concluded that the average effect size was significantly larger among older children (6 years through adolescence) than younger children (two and a half to five years), and there was a greater likelihood of finding significant effects in older samples (Rothbaum & Weisz, 1994).

The meta-analysis failed to find a significant sex difference in the association between parent behaviors and externalizing (Rothbaum & Weisz, 1994). When examining only mothers of preadolescent children, a significant effect across studies was reported, with mother behavior having a stronger influence on boys. In general mothers have considerably more frequent interactions with children than do fathers, a pattern that extends from preschool into adolescence (Lamb, Ketterlinus, & Fracasso, 1992). Although the mother typically spends more time with the child, fathers may have an equal or stronger effect on the child's behavior (MacDonald, 1992). Research in general is mixed as to whether warmth differs by sex of child (Eisenberg &

Fabes, 1998; Isley et al., 1996, 1999; Shaw, Keenan & Vondra, 1994; Shaw et al., 1998).

Last, questionnaire measures of caregiving often resulted in smaller effect sizes than interview or observational measures, though there was no significant difference in the percentage of significant effects yielded by each type of study (Rothbaum & Weisz, 1994), perhaps as a function of the larger sample size of most studies using questionnaire measures. In general, Rosenbaum and Weisz (1994) found that longitudinal studies were scarce and often had long lag times between the measurement of parent behavior and subsequent measurement of child/adolescent outcomes, making it difficult to compare studies and potentially overlooking short-term effects.

In summary, there is a need for additional studies of warmth/rejection that consider mother and father warmth/rejection separately, span developmental periods, measure parenting and child outcomes concurrently, and examine ways in which genetic influences on individual differences shift in response to parental warmth/rejection. The proposed study addresses these needs, and furthermore, combines behavior genetic methodology with a continuum approach to measuring warmth/rejection and externalizing/competence.

Although it is true that studies that fail to take into account the heritable nature of behavior often conflate genetic and environmental sources of information (Lindzey, Lykken, & Winston, 1960; Turkheimer & Gottsman, 1996), behavior genetic methodology also has limitations. Chiefly, twin and family design studies rely on inferences of heritability and do not show direct

evidence of genetic association. New technologies make it possible to measure the relations of specific genes to a given trait or outcome. Researchers are no longer limited to the "black box" explanation for the processes through which genes and experiences interact to influence behavior (Rutter, 2008).

In terms of specifics for studies of measured genes, genes organize on 23 pairs of chromosomes, with each gene consisting of pairs of alleles, one of each pair inherited from each parent. The expression of alleles is typically either dominant (the heterozygote is indistinguishable from the homozygous dominant) or additive (the homozygous dominant and the homozygous recessive are equal distance from the heterozygote, or midpoint on the trait.) Quantitative trait loci (QTL) are gene segments underlying the genetic contribution to quantitative traits. Segments of interest may be of differing lengths. Many molecular genetic studies focus on single nucleotide polymorphism (SNPs). SNPs occur when a nucleotide (cytosine [C], guanine [G], adenine [A], and thymine [T]) differs between individuals in a population. Studies can also estimate differences in VNTRs. A VNTR occurs when a set of nucleotides have a varying number of possible tandem repeats. Individual variation is then the number of repeats, or the length of the VNTR. In this study, two VNTRs are also examined, one each relating to dopamine and vasopressin expression, and a series of SNPs (analyzed in haplotypes) related to neuropeptide-Y. A haplotype is a group of nucleotides (SNPs) that are transmitted together from parent to child. These segments are not anticipated to be solely predictive of any given trait – instead they are expected to function in conjunction with other genes and experiences (Plomin,

DeFries, McClearn, & McGuffin, 2000). Genetic effects are probabilistic and involve multiple genes acting in combination, having indirect effects, and affecting outcomes along a dimension (Plomin & Rutter, 1998). Individual differences stem from normal variants of a gene. Because disorders and behavioral outcomes are a result of a complex system of multiple genes and environmental influences, candidate gene studies often yield conflicting and insubstantial results (Faraone, 2008). Given that the effect of any one gene on a behavioral outcome is small, to the extent that there are many genes that influence a trait an individual QTL is likely to have a small effect, resulting in a common criticism of molecular genetic research: poor replication. QTL nevertheless provide an important piece of information to behavior genetic studies by adding a measured gene component that can illuminate the "black box."

In this study, three functional QTL located along the dopamine, vasopressin, and neuropeptide-Y genes were considered. These genes fall within three separate biological systems of adaptation (reward response, pair bonding, and stress response; respectively), with each QTL potentially predisposing an individual to either adaptive or maladaptive outcomes, dependent upon environmental circumstances. These were putative differential susceptibility QTL, expected to interact with parent warmth such that certain variants increased the individual's responsiveness to his or her environment.

Given the difficulties in conducting molecular genetic research, it is particularly important to utilize findings from behavior genetic studies as

well as what is known about biology and brain function to inform the selection of QTL. Genes influencing dopamine levels in the brain are one example of where known biology and brain functionality lead to the consideration of certain gene segments as QTL for externalizing/competence.

Dopamine is a key component of the psychobiology of the reward system. Dopamine is expressed in the brain through the meso-limbic pathway, which begins in the group of neurons located in the ventral tegmental area (VTA) of the midbrain and extends to the ventral striatum, which contains the nucleus accumbens (Ikemoto, 2007) and projects to the prefrontal cortex (Baskerville & Douglas, 2010). All brain areas discussed are illustrated in Figure 1. The activation of this pathway underlies an individual's response to reward and reinforcement (Ikemoto, 2007; Schultz, 2007). VTA dopamine neurons also extend (to a lesser extent) to other structures within the brain, including the hippocampus and amygdala (Ikemoto, 2007).

The dopaminergic system has been repeatedly shown to be of importance in relation to behavioral outcomes. Dopamine is expressed at a higher concentration in the brain than most other neurotransmitters and has broad receptor distribution. As a result of this innervation, dopamine is particularly influential in the pre-frontal and frontal cortex (Baskerville & Douglas, 2010; Bell & Deater-Deckard, 2007) and prefrontal cortex functioning has been found to operate optimally within a narrow band of dopaminergic activity (Arnsten, 1998). Stressful events disturb the balance of dopamine, particularly in the prefrontal cortex, nucleus accumbens, and amygdala (see Figure 1; Mizoguchi, Shoji, Ikeda, Tanaka, & Tabira, 2008). Neurons in the

VTA increase dopamine release in the prefrontal cortex during stressful events (Ingram, 2005). Chronic stress may decrease dopamine concentrations in the brain (Mizoguci et al., 2008). Decreases in dopamine been implicated in relation to depression, and in fact dopamine agonists have been shown to have efficacy as antidepressants (Breuer et al., 2009; Mizoguci et al., 2008).

In addition to the meso-limbic pathway, the nigrostriatal pathway is another major dopamine pathway in the brain that connects the striatum, prefrontal cortex, nucleus accumbens, and amygdala (Baskerville & Douglas, 2010). Activation of the lateral frontal and midline areas of the brain are often implicated in the functioning of the executive attention network (Fan et al., 2001). *In vivo* analyses have shown 20-100% increases in dopamine concentration in the nucleus accumbens and prefrontal cortex in response to behavioral events (Abercrombie, Keefe, DiFrischia, & Zigmond, 1989; Feenstra, Botterblom, & Mastenbroek, 2000).

Frequently implicated genes related to behavioral outcomes in the dopaminergic system are the dopamine transporter gene (DAT1), which clears dopamine from the synapses, and the five dopamine receptors. The dopamine receptor D_4 (DRD4) is located on chromosome 11 and contains a highly polymorphic 48 base pair VNTR (common repeats are four and seven) on the third exon that influences ligand binding (wherein a ligand binds to a receptor with varying degrees of affinity, changing the chemical conformation of that receptor; Schmidt, Fox, Perez-Edgar, Hu, & Hamer, 2001). DRD4 belongs to the D_2-like family of dopamine receptors.

Functionality of the DRD4 exon 3 VNTR has been further shown in relation to cyclic adenosine monophosphate (cAMP), an intracellular messenger in the brain. The cAMP pathway connects G protein-coupled receptors in the brain (those which are responsive to extracellular stimuli). Dopamine in the DRD4 receptor inhibits cAMP levels. The seven-allele repeat of the VNTR is related to a twofold reduction in the ability of dopamine to inhibit cAMP formation (Asghari et al., 1995), implicating it as a risk variant. Longer versions of the allele result in less efficient receptor binding of dopamine (Plomin & Rutter, 1998).

The longer allele of DRD4 is associated with higher scores on the personality trait of novelty seeking. Novelty seeking is indexed by high levels of exploratory, impulsive, extravagant, and excitable behaviors (Schmidt et al., 2001). The trait of novelty-seeking is hypothesized to underlie externalizing behaviors by increasing impulsiveness and stimulation-seeking (Barnow, Lucht, & Freyberger, 2005). DRD4 knock-out mice (mice that are genetically engineered to turn off a given gene) show an inability to explore novel stimuli (Dulawa, Grandy, Low, Paulus, & Geyer, 1999), though a review of DRD4 and dimensions of normal personality including novelty seeking has failed to find an association (Paterson, Sunohara, & Kennedy, 1999) and findings in general relating DRD4 to psychiatric disorders have been mixed (Serretti, Lilli, Lorenzi, Lattuada, & Smeraldi, 2001). In a longitudinal non-clinical sample of children with mother report of attention problems, the long version of the DRD4 polymorphism predicted increases in attention problems at ages four and seven, and a small but significant

proportion of the temporal stability of attention problems across early childhood (Schmidt, Fox, Perez-Edgar, Hu, & Hamer, 2001). McGue (2001) found a mean effect size of .21 of the DRD4 polymorphism on personality in a meta-analysis of 17 diverse studies. The 7-repeat allele has also been associated with slower cognitive responses in a community sample of older adolescents (Szekely et al., 2010), and has been associated with ADHD more often than any other individual gene variant (Li, Sham, Owen, & He, 2006).

The long version of DRD4 may not always be maladaptive, raising the possibility of a differential susceptibility explanation. In newborns, the long variant of DRD4 has been positively associated with orientation, regulation of state, range of state, and motor organization (Auerbach et al., 1999; Ebstein et al., 1998). Infants with higher orientation skills experienced less negative emotionality when they were two months of age. These infants with a long allele for DRD4, in addition to having less negative emotionality at two months, also had diminished distress to limitations. At one year of age, the long allele related to temperament dimensions such as higher activity level, lower interest, and lower anger (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001). In infancy approach behaviors such as quicker movement toward novel toys, energetic play, and more frequent movement shifts may result in increased exploration (Auerbach et al., 2001), but in adulthood these behaviors may index risk for novelty seeking and disordered personality. In toddlerhood, warm and responsive parenting related to decreased externalizing behavior only among African American children with the short version of DRD4 (Propper et al., 2007).

A study of slightly older children (18-21 months) found an interaction between the DRD4 VNTR and parenting. Children with the 7-repeat allele (decreased response to dopamine) were influenced by parenting quality such that lower quality parenting related to higher levels of sensation seeking (activity level, high intensity play, and impulsivity) and higher quality parenting was related to lower levels of sensation seeking. Children with the 4-repeat allele were not significantly affected by the risk factor of lower quality parenting (Sheese, Voelker, Rothbart, & Posner, 2007). This study supported the differential susceptibility hypothesis, as children with the 4-repeat version were not at risk for environmental stressors, but children with the 7-repeat allele displayed a more flexible genotype that allows for environmental interventions (Sheese et al., 2007), which may better illuminate how parenting and experiential factors contribute to externalizing.

A gene-environment interaction has also been found for the DRD4 VNTR and maternal insensitivity (Bakermans-Kranenburg & van IJzendoorn, 2006). In preschool-age children, maternal insensitivity related to a six-fold increase in externalizing behaviors only for children carrying the long variant of DRD4. When exposed to high levels of maternal sensitivity, children with the long variant appeared to have fewer symptoms of externalizing (though the difference was nonsignificant) than children with the short variant, again indicating differential susceptibility.

The literature on the DRD4 VNTR is mixed, as is often the case in studies of measured genes. In addition to many studies that have failed to find an effect (see Gizer, Ficks, & Waldman, 2009 for a recent review of DRD4 and

ADHD), some studies yield contradictory findings. The longer version (6+ repeats) of DRD4 is typically implicated as the risk variant, but, as reviewed earlier, it has also been found to be adaptive (Auerbach et al., 1999; Ebstein et al., 1998). A prevailing theme in many studies with null or contradictory results is the lack of a measure of experience (Gizer et al., 2009; Propper et al., 2007).

In addition to its role in the reward system, dopamine also plays a part in pair bonding. This may indicate that dopamine is particularly influential in the context of parent-child relations (such as parental warmth/rejection). In rats, mothers with better maternal behaviors (indexed by grooming) have more dopamine in the nucleus accumbens, supporting the hypothesis that dopamine relates to bonding (Shakrok & Meaney, 2010). In prairie voles, mating increases dopamine turnover in the nucleus accumbens (Hammock & Young, 2006) and dopamine seems to act to facilitate bonding during sexual activity (Melis, Succu, Sanna, Boi, & Argiolas, 2009). In addition to dopamine, a second neuropeptide has been shown to play a key role in social recognition and pair bonding: vasopressin (Hammock & Young, 2006). Vasopressin neurons express D_2-like family receptors similar to DRD4 (Baskerville, Allard, Wayman, & Douglas, 2009). It has been hypothesized that dopamine may act to control vasopressin release or vasopressin may partly control dopamine release (Baskerville & Douglas, 2010).

Arginine vasopressin is a peptide produced in the hypothalamus, the stria terminalis, and the medial amygdala (see Figure 1; Meyer-Lindenberg et al., 2009). The Arginine Vasopressin Receptor 1A (AVPR1a) is one of the three

major receptor genes for vasopressin (along with AVPR1b and AVPR2) and contributes to vasopressin present in the brain as well as the liver, kidney, and vasculature (Caldwell, Lee, MacBeth, & Young, 2008). Located on chromosome 12, AVPR1a is, like DRD4, a g-protein coupled receptor (found only in eukaryotes); these receptors are involved in many diseases and are the target of 30% of modern drug treatments for psychopathology (Filmore, 2004). AVPR1a contains a polymorphism in the promoter region (R3) that is located 3625 base pair (bp) upstream from the transcription start site with a complex repeat that is often broken into long (more than 325 bp) and short (325 or fewer bps) variants (Knafo et al., 2008) that is examined in the current study. The long allele has been previously implicated as the protective allele for affiliative behaviors (Hammock & Young, 2002, 2004, 2005, 2006) as well as a risk for autism and marital discord (Kim et al., 2002; Walum et al., 2008). The longer variant relates to higher AVPR1a mRNA levels using human postmortem hippocampal samples (Knafo et al., 2008). To the extent that the long variant of AVPR1aRS3 is protective in advantaged environments and increases risk in disadvantaged environments, this VNTR also fits with the differential susceptibility hypothesis.

To date, much of the literature on vasopressin genes examines pair-bonding in prairie voles. Vasopressin (in addition to oxytocin) is released during mating and cohabitation and influences dopamine levels in the nucleus accumbens (Hammock & Young, 2006; Young, 2009). This means that vasopressin has the ability to alter the reinforcing properties of dopamine during social interactions with a mate, and can influence the

probability of future contact with that mate. Mating among male prairie voles leads to both social bonding and increased paternal care behaviors (Lonstein, 2002; Bales, Kim, Lewis-Reese, & Carter, 2004). Male marmosets show increased AVPR1a receptors in the prefrontal cortex (Kinsley & Lambert, 2008). Furthermore, selective AVPR1a blockade in the ventral pallidum prevents partner preference – indicating that the receptors in the ventral forebrain region are crucial for pair bonding (Lim & Young, 2004).

Humans do not have the same sequence in the AVPR1a gene that predicts monogamous behavior in prairie voles; however, the RS3 repeat in adult twins predicts similar pair-bonding patterns of behavior in humans (Walum et al., 2008). Extending the literature on prairie voles to humans, Walum and colleagues (2008) reported that a longer variant of RS3 (i.e., 334 bp) was associated with decreases in perceived pair bonding and likelihood of being married, but only among males. Males homozygous for the 334 bp allele were at double the risk for marital crisis than males with no 334 bp allele. Further, men with this risk allele were more likely to be in a relationship than a marriage, perhaps indicating diminished pair-bonding.

From the evolutionary perspective, a mutation in the vasopressin gene in a common primate ancestor may help explain human prosocial behavior. The RS3 polymorphism of AVPR1a is found in both humans and bonobos (an ape species related to the chimpanzee) but it is not found in the chimpanzee genome. Bonobos, like humans, are known for social reciprocity, empathy, and bonding – more so than the chimpanzee. These differences have been attributed in part to the R3 polymorphism (Hammock & Young, 2005).

Increased vasopressin from the AVPR1a receptor in the brain bolsters social recognition and decreasing vasopressin decreases social recognition (Hammock & Young, 2006).

Mating dyad bonding and parent-child dyad bonding have similarities. An fMRI study of oxytocin receptor distribution (a neuropeptide closely tied to both dopamine and vasopressin) and brain activation of adults while viewing photos of romantic partners and offspring found that the patterns of activation were considerably similar for both types of photos (Bartels & Zeki, 2004). Vasopressin in the offspring can also to the parent-child bonding experience. A demonstration of this can be found in rats, where odor cues stimulate vasopressin release, which in turn allow the offspring to show a preference for the maternal odor, facilitating social recognition (Hammock & Young, 2006). Similar findings have been found with prairie voles (Curtis, Liu, & Wang, 2001; Kirkpatrick, Williams, Slotnick, & Carter, 1994). Vasopressin in humans may stimulate multiple sensory and cortical systems, in addition to the olfactory (Hammock & Young, 2006).

Similar to the relations of this peptide to pair bonding and prosocial behaviors, investigators have linked vasopressin to externalizing behaviors in hamsters (Ferris & Potegal, 1988; Ferris et al., 1997), rats (Koolhaus, Van den Brink, Roozendal, & Boorsma, 1990), and prairie voles (Winslow, Hastings, Carter, Harbaugh, & Insel, 1993). Among adults with a diagnosed personality disorder, higher cerebrospinal fluid arginine vasopressin concentrations were positively correlated with increased general aggression, with stronger relations for men than for women (Coccaro, Kavoussi, Hauger,

Cooper, & Ferris, 1998). Studies of vasopressin often yield sexually dimorphic results, with men having higher base levels and larger behavioral effects related to vasopressin (Meyer-Lindenberg et al., 2009).

Thus far, no direct relationship between AVPR1a and externalizing behaviors has been found in humans (Craig & Halton, 2010), despite the interesting relations of vasopressin to aggressive behaviors. In a study utilizing imaging genetics, carriers of the long variant for RS3 had differential activation of the amygdala (Meyer-Lindenberg et al., 2009). Meyer-Lindenberg and colleagues (2009) found that overtransmission of the long allele of RS3 was associated with higher amygdala activation in response to an emotional face-matching paradigm. There were no sex differences in this study. In a previous study, the authors had shown that decreased amygdala function related to increased externalizing behaviors (Meyer-Lindenberg et al., 2005).

AVPR1aRS3 is a promising QTL for externalizing and competence behaviors in childhood and adolescence. To date, no one has investigated the relations of this gene variant to developmental behavioral outcomes. Additionally, the literature on the importance of vasopressin in pair bonding is of interest. As parental warmth bolsters the parent-child bond, I hypothesize that the AVPR1aRS3 VNTR should moderate the relations between parent warmth/rejection and child externalizing/competence. Children with one or more long repeats on AVPR1aRS3 should be better able to form dyadic bonds and thus should experience stronger benefits from

parental warmth and, conversely, may be more at-risk under conditions of high parental rejection.

Thus far, I have proposed that a polymorphic variant of a gene in the reward response system (DRD4) and a second variant in a gene implicated in pair-bonding, prosocial behaviors, and aggression (AVPR1a) may moderate the relations between parental warmth/rejection and child externalizing/behavior competence. Both of these gene variants are hypothesized to support the differential susceptibility hypothesis, with the long variants of each making the child more reactive to his or her environment. A third system of interest in understanding the relations between parenting and child behavior is the stress-response system. Neuropeptide-Y (NPY) is a relatively small, infrequently-studied gene on chromosome 7 but it is a gene with an important function in the stress-response system.

The neuropeptide NPY is encoded by the NPY gene and is commonly expressed in the central nervous system. NPY, like vasopressin, impacts a wide range of physiological processes including circadian rhythm, food intake, cortical excitability, and cardiovascular function. Importantly for the current study, NPY is released in the brain during times of stress (Heilig, 2004). After experiencing a stressor, there is an initial phase characterized by increased emotionality and anxiety, as the individual rallies to respond. This initial phase is marked by a cortisol release within the amygdala. Equally important as mounting a stress response is the ability of the organism to regain equilibrium afterwards. Neuropeptide-Y is released during the second

phase of the stress-response and decreases fear- and stress- related behaviors as well as affect, allowing the individual to revert to baseline functioning (Heilig, 2004). Lower NPY expression correlates with higher emotion-induced amygdala activation. Higher NPY expression conversely correlates with diminished negative affect and behavior. The current study included seven SNPs (rs16475, rs16147, rs16139, rs17149106, rs3037354, rs5573, and rs5574) in the NPY gene. This grouping of SNPs within the NPY gene captures major five haplotypes and, through linkage disequilibrium, accounts for approximately 70% of the variation within the gene (Zhou et al., 2008), though rs16475 does not distinguish between haplotypes. Haplotypes are specific combinations of groups of SNPs transmitted from each parent (i.e., on one chromosome) that are in linkage disequilibrium with each other (Morris, 2008). Rs16147 accounts for between 30-50% of the expression of NPY in vivo (Zhou et al., 2008). A deletion on rs3037354 may increase expression of NPY through limiting binding sites for the transcription factor. Zhou and colleagues (2008), however, found that the TG allele at rs3037354 may actually decrease expression, though their findings did not reach statistical significance. TG in combination with a C allele on rs16147 reduced the expression of NPY by 47%.

NPY is often explored in relation to internalizing disorders like depression and anxiety. When injected, NPY acts as a sedative/anti-anxiety agent, similar to benzodiazepines or barbiturates (Amstadter et al., 2010). When NPY is injected directly into the baso-lateral amygdala, it blocks both chemical and physical anxiety responses for up to eight weeks post-injection

(Sajdyk, Shekhar & Gehlert, 2004; Sajdyk et al., 2008). Using haplotype analysis, Zhou and colleagues (2008) found that greater NPY expression predicts more efficient brain responses (quicker recovery) to emotions and stress. Amygdala activation to threatening facial expressions (indexing the strength of an individual's affective response) was higher among adults with low NPY expression. NPY also accounts for a significant amount of variance in response to moderate muscular pain in the prefrontal cortex, nucleus accumbens, and posterior insular cortex (Zhou et al., 2008).

As is the case with QTL research, studies using NPY often fail to replicate significant findings. The C allele of rs16139 has been previously linked to increased alcohol dependence (Lappalainen et al., 2002). In a later study that considered multiple SNPs, both the C alleles of rs16139 and rs16147 were only marginally related to alcohol dependence (Mottagui-Tabar et al., 2005), however, neither rs16139 or rs5574 was related to alcohol and cocaine dependence and withdrawal behaviors (Wetherill et al., 2008), nor was rs16147 a significant predictor of methamphetamine dependence (Okahisa et al., 2009). Additionally, no association was found between either rs16147 or rs16139 and panic disorder among adults (Domschke et al., 2008), or between rs16139 and sports performance (Buxens et al., 2010). The T allele of rs16147 was related to increased frequency of schizophrenia (Itokawa et al., 2003), but a replication failed to find an association with schizophrenia, depression, or panic disorder (Lindberg et al., 2006).

In a follow-up to the Zhou and colleagues (2008) functionality study, the functionality of rs16147 in the anterior cingulate cortex was examined.

Sommer and colleagues (2010) found that the C allele has reduced protein binding affinity and increased NPY expression in the anterior cingulated cortex, resulting in a greater risk for psychopathology (trait anxiety and depression) in young adulthood for individuals exposed to early childhood risk. The conclusion that the C allele increases NPY expression (Sommer et al., 2010) is inconsistent with earlier findings that the C allele actually decreases NPY expression (Zhou et al., 2008). The earlier study drew NPY samples during a non-stressful task, while the more recent study (Sommer et al., 2010) collected samples when participants were in a stressful situation. Both studies found the C allele to be maladaptive, but allelic variations in the expression of NPY seem to depend upon environmental conditions.

The finding that gene expression is moderated by the environment, in conjunction with the null main effect results from most of the literature, raises the possibility that variants within NPY influence behavior through a process of gene-experience interaction. Although there is no main effect of rs16147 on depression, the variant does interact with early adversity to predict later depression such that carriers of the C allele experience higher levels of depression when exposed to early adversity (Sommer et al., 2010). NPY (rs16147) has also been found to moderate the relationship between stress exposure (i.e., hurricane exposure) and generalized anxiety disorder in adults (Amstadter et al., 2010). These two studies are the only to date to consider NPY in gene-experience interaction models.

Most studies of the NPY gene are conducted using adult samples and focus on the ability of NPY to predict mood disorders such as depression and

anxiety; however, NPY has been shown to underlie stress-related behaviors as well as affect (Heilig, 2004), and thus I hypothesize that it will predict both child and adolescent externalizing and behavioral competence. Given the dearth of main effect findings in the literature, examining NPY variants in moderation models is hypothesized to extend earlier moderation findings (Amstadter et al., 2010; Sommer et al., 2010) with adults and internalizing to a childhood sample and externalizing behaviors.

Based on the supporting literature reviewed above, five distinct hypotheses were tested. First, externalizing/competence was hypothesized to have significant heritability at each age (toddlerhood, middle childhood, and middle adolescence) with a decreasing influence of the shared environment with age.

Second, parental warmth was hypothesized to be a significant predictor of externalizing/competence at each age. Warmth was predicted to relate to fewer behavior problems and more behavior competencies at each age, with warmth at an earlier time point predicting change in externalizing/competence from that time point to the next. Mean levels of parental warmth should decrease over time. Paternal warmth is rarely examined (Smetana, Campione-Barr, & Metzger, 2006) and may have added evolutionary benefits (MacDonald, 1992) and thus was hypothesized to be a stronger predictor of behavior than maternal warmth. Research is mixed as to whether warmth differs by the sex of child (Eisenberg & Fabes, 1998; Isley et al., 1996, 1999; Shaw et al., 1994, 1998), thus mean differences in warmth

and differences in covariances between warmth and child behavior resulting from the sex of the child was examined as a general research question.

In addition to main effects on behavioral outcomes, the third hypothesis was that parental warmth would moderate the heritability of behavior problems and competencies. Following the average expectable environment concept (Plomin, 1986; Scarr, 1992, 1993) a "normal" environment should allow for full expression of genetic variability. Low warmth/high rejection environments should function to overwhelm the genetic predisposition of an individual; therefore externalizing and competence behaviors were predicted to be more heritable under conditions of higher parental warmth and more environmentally-influenced under conditions of lower parental warmth. Externalizing and competence often become more heritable with age, and so the ability of warmth to act as a moderator of heritability was hypothesized to diminish with age.

Measured gene variants were hypothesized to be associated with behavior, through gene-experience interactions. Based on the above review, parental warmth and rejection were hypothesized to be more impactful for children with the long version of DRD4 than for children with the short version. Children with the short version should not be as susceptible to environmental influences, instead reflecting their genetic predisposition to externalizing/competence. Parallel to increasing heritabilities, measured gene effects were hypothesized to increase with child age.

A second gene variant that was hypothesized to moderate the relations between parenting and behavioral outcomes is AVPR1a. In line with the

environmental susceptibility hypothesis for DRD4, children with the long allele of AVPR1aRS3 were predicted to be more influenced by parental warmth and rejection than children with the long/short or short/short genotypes.

Lastly, seven SNPs within the NPY gene are also hypothesized to moderate the relations between parent warmth/rejection and child externalizing/competence. A stressor results in a cortisol release, stimulating the organism to respond to the threat. NPY is then released to regaining equilibrium following a stressor (Heilig, 2004). Given the function of NPY in the stress-response system, the variants of the SNPs that code for higher NPY expression in the brain should be more adaptive for individuals experiencing stress (i.e., low parental warmth and high rejection), buffering these individuals from negative outcomes. The effect of these SNPs on child externalizing and competence behaviors are tested using haplotype analysis. Because NPY decreases emotionality and related behaviors (Heilig, 2004), high expression of NPY in the absence of the experience of stress (i.e., high parental warmth and low rejection) may be maladaptive.

Method

Participants

Participants come from two cohorts from the ongoing longitudinal Wisconsin Twin Project, spread across three time points (see Lemery-Chalfant, Goldsmith, Schmidt, Arneson, & Van Hulle, 2006 for details about the sample and recruitment). At Time 1 (T1; family N = 762; M age = 2.25 years, SD = .24; 48% female; 35% MZ, 33% DZ same sex, 31% DZ opposite

sex), the sample was representative of young twins born in the state of Wisconsin. Follow-ups were conducted at two time points; Time 2 (T2; family *N* = 806 families; *M* age = 7.40 years, *SD* = .84; 48% female; 38% MZ, 31% DZ same sex, 31% DZ opposite sex) and Time 3 (T3; family *N* = 392 families; *M* age = 14.00 years, *SD* = 1.64; 52% female; 38% MZ, 34% DZ same sex, 28% DZ opposite sex). Cohort 1 spans Times 1 and 2, and Cohort 2 spans Times 2 and 3. Cohort 2 did not begin the study until middle childhood, and Cohort 1 (consisting of those who began with the toddler assessment) is only just now beginning Time 3 data collection. Additionally, although biological samples for genotyping have been collected from all participants, DNA has been extracted and typed for only a subset of participants (primarily those in Cohort 2) to date. Cohort 1 will only be included in analyses for a brief examination of the relations of warmth to externalizing/competence in toddlerhood and the relations of parent warmth in toddlerhood to change in externalizing/competence from toddlerhood to middle childhood.

Given the on-going data collection and the cohort nature of the study, longitudinal sample sizes are modest and final attrition rates are unknown. Specifically, 195 families have parenting data at both T1 and T2, 277 families have parenting data at both T2 and T3, and 23 families have parenting data at both T1 and T3. Currently 56 families have genetic data and T1 parenting, 369 have genetic data and T2 parenting, and 212 families have genetic data and T3 parenting. As a result, longitudinal models were only fit from T1-T2 and T2-T3, and models including measured genes (encompassing the majority of the study hypotheses) were only fit in T2 and T3.

Attrition analyses were conducted at the family level using a series of independent samples t-tests. If Levene's test for equality of variances was significant, the t-test results from the *equal variances not assumed* condition are provided (denoted by decimalized degrees of freedom). Differences were examined among families within each time with and without father data (among families in which the father was recruited) and within families that have completed Time 1 but not Time 2 or that have completed Time 2 but not Time 3 (mother or father report). Potential differences were also examined between the genetic subsample and the full sample at Time 2 and Time 3.

At Time 1, 13.61% of fathers did not complete reports of child behavior (Father family N = 660, Mother family N = 764). Families with missing father data did not differ from those with father data on child age, ethnicity, mother warmth or externalizing/competence, although families missing father data did report a lower socioeconomic status [SES; $t(270.62) = 3.62$, $p < .01$]. Families from Time 1 who completed the Time 2 assessment did not differ from those who have not yet completed the Time 2 assessment on Time 1 demographics, child age, ethnicity, or Time 1 warmth or externalizing/competence. Families who were missing father data at Time 2 (21.96%) and Time 3 (27.82%) also had a lower SES [$t(290.44) = 4.25$, $p < .01$ and $t(335) = 3.48$, $p < .01$, respectively] than those with father data. Families who completed Time 2 but not Time 3 differed from those who had completed both on child age. Families whose children were younger at the middle childhood assessment were more likely to have not yet completed the adolescent assessment [$t(814.62) = 4.46$, $p < .01$]. The mean age of the genetic

subsample was not different from the mean age of the full sample, nor did the genetic subsample report significantly different ethnicity or levels of externalizing/competence or father report of warmth, though the subsample did report less mother warmth [$t(802) = -2.11$, $p = .04$] and a lower SES [$t(689.77) = -4.42$, $p < .01$] than the full sample at Time 2. These differences between the genetic subsample and the full sample were not replicated at Time 3. Overall, there was no coherent pattern attributable to missingness. Missing data were addressed using maximum likelihood estimation with robust standard errors, which provides reliable estimates of parameter estimates when data are missing at random and missing completely at random, and only biases a subset of estimates if data are missing not at random (Enders, 2010).

Measures

Socioeconomic status. SES was indexed by mother and father education, employment and total family income. Educational level was assessed along a 20 point scale ranging from "grade school" to "graduate degree." Both mothers and fathers averaged "trade, tech, or some college." Employment was measured using the occupational factor on the Hollingshead index (Hollingshead, 1975). When the twins were toddlers 66.1% of mothers and 94.9% of fathers were employed, in middle childhood 77.9% of the mothers and 93.8% of the fathers were employed, and in adolescence 81.9% of the mothers and 89% of the fathers were employed. The occupational factor on the Hollingshead index is rated on a 9 point scale where a low score represents a low status occupation and a high score represents a high status

occupation. At all three phases, both parents averaged a five on this factor (i.e., "clerical and sales workers, small farm and business owners"). Total family income was measured by the total household income for the last year prior to taxes, with income ranges broken into 17 categories ranging from *$10,000 or less* to *Over $200,000*. Families averaged *$45,001 to $50,000* at T1 and *$50,001 to $60,000* at T2 and T3. Variables were standardized and a mean composite, *SES,* was formed for each Time. See Table 1 for descriptive statistics. *SES* was normally distributed, with no outliers.

Parental warmth/rejection. Parental warmth/rejection was assessed via self-report at each time point for mothers and fathers using the warmth composite of the Child Rearing Practices Report (CRPR; Block, 1965). This composite consists of 20 items drawn from the subscales of encouraging independence, open expression of affect, encouraging openness of expression, and rational guiding of the child (see Appendix A). Parents rated statements on subscale ranging from 1 (*Strongly disagree*) to 6 (*Strongly agree*). Example items are: "I respect my twins' feelings and opinions and encourage the twins to express them," and "My twins and I have warm, intimate times together." The reliability and validity of the CRPR has been previously demonstrated (Block, 1965; Block & Block, 1969; Jones, Rickel & Smith, 1980; Kochanska, Kuczynski, & Radke-Yarrow, 1989). A mean composite of each of the four subscales was formed for each parent at each time period; all items were significantly ($p < .05$) inter-correlated within reporter. Warmth/rejection was created for each parent from a mean of the subscales where higher values index greater warmth and lower values index greater rejection. Scale

reliability at T1, T2 and T3 was .67, .76, and .79 respectively for mother report, and .75, .83 and .83 for father report.

Outliers were identified as values outside three standard deviations of the mean of the warmth/rejection continuum at each time. Mother report at T1 had three outliers outside the lower bound and father report at T1 had one outlier outside the lower bound. Mother report at T2 had one outlier outside the lower bound and father report at T2 had three outliers outside the lower bound. Similarly, T3 had one mother report outlier and two father report outliers below the lower bound. These 11 cases were winsorized, a technique whereby variables outside of the third standard deviation are replaced with the value at the 1st (lower bound) or 99th (upper bound) percentile. See Table 2 for descriptive statistics for warmth/rejection.

Externalizing and competence behaviors. The twins' externalizing (i.e., aggression, defiance, and peer aggression) and competence behaviors (i.e., compliance, attention, and prosocial peer relations) were reported on by both mothers and fathers at T1 using the Brief Infant-Toddler Social and Emotional Assessment (BITSEA; Briggs-Gowan & Carter, 2002). The BITSEA contains six items that tap pure externalizing, free of impulsivity/inattention (e.g., "Cries or tantrums until s/he is exhausted"), and seven that tap behavior competence (e.g., "Plays well with other children [not including brother/sister]). See Appendix B. Each parent rated questions pertaining to his or her twins' behavior on a three point scale consisting of 0 (*Not true/rarely*), 1 (*Somewhat true/sometimes*), and 2 (*Very true/often*).

To test whether the externalizing and competence items could be combined into one dimension, all 13 items were entered into a principal component analysis (separately for each parent) specifying a one component solution. Parent reports of behavior were kept separate at T1 because of disparate sample sizes; 2116 families had mother report of BITSEA, fathers were only requested to complete the BITSEA in a subsample of families, yielding 660 families with father report. For mother report, the one factor solution accounted for 19.84% of the total variance with an eigen value of 2.78, and most items loaded above .30, which is considered significant in a sample larger than 100 (Kline, 2002, pp. 52-53). One item had a lower loading (.23), but was retained to correspond to the current literature. The one factor solution accounted for 24.85% of the variance in father report of externalizing/competence, with an eigen value of 3.73. Three items had loadings less than .30 (ranging from .16 to .27) but were also retained for replication purposes and consistency across reporter. Competence items were reverse-coded and a mean composite of externalizing/competence was created for each parent.

The BITSEA externalizing and competence scales have good test-retest reliability, inter-rater agreement, and construct and criterion validity (Briggs-Gowan, Carter, Irwin, Wachtel, & Cicchetti, 2004). Cronbach's alphas in the current sample were acceptable (mother report = .73; father report = .75). At T1, mother report of the behavioral continuum yielded 28 scores (1.32% of scores) that were above the 99th percentile and father report added an additional six scores (.91% of scores). These were winsorized.

At T2, comparable measures of externalizing (i.e., aggression, conduct problems, and oppositional defiant problems) and competence (i.e., prosocial behaviors and social relations) were assessed using mother and father report on the MacArthur Health and Behavior Questionnaire (HBQ; Armstrong, Goldstein, & The MacArthur Working Group on Outcome Assessment, 2003). The HBQ contains 25 items that tap pure externalizing, free of impulsivity/inattention (e.g., "Has temper tantrums or hot temper"), and 23 that tap behavior competence (e.g., "Can work easily in a small peer group"). See Appendix C. Test-retest reliability and validity of the HBQ has been demonstrated across multiple reporters in middle childhood (Ablow et al., 1999; Essex et al., 2002; Lemery-Chalfant et al., 2007). Parents rated the twins' behavior on a three point scale of 0 (*Rarely applies*), 1 (*Applies somewhat*), and 2 (*Certainly applies*). The externalizing items had a reliability of .91 for both mother and father report, while the reliability for the competence items was .90 for both mother and father report.

To be consistent with T1, mother and father report were kept separate at T2 as well. The externalizing and competence items from the HBQ were entered into a principle components analysis specifying a one factor solution. For mother report, this factor accounted for 26.29% of the total variance with an eigen value of 12.62. Two items loaded under the .30 threshold (at .17 and .22), but were retained to remain consistent with the literature. For father report, one factor accounted for 24.16% of the total variation with an eigen value of 11.60. Again, two items loaded under the .30 threshold (.19 and .22) but were retained for consistency. For the combined externalizing/competence

scale, the reliability was .94 for mother report and .93 for father report. A mean composite of externalizing/competence for each parent was formed where lower scores indicate more competence behavior and high scores indicate more externalizing behavior. At T2, there were 15 outliers for mother report and 17 for father report, all above the upper bound. These cases were winsorized to the 99th percentile.

At T3, both parents again completed the externalizing and competence scales of the HBQ. The principal components analyses (separate for each reporter) again supported combining externalizing and competence. For mother report, a one factor solution accounted for 28.00% of the variance with an eigen value of 12.88. Four mother report items loaded only marginally (ranging from .15 to .24), these were retained for consistency across waves and previous studies. A one factor solution accounted for 25.86% of the total variance with an eigen value of 13.19. Four items loaded below the .30 threshold (ranging from .15 to .25) for father report, these were also retained. The reliability for the competence items was .93 for mother report and .92 for father report. Alphas for the externalizing items were .91 for mother report and father report. Competence items were reverse-coded and a mean composite of externalizing/competence for each parent was formed, where lower scores index competence behaviors and higher scores index externalizing behaviors. Reliability for the full continuum was .94 for mother and father report. At T3, there were 10 cases above the upper bound for mother report and seven above the upper bound for father report, these cases were also winsorized to the 99th percentile.

See Table 3 for phenotypic correlations between warmth/rejection and externalizing/competence at each time point. Correlations were computed using the cluster option in Mplus v. 5.1 (Muthén & Muthén, 2008) to account for clustering due to twin dependence. Correlations between T1 and T3 were not calculable, due to low *N*s, and so are not shown. All variables were significantly correlated in the expected direction (i.e., higher *SES* related to more parental warmth and higher *SES* and warmth related to fewer externalizing problems) within time.

Dopamine

Genotypes on the dopamine receptor gene VNTR were normally distributed (skew less than two and kurtosis less than seven), and ranged from 2/2 to 7/8 where each number represents the number of repeats of DRD4 on one allele for a given individual. DRD4 was coded and analyzed dominantly for the long allele (i.e., equal to or greater than six repeats) with heterozygotes expected to be indistinguishable from individuals with two long alleles (Plomin & Rutter, 1998). Under dominant coding all individuals with one allele equal to or greater than six repeats were compared to those with both alleles having fewer than six repeats. Individuals with two short alleles (2, 3, 4, 5) on DRD4 are typically compared to those with one or two long alleles (6, 7, 8). Approximately 70% of individuals in the population have a short/short genotype and 30% have a long/short or long/long genotype (Plomin & Rutter, 1998). In the current sample, 68.2% of children had the short/short genotype compared to 31.8% with at least one long allele. See Table 4 for sample sizes of all gene variants.

Vasopressin

AVPR1a was analyzed both dominantly for the short allele (i.e., less than or equal to 325 bps) compared to individuals with two long alleles (greater than 326 bps) to replicate previous literature (Knafo et al., 2008) and continuously, using a sum score across both alleles to investigate whether the highly polymorphic nature of the variant should be taken into account when analyzing this gene variant. Summing across both alleles resulted in a normal distribution ranging from 637.90 bps to 687.70 bps. Seven scores were outside of the lower bound and three were outside of the upper bound, these were winsorized and a Z score was formed to create a centered predictor. AVPR1a dominant coding stipulates coding those with 326 bps as missing, thus for the continuous coding, the sample size increased from 760 to 867.

Neuropeptide-Y

For NPY, I replicated Zhou and colleagues' (2008) haplotype coding for the six NPY SNPs, resulting in five common haplotypes. See Table 5 for haplotype details and frequencies from the current study as well as from the study by Zhou and colleagues (2008). Of individuals who had any SNP data for NPY, 82.3% had complete data across all six SNPs for haplotype determination.

Genotyping methods did not include information on parent-child transmission of alleles (i.e., gametic phase), therefore NPY haplotypes were estimated using the ELB algorithm (Excoffier et al., 2003) in Arlequin 3.5.1.2 (Excoffier & Lischer, 2010). In this program, the algorithm is a pseudo-Bayesian approach that estimates gametic phase and allows for further

analysis of genotype data as if phase was known. This algorithm uses nearby loci as well as the level of linkage disequilibrium (the nonrandom association of alleles at various loci) to estimate phase. Since phase is unknown, linkage disequilibrium is tested using a likelihood-ratio test with a permutation procedure that corrects for biases from small Ns and large number of variants. The purpose of using this algorithm is to assign the two variants on each of the six SNPs within the NPY gene to one haplotype resulting in two six SNP haplotypes, one inherited from each parent.

Within the ELB framework, I specified the recommended dirichlet prior alpha value (the parameter of the symmetric prior distribution of the haplotype frequencies, with smaller values indicating less chance of choosing a haplotype pair that includes an unobserved haplotype) of .01 which has been found to work well with all types of data (Excoffier et al., 2003), an epsilon value (amount a haplotype differing by one variant from present haplotypes is weighted) of .01, and a sampling interval between steps in the Gibbs chain of 500. Giving the distribution time to normalize, after 100,000 iterations (the burnin), every 2000th sample in the Gibbs chain was drawn to create the posterior distribution of haplotype frequencies for each twin. The overall distribution of these five haplotypes in the current study was not significantly different from previously published estimates (χ^2 [df= 4, individual child N= 1505] = 8.62, p = .07).

The ELB algorithm also assumes Hardy-Weinberg equilibrium (a state in which both allele and genotype frequencies are constant across generations) across the variants (Excoffier & Slatkin, 1998), which is expressed as $p^2 + 2pq$

+ q^2 where the frequency of the dominant homozygote is p^2, the frequency for a heterozygote is 2pq and the frequency for a recessive homozygote is q^2. This is calculated in Arlequin using a Markov chain (for all loci) to compare observed heterozygotes to expected heterozygotes using the chi-square distribution. All NPY SNP frequencies were in Hardy-Weinberg Equilibrium (*p* values greater than .05) with the exception of the insertion/deletion polymorphism rs3037354 (expected heterozygotes: .50, observed heterozygotes: .34; $p = .048$). Although this may have a slight impact upon the formation of the NPY haplotypes, the primary implication of a SNP out of Hardy-Weinberg Equilibrium for Arlequin is for assigning phase to a posterior distribution of samples for further genetic analyses within the program. I did not use Arlequin for further analyses and did not treat haplotypic data as phased (i.e., haplotype 1 and 2 for each individual were treated as interchangeable).

Neuropeptide-Y haplotypes were analyzed at the diplotypic level. Diplotypes are composed of an individual's two inherited haplotypes. The three most frequent haplotypes accounted for 91.3% of the haplotypic data; these haplotypes were recoded into the six possible diplotypes (i.e., H1/H1, H2/H2, H3/H3, H1/H2, H1/H3, H2/H3). See Table 6 for diplotype *N*s and frequencies. The previous study using NPY diplotypes (Zhou et al, 2008) examined NPY mRNA levels across diplotypes and found that the six common diplotypes combine into three groups: low NPY expression in the brain (diplotype 1), medium expression (diplotypes 3, 4, and 5) and high expression (diplotypes 2 and 6). In the current sample, there were 160 low

expression children, 365 medium expression children, and 106 high expression children. Diplotypes were coded as -1 (low expression), 0 (medium expression), or 1 (high expression).

Procedure

Using birth records, all twins born within the state of Wisconsin were invited to participate in the Wisconsin Twin Project. Importantly, at the start of the project *all* twins who would age into either a toddler or middle childhood (age eight) assessment were invited to participate. As a result, there are two separate cohorts within the project; children who began with the project in toddlerhood and who are currently leaving middle childhood (Cohort 1), and children whose first assessment was in middle childhood and who are now in adolescence (Cohort 2). Adolescent data collection is currently underway for Cohort 1. Genetic data are currently only available for Cohort 2, although DNA extraction and typing for Cohort 1 is also underway. Van Hulle and colleagues (2002) reported that, of the 750 twin births occurring yearly in the state, 60-80% of families responded to the initial recruitment letter, with an additional 10-15% responding to the follow-up letter. Families who did not respond to the mailed recruitment letters were telephoned, approximately 70% of those families agreed to take part in the project.

I am using data from both Cohort 1 and 2 for this study. T1 data were collected when the twins were approximately two years of age. Mothers and fathers completed mailed questionnaires, and trained research assistants conducted telephone interviews with primary caregivers. At T2, the primary caregiver of the twins (now approximately eight years of age) was

interviewed via telephone for internalizing, externalizing, and attention problem scales using the HBQ. If either twin scored at or above 1.5 standard deviations above the mean on a given problem behavior scale, that family was placed in an at-risk group for that respective problem behavior. A comparison group was also formed with twins who scored below the mean on all of the three problem behavior scales. Approximately one quarter of the available T1 families qualified for an at-risk group at T2, with another quarter meeting the criteria for the comparison group. All cotwins of at-risk and comparison twins were included in the sample, regardless of whether they met criteria, with 95% retention of selected families (Van Hulle et al., 2002).

This selection procedure at T2 was intended to provide a range of behavior by enriching the sample with high-risk children, but analyses in selected samples can yield biased estimates (Heckman, 1979). Potential biases were addressed by including all cotwins of selected children, and additionally by sampling a comparison group of competent children and their cotwins. In addition, sample selection occurred only at T2; so potential biases should be diminished by T3. Furthermore, there was no selection at T1, and thus estimates of externalizing/competence and parent warmth at T1 can be compared to T2 estimates to gauge if there is a selection bias at work.

T2 consisted of mailed questionnaires for the parents, telephone interviews, and a five hour home visit. During this visit, buccal cells were obtained using cheek swabs for each family member for later DNA analysis. DNA extraction from the cheek swabs was completed on MasterPure DNA

kits from Epicentre Biotechnologies at the Translational Genomics Research Institute (SNP data) and at the University of Wisconsin-Madison Biotechnology Center (VNTR data). For SNP data, individuals were genotyped using Sequenom technology, which employs mass spectrometry as well as second generation DNA sequencing, a technology which allows for the direct reading of the sequence of bases that make up the strands of DNA. Mass spectrometry sequencing yields high resolution fragments, rapid separation of strands, and is well-equipped to identify mutations and single nucleotide polymorphisms (Edwards, Ruparal, & Ju, 2005). A total of 11 plates on Direct Lysis Plasmid96 DNA Purification Kits were used; each included two positive CEPH (Centre d'etude du polymorphisme humain) controls from the Utah reference sample (CEU) of the International HapMap Consortium (2005) and four randomly chosen intraplate replicates. CEPH controls were dispensed in two wells in each of the 11 plates to ensure a positive DNA control and to aid in future DNA target amplification. Each plex included a plate of CEPH trios to determine HAPMAP concordance and to identify Mendelian errors. Hardy-Weinberg results (a Mendelian check) were reported above (see the Neuropeptide-Y section). VNTR data were extracted and sequenced using Sanger sequencing on 96-well plates on three Applied Biosystems 3730xl DNA analyzers. Further information on current genetic typing methodologies can be found in Edwards and colleagues (2005) paper on mass-spectrometry DNA sequencing or Tang and colleagues (1999) paper on chip-based genotyping methods.

At T3 (when the twins were approximately 14 years of age), questionnaire packets were again mailed to mothers, fathers, and adolescents and telephone interviews were conducted with primary caregivers. An additional home visit was conducted with all participating family members. Data collection is currently ongoing. At present, 392 families have participated in this wave of data collection.

Statistical Analyses

Heritability of externalizing/competence at each age (Hypothesis 1) was tested by fitting six univariate Cholesky models (ACE models), one for each parent report of externalizing/competence at each time: T1, T2, and T3. These models decompose the variance in externalizing/competence at each time point for each reporter into latent genetic (A), shared environment (C), and nonshared environment (E) components using the structural equation modeling package Mx version 1.7.03 (Neale, Boker, Xie, & Maes, 1999). These latent factors are defined *a priori* by structural constraints in the multigroup model. Genetic effects are a result of shared genes between the twins whereas shared environment effects are aspects of the environment that serve to make the twins more similar and nonshared environment effects are those that create differences between the twins. The nonshared environment component also contains measurement error. See Figure 2 for an illustration of the basic twin model. In essence, this univariate Cholesky model is a multigroup structural equation model that estimates the path coefficients a^2, c^2 and e^2 for the latent variables A, C, and E on a measured trait in both twins by programming matrices and pathways to fit the assumptions of the

twin model. Specifically, in Group 1 (MZ twins), the twin pair is assumed to share 100% of the genetic component (A) and 100% of the shared environment component (C), so both of these paths between twin 1 and twin 2 in Group 1 are set to 1.0. In Group 2 (DZ twins), the twin pair is assumed to share 50% of the genetic component (A) and 100% of the shared environment component (C), so these paths are set to 0.5 and 1.0, respectively. In both groups, the E paths are allowed to freely vary, to account for unshared environmental influences and error. The most parsimonious model for the data is determined using a nested modeling approach, wherein the path coefficients are first fully estimated across both groups for ACE and then a series of nested models (AE, CE, and E) are fit. The E pathway is never dropped as it contains measurement error. The best fitting model is then the most parsimonious model that is not a significantly worse fit to the data (gauged via the chi-square for the nested model and the Akaike's Information Criterion). If, for example, a CE model emerges as the most parsimonious fit to the data, the conclusion drawn would be that externalizing/competence is solely a function of shared and nonshared environmental effects, and that there is no genetic component to externalizing/competence.

In the presence of an unmeasured gene-environment interaction, the heritability estimate (a^2) of a given trait would be an average of the actual heritability estimates for all individuals in the population (e.g., if in half the population the heritability is 50% and in the other half of the population the heritability is 100%; the overall heritability estimate would appear to be 75%). The ability of parental warmth to moderate the heritability of

externalizing/competence (Hypothesis 3) was examined using latent genetic effect (*G*) by continuous environment (*E*) gene-environment interaction variance components models in Mx (Neale et al., 1999). A continuous environmental moderator tests whether heritability increases or decreases as a linear function of the moderator (Purcell, 2002), additionally warmth is tested as a moderator of the shared environment and nonshared environment variables. Importantly, the linearity modeled is at the level of the effect, not the variance component. In the moderated model, the path coefficients a^2, c^2 and e^2 become $(a + B_XM)^2$, $(c + B_YM)^2$, and $(e + B_ZM)^2$ where B_X represents the effects of the moderator (parental warmth) on additive genetic variance, B_Y represents the effects of the moderator on shared environment variance, and B_Z represents the effects of the moderator on nonshared environmental variance. Each source of influence is now represented by a constant plus a coefficient multiplied by parent warmth. When combined, these three pathways sum to the total variance in any given twin's externalizing/competence. An interaction of A, C, or E is represented by a non-zero B_X, B_Y, or B_Z, respectively. Main effects are tested in the presence of interaction effects by allowing warmth in the means model (B_M).

In order to probe moderation effects the data were re-centered at three different levels of the moderator; +1 *SD* warmth (mother or father, dependent upon the interaction), mean level warmth, and -1 *SD* warmth. Within these three levels of centering, the best-fitting model from the univariate analyses was again fit to the data with nested models dropping the moderation term (B_X, B_Y, B_Z) from the model. If an AE model was the most parsimonious fit to

the data, for example, moderation was tested by allowing both the B_X and B_Z terms in the model, then removing each in two separate nested models, and finally removing both terms at the same time. If removing either or both moderation terms resulted in a significantly worse fit to the data then moderation was significant.

Given that the three moderated pathways are parts of a proportion score and must sum to 1.0, moderation of one pathway could be misspecified. For example, the heritability of externalizing/competence (B_X) could appear as a non-zero B_Y or B_Z. Purcell (2002) tested this idea using a series of three simulated data sets of MZ and DZ twins (each set specifying a true model with one moderated pathway [B_X, B_Y, or B_Z], replicated 50 times) and found that the correct model was selected the majority of the time using the lowest AIC for each simulated set. Although misspecification of the effect did occur (e.g.; when the moderation was on B_Z it was attributed to B_Y and B_Z in 12 instances), it was not considered problematic because the unmoderated model was never selected.

All other hypotheses were tested using the methodology set forth by Olsen and Kenny (2006) for structural equation modeling with interchangeable dyads in Mplus v. 5.1 (Muthén & Muthén, 2008); maximum likelihood estimation with robust standard errors was used to account for missing data. Twin pairs are considered interchangeable dyads because there is no consistent way to order Twin A and Twin B (Olsen & Kenny, 2006). Under this framework, each twin's predictor variable is estimated on both their own outcome and their cotwin's outcome. To estimate corresponding models for

each twin, equality restrictions are placed upon parameters. Regression coefficients are constrained to be equal across twin and cotwin externalizing/competence. For example, the effect of SES on one twin's externalizing/competence is held to be the same as the effect of SES on the cotwin's externalizing/competence. Additionally, the predictor means, variances and covariances of twin-specific predictors are held to be equal. SES and mother and father warmth are not twin-specific (i.e., they do not differ across twin and cotwin pairs), but sex (in opposite-sex DZ pairings) and gene variant (in DZ twins only) can differ across dyad members and thus the means, variances, and covariances are constrained to be equal when included in a model. Finally, equality restrictions across the twin and cotwin are also placed on the intercepts and error variances of externalizing/competence. Using these constraints, externalizing/competence was predicted from SES, child sex, cotwin sex, mother warmth, and father warmth in the concurrent main effects models.

When measured genes were included in the model, externalizing/competence was also predicted by the twin's and the cotwin's gene variant and the interaction between the twin's variant and warmth and the cotwin's variant and warmth. A limitation of this method is that the twin's GxE interaction is considered in the context of the cotwin's GxE interaction (a highly related term given that the environment is fully shared between the twins and the gene variants are fully shared among the MZ subset and similar among the DZ subset of twins). The possible significance of the cotwin's genotype by warmth interaction on the twin's behavior was

not hypothesized and for the purpose of this study is considered a statistical artifact. These terms will not be interpreted. Longitudinally, an individual's externalizing/competence was predicted from concurrent SES, child sex, cotwin sex, warmth at the previous time point, externalizing/competence at the previous time point, and both the child and the cotwin's genotype.

There was little ethnic variability in the sample (92% Caucasian), thus, ethnicity did not have enough variability to be included in the models as a covariate. To account for population stratification (differences in genetic association across ethnic groups), all models including measured gene variants were tested first in the full sample and then retested using the Caucasian-only subsample. Any differences were noted in the respective table.

Non-dichotomous predictor variables were grand mean centered if used as part of an interaction effect. Interactions were computed and probed using guidelines set out by Aiken and West (1991). Figure 4 illustrates an example of an interaction effect between T2 mother warmth and DRD4 on T2 mother report externalizing/competence with these constraints. Standardized estimates were reported from all analyses.

Results

Hypothesis 1

The first hypothesis was that externalizing/competence would be heritable at all ages (toddlerhood, middle childhood, and early adolescence), and that genetic influences would increase with age while shared environmental influences would decrease with age. This hypothesis was

tested by fitting six ACE models decomposing the variance in the externalizing/competence continuum at each time point (T1, T2, and T3) for each reporter into genetic (A), shared environment (C), and nonshared environment (E) components using the structural equation modeling package Mx version 1.7.03 (Neale et al., 1999). See Statistical Approach above for a description of the ACE model.

Across all three time points and mother and father reporters, the AE model was the best fit to the data (see Table 7), as this model did not fit significantly worse than the ACE model gauged by a nonsignificant chi square change and the lowest Akaike's Information Criterion fit index. Behavior was found to be a function of additive genetic and nonshared environmental influences, with no noticeable contribution of the shared environment. Genetic influences were relatively stable across the time points and there was never significant contribution of the shared environment. Hypothesis 1 was partially supported; externalizing and competence behaviors were significantly heritable at all ages; however, there was no evidence of increasing heritability with age or of a shared environmental component.

Hypothesis 2

My second hypothesis was that parental warmth/rejection would be a significant predictor of change in externalizing/competence, with higher levels of warmth (lower rejection) at one age predicting fewer externalizing behaviors and more competencies at the next time point controlling for earlier externalizing/competence. Constrained structural equation models (as

described under Statistical Approach and illustrated in Figure 4) were fit to the data to examine both concurrent relations of warmth to externalizing/competence and the longitudinal relations of warmth to change in externalizing/competence from one time to the next. Child sex, cotwin sex, and *SES* were included as covariates.

Sex consistently related to differences in externalizing/competence across models and times, with boys displaying higher levels of externalizing than girls (boys were coded as 0, girls as 1). Concurrently, children with a lower *SES* were also higher on externalizing in all 18 models. Longitudinally, *SES* remained a significant predictor of mother report externalizing/competence, but not of father report externalizing/competence. Mother warmth was concurrently related to externalizing/competence in all models when tested independent of father warmth, and remained significant in five out of six models when tested together with father warmth. Father warmth was significant in five out of six concurrent models when tested independent of mother warmth, and remained significant in four out of six models when tested together with mother warmth (see Table 8).

Toddlerhood warmth predicting middle childhood behavior was tested, as was middle childhood warmth predicting adolescent behavior, but toddlerhood warmth to adolescent behavior was not tested due to sample size limitations (see Method). Father warmth was hypothesized to be a stronger predictor than mother warmth, and warmth was hypothesized to decrease with age. Contrary to my hypothesis, mean level mother and father warmth did not decrease with age (see Table 2). Warmth appears to increase with

child age, but these differences were not significant when examined using a univariate ANOVA with a Bonferroni post-hoc adjustment to examine group differences (e.g., T1 warmth compared to T2 warmth).

Longitudinally, middle childhood externalizing/competence was most strongly predicted across reporter by toddler externalizing/competence (see Table 9). T1 father warmth was a marginal predictor in all four models tested of externalizing/competence in middle childhood, over and above the influence of toddlerhood behavior (both mother and father report) such that more father warmth in toddlerhood predicted marginally fewer externalizing problems and more competencies in middle childhood. In middle childhood, mother warmth predicted decreased adolescent externalizing (mother report) in two of four models testing mother warmth (see Table 10). Hypothesis 2 was also partially supported in that warmth generally related to decreased externalizing both within and across times. Contrary to my hypothesis, parental warmth did not decrease with increased child age, nor did parents report differing levels of warmth dependent upon child sex.

Hypothesis 3

Having tested the main effects of mother and father warmth on child behavior, the next hypothesis was that warmth/rejection would moderate the heritability of child behavior (see Figure 3). Purcell's (2002) gene-experience interaction model in Mx was used to test this hypothesis, as it is appropriate for continuous variables and non twin-specific moderators (Medland, Neale, Eaves, & Neale, 2009; see Statistical Approach, above). Since the AE model was the best-fitting model for all time points and reporters, a series of nested

models were fit comparing the AE model with moderation (full model) to nested models— AE with only the A path moderated, AE with only the E path moderated, and AE with no moderation.

It is important to note that warmth/rejection could appear to be a significant moderator of the heritability of externalizing/competence as a result of limited phenotypic variability in externalizing/competence in some environmental contexts. Before testing moderational models, homogeneity of variance by environmental condition across the outcome variables was tested. A series of dummy codes were created for each index of the environment (i.e., mother and father warmth/rejection at T1, T2, and T3) where indices of parental rejection (-1 SD on warmth) were given a 0 and all other environments (average to high warmth) were designated with a 1. Randomly selecting one twin from each pair and then repeating analyses with cotwins, the Levene's test for equality of variance was nonsignificant for differences across rejecting and warmer environments on all reports of externalizing/competence except father report of externalizing/competence in toddlerhood as predicted by father report of warmth ($F[2,390] = 4.38$, $p = .01$). For father report of externalizing/competence, there was significantly less variance in the rejecting environment (0.05) than in warmer environments (0.06). This significant difference in variance could have led to an underestimation of the C component at T1; however, given that mother report of externalizing/competence at T1 also fit an AE model and the parenting environment did not relate to limited variability in mother report of externalizing/competence, this is unlikely to be a significant limitation.

Additionally, warmth at an earlier time point was not expected to moderate the heritability of externalizing/competence at the next time point, however these models (T1 warmth moderating T2 externalizing/competence and T2 warmth moderating T3 externalizing/competence) were tested within mother and father report to determine if it was necessary to control for the effect of earlier warmth in moderation models. Using the same model-fitting procedure described in the Statistical Analyses, T1 warmth was not a significant moderator of T2 externalizing/competence. T2 warmth was, however, a significant moderator of T3 externalizing/competence (with the exception of mother warmth on father report of behavior), and so T2 warmth was included as a covariate in analyses of T3 warmth moderating the heritability of T3 behavior.

See Table 11 for parent warmth moderated model-fitting results. Neither mother nor father warmth moderated the genetic or nonshared environment influence on externalizing/competence in toddlerhood. Mother warmth was a significant moderator for mother report of externalizing/competence at T2; however, power was not sufficient to distinguish between A only and E only moderation (both nested models yielded nonsignificant decreases in fit in comparison to the full AE moderated model). Examining the A, C, and E variance components across three different levels of mother warmth (-1 *SD*, mean level, and +1 *SD* warmth) showed that externalizing/competence was largely a function of the nonshared environment when mother warmth was low (E = 0.76) whereas behavior was primarily a result of additive genetic influences when mother warmth was average or high (A = 0.73 at both

average and high mother warmth; see Figure 5). In middle childhood, father warmth moderated the E path for mother report of externalizing/competence such that in a more negative environment (-1 *SD* on father warmth), the nonshared environment had less influence on child behavior (E = 0.23) than it did in a more positive (mean warmth or +1 *SD* warmth) environment (E = 0.27 and 0.31, respectively; see Figure 6). Neither mother nor father warmth significantly moderated father report of child externalizing/competence. Note that differences between estimates of the AE model with no moderation across reporter in Table 11 and the AE model with no moderation in Table 7 are a result of slightly different samples (i.e., the *N* for individuals with mother report of externalizing/competence and mother warmth at T1 differs slightly from the *N* for individuals with mother report of externalizing/competence and father warmth at T1).

In adolescence, mother warmth moderated both the A path on mother report of externalizing/competence and the E path on father report of externalizing/competence. For mother report of externalizing/competence (see Figure 7), in a negative environment (-1 SD on mother warmth) adolescent behavior was driven more by nonshared environmental influences (E = 0.42), in an average environment (mean level mother warmth) the A and E components shared the variance (0.66 and 0.34, respectively), while in an advantaged environment (+1 SD on warmth) adolescent behavior was largely a function of additive genetic influences (A = 0.73). Father report of externalizing/competence showed the opposite pattern of results (see Figure 8). When mother warmth was low, behavior was largely determined by

additive genetic influences (A = 0.81). The nonshared environment component increased as mother warmth increased, and shared variance fairly equally with the genetic component in a positive environment (high mother warmth); A = 0.57, E = 0.43.

Hypothesis 3 was largely supported for mother warmth on mother report of outcome in T2 and T3, with low warmth/high rejection environments lending themselves to behavioral outcomes that are more driven by environmental than additive genetic influences. When father warmth was the moderator or when the outcome was father report, the patterns reversed. Four out of 12 moderated models were significant (33%). There was no evidence of warmth moderating the heritability of externalizing/competence in toddlerhood.

Hypothesis 4

A VNTR in the dopamine receptor gene was next examined in interaction with warmth to predict behavior in children and adolescents. I hypothesized that individuals with a long allele on DRD4 (i.e., equal to or greater than six repeats) would be more susceptible to environmental influences than those with the short version. When exposed to low parental warmth, children with a long allele on DRD4 should display significantly more externalizing than children with two short alleles, but when exposed to high parental warmth, children with a long allele on DRD4 should display significantly more competence behaviors than children with two short alleles.

Gene variants were not tested in toddlerhood due to cohort constraints (the toddlerhood sample did not have sufficient overlap with the genotyped

subsample, see Method). Main effects of the measured genes were tested separate for each gene in constrained structural equation models that included SES, child and cotwin sex, and child and cotwin gene. Warmth/rejection by measured gene interactions were examined by extending the constrained structural equation models to include an interaction term for twin genotype by warmth and one for cotwin genotype by warmth, see Statistical Approach in Method

Controlling for twin and cotwin sex and *SES*, there was no main effect of twin DRD4 on mother or father report of behavior at T2 or T3 (see Table 12). At T3, twin DRD4 marginally predicted mother report of behavior, only in the Caucasian subsample (*Estimate* = 0.07, *SE* = 0.04, *p* = .09). Having the longer variant on DRD4 marginally related to increased mother report of externalizing behaviors and fewer competencies in the Caucasian subsample of one out of four main effect models. DRD4 did not predict father report of adolescent behavior.

See Table 12 for concurrent gene x environment interactions in both childhood and adolescence. Although there were no main effects of DRD4 in childhood, there was a marginal interaction at T2 with concurrent mother warmth by twin DRD4 on mother report of externalizing/competence in the Caucasian-only subsample (see Figure 9). Using guidelines set forth by Aiken and West (1991), simple slopes were estimated for each group on the dichotomous moderator using the maximum likelihood estimates (children with two short alleles on DRD4 and children with at least one long allele on DRD4). As illustrated in Figure 9, children with a long allele on DRD4 were

more susceptible to their environment (mother warmth) than children with two short alleles [s/s: B = -0.15 (SE = .02); s/l and l/l: B = -0.27 (SE = .04)].

There was also a significant interaction in the Caucasian-only subsample of concurrent father warmth by twin DRD4 on mother report externalizing/competence (see Figure 10). This mirrors the earlier interaction of mother warmth by twin DRD4 (Figure 9), with the long allele relating to more susceptibility to father warmth [s/s: B = -0.10 (SE = .02); s/l and l/l: B = -0.22 (SE = .04)]. Concurrent father warmth by twin DRD4 also interacted in relation to father report externalizing/competence, again in the Caucasian subsample (see Figure 11). This interaction is consistent with the two previous interactions, with the long allele relating to increased susceptibility to father warmth [s/s: B = -0.14 (SE = .02); s/l and l/l: B = -0.23 (SE = .04)].

Concurrently at Time 2, three out of four interactions between warmth and twin DRD4 status were significant in the Caucasian subsample. Concurrently at Time 3, there was no significant interaction of twin DRD4 and warmth (see Table 12). Longitudinally, there were no significant interactions with middle childhood warmth by DRD4 to predict adolescent behavior (see Table 13). A total of 12 interactions were tested with DRD4, three were significant (25%). Hypothesis 4 was partly supported, in that the long allele of DRD4 related to increased plasticity in middle childhood only.

Hypothesis 5

I next tested whether a highly polymorphic VNTR in the vasopressin receptor gene predicted child or adolescent behavior. Warmth/rejection by measured gene interactions were tested in the same fashion as with DRD4.

AVPR1a was coded both dominantly for the short allele (i.e., less than or equal to 325 bps) compared to individuals with two long alleles (greater than 326 bps) to replicate previous literature (Knafo et al., 2008) and continuously, using a sum score across both alleles to investigate whether the highly polymorphic nature of the variant should be taken into account when analyzing this gene variant, see description given in Method. Again, children with longer variants of AVPR1a were hypothesized to be more susceptible to parental warmth/rejection at all ages than children with shorter variants.

Controlling for twin and cotwin sex and *SES*, neither dominantly- nor additively-coded AVPR1a predicted mother or father report of behavior at T2 or T3. There were no main effects of the gene on behavior. Table 14 contains estimates for concurrent relations of T2 warmth by AVPR1a (dominantly coded) on externalizing/competence in both childhood and adolescence. There were no significant interactions between T2 warmth and twin AVPR1a (dominantly-coded) on child behavior. There were also no significant interactions between T3 warmth and twin AVPR1a (dominantly coded).

When AVPR1a was coded additively (see Table 15), one interaction was significant across four models in middle childhood and four models in adolescence. Figure 12 depicts a significant interaction in the Caucasian subsample of mother warmth by twin AVPR1a (additively coded) on mother report of externalizing/competence at Time 2. All groups of AVPR1a started high on externalizing under conditions of low mother warmth, but the decline of the simple slope became increasingly sharper as the length of the twins'

AVPR1a alleles increased [-1 SD: B = -0.08 (SE = .02); mean length: B = -0.12 (SE = .02); +1 SD: B = -0.15 (SE = .03)].

Longitudinally, there were no significant interactions of T2 mother warmth by twin AVPR1a (dominantly coded; see Table 16). When AVPR1a was coded additively (Table 17), the interaction between T2 father warmth and twin AVPR1a on mother report of externalizing/competence was significant (see Figure 13) in the Caucasian subsample. It appears that adolescents with longer AVPR1a variants show more externalizing as a result of low father warmth in childhood and less externalizing as a result of high father warmth in childhood, and adolescents with shorter AVPR1a variants seem to display less externalizing when exposed to low father warmth in childhood and more externalizing when exposed to high father warmth but none of the simple slopes were statistically significant. Under the dominant and additive coding systems, zero and one out of four longitudinal models, respectively, yielded a significant interaction.

Hypothesis 5, that the long variant of AVPR1a would convey additional plasticity to the environment, was moderately supported. A total of 24 interactions were tested with AVPR1a (12 for each of the two coding schemes), two were significant (8%), with an additional two showing trends (8%). Concurrent (in middle childhood only) mother warmth and longitudinal father warmth interact with AVPR1a (additive coding) to predict mother report of externalizing/competence.

Hypothesis 6

The final hypothesis was that individuals with variants of the NPY gene that code for higher levels of neuropeptide-Y in the brain would display fewer externalizing behaviors overall and be more resilient in the face of parental rejection. Zhou and colleagues (2008) diplotype coding was used, combining each individual's haplotypic data, such that diplotypes that relate to low NPY expression in the brain were coded as -1, diplotypes that relate to average NPY expression were coded as 0, and diplotypes that relate to high NPY expression were coded as 1, see Method.

Using the diplotype coding, there was a main effect of NPY on mother report of externalizing/competence in middle childhood, controlling for twin and cotwin sex and *SES* (*Estimate* = -0.09, *SE* = 0.04, $p = .04$), such that children with diplotypes that code for higher NPY expression display fewer externalizing and more competence behaviors. This effect became marginal in the Caucasian-only subsample. There was a marginal main effect of NPY on father report of externalizing/competence in middle childhood in the full sample (*Estimate* = -0.06, *SE* = 0.04, $p = .09$), that remained marginal in the Caucasian subsample. There were no significant main effects of NPY in adolescence.

There were no significant NPY x parenting interactions on externalizing/competence concurrently in middle childhood or adolescence or longitudinally (24 interactions tested). Hypothesis 6, that individuals with higher functioning NPY diplotypes would display fewer externalizing/competence behaviors in general and be more resilient in the

face of low parent warmth was largely not supported. NPY diplotypes only had a main effect on externalizing/competence in childhood, not in adolescence, and NPY diplotypes did not interact with parent warmth.

Discussion

Placing the Results in Context

The overarching aim of this study was to examine the gene-environment interplay among mother and father warmth and variants in the DRD4, AVPR1a, and NPY genes on externalizing/competence behavior across development. Traditionally, research in the fields of clinical and developmental psychopathology has been informed by the diathesis-stress hypothesis, and in this study the efficacy of an alternative approach, the differential susceptibility hypothesis (Belsky & Pluess, 2009) was tested by creating continuums of both parental warmth (warmth to rejection) and child behavior (externalizing behaviors to competence behaviors). This approach circumvents the search for 'risk' factors by reframing individuals as either more or less susceptible to their environment, both beneficially and detrimentally. This section parallels the Results by first framing the heritability results, followed by the moderated heritability models, and last the phenotypic relations of warmth to externalizing/competence and the measured gene-experience interactions with DRD4, AVPR1a, and NPY in the context of the differential susceptibility hypothesis.

First, the heritability of behavior at each time point was examined in both univariate main effect and parental warmth moderated models. In toddlerhood, middle childhood, and early adolescence, for both mother and

father report of behavior, externalizing/competence was a function of additive genetic and nonshared environmental influences, with no evidence of a shared environmental influence. A strength of the current findings is the across-reporter consistency. Previous twin studies using the same measure of behavior (but examining externalizing and competence in separate models) have indicated that in toddlerhood competence behaviors are more likely to be influenced by the shared environment (C) early in life (Saudino et al., 2008; Van Hulle et al., 2007) while externalizing is a function of additive genetic and nonshared environmental influences (Van Hulle et al., 2007). Studies of older children find that the influence of the shared environment drops off with age (Bell & Deater-Deckard, 2007; Deater-Deckard et al., 2005; Rushton et al., 1986; Zahn-Waxler et al., 1992).

The lack of a significant influence of the shared environment is typical in the behavior genetics literature across a multitude of behaviors (e.g., prosocial behavior: Gregory, Light-Hausermann, Rijsdijk, & Eley, 2009; effortful control: Lemery-Chalfant et al., 2008; self-esteem: Neiss et al., 2002). Studies typically agree that the significant effect of the environment is in explaining why twins differ (E) not why they are similar (C) and that these environmental effects are time specific; that is, they do not contribute to the continuity of behavior but instead account for variance within time and the environmental effects that are important at one time are not necessarily important at later times (Hoekstra et al., 2007). Often heritabilities emerge or notably increase as children enter the school environment (Deater-Deckard et al., 2005) leading to the explanation that the early rearing environment is

particularly important for behavior (Hoekstra et al., 2007) or to the alternative explanation that standardized schooling creates a standard environment for children, diminishing variance, which inflates the genetic component since A, C, and E represent the three parts of a proportion score. As twin models apportion the total variance of any trait into the three variance components, if one component increases or decreases for any reason, the variance attributed to the other two components must adjust to maintain the proportion.

A further layer of complexity is that variation due to the shared environment is difficult to properly model using current techniques (Posthuma & Boomsma, 2000), and when additive genetic influences are greater than shared environment influences, variance attributable to the shared environment is particularly hard to detect. A simulation study of the power of family designs found that for a twin sample with a true effect of A = 0.50, C = 0.20, and E = 0.30; a sample size of just under 330 individuals was required for 80% power to detect A whereas a sample size of just over 2000 individuals would be required to properly estimate C (Posthuma & Boomsma, 2000). With power in the current samples, a true C effect of 0.30 could have been detected, but a lesser influence of C may have been missed. Power for detecting a C effect can be increased by adding an additional sibling to the model (Posthuma & Boomsma, 2000). Future studies could benefit from collecting data on all children in the family.

Given the low power to detect a significant C, some behavior genetics theorists question even the basic assumptions of the ACE model, with one

going so far as to offer a prize to the first person to demonstrate that C and E are legitimate variables in their own right (Carey, 2009) and can be teased apart. Carey's point regarding whether the environment can be separated into two components (C and E) is a good one, in light of the difficulty of modeling a true C effect. Regardless of whether or not C and E environment effects can be properly distinguished, the ACE model is useful for separating basic genetic and environmental effects. Of interest in the current study is not whether parenting as a measure of the environment belongs on the C or E latent variable, but whether or not the environment in general (either C or E) has a significant influence on behavior.

Regardless of whether or not parent warmth falls under the shared or nonshared environment, parenting in the current study emerged as both a significant predictor of child and adolescent behavior in phenotypic models, as well as a moderator of the heritability of externalizing/competence in twin models. The purpose of this study was not to enter the debate over the assumptions of twin modeling but instead to use the best available models to examine the interplay between genetic and environmental factors.

The ACE moderation models presented a less clear picture than the univariate ACE models. In middle childhood, mother warmth moderated mother report of externalizing/competence such that at low levels of mother warmth, behavior was largely a function of the nonshared environment with a small additive genetic influence, whereas in average or advantaged environments (high warmth) externalizing/competence was highly heritable (see Figure 5). This finding supports the general hypothesis that the strength

of the effect of a negative environment is expected to overwhelm the influence of genetic predispositions. In a positive environment, the full genetic potential of the individual can be expressed, lending to behavior that is more heritable (Plomin, 1986; Scarr, 1992, 1993). This conclusion was supported by similar findings in adolescence, where mother warmth again moderated mother report of externalizing/competence such that in a more negative environment externalizing/competence had a larger nonshared environmental component, which diminished as the environment became progressively more positive (i.e., with increased mother warmth; see Figure 7).

Father report variables presented a different picture. In middle childhood, father warmth significantly moderated mother report of externalizing/competence such that when the environment was negative (low father warmth), behavior was largely heritable with an increasing nonshared environmental influence with increasing father warmth (see Figure 6). The same pattern presented itself in early adolescence, with mother warmth on father report of externalizing/competence (see Figure 8). These results were not predicted and are difficult to interpret.

Few studies examining environmental moderators of the heritability of behavior are available and fewer yet yield significant gene-environment interactions in human populations (Shanahan & Hofer, 2005), although significant gene-environment interactions have been found in animal populations, where genes and environments are more readily manipulated (McClearn, 2004; Suomi, 2004). One way in which genes and environments

can interact is by way of contextual triggering (i.e., when a certain environment triggers genetic dispositions; Shanahan & Hofer, 2005). It may be that father warmth as an environmental influence and father observation of child behavior reflect contextual triggering, wherein the negative environment is triggering a genetic response, increasing heritability. An example of contextual triggering can be found in the adolescent substance abuse literature. In an adolescent twin sample, additive genetic factors were found to account for 60% of the variance in smoking when adolescents were exposed to low parental monitoring but less than 15% of the variance in smoking when adolescents encountered higher levels of parental monitoring (Dick et al., 2007). Dick and colleagues concluded that when the environment afforded the opportunity (low levels of parental monitoring), genetic influences on adolescent smoking were more fully expressed.

Under the evolutionary perspective (MacDonald, 1992) father warmth indexes a special benefit to the offspring and is expected to have differential patterns of relations to child outcomes. If father warmth is acting as a buffer against externalizing behaviors, this may explain why in the current study externalizing/competence was more heritable under conditions of low father warmth – the lack of father warmth allowed for the full expression of the heritability of externalizing/competence. Why the model with mother warmth on father report of externalizing/competence would show differing patterns of heritability than the mother report model is unclear. Results are, however, indicative of differences in the pattern of relations across both reporter and time, or developmental stage. More research is needed to clarify how mother

and father warmth differentially relate to child and adolescent behavior and results from the current study should be interpreted with caution as a result of the unpredicted differences in the moderation of heritability in mother and father report. Studies examining whether related aspects of parenting such as involvement (both mother and father report) that could indicate father investment in his offspring, would help clarify whether or not the differing patterns of heritability in the current study result from systematic underlying differences between mothering and fathering.

The moderated heritability models demonstrate that parental warmth, a measure of the environment, interacts with the latent, unmeasured additive genetic and nonshared environmental influences on behavior. This finding supports the many phenotypic studies reporting significant associations between parent warmth and child behavior (see Eisenberg et al., 2005 for a three wave longitudinal study in which parent warmth predicts diminished externalizing).

One way in which the current study advances the field is by considering both parent warmth as a moderator of the heritability of behavior as well as specific gene variants as moderators of the phenotypic relations of warmth and externalizing/competence. A strong additive genetic component (A) is often considered evidence for a biological basis of behavior, and justifies the search for candidate genes (Kovas & Plomin, 2006; Petrill, 1997). Results from the moderated ACE models demonstrated that there is some significant interplay between the ACE estimates and parental warmth on externalizing/competence in a series of latent genetic effects by measured

environment models. The measured gene analyses followed this line of inquiry by testing specific measured gene effects by measured environment interactions.

Phenotypically, the continuum of warmth was significantly related to concurrent externalizing/competence in toddlerhood, middle childhood, and early adolescence, consistent with previous literature (Deater-Deckard et al., 1998; Maccoby & Martin, 1983; Ruchkin et al., 1998). However, only father warmth in toddlerhood marginally predicted change in mother and father report of childhood externalizing in middle childhood. This finding was consistent across mother and father report of externalizing, and remained when tested simultaneously with mother warmth, lending it credence. Father warmth was hypothesized to be particularly beneficial for offspring behavior, following the evolutionary perspective that while mother warmth is normative, father warmth indexes the father's level of investment in the child, making the offspring of involved fathers more competitive in their environment (MacDonald, 1992). The differing longitudinal patterns of mother and father warmth may stem from this added benefit of father involvement (as indexed by warmth).

In adolescence, father warmth was significantly related to externalizing/competence within reporter but not across, and father warmth in middle childhood did not predict a change in externalizing/competence in adolescence. Mother warmth in middle childhood predicted a significant change in externalizing/competence in adolescence on mother report only of externalizing/competence. Externalizing behaviors evidence high rank-order

stability from middle childhood to adolescence (Campbell et al., 1996), and thus middle childhood externalizing/competence was a large predictor of adolescent behavior in the models. In addition, the behavior genetic literature tends to give more emphasis to the early rearing environment as an important predictor of behavior (Hoekstra et al., 2007), and the larger literature has confirmed that the early environment (parenting in toddlerhood) predicts externalizing behavior up through adolescence (Olson, Bates, Sandy, & Lanthier, 2000), whereas estimates of parental warmth in middle childhood have accounted for a very small proportion of the variance in adolescent externalizing (1%; Scaramella, Conger & Simons, 1999). The lack of significance of most of the middle childhood to early adolescence models in predicting change in externalizing/competence may stem from this small effect size (a lessening of the importance of parental warmth in middle childhood) in predicting change in externalizing/competence at later ages. Further research could examine the relative importance of early warmth compared to warmth in middle childhood on adolescent outcomes by comparing the predictive ability of the two.

Moving beyond the additive influence of warmth, this study represents a significant contribution to the literature as the first to examine gene-environment interactions with dopamine, vasopressin, and neuropeptide-Y genes by mother and father warmth on the change in externalizing/competence from childhood to adolescence. The VNTR in the DRD4, the variant most supported by the existing literature (Baskerville & Douglas, 2010), emerged as the most consistent indicator of developmental

plasticity supporting the role of the meso-limbic dopamine pathway in child and adolescent behavior. In addition to a marginal main effect of the DRD4 genotype wherein the long variant related to marginally more externalizing/competence (mother report), in three out of four interactions in middle childhood, across reporters of warmth and child behavior, the long allele related to an increase in plasticity or susceptibility to the environment (see Figures 9, 10, and 11). Children with the long/long genotype exposed to low parental warmth displayed higher levels of externalizing behavior than did children with a short variant; but when exposed to high parental warmth, children with the long/long genotype displayed more behavior competencies.

These significant cross-reporter interactions support the differential susceptibility hypothesis and further our understanding of this particular QTL. Previous research had labeled the long version the *risk* variant (McGue, 2001; Schmidt et al., 2001) as well as the *protective* variant (Auerbach et al., 1999; Ebstein et al., 1998). By examining the full range of behavior and parenting, the picture becomes clearer: the long variant of DRD4 creates greater susceptibility to both positive and negative aspects of the environment. This finding emphasizes the importance of measured gene research for understanding the etiology of externalizing/competence behaviors. Which gene variant an individual inherits is unalterable, but if certain gene variants confer susceptibility to the environment rather than risk as previously supposed, intervention and prevention efforts can be better targeted towards individuals who are likely to benefit the most.

Importantly, these three interactions only emerged when effects were examined in the Caucasian subsample. Despite the minimal ethnic diversity in the sample (92% Caucasian), population stratification, or systematic differences in allele frequencies across different racial or ethnic subgroups within the population, appears to have masked significant interactions when not properly controlled.

The DRD4 VNTR by parent warmth effect did not hold for externalizing/competence behaviors in early adolescence or for the change in behavior from childhood to adolescence. Effects of measured genes are small, and there was a considerable drop in the sample size from middle childhood to adolescence, as the adolescent data collection is currently still underway (see Method). An important future direction is to replicate the adolescent analyses with the full sample to more powerfully examine if there is a gene-environment interaction with dopamine in early adolescence.

Similar to DRD4, the long allele of the VNTR within the vasopressin receptor gene (AVPR1a) was hypothesized to be the plasticity allele. Fewer studies have examined the AVPR1a VNTR in human populations, and these studies typically followed the dichotomous coding scheme set forth by Knafo and colleagues (2008) in which polymorphisms composed of fewer than 325 nucleotide base pairs are grouped as *short*, and polymorphisms with more than 325 base pairs are *long*. This classification system is based upon a functional examination of vasopressin in the human brain, found predominantly in the hypothalamus, the stria terminalis, and the medial amygdala (Meyer-Lindenberg et al., 2009). Despite the functional basis for

the grouping, this classification system still acts to condense a highly variable polymorphism into a dichotomous predictor. To test the efficacy of this coding, the current study examined AVPR1a both dichotomously and additively. Interestingly, the additive coding provided two significant and two marginal interactions while the dichotomous coding yielded no significant effects. Although there may be a functional rational for condensing this highly-polymorphic VNTR into a short/long classification system, there is also utility in preserving variability. Future studies should consider additive coding, or likewise test both the prevailing field standard dichotomous coding in conjunction with a more variable coding scheme.

Unlike with DRD4, the AVPR1a interactions do not provide compelling evidence for the differential susceptibility hypothesis. In middle childhood, there was a significant interaction in the Caucasian subsample with mother warmth in relation to mother report externalizing/competence. When mothers were low on warmth, all children displayed more externalizing, regardless of AVPR1a genotype. When mothers were average or high on warmth, however, children with all AVPR1a variants displayed fewer externalizing behaviors and more competencies, with the most negative simple slope belonging to children with long AVPR1a variants (see Figure 12). This interaction did not fully fit with the differential susceptibility hypothesis. When mothers were warm, there were differences dependent upon the length of the AVPR1a VNTR, which disappear when children were exposed to low mother warmth. The other significant interaction finding with AVPR1a was an interaction with father warmth in childhood predicting

change in externalizing/competence from childhood to adolescence (mother report; see Figure 13). Although the interaction was statistically significant, none of the simple slopes reached significance. As with DRD4, the limited adolescent sample size may restrict significance. The AVPR1a by parent warmth interactions, although they do not reflect differential susceptibility, inform the study of individual differences under conditions of high parent warmth.

The final QTL examined was a series of SNPs within the neuropeptide Y gene, which is part of the stress-response system, analyzed at the level of the diplotype. These analyses were highly exploratory, as the existing literature does not associate this gene with children's behaviors or externalizing/competence in general. Two previous studies of NPY by stress predicting internalizing behaviors in adults were encouraging (Amstadter et al., 2010; Sommer et al., 2010). Functionally, neuropeptide Y plays a key role in the stress response system by helping the organism return to equilibrium following a stressful event, and is hypothesized to underlie stress-related behaviors as well as affect (Heilig, 2004). Additionally, NPY gene expression is moderated by the environment (Sommer et al., 2010), lending credence to the possibility that the influence of NPY gene variants is through gene-environment interaction. In the first study to create haplotypes and diplotypes within the NPY gene, Zhou and colleagues (2008) found that the variants used in the current study related to response to anxiety in adults.

Despite replicating their procedures for forming haplotypes and diplotypes, there were no significant NPY by parenting interactions on

externalizing/competence in childhood or adolescence although there was a consistent main effect across reporter in middle childhood such that children with NPY diplotypes that code for higher NPY brain expression display fewer externalizing behaviors. This finding did not hold in the adolescent sample. The previous research with this haplotype also found a main effect in adults such that diplotypes that coded for lower expression of NPY related to higher emotion-induced amygdala response (Zhou et al., 2008). Interactions with parent warmth were expected in the current study given the function of neuropeptide-Y in the brain. Following a stressful event and a cortisol release NPY is released to return the system functioning to normal (Heilig, 2004). Higher levels of NPY in the brain have a tranquilizing effect that is well-documented in relation to anxiety disorders (Eaton, Sallee, & Sah, 2007; Heilig, 2004). In the current study, NPY diplotypes did not interact with parent warmth but instead related to a blanket decrease in externalizing behaviors, extending the existing literature on adult internalizing to childhood externalizing/competence. This main effect has pharmacological significance and does not rule out the possibility of the NPY diplotypes interacting with a different index of the family environment.

Considerable follow-up with this gene is required. The two published NPY interaction studies considered more extreme environments (i.e., hurricane exposure and retrospective childhood abuse). Perhaps the nonadditive effect of NPY is only to be found under conditions of extreme stress, not the more normative variation offered by parent warmth/rejection. NPY has been found to operate differently under conditions of acute and chronic stress (Eaton et

al., 2007). In the animal literature, acute exposure to stress relates to increased NPY mRNA in the hippocampus in rats (Sergeyev et al., 2005) while exposure to chronic stress relates to increased NPY in the amygdala (Thorsell, Carlsson, Ekman, & Heilig, 1999). Low parent warmth may follow a pattern of chronic stress exposure. Aspects of the family environment that reflect more acute or transitory stressors may interact with NPY diplotypes via an endophenotype marked by increased NPY levels in different areas of the brain (e.g., the hippocampus). Alternatively, although the understanding of the role of NPY in stress-related affect and behaviors extends to externalizing, perhaps the true effects of NPY are specific to depression and anxiety.

The first part of the current study examined the heritability of externalizing/competence across toddlerhood, middle childhood, and early adolescence. Behavior was found to be heritable across ages and reporters, with the heritability moderated by parent warmth in middle childhood and adolescence. There was no main effect of the shared environment on behavior, which may have resulted from either methodological limitations or a circumstance in which the effect of the environment is through interactions with genes. Specific gene variants were investigated as moderators of the relations between parent warmth and child and adolescent externalizing/competence. VNTRs in DRD4 and AVPR1a emerged as significant moderators, implicating the long allele in DRD4 as a susceptibility variant that increases an individual's responsiveness to the environment and the long allele in AVPR1a as a protective variant. These results should be

viewed as encouraging to the field. The genetic expression of externalizing/competence can be altered by the environment (i.e., parent warmth), and genetic variants previously conceived of as risk variants can, in the proper environment, result in above-average functioning. Some developmental theorists have argued to de-emphasize the role of the family environment (Harris, 1998; Scarr, 1992); postulating that studies showing the effects of the family environment are merely reflecting underlying gene-environment correlations. Under this theory, parenting appears to relate to child behavior, for example, because of shared genes between the parent and child. The current study contradicts the argument that the family environment does not matter with the take home message very much being one of gene-environment interplay. Parental warmth predicted change in child behavior over time, and interacted with the heritability of externalizing/competence as well as with measured gene variants. Findings varied by reporter as well as child age and so caution should be used in attempts to generalize current findings to other ages.

Limitations of the Current Study

Results provide a preliminary picture regarding the gene-experience interplay between parent warmth and three gene variants tested under the differential susceptibility hypothesis across multiple time points. This study was limited, however, in that data collection is ongoing. The cohort with parenting and externalizing/competence data in toddlerhood is currently being genotyped and the cohort that has been genotyped is still undergoing adolescent data collection. This limits the ability of the current study to

examine change in externalizing/competence from middle childhood to early adolescence.

An alternative analytic approach would have been the cohort sequential design (CSD; Duncan, Duncan, & Hops, 1996; Nesselroade & Baltes, 1979). With this strategy, several short-term longitudinal studies with participants of varying ages are combined to simulate a substantially longer longitudinal study. In the current study, Cohort 1 (data at ages two and eight) would be combined with Cohort 2 (data at ages eight and 14, as well as genotype data) to simulate a full longitudinal study spanning toddlerhood to early adolescence. This approach, which utilizes maximum likelihood estimation of missing data, can provide as much power as collecting the full sample at all waves and reduce standard errors (Enders, 2010). CSD can also reduce standard errors. A limitation of CSD in terms of the current study is that it would not have contributed to comparisons between Time 1 and Time 3 or Time 1 genotype data on Time 3 externalizing/competence (Enders, 2010). Since there is no cohort that has both Time 1 and Time 3, there is full missingness for this comparison – CSD does not allow for correlational analyses under conditions of full missingness, thus the relation between Time 1 parenting and Time 3 externalizing/competence could not be calculated. Despite these restrictions in correlations, CSD would have made possible growth curve analyses, as the study has three separate time points. Growth curve analyses offer a more sophisticated way of measuring change, or growth, across time points on both the intercept (the mean level of externalizing/competence at each age) and the slope (the amount of

curvature, or the mean rate of growth; Raudenbush & Chan, 1992). With growth curve analyses, the change in externalizing/competence across all three time points, influenced by parent warmth and genotype, could have been modeled both linearly and nonlinearly, which may have yielded additional findings. CSD was not implemented in the current study in part because of the correlation limitation; additionally, these techniques have been found to be most efficacious and reliable when the cohorts overlap more times than they do not (Raudenbush & Chan, 1992). The current study, with only two cohorts that each have one individual time point and one overlapping time point (though not for genotype data), was not considered optimal for these analyses. The full adolescent sample and a more complete genetic sample will soon be available, and an important future direction will be to rerun the models in the complete sample. The current findings, however, are still important. This is the first time two of these gene variants have been examined in a developmental sample (AVPR1a and NPY), and the multiple reporter setup of the current study lends credence to reported gene-experience interactions.

Furthermore, as parent genotype data are currently unavailable, there is the possibility that significant gene-experience interactions (moderated heritability) are a result of shared genes between the parent and child (see Deater-Deckard et al., 1999; Losoya et al., 1997). A passive gene-environment correlation through which children's level of externalizing/competence is similar to parent warmth as a result of shared genes that either increase externalizing/decrease warmth or decrease externalizing/increase warmth

would increase similarity among twins on a given outcome. This similarity would be modeled on the additive genetic component. An evocative gene-environment correlation, on the other hand, would lead to the child's heritable externalizing/competence eliciting different levels of parental warmth, and as MZ twins are more similar on externalizing/competence (to the extent that it is a heritable behavior) they would elicit more similar parental warmth than would DZ twins. This would again increase the additive genetic component. Active gene-environment correlations, in which the child seeks out an environment that suits his or her genotype, are not thought to be relevant to parent warmth but can come into play with externalizing behaviors, particularly in adolescence (Moffit, 2005). With the current measure of the environment (parent warmth), passive and evocative gene-environment correlations are more likely sources of confounding effects.

The measure of warmth/rejection used in this study (the Child Rearing Practices Report; Block, 1965), though a field standard with proven reliability and validity, is not twin specific. A different measure of parenting that distinguishes warmth and rejection provided to each twin individually would enable the testing of gene-environment interaction in the presence of potential gene-environment correlation, strengthening the findings (Johnson, 2007). These models require twin-specific measures of both the moderator and the outcome as they entail decomposing the variance into A, C, and E components that are a) unique to parent warmth, b) shared between parent warmth and externalizing/competence, and c) unique to externalizing/competence. For b) and c) above, each variance component is a

function of a constant as well as a coefficient multiplied by the effect of the moderator (Johnson, 2007). Gene-environment correlation is modeled on the a^2, c^2, and e^2 warmth-moderated paths shared between warmth and externalizing/competence while gene-environment interaction is modeled on the a^2, c^2, and e^2 warmth-moderated paths specific to externalizing/competence.

A statistical limitation stems from the number of analyses conducted. The current study was very exploratory and descriptive, providing a first examination of the relations between parent warmth, DRD4, AVPR1a, and NPY on externalizing/competence at multiple ages. Accordingly, the percent of interactions that were significant from all tested interactions was carefully noted throughout the Results.

Finally, the current sample was not very racially or ethnically diverse (92% Caucasian). In most instances, this homogeneity limits generalizability. The current study, however, was largely focused on measured gene variants. Different ethnic groups can have different base allelic frequencies (population stratification) and this can add an additional and unwelcome source of variability (Lander & Schork, 1994). In fact, when population stratification was controlled for (by repeating analyses within the Caucasian subsample), significant interactions of gene variants by parent warmth emerged. Therefore, for the sake of studies including measured genes, a lack of diversity should not be considered a limitation but rather a necessity. Replications should, however be attempted within other ethnic groups.

Future Directions

A future direction would be to attempt a replication of current findings with internalizing. In the current study only externalizing/competence was considered, as parenting that leaves a child aroused and unresolved (i.e., parental rejection) leads to externalizing behavior (Eisenberg et al., 1999). Kochanska's work (1991, 1995, 1997) also supports examining warmth as a predictor of externalizing/competence, reporting that less fearful children (those higher in externalizing) are more responsive to high levels of parental warmth and positive affect than to parental control. Despite a theoretical rationale for examining externalizing, the gene variants in the current study have also been found to be important for internalizing, however (e.g., DRD4 and internalizing, Propper et al., 2007). NPY in particular has been found to interact with the environment to predict internalizing disorders in adults (i.e., generalized anxiety disorder and depression; Amstadter et al., 2010; Heilig, 2004; Sommer et al., 2010) and replicating the analyses with internalizing behaviors would be of interest.

A future direction would be to replicate the current analyses with a twin-specific measure of parent warmth which would enable the testing of more complex gene-experience models that attempt to separate gene-environment correlation from gene-environment interaction (Johnson, 2007). Fitting these more complex models would strengthen the findings that parent warmth moderates the heritability of externalizing/competence.

Along the same vein, in the current study warmth-moderated heritabilities of externalizing/competence resulted in different patterns

across reporter and age. Future studies should carefully examine differences in the relations of parent warmth as well as the measured gene variants across different ages. Parent warmth is only one of many aspects of the environment that may interact with genetic variants as well as moderate the heritability of behavior. Future studies could examine different aspects of parenting that developmental studies have deemed important, such as harsh discipline (Weiss, Dodge, Bates, & Pettit, 1992) intrusiveness, hostility, and control (Pettit, Laird, Dodge, Bates, & Criss, 2001; Rubin, Burgess, Dwyer, & Hastings, 2003).

The current study examined gene variants within DRD4, AVPR1a, and NPY. These variants were hypothesized to interact with parent warmth under the differential susceptibility framework to predict externalizing/competence. The idea that gene variants can increase susceptibility to the environment, however, does not presuppose that there are a fixed set of variants that do so. There are other genes that lie within these same systems (e.g.; DAT1, the dopamine transporter gene) that may result in the same endophenotype (e.g., a given level of dopamine in the brain that leads to certain behavioral outcomes). There are also genes that code for neurotransmitters not discussed in the current study that may play an important role in child and adolescent externalizing/competence behaviors. For example, AVPR1a was selected for inclusion in the current study in part because of the relation between vasopressin and pair bonding (Hammock & Young, 2002, 2004, 2005, 2006). The oxytocin receptor (OXTR) which codes for the neurotransmitter oxytocin similarly relates to bonding and affiliation,

as well as aggression (Caldwell & Young, 2006; Veenema & Neumann, 2008) and is an excellent candidate to interact with parent warmth to predict externalizing/competence. As the measured gene literature expands, extensions of the current work with other functional gene variants should be tested.

Finally, a future direction would be to utilize methods of family based association testing (FBAT). These methods test association (the covariance between specific gene variants and phenotypic variation; Ianocci et al., 2007) in the presence of linkage (transmission of a disorder or behavior through the family pedigree linked to a gene variant) by parsing variance into between- and within- twin pair components (Fulker, Cherny, Sham, & Hewitt, 1999). Compared to the univariate analyses utilized in the current study, FBAT methods can yield power gains as high as 200% (Lange, DeMeo, Silverman, Weiss, & Laird., 2004). For full effectiveness, FBAT procedures require a triad, although when parent genotypes are unavailable (as in the current sample), expectation of a specific genotype in the offspring can be conditioned on other information (Lange et al., 2004). Having tested the associations in the univariate framework of the current study, FBAT analyses would be both appropriate and potentially further illuminate the relations between parent warmth and DRD4, AVPR1a, and NPY on externalizing/competence. An additional benefit of the FBAT approach is that it increases the power of detecting a QTL when the gene variant under study is not the actual QTL but is in linkage disquilibrium with it (Fulker et al., 1999).

Conclusion

Parental warmth, a significant predictor of externalizing/competence behaviors across development, moderates the heritability of externalizing/competence in middle childhood and early adolescence. The differential susceptibility hypothesis is a viable alternative to the diathesis-stress hypothesis in the context of gene-environment interactions. Although genes are fixed and unchangeable, their effects are not deterministic but rather depend upon salient environmental influences, one of which is parent warmth. Reframing the genetic literature away from *risk* variants and toward *susceptibility* variants will encourage further investigations into the complex interrelations of genes and environments that underlie behavior. Examining just externalizing, for example, and not competence would have (falsely) confirmed the long variants of the DRD4 VNTR as *risk*, when it in fact also relates to greater competence under conditions of high parent warmth. Current findings offer an interesting and hopeful picture of the relations between parent warmth and gene variants on child and adolescent externalizing/competence behaviors.

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Table 1

Descriptive Demographic Statistics

	M	*SD*	Minimum	Maximum	*N*
Toddlerhood					
Income	9.66	3.72	1.00	17.00	738
Mother education	14.95	2.23	8.00	20.00	760
Father education	14.73	2.52	6.00	20.00	755
Mother employment	5.69	1.87	1.00	9.00	501
Father employment	5.79	2.13	1.00	9.00	726
SES	0.00	1.00	-2.42	2.45	760
Twin pair age (years)	2.25	0.24	1.50	3.08	762
Middle Childhood					
Income	10.47	3.85	1.00	17.00	779
Mother education	14.98	2.31	6.00	20.00	806
Father education	14.47	2.54	6.00	20.00	787
Mother employment	5.77	1.93	1.00	9.00	648
Father employment	5.64	2.11	1.00	9.00	740
SES	0.00	1.00	-2.14	1.81	806
Twin pair age (years)	7.40	0.84	5.42	11.83	806
Early Adolescence					
Income	10.63	4.12	1.00	17.00	369
Mother education	14.67	2.26	9.00	20.00	385
Father education	14.10	2.42	8.00	20.00	368
Mother employment	5.62	1.78	1.00	9.00	306
Father employment	5.39	2.08	1.00	9.00	316
SES	0.00	1.00	-2.80	2.66	390
Twin pair age (years)	14.00	1.64	11.17	18.08	392

Note. Sample sizes reported at the family level. SES = Socioeconomic status.

Table 2

Descriptive Statistics for Parental Warmth and Child Behaviors

	M	*SD*	Minimum	Maximum	*N*
Toddlerhood					
Mother Warmth	5.28	0.31	4.33	6.00	764
Father Warmth	5.13	0.40	3.93	5.89	392
Externalizing/Competence: MR	0.51	0.27	0.00	1.25	764
Externalizing/Competence: FR	0.57	0.27	0.00	1.37	660
Middle Childhood					
Mother Warmth	5.30	0.36	3.86	5.96	805
Father Warmth	5.11	0.44	3.73	6.00	618
Externalizing/Competence: MR	0.03	0.51	-0.77	1.62	806
Externalizing/Competence: FR	0.00	0.47	-0.80	1.46	629
Early Adolescence					
Mother Warmth	5.39	0.36	4.29	5.96	292
Father Warmth	5.22	0.42	3.90	6.00	241
Externalizing/Competence: MR	0.00	0.52	-0.96	1.62	435
Externalizing/Competence: FR	0.00	0.49	-1.09	1.53	314

Note. Sample sizes reported at the family level. Predictor variables were centered prior to analyses. FR = Father Report; MR = Mother Report.

Table 3

Correlations between Phenotypic Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. T1 SES	-													
2. T1 Mother Warmth	.11**	-												
3. T1 Father Warmth	.08*	.18**	-											
4. T1 MR Behavior	-.14**	-.16**	-.10*	-										
5. T1 FR Behavior	-.13**	-.11**	-.23**	.55**	-									
6. T2 SES	.92**	.10**	.12**	-.13**	-.13**	-								
7. T2 Mother Warmth	.17**	.38**	.02	-.15**	-.11	.15**	-							
8. T2 Father Warmth	.13*	.11*	.51**	-.06	-.20**	.14**	.24**	-						
9. T2 MR Behavior	-.17**	-.11*	-.20*	.46**	.32**	-.20**	-.24**	-.21**	-					
10. T2 FR Behavior	-.16**	-.03	-.26**	.28**	.40**	-.15**	-.14**	-.31**	.62**	-				
11. T3 SES	-	-	-	-	-	.92**	.14**	.18**	-.20**	-.17**	-			
12. T3 Mother Warmth	-	-	-	-	-	.14**	.49**	.14**	-.25**	-.19**	.10*	-		
13. T3 Father Warmth	-	-	-	-	-	.19**	.19**	.53**	-.10*	-.24**	.18**	.21**	-	
14. T3 MR Behavior	-	-	-	-	-	-.21**	-.22**	-.10**	.63**	.45**	-.22**	-.32**	-.13**	-
15. T3 FR Behavior	-	-	-	-	-	-.17**	-.14**	-.24**	.52**	.68**	-.19**	-.23**	-.27**	.64**

Note. Correlations were computed using the cluster option in Mplus v. 5.1 (Muthén & Muthén, 2008) to account for twin dependence; correlations not shown were incalculable due to sample size. FR = Father Report; MR = Mother Report; SES = Socioeconomic status; T1 = Time 1 (Toddlerhood); T2 = Time 2 (Middle Childhood); T3 = Time 3 (Early Adolescence). $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 4

Allelic Frequencies of Dopamine (DRD4), Vasopressin (AVPR1a), and Neuropeptide-Y (NPY) Gene Variants

DRD4	Short/Short	Short/Long	Long/Long	Total N
48 base pair VNTR	562	219	43	824
AVPR1a	Short/Short	Short/Long	Long/Long	
R3 VNTR	13	126	621	760
Neuropeptide-Y	A/A	A/G	G/G	
rs16139	679	57A	3	739
rs16147	211	379	166	756
rs5573	166	389	191	746
	T/T	C/T	C/C	
rs5574	134	336	218	688
	G/G	G/T	T/T	
rs17149106	621	68	3	692
	TG/TG	TG/DEL	DEL/DEL	
rs3037354	362	270	49	681

Note. All Single Nucleotide Polymorphisms (SNPs) were measured on the forward allele and are designated by the appropriate refSNP (rs) number. Reverse strand measurement results can be interpreted as C=G, A=T for these SNPs. rs17149106 corresponds to A/C on the reverse allele and rs3037354 corresponds to CA/DEL. All SNPs are in Hardy-Weinberg Equilibrium. DEL = Deletion; VNTR = Variable Number Tandem Repeat.

Table 5

Frequencies and Clustering of Major Neuropeptide-Y Haplotypes in 516 Finnish Caucasians and in the Current Sample

rs3037354	rs17149106	rs16147	rs16139	rs5573	rs5574	Haplotype	Freq. Reported in Zhou et al., 2008	Freq. in Current Sample
TG	G	A	A	G	C	H3	0.178	0.179
Del	G	A	A	G	C	H2	0.224	0.263
TG	T	A	G	G	C	H5	0.046	0.043
TG	G	G	A	A	C	H4	0.043	0.037
TG	G	G	A	A	T	H1	0.447	0.471
Percent of chromosomes accounted for by five haplotype block:							93.8%	99.8%

Note. Freq. = Frequency; H = Haplotype. Source for comparison sample: Zhou et al., 2008.

Table 6

Creating Neuropeptide-Y (NPY) Diploypes from the Three Most Common Haplotypes

	Allele 1			Allele 2			*N*	Frequency	NPY Expression
	H1	H2	H3	H1	H2	H3			
Diplotype 1 (D1)	1	0	0	1	0	0	160	25.4%	Low
Diplotype 2 (D2)	0	1	0	0	1	0	51	8.1%	High
Diplotype 3 (D3)	0	0	1	0	0	1	28	4.4%	Medium
Diplotype 4 (D4)	1	0	0	0	1	0	201	31.9%	Medium
Diplotype 5 (D5)	1	0	0	0	0	1	136	21.6%	Medium
Diplotype 6 (D6)	0	1	0	0	0	1	55	8.7%	High

Note. Where allele 1 and allele 2 are randomly assigned, and are cross-calculated (i.e., D4 consists of individuals with H1/H2 and H2/H1). D = Diplotype; H = Haplotype; 1 = presence of haplotype in the diplotype, 0 = absence of haplotype in the diplotype.

Table 7

ACE Model Fit Statistics for Externalizing/Competence

	Model	-2LL	df	AIC	Δdf	$\Delta\chi^2$	p	a^2	c^2	e^2
Toddlerhood										
Mother	ACE	57.62	1509	-2960.38	-	-	-	.68	0	.32
Report	**AE**			**-2962.38**	**1**	**0.00**	**0.99**	**.68**	-	**.32**
	CE			-2864.53	1	97.85	0.00	-	.30	.70
	E			-2746.44	2	217.94	0.00	-	-	1
Father	ACE	-19.28	863	-1745.28	-	-	-	.67	0	.33
Report	**AE**			**-1747.28**	**1**	**0.00**	**0.99**	**.67**	-	**.33**
	CE			-1688.45	1	58.83	0.00	-	.25	.75
	E			-1642.73	2	106.56	0.00	-	-	1
Middle Childhood										
Mother	ACE	1948.98	1554	-1159.03	-	-	-	.73	0	.27
Report	**AE**			**-1161.03**	**1**	**0.00**	**0.99**	**.73**	-	**.27**
	CE			-1008.80	1	152.22	0.00	-	.31	.69
	E			-899.42	2	263.61	0.00	-	-	1
Father	ACE	1350.87	1188	-1025.13	-	-	-	.71	0	.29
Report	**AE**			**-1027.13**	**1**	**0.00**	**0.99**	**.71**	-	**.29**
	CE			-945.16	1	81.97	0.00	-	.34	.66
	E			-834.59	2	194.54	0.00	-	-	1
Early Adolescence										
Mother	ACE	666.67	575	-483.33	-	-	-	.67	0	.33
Report	**AE**			**-485.33**	**1**	**0.00**	**0.99**	**.67**	-	**.33**
	CE			-441.15	1	44.18	0.00	-	.31	.69
	E			-399.00	2	88.33	0.00	-	-	1
Father	ACE	494.37	443	-391.63	-	-	-	.66	0	.34
Report	**AE**			**-393.63**	**1**	**0.00**	**0.99**	**.66**	-	**.34**
	CE			-363.68	1	29.95	0.00	-	.34	.66
	E			-327.05	2	68.58	0.00	-	-	1

Note. -2LL = -2 times the log likelihood (fit statistic); df = degrees of freedom; AIC = Akaike's Information Criterion (fit index); $\Delta\chi^2$ = change in Chi-square value from the full model to reduced model; p = probability; a^2 = heritability estimate; c^2= shared environment estimate; e^2= non-shared, or unique, environment estimate.

Table 8

Concurrent Relations of Parental Warmth to Externalizing/Competence Behaviors

	Toddlerhood Externalizing/Competence Behaviors				Middle Childhood Externalizing/Competence Behaviors				Early Adolescence Externalizing/Competence Behaviors			
	Mother Report		Father Report		Mother Report		Father Report		Mother Report		Father Report	
	Estimate	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*
Model 1	R^2_A = .09, R^2_B = .08		R^2_A = .10, R^2_B = .10		R^2_A = .14, R^2_B = .12		R^2_A = .07, R^2_B = .07		R^2_A = .20, R^2_B = .18		R^2_A = .12, R^2_B = .11	
Intercept	0.50	63.69**	0.65	48.12**	0.13	5.54**	0.10	3.88**	0.11	3.27**	0.11	3.00**
Child sex[1]	-0.11	-13.72**	-0.14	-9.59**	-0.23	-9.28**	-0.18	-7.09**	-0.23	-6.02**	-0.13	-3.41**
Cotwin sex	0.02	2.06*	-0.01	-0.44	0.01	0.58	-0.02	-0.75	0.01	0.24	-0.06	-1.56
SES	-0.04	-6.40**	-0.03	-2.48*	-0.09	-5.70**	-0.05	-2.68**	-0.10	-4.52**	-0.08	-2.58*
Mother warmtl	-0.13	-5.31**	-0.03	-2.59*	-0.11	-7.44**	-0.06	-3.90**	-0.15	-7.10**	-0.11	-3.46**
Model 2	R^2_A = .07, R^2_B = .07		R^2_A = .13, R^2_B = .14		R^2_A = .07, R^2_B = .07		R^2_A = .15, R^2_B = .14		R^2_A = .12, R^2_B = .10		R^2_A = .14, R^2_B = .12	
Intercept	0.50	62.80**	0.64	47.67**	0.13	5.36**	0.10	4.48**	0.13	3.54**	0.12	3.25**
Child sex	-0.11	-13.60**	-0.14	-9.49**	-0.23	-9.34**	-0.19	-7.62**	-0.24	-6.33**	-0.15	-3.83**
Cotwin sex	0.02	2.12*	-0.01	-0.35	0.01	0.50	-0.03	-1.08	-0.01	-0.21	-0.08	-2.02*
SES	-0.04	-6.67**	-0.03	-2.58**	-0.09	-5.55**	-0.04	-2.30*	-0.11	-4.89**	-0.07	-2.27*
Father warmtl	-0.03	-2.60**	-0.06	-5.96**	-0.10	-6.08**	-0.15	-9.31**	-0.04	-1.40	-0.12	-4.77**
Model 3	R^2_A = .09, R^2_B = .09		R^2_A = .14; R^2_B = .14		R^2_A = .16, R^2_B = .14		R^2_A = .16; R^2_B = .14		R^2_A = .20, R^2_B = .18		R^2_A = .17; R^2_B = .15	
Intercept	0.50	63.09**	0.65	48.27**	0.13	5.74**	0.10	4.54**	0.11	3.39**	0.11	3.13**
Child sex	-0.11	-13.62**	-0.14	-9.52**	-0.23	-9.55**	-0.19	-7.68**	-0.23	-6.06**	-0.14	-3.66**
Cotwin sex	0.02	2.08*	-0.01	-0.33	0.01	0.41	-0.03	-1.14	0.01	0.23	-0.07	-1.76+
SES	-0.03	-6.09**	-0.03	-2.31*	-0.08	-5.16**	-0.04	-2.10*	-0.10	-4.51**	-0.07	-2.22*
Mother warmtl	-0.04	-4.79**	-0.02	-1.85+	-0.09	-5.75**	-0.03	-2.00*	-0.15	-6.77**	-0.11	-4.17**
Father warmth	-0.02	-1.72+	-0.06	-5.51**	-0.08	-4.50**	-0.14	-8.68**	-0.01	-0.29	-0.08	-2.91**

Note. [1] boys = 0, girls = 1; SES = Socioeconomic status. $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 9

Longitudinal Relations of Toddlerhood Parental Warmth to Change in Externalizing/Competence Behaviors from Toddlerhood to Middle Childhood

	Externalizing/ Competence Mother Report		Externalizing/ Competence Father Report	
	Estimate	*Estimate/ SE*	*Estimate*	*Estimate/ SE*
Model 1	R^2_A = .26, R^2_B = .25		R^2_A = .25, R^2_B = .23	
Intercept	0.07	2.72**	0.03	0.83
Child sex[1]	-0.17	-6.25**	-0.09	-3.11**
Cotwin sex	0.01	0.34	0.01	0.16
T2 SES	-0.07	-4.48**	-0.01	-0.65
T1 Twin Behavior	0.74	9.24**	0.70	7.99**
T1 Cotwin Behavior	0.13	1.66+	0.24	2.81**
T1 Mother warmth	-0.02	-0.80	0.03	0.71
Model 2	R^2_A = .27, R^2_B = .27		R^2_A = .28, R^2_B = .27	
Intercept	0.08	2.83**	0.05	1.35
Child sex	-0.18	-6.15**	-0.12	-3.32**
Cotwin sex	0.00	-0.05	-0.02	-0.63
T2 SES	-0.06	-2.64**	0.00	-0.16
T1 Twin Behavior	0.75	-9.32**	0.63	6.48**
T1 Cotwin Behavior	0.14	1.82+	0.17	1.70+
T1 Father warmth	-0.08	-1.78+	-0.10	-1.89+
Model 3	R^2_A = .28, R^2_B = .27		R^2_A = .29; R^2_B = .28	
Intercept	0.08	2.76**	0.05	1.36
Child sex	-0.18	-6.16**	-0.12	-3.35**
Cotwin sex	0.00	-0.05	-0.02	-0.62
T2 SES	-0.06	-2.67**	0.00	-0.16
T1 Twin Behavior	0.75	9.20**	0.63	6.49**
T1 Cotwin Behavior	0.14	1.79+	0.17	1.71+
T1 Mother warmth	-0.01	-0.46	0.03	0.94
T1 Father warmth	-0.08	-1.81+	-0.10	-1.93+

Note. [1] boys = 0, girls = 1; SES = Socioeconomic status, T1 = Time 1 (Toddlerhood), T2 = Time 2 (Middle Childhood). $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 10

Longitudinal Relations of Middle Childhood Parental Warmth to Change in Externalizing/Competence Behaviors from Middle Childhood to Early Adolescence

	Externalizing/ Competence Mother Report		Externalizing/ Competence Father Report	
	Estimate	*Estimate/ SE*	*Estimate*	*Estimate/ SE*
Model 1	R^2_A = .47, R^2_B = .40		R^2_A = .48, R^2_B = .45	
Intercept	0.06	2.06*	0.03	0.84
Child sex[1]	-0.13	-4.19**	-0.02	-0.74
Cotwin sex	-0.01	-0.43	-0.03	-0.92
T3 SES	-0.04	-2.36*	-0.03	-1.33
T2 Twin Behavior	0.55	14.06**	0.65	15.18**
T2 Cotwin Behavior	0.07	1.82+	0.07	1.98*
T2 Mother warmth	-0.05	-2.79**	-0.02	-0.88
Model 2	R^2_A = .46, R^2_B = .39		R^2_A = .48, R^2_B = .45	
Intercept	0.06	1.99*	0.03	0.90
Child sex	-0.13	-3.96**	-0.02	-0.77
Cotwin sex	-0.01	-0.27	-0.03	-0.94
T3 SES	-0.05	-2.58*	-0.03	-1.35
T2 Twin Behavior	0.56	14.48**	0.64	14.95**
T2 Cotwin Behavior	0.09	2.15*	0.07	1.79+
T2 Father warmth	0.01	0.29	-0.02	-0.82
Model 3	R^2_A = .47, R^2_B = .40		R^2_A = .48; R^2_B = .45	
Intercept	0.05	1.97*	0.03	0.91
Child sex	-0.13	-4.12**	-0.03	-0.79
Cotwin sex	-0.01	-0.37	-0.03	-0.97
T3 SES	-0.05	-2.54*	-0.03	-1.30
T2 Twin Behavior	0.55	13.98**	0.64	14.97**
T2 Cotwin Behavior	0.07	1.87+	0.07	1.78+
T2 Mother warmth	-0.05	-2.92**	-0.01	-0.65
T2 Father warmth	0.02	0.81	-0.01	-0.56

Note. [1] boys = 0, girls = 1; SES = Socioeconomic status, T2 = Time 2 (Middle Childhood), T3 = Time 3 (Early Adolescence). $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 11

AE with Moderation Fit Statistics for Externalizing/Competence Behaviors

Model	Moderator	-2LL	df	AIC	Δdf	$\Delta\chi^2$	p
Toddlerhood Behavior (Mother Report)							
AE full moderation		56.74	1508	-2959.26	-	-	-
Only A moderation	Mother			-2960.46	1	0.80	0.37
Only E moderation	Warmth			-2961.23	1	0.03	0.86
AE no moderation				**-2962.38**	**2**	**0.88**	**0.64**
Toddlerhood Behavior (Mother Report)							
AE full moderation		9.56	754	-1498.44	-	-	-
Only A moderation	Father			-1497.04	1	3.40	0.07
Only E moderation	Warmth			-1500.30	1	0.14	0.71
AE no moderation				**-1498.78**	**2**	**3.66**	**0.16**
Toddlerhood Behavior (Father Report)							
AE full moderation		-23.90	866	-1755.90	-	-	-
Only A moderation	Mother			-1756.32	1	1.58	0.21
Only E moderation	Warmth			-1754.46	1	3.44	0.06
AE no moderation				**-1756.31**	**2**	**3.59**	**0.16**
Toddlerhood Behavior (Father Report)							
AE full moderation		-57.32	754	-1567.32	-	-	-
Only A moderation	Father			-1567.75	1	1.57	0.21
Only E moderation	Warmth			-1566.77	1	2.55	0.11
AE no moderation				**-1568.59**	**2**	**2.73**	**0.26**
Middle Childhood Behavior (Mother Report)							
AE full moderation		1942.70	1553	**-1163.30**	**-**	**-**	**-**
Only A moderation	Mother			-1163.31	1	1.99	0.16
Only E moderation	Warmth			-1163.71	1	1.59	0.21
AE no moderation				-1161.03	2	6.28	0.04
Middle Childhood Behavior (Mother Report)							
AE full moderation		1322.60	1179	-1035.40	-	-	-
Only A moderation	Father			-1029.85	1	7.54	0.01
Only E moderation	Warmth			**-1037.37**	**1**	**0.03**	**0.87**
AE no moderation				-1030.60	2	8.80	0.01
Middle Childhood Behavior (Father Report)							
AE full moderation		1348.18	1187	-1025.82	-	-	-
Only A moderation	Mother			-1028.98	1	1.84	0.18
Only E moderation	Warmth			-1027.77	1	0.05	0.82
AE no moderation				**-1027.13**	**2**	**2.69**	**0.26**
Middle Childhood Behavior (Father Report)							
AE full moderation		1278.42	1193	-1107.58	-	-	-
Only A moderation	Father			-1106.30	1	3.28	0.07
Only E moderation	Warmth			-1109.58	1	0.00	0.99
AE no moderation				**-1106.96**	**2**	**4.62**	**0.10**
Early Adolescence Behavior (Mother Report)							
AE full moderation		613.04	548	-482.96	-	-	-
Only A moderation	Mother			**-484.96**	**1**	**0.00**	**0.96**
Only E moderation	Warmth			-482.12	1	5.76	0.02
AE no moderation				-479.51	2	7.46	0.02
Early Adolescence Behavior (Mother Report)							
AE full moderation		442.70	412	-381.30	-	-	-
Only A moderation	Father			-383.30	1	0.01	0.99
Only E moderation	Warmth			-382.08	1	1.22	0.27
AE no moderation				**-383.76**	**2**	**1.55**	**0.46**
Early Adolescence Behavior (Father Report)							
AE full moderation		455.52	426	-396.49	-	-	-
Only A moderation	Mother			-376.51	1	21.97	0.00
Only E moderation	Warmth			**-398.02**	**1**	**0.47**	**0.49**
AE no moderation				-373.68	2	26.81	0.00
Early Adolescence Behavior (Father Report)							
AE full moderation		445.35	420	-394.66	-	-	-
Only A moderation	Father			-393.45	1	3.20	0.07
Only E moderation	Warmth			-396.62	1	0.03	0.86
AE no moderation				**-394.79**	**2**	**3.87**	**0.14**

Note. Bold font indicates the best-fitting model; -2LL = -2 times the log likelihood (fit statistic); df = degrees of freedom; AIC = Akaike's Information Criterion (fit index); $\Delta\chi^2$ = change in Chi-square value from full model to reduced model; p = probability; a^2 = heritability estimate; c^2= shared environment estimate; e^2= non-shared, or unique, environment estimate.

Table 12

Concurrent Relations of Parental Warmth and Dopamine (DRD4) to Externalizing/Competence Behaviors in Middle Childhood and Early Adolescence

	Middle Childhood Externalizing/Competence Behaviors				Early Adolescence Externalizing/Competence Behaviors			
	Mother Report		Father Report		Mother Report		Father Report	
	Estimate	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*
Model 1	$R^2_A = .14$, $R^2_B = .12$		$R^2_A = .07$, $R^2_B = .07$		$R^2_A = .20$, $R^2_B = .18$		$R^2_A = .14$, $R^2_B = .13$	
Intercept	0.14	4.62**	0.08	2.62**	0.10	2.54*	0.10	2.46*
Twin sex[1]	-0.23	-9.23**	-0.18	-6.96**	-0.23	-6.09**	-0.13	-3.48**
Cotwin sex	0.02	0.60	-0.02	-0.69	0.01	0.36	-0.06	-1.61
SES	-0.09	-5.74**	-0.05	-2.66**	-0.10	-4.44**	-0.08	-2.60** [b]
Mother warmth	-0.12	-5.48**	-0.07	-2.80**	-0.15	-7.22**	-0.10	-3.35**
Twin DRD4	0.00	-0.02	0.04	0.88	0.06	1.34	0.00	-0.04
Cotwin DRD4	-0.03	-0.71	0.00	-0.01	-0.03	-0.71	0.01	0.30
Warmth * Twin DRD4	-0.03	-0.83 [c]	-0.02	-0.42	-0.01	-0.11	-0.06	-1.34
Warmth * Cotwin DRD4	0.06	1.60 [c]	0.04	0.93	0.04	0.88	-0.07	-1.53
Model 2	$R^2_A = .14$, $R^2_B = .12$		$R^2_A = .15$, $R^2_B = 14$		$R^2_A = .12$, $R^2_B = .11$		$R^2_A = .14$, $R^2_B = .12$	
Intercept	0.14	4.69**	0.10	3.36**	0.12	2.75**	0.11	2.53*
Twin sex[1]	-0.23	-9.36**	-0.19	-7.53**	-0.25	-6.36**	-0.15	-3.78**
Cotwin sex	0.01	0.49	-0.03	-1.08	-0.01	-0.14	-0.07	-1.91+
SES	-0.09	-5.54**	-0.04	-2.28* [b]	-0.12	-4.92**	-0.07	-2.56* [a]
Father warmth	-0.08	-3.34**	-0.13	-5.37**	-0.04	-1.14	-0.12	-4.59**
Twin DRD4	-0.01	-0.33	0.02	0.55	0.06	1.42	0.01	0.32
Cotwin DRD4	-0.04	-1.16	-0.02	-0.53	-0.04	-0.90	0.02	0.35
Warmth * Twin DRD4	-0.06	-1.06 [c]	-0.01	-0.17 [c]	-0.03	-0.62	0.01	0.12
Warmth * Cotwin DRD4	-0.02	-0.46	-0.05	-1.13	-0.05	-1.11	-0.03	-0.65

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. All models tested in full sample and Caucasian-only subsample to control for population stratification, differences are noted with subscripted letters; [1] boys = 0, girls = 1; SES = Socioeconomic status. $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 13

Longitudinal Relations of Parental Warmth in Middle Childhood and Dopamine (DRD4) to Change in Externalizing/Competence Behaviors from Middle Childhood to Early Adolescence

	Externalizing/ Competence Mother Report		Externalizing/ Competence Father Report	
	Estimate	*Estimate/ SE*	*Estimate*	*Estimate/ SE*
Model 1	R^2_A = .50, R^2_B = .38		R^2_A = .48, R^2_B = .45	
Intercept	0.05	1.56	0.02	0.55
Child sex[1]	-0.13	-4.04**	-0.02	-0.76
Cotwin sex	-0.02	-0.63	-0.03	-0.86
T3 SES	-0.05	-2.54*	-0.03	-1.32
T2 Twin Behavior	0.55	12.94**	0.65	14.98**
T2 Cotwin Behavior	0.05	1.17	0.07	1.96+ [a]
T2 Mother warmth	-0.05	-2.94**	-0.02	-0.88
Twin DRD4	0.06	1.58 [c]	-0.01	-0.36
Cotwin DRD4	-0.04	-1.06	0.03	0.82
T2 Warmth * Twin DRD4	0.02	0.44	-0.02	-0.82
T2 Warmth * Cotwin DRD4	-0.06	-1.15	0.02	0.74
Model 2	R^2_A = .48, R^2_B = .37		R^2_A = .48, R^2_B = .45	
Intercept	0.05	1.42	0.02	0.60
Child sex[1]	-0.13	-4.03**	-0.02	-0.75
Cotwin sex	-0.01	-0.19	-0.03	-0.90
T3 SES	-0.05	-2.50*	-0.03	-1.34
T2 Twin Behavior	0.56	14.40**	0.64	15.10**
T2 Cotwin Behavior	0.09	2.19*	0.07	1.80+ [a]
T2 Father warmth	0.01	0.47	-0.02	-0.87
Twin DRD4	0.05	1.45 [b]	-0.01	-0.33
Cotwin DRD4	-0.03	-0.74	0.03	0.80
T2 Warmth * Twin DRD4	-0.03	-0.96	-0.01	-0.29
T2 Warmth * Cotwin DRD4	0.00	-0.07	0.04	1.14

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. [1] boys = 0, girls = 1; SES = Socioeconomic status, T2 = Time 2 (Middle Childhood), T3 = Time 3 (Early Adolescence).
$^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$

Table 14

Concurrent Relations of Parental Warmth and Vasopressin (AVPR1a; dominant coding) to Externalizing/Competence Behaviors in Middle Childhood and Early Adolescence

	Middle Childhood Externalizing/Competence Behaviors				Early Adolescence Externalizing/Competence Behaviors			
	Mother Report		Father Report		Mother Report		Father Report	
	Estimate	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*
Model 1	R^2_A = .14, R^2_B = .12		R^2_A = .07, R^2_B = .07		R^2_A = .21, R^2_B = .18		R^2_A = .12, R^2_B = .11	
Intercept	0.15	3.11**	0.13	2.31*	0.14	1.92+	0.12	1.81+
Twin sex[1]	-0.23	-9.28**	-0.19	-7.07**	-0.23	-5.93**	-0.13	-3.36**
Cotwin sex	0.02	0.58	-0.02	-0.76	0.01	0.33	-0.06	-1.55
SES	-0.09	-5.72**	-0.05	-2.71**	-0.10	-4.59**	-0.08	-2.60* [b]
Mother warmth	-0.06	-1.63	-0.07	-1.21	-0.18	-6.84**	-0.12	-1.72+ [a]
Twin AVPR1a	0.04	0.51	0.01	0.19	0.00	-0.02	0.01	0.17
Cotwin AVPR1a	-0.06	-0.79	-0.06	-0.98	-0.04	-0.55	-0.03	-0.41
Warmth * Twin AVPR1a	-0.09	-1.25	0.00	0.00	0.07	1.44	0.02	0.46
Warmth * Cotwin AVPR1	-0.03	-0.40	0.00	0.04	0.08	1.74+ [a]	-0.01	-0.09
Model 2	R^2_A = .13, R^2_B = .12		R^2_A = .15, R^2_B = 14		R^2_A = .12, R^2_B = .10		R^2_A = .13, R^2_B = .12	
Intercept	0.14	3.03**	0.14	2.65**	0.17	2.13*	0.16	2.39*
Twin sex[1]	-0.23	-9.31**	-0.19	-7.67**	-0.24	-6.29**	-0.15	-3.77**
Cotwin sex	0.01	0.46	-0.03	-1.12	-0.01	-0.23	-0.08	-2.06* [b]
SES	-0.09	-5.41**	-0.04	-2.29*	-0.12	-4.95**	-0.07	-2.40* [b]
Father warmth	-0.04	-0.95	-0.11	-1.88+ [a]	-0.04	-1.38	-0.09	-1.68+
Twin AVPR1a	0.05	0.67	0.01	0.15	-0.01	-0.08	0.02	0.44
Cotwin AVPR1a	-0.07	-0.88	-0.06	-1.01	-0.05	-0.68	-0.07	-1.34
Warmth * Twin AVPR1a	0.06	0.78	-0.01	-0.11	0.00	0.06	-0.08	-0.74
Warmth * Cotwin AVPR1	-0.14	-2.00*	-0.03	-0.60	0.00	-0.26	0.04	0.32

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. All models tested in full sample and Caucasian-only subsample to control for population stratification, differences are noted with subscripted letters; [1] boys = 0, girls = 1; SES = Socioeconomic status. $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 15

Concurrent Relations of Parental Warmth and Vasopressin (AVPR1a; additive coding) to Externalizing/Competence Behaviors in Middle Childhood and Early Adolescence

	Middle Childhood Externalizing/Competence Behaviors				Early Adolescence Externalizing/Competence Behaviors			
	Mother Report		Father Report		Mother Report		Father Report	
	Estimate	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*
Model 1	$R^2_A = .14$, $R^2_B = .12$		$R^2_A = .07$, $R^2_B = .07$		$R^2_A = .21$, $R^2_B = .18$		$R^2_A = .13$, $R^2_B = .12$	
Intercept	0.13	5.66**	0.10	3.89**	0.11	3.27**	0.11	3.04**
Twin sex[1]	-0.23	-9.30**	-0.19	-7.09**	-0.23	-6.02**	-0.13	-3.43**
Cotwin sex	0.01	0.45	-0.02	-0.74	0.01	0.26	-0.06	-1.57
SES	-0.09	-5.81**	-0.05	-2.73**	-0.10	-4.52**	-0.08	-2.62** [b]
Mother warmth	-0.11	-7.57**	-0.06	-3.88**	-0.15	-7.18**	-0.10	-3.39**
Twin AVPR1a	-0.01	-0.76	-0.01	-0.57	0.01	0.48	0.00	-0.07
Cotwin AVPR1a	-0.01	-0.29	0.00	-0.20	-0.01	-0.50	0.01	0.33
Warmth * Twin AVPR1a	-0.03	-1.76+ [c]	0.00	0.13	0.00	0.22	-0.03	-1.25
Warmth * Cotwin AVPR1a	-0.01	-0.50	-0.01	-0.25	-0.02	-0.93	-0.02	-1.22
Model 2	$R^2_A = .13$, $R^2_B = .12$		$R^2_A = .15$, $R^2_B = 14$		$R^2_A = .15$, $R^2_B = .13$		$R^2_A = .14$, $R^2_B = .13$	
Intercept	0.13	5.34**	0.10	4.44**	0.12	3.34**	0.12	3.17**
Twin sex[1]	-0.23	-9.41**	-0.19	-7.54**	-0.24	-6.15**	-0.14	-3.78**
Cotwin sex	0.02	0.60	-0.03	-1.08	0.00	-0.01	-0.07	-1.93+
SES	-0.09	-5.63**	-0.04	-2.34*	-0.11	-5.07**	-0.07	-2.34* [b]
Father warmth	-0.10	-5.90**	-0.14	-9.08**	-0.04	-1.29	-0.12	-4.80**
Twin AVPR1a	-0.00	-0.06	-0.02	-0.67	0.01	0.33	-0.01	-0.36
Cotwin AVPR1a	-0.02	-1.00	0.00	-0.06	-0.01	-0.50	0.00	0.10
Warmth * Twin AVPR1a	0.01	0.50	-0.01	-0.28	-0.06	-1.75+	-0.02	-0.77
Warmth * Cotwin AVPR1a	-0.04	-1.68+ [a]	0.00	-0.07	-0.06	-1.81+ [c]	-0.03	-0.86

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. All models tested in full sample and Caucasian-only subsample to control for population stratification, differences are noted with subscripted letters; [1] boys = 0, girls = 1; SES = Socioeconomic status. $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 16

Longitudinal Relations of Parental Warmth in Middle Childhood and Vasopressin (AVPR1a; dominant coding) to Change in Externalizing/Competence Behaviors from Middle Childhood to Early Adolescence

	Externalizing/ Competence Mother Report		Externalizing/ Competence Father Report	
	Estimate	*Estimate/ SE*	*Estimate*	*Estimate/ SE*
Model 1	R^2_A = .48, R^2_B = .40		R^2_A = .48, R^2_B = .45	
Intercept	0.04	0.62	0.00	0.02
Child sex[1]	-0.13	-4.05**	-0.02	-0.72
Cotwin sex	-0.02	-0.46	-0.03	-0.94
T3 SES	-0.05	-2.43*	-0.03	-1.28
T2 Twin Behavior	0.55	14.24**	0.65	15.28**
T2 Cotwin Behavior	0.07	1.93+ [c]	0.07	2.01* [a]
T2 Mother warmth	-0.07	-3.40**	-0.01	-0.72
Twin AVPR1a	0.00	-0.01	0.02	0.56
Cotwin AVPR1a	0.02	0.25	0.00	0.09
T2 Warmth * Twin AVPR1a	0.04	1.16	-0.01	-0.18
T2 Warmth * Cotwin AVPR1a	0.07	2.31*	-0.01	-0.21
Model 2	R^2_A = .47, R^2_B = .39		R^2_A = .48, R^2_B = .45	
Intercept	0.05	0.68	0.01	0.18
Child sex[1]	-0.13	-3.98**	-0.02	-0.71
Cotwin sex	-0.01	-0.22	-0.03	-0.91
T3 SES	-0.04	-2.02* [b]	-0.02	-0.94
T2 Twin Behavior	0.57	14.40**	0.64	15.21**
T2 Cotwin Behavior	0.09	2.27*	0.07	1.91+
T2 Father warmth	-0.02	-0.85	-0.03	-1.26
Twin AVPR1a	-0.01	-0.08	0.02	0.36
Cotwin AVPR1a	0.02	0.21	0.00	0.03
T2 Warmth * Twin AVPR1a	0.07	1.92+ [a]	0.04	1.06
T2 Warmth * Cotwin AVPR1a	0.05	1.10	0.05	1.28

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. [1] boys = 0, girls = 1; SES = Socioeconomic status, T2 = Time 2 (Middle Childhood), T3 = Time 3 (Early Adolescence).
$^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$

Table 17

Longitudinal Relations of Parental Warmth in Middle Childhood and Vasopressin (AVPR1a; additive coding) to Change in Externalizing/Competence Behaviors from Middle Childhood to Early Adolescence

	Externalizing/ Competence Mother Report		Externalizing/ Competence Father Report	
	Estimate	*Estimate/ SE*	*Estimate*	*Estimate/ SE*
Model 1	R^2_A = .48, R^2_B = .39		R^2_A = .48, R^2_B = .45	
Intercept	0.06	1.97*	0.03	0.87
Child sex[1]	-0.13	-4.17**	-0.02	-0.66
Cotwin sex	-0.01	-0.41	-0.03	-1.02
T3 SES	-0.04	-2.17*	-0.03	-1.37
T2 Twin Behavior	0.55	14.24**	0.65	15.15**
T2 Cotwin Behavior	0.07	1.81+ [c]	0.07	1.98* [a]
T2 Mother warmth	-0.05	-2.83** [c]	-0.02	-0.87
Twin AVPR1a	0.02	0.92	0.00	-0.05
Cotwin AVPR1a	0.00	0.17	0.01	0.26
T2 Warmth * Twin AVPR1a	0.02	1.08	-0.02	-0.99
T2 Warmth * Cotwin AVPR1a	-0.01	-0.83	0.01	0.81
Model 2	R^2_A = .46, R^2_B = .40		R^2_A = .48, R^2_B = .45	
Intercept	0.06	2.00*	0.03	0.88
Child sex[1]	-0.13	-3.91**	-0.02	-0.74
Cotwin sex	-0.01	-0.26	-0.03	-0.92
T3 SES	-0.05	-2.50[c]	-0.03	-1.36
T2 Twin Behavior	0.56	14.49**	0.64	14.97**
T2 Cotwin Behavior	0.08	2.13*	0.07	1.78+ [a]
T2 Father warmth	0.01	0.34	-0.02	-0.79
Twin AVPR1a	0.01	0.64	0.00	0.08
Cotwin AVPR1a	0.01	0.40	0.00	0.07
T2 Warmth * Twin AVPR1a	-0.02	-0.74 [c]	-0.01	-0.50
T2 Warmth * Cotwin AVPR1a	-0.02	-0.75	-0.01	-0.41

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. [1] boys = 0, girls = 1; SES = Socioeconomic status, T2 = Time 2 (Middle Childhood), T3 = Time 3 (Early Adolescence).
$^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$

Table 18

Concurrent Relations of Neuropeptide-Y (NPY) Diplotypes and Parental Warmth to Externalizing/Competence Behaviors in Middle Childhood and Early Adolescence

	Middle Childhood Externalizing/Competence Behaviors				Early Adolescence Externalizing/Competence Behaviors			
	Mother Report		Father Report		Mother Report		Father Report	
	Estimate	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*	*Estimate*	*Estimate/SE*
Model 1	R^2_A = .15, R^2_B = .13		R^2_A = .08, R^2_B = .07		R^2_A = .20, R^2_B = .17		R^2_A = .12, R^2_B = .11	
Intercept	0.13	5.39**	0.09	3.78**	0.11	3.20**	0.11	2.94**
Twin sex[1]	-0.22	-8.32**	-0.18	-6.64**	-0.23	-5.93**	-0.13	-3.20**
Cotwin sex	0.00	0.00	-0.03	-1.02	0.01	0.20	-0.07	-1.70+ [a]
SES	-0.09	-5.59**	-0.05	-2.63**	-0.10	-4.46**	-0.08	-2.56* [b]
Mother warmth	-0.11	-6.89**	-0.06	-3.74**	-0.15	-6.62**	-0.11	-3.40**
Twin NPY	-0.09	-2.02* [b]	-0.06	-1.65+ [a]	-0.02	-0.50	-0.04	-0.97
Cotwin NPY	0.06	1.32	0.04	1.08	0.01	0.19	0.03	0.61
Warmth * Twin NPY	-0.05	-0.90	0.01	0.21	0.03	0.56	-0.02	-0.26
Warmth * Cotwin NPY	0.08	1.57	0.00	0.01	0.01	0.11	0.01	0.12
Model 2	R^2_A = .15, R^2_B = .13		R^2_A = .16, R^2_B = 15		R^2_A = .13, R^2_B = .11		R^2_A = .14, R^2_B = .13	
Intercept	0.13	5.32**	0.10	4.42**	0.13	3.63**	0.12	3.25**
Twin sex[1]	-0.22	-8.32**	-0.18	-7.09**	-0.24	-6.31**	-0.14	-3.56**
Cotwin sex	0.00	0.04	-0.04	-1.34	-0.01	-0.17	-0.08	-2.11* [b]
SES	-0.08	-5.28**	-0.04	-2.16*	-0.11	-4.50**	-0.07	-2.19* [a]
Father warmth	-0.12	-6.50**	-0.15	-9.57**	-0.05	-1.71+	-0.13	-4.57**
Twin NPY	-0.11	-2.34*	-0.07	-1.71+	0.00	0.04	-0.03	-0.61
Cotwin NPY	0.07	1.57	0.05	1.23	-0.01	-0.19	0.05	0.74
Warmth * Twin NPY	0.04	0.64	-0.02	-0.43	-0.06	-0.61	0.01	0.15
Warmth * Cotwin NPY	-0.11	-2.14*	-0.03	-0.64	-0.02	-0.25	-0.03	-0.42

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. All models tested in full sample and Caucasian-only subsample to control for population stratification, differences are noted with subscripted letters; [1] boys = 0, girls = 1; SES = Socioeconomic status. $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

Table 19

Longitudinal Relations of Neuropeptide-Y (NPY) Diplotypes and Parental Warmth in Childhood to Change in Externalizing/Competence Behaviors from Middle Childhood to Early Adolescence

	Externalizing/ Competence Mother Report		Externalizing/ Competence Father Report	
	Estimate	*Estimate/ SE*	*Estimate*	*Estimate/ SE*
Model 1	R^2_A = .48, R^2_B = .40		R^2_A = .48, R^2_B = .45	
Intercept	0.06	2.10*	0.03	0.90
Child sex[1]	-0.14	-4.27**	-0.02	-0.55
Cotwin sex	-0.01	-0.32	-0.03	-0.98
T3 SES	-0.05	-2.46*	-0.03	-1.36
T2 Twin Behavior	0.55	14.23**	0.65	15.07**
T2 Cotwin Behavior	0.06	1.72+ [c]	0.07	2.04* [a]
T2 Mother warmth	-0.04	-2.60**[c]	-0.02	-0.94
Twin NPY	0.01	0.29	-0.01	-0.31
Cotwin NPY	0.01	0.14	0.03	0.89
T2 Warmth * Twin NPY	0.08	1.23	-0.03	-0.98
T2 Warmth * Cotwin NPY	-0.03	-0.54	0.01	0.32
Model 2	R^2_A = .46, R^2_B = .40		R^2_A = .48, R^2_B = .46	
Intercept	0.06	2.10*	0.03	0.99
Child sex[1]	-0.13	-3.89**	-0.02	-0.62
Cotwin sex	-0.01	-0.22	-0.03	-1.02
T3 SES	-0.05	-2.38*	-0.03	-1.21
T2 Twin Behavior	0.56	13.74**	0.64	14.79**
T2 Cotwin Behavior	0.08	1.94+ [c]	0.07	1.81+ [a]
T2 Father warmth	-0.01	-0.36	-0.02	-1.13 [c]
Twin NPY	0.01	0.15	-0.02	-0.51
Cotwin NPY	0.01	0.22	0.04	1.12
T2 Warmth * Twin NPY	-0.02	-0.56	-0.03	-0.81
T2 Warmth * Cotwin NPY	-0.05	-1.36	-0.01	-0.29

[a]Nonsignificant in the subsample.
[b]$p < .10$ in the subsample.
[c]$p < .05$ in the subsample.
Note. [1] boys = 0, girls = 1; SES = Socioeconomic status, T2 = Time 2 (Middle Childhood), T3 = Time 3 (Early Adolescence).
$^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$

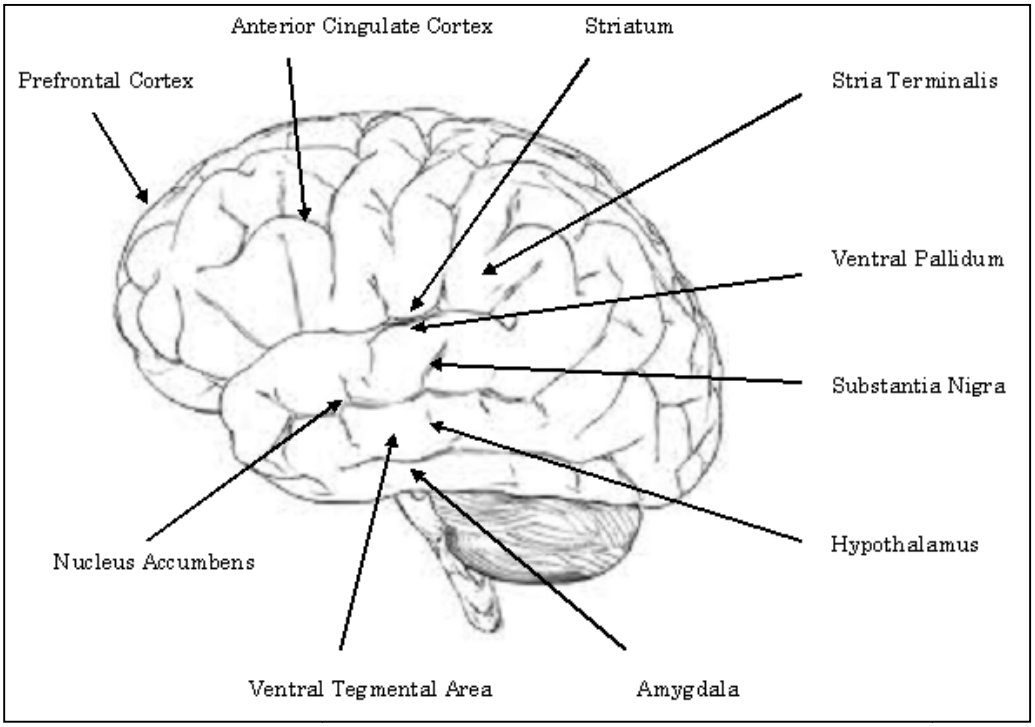
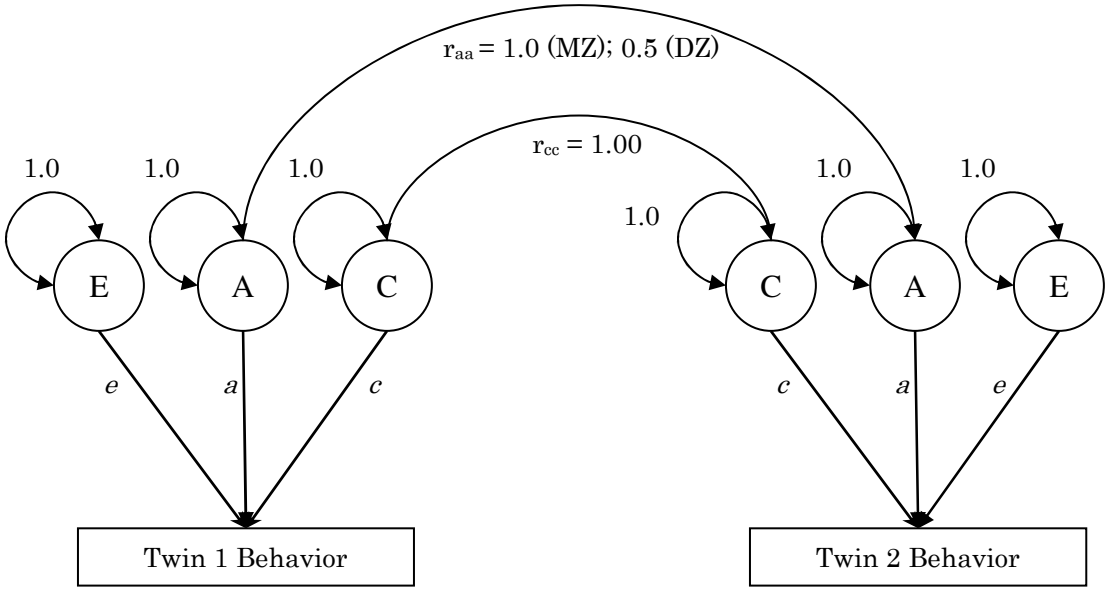


Figure 1. Relevant structures and areas of left hemisphere of human brain.



$r_{MZ} = a^2 + c^2$; $r_{DZ} = .5a^2 + c^2$

Figure 2. Pictoral model of the basic twin decomposition model (multigroup structural equation model), estimating the genetic, shared environment, and unique environment path coefficients (*a, c,* and *e*; respectively) for three latent variables (A, C, and E) on a measured phenotype; dominant genetic effects (D) can be tested in place of C by adjusting the DZ r_{aa} to 0.25.

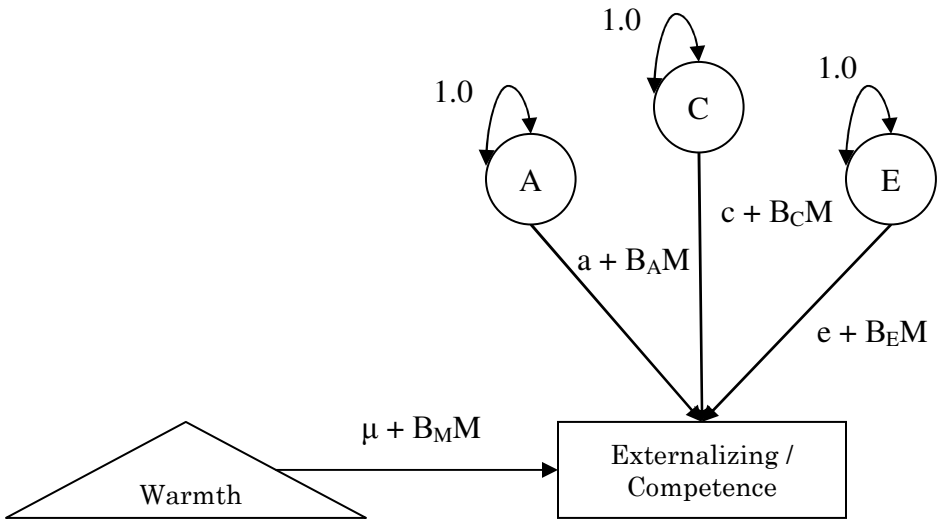


Figure 3. Partial moderation model (shown for one twin only) decomposing variances and covariances of observed parental warmth/rejection as a moderator of the heritability of observed child externalizing/competence, shown for one twin only. The model simultaneously estimates genetic and experiential effects specific to externalizing/competence (a, c, e) as well as the possibility that parental warmth/rejection moderates the influences unique to externalizing/competence. Paths to externalizing/competence contain three components; a, c and e which equal, respectively, the parameters for shared genetic, shared experiential, and unique experiential influence on externalizing/competence, B = regression coefficient, M = level of warmth/rejection. Moderation is tested by comparing the full model to a model in which the four moderation parameters are fixed at zero.

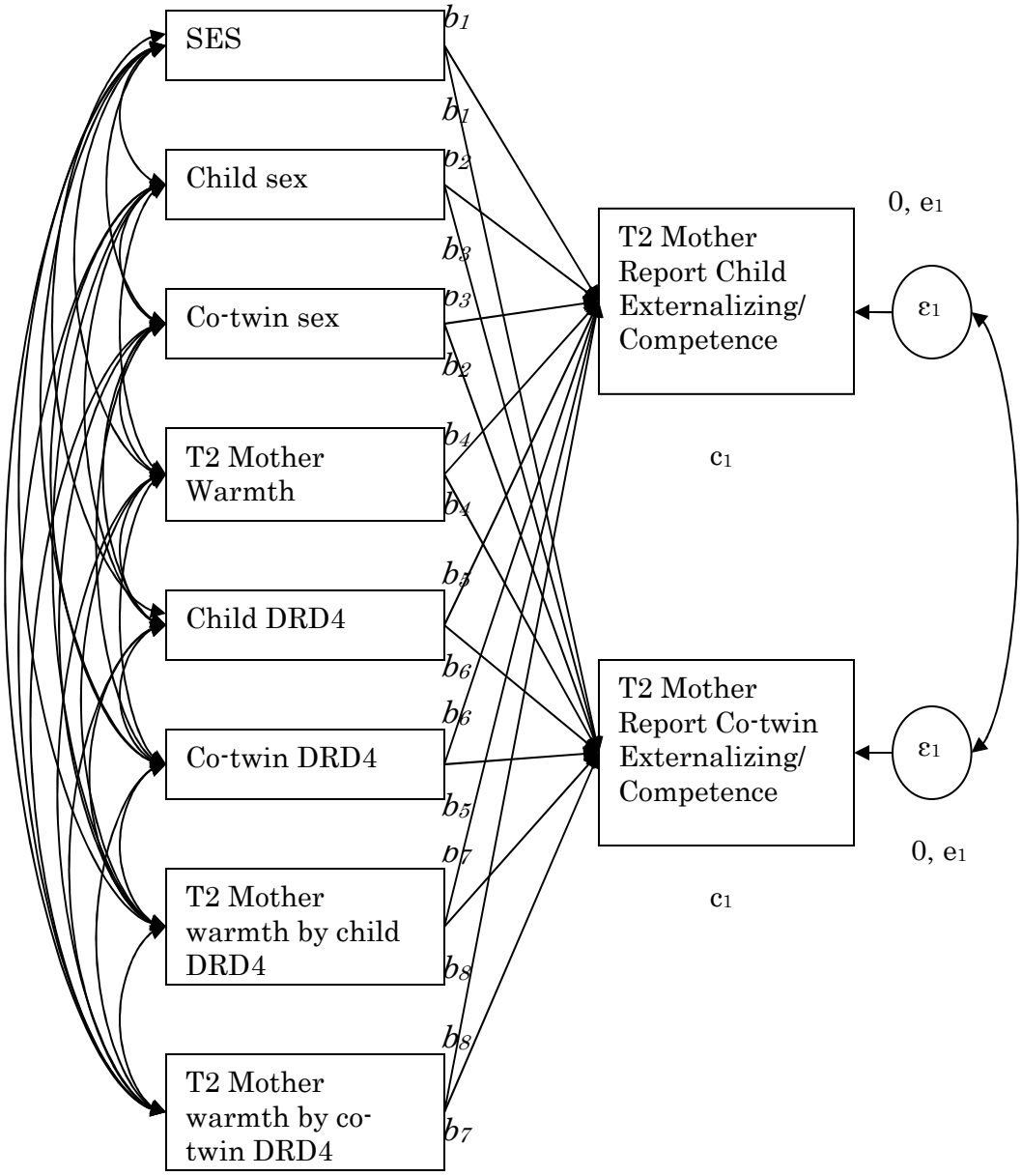


Figure 4. The twin-cotwin interchangeable structural equation model with an interaction effect. Simple predictor effects (b_1, b_2, b_4, b_5, b_7), twin on cotwin and cotwin on twin effects (b_3, b_6, b_8), predictor means, predictor variances and covariances, outcome intercepts (c), and residual variances (e) were constrained according to guidelines for the twin-cotwin interdependence model for interchangeable dyads (Olsen & Kenny, 2006).

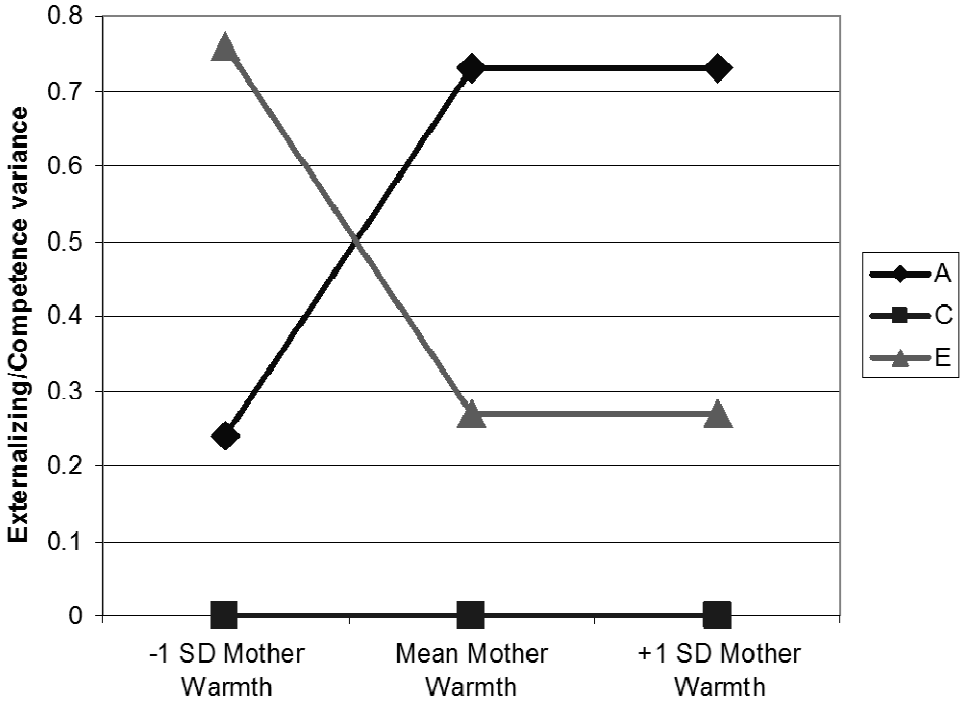


Figure 5. Significant moderation of mother warmth on the A and E variance path coefficients of mother report externalizing/competence at Time 2 (middle childhood); A = genetic influence, C = shared environment influence, E = unique environment influence.

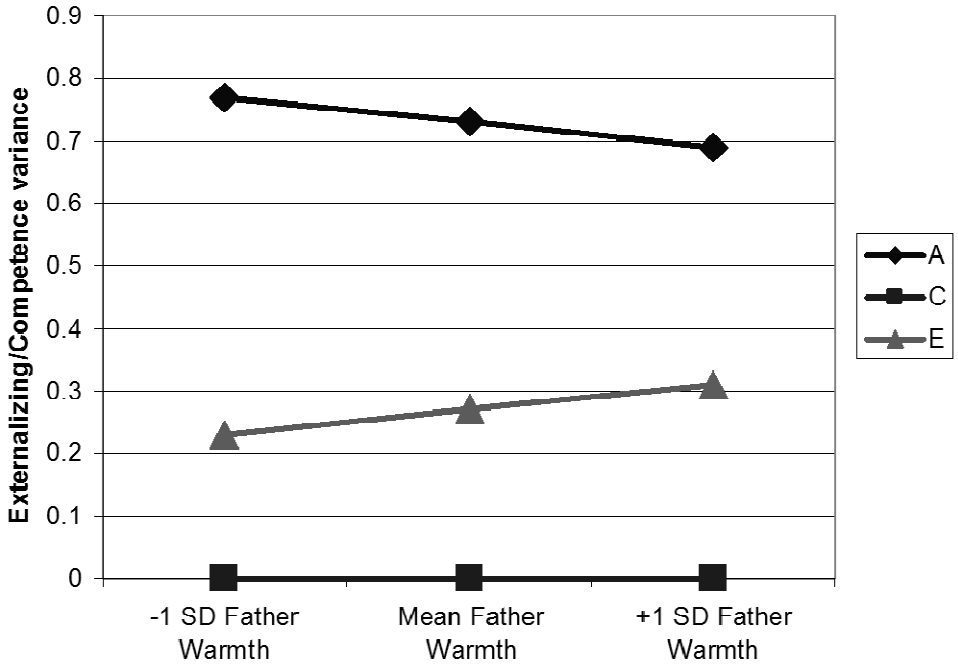


Figure 6. Significant moderation of father warmth on the E variance path coefficient of mother report externalizing/competence at Time 2 (middle childhood); A = genetic influence, C = shared environment influence, E = unique environment influence.

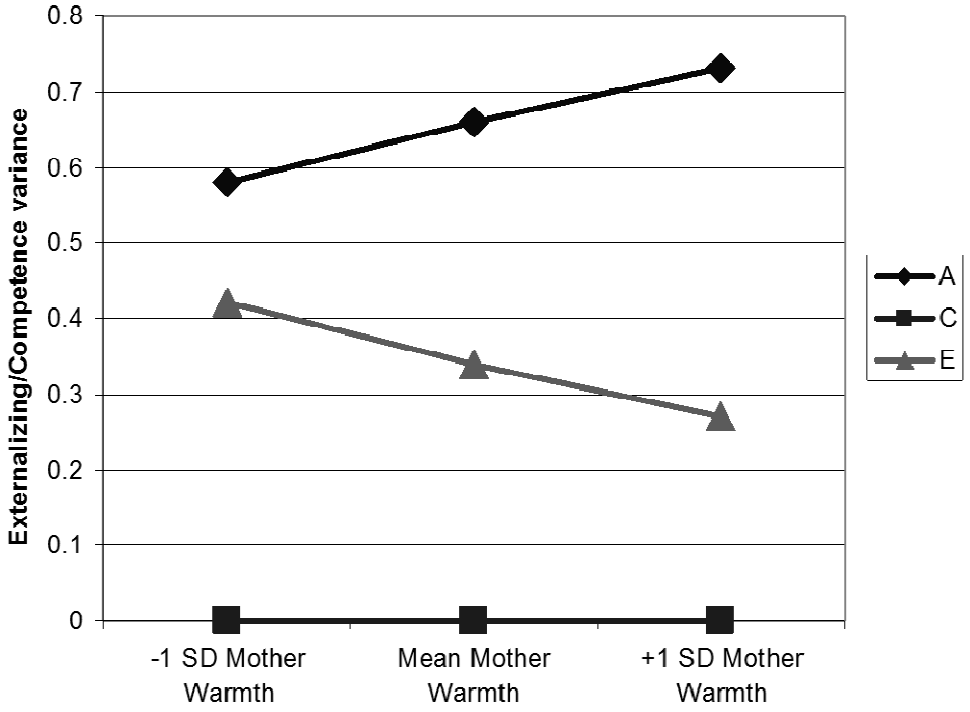


Figure 7. Significant moderation of mother warmth on the A variance path coefficient of mother report externalizing/competence at Time 3 (early adolescence); A = genetic influence, C = shared environment influence, E = unique environment influence.

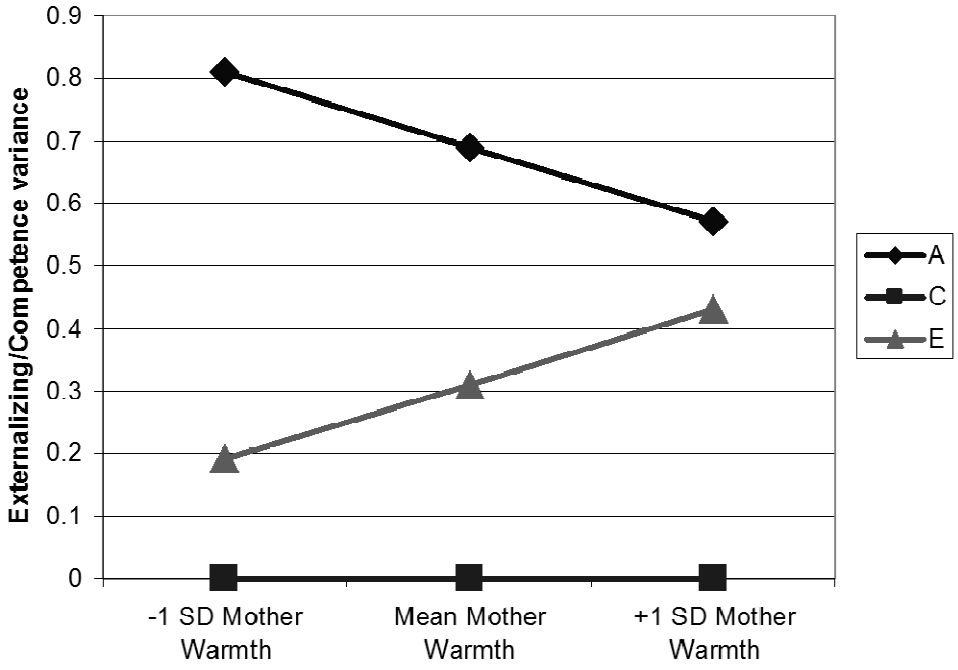


Figure 8. Significant moderation of mother warmth on the E variance path coefficient of father report externalizing/competence at Time 3 (early adolescence); A = genetic influence, C = shared environment influence, E = unique environment influence.

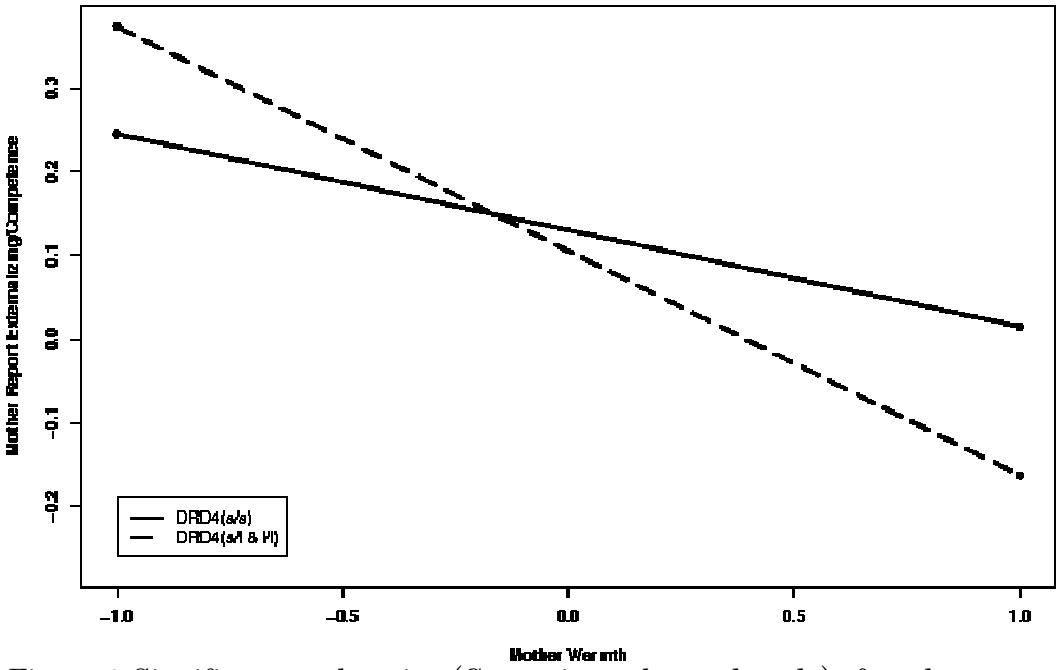


Figure 9. Significant moderation (Caucasian subsample only) of mother warmth by dopamine (DRD4) on mother report of externalizing/competence at Time 2.

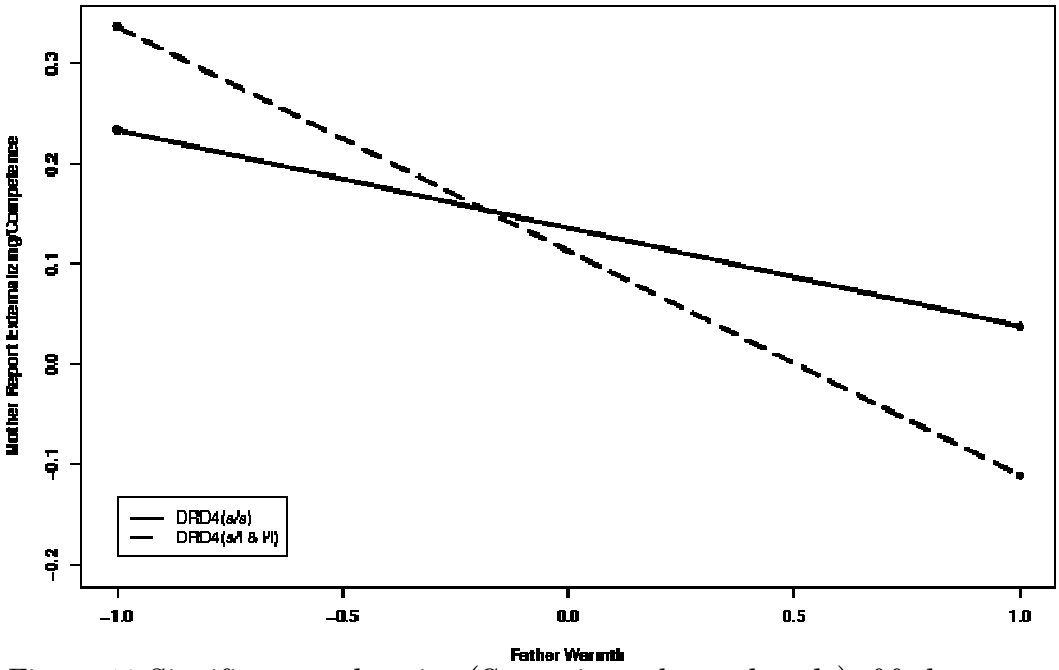


Figure 10. Significant moderation (Caucasian subsample only) of father warmth by dopamine (DRD4) on mother report of externalizing/competence at Time 2.

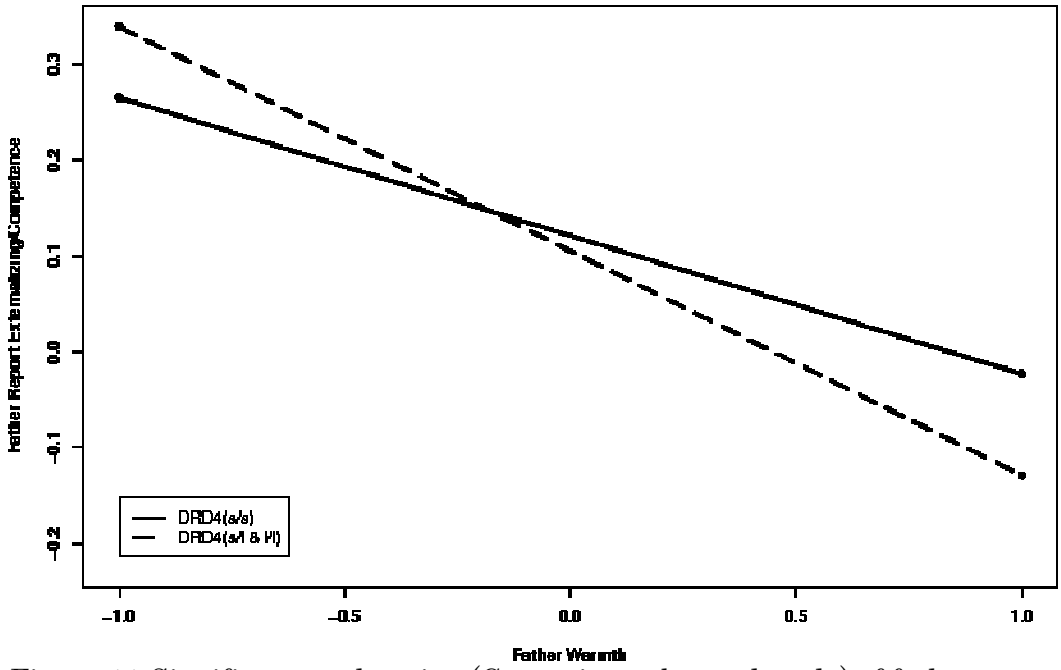


Figure 11. Significant moderation (Caucasian subsample only) of father warmth by dopamine (DRD4) on father report of externalizing/competence at Time 3.

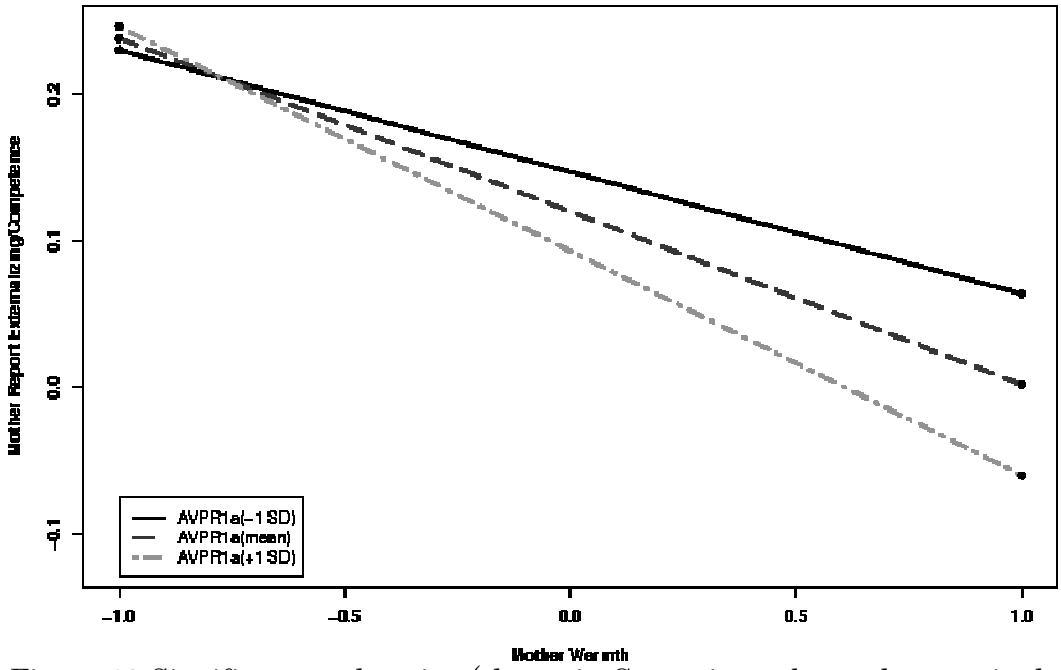


Figure 12. Significant moderation (shown in Caucasian subsample, marginal in full sample) of mother warmth by vasopressin (AVPR1a; additively coded) on mother report of externalizing/competence at Time 2.

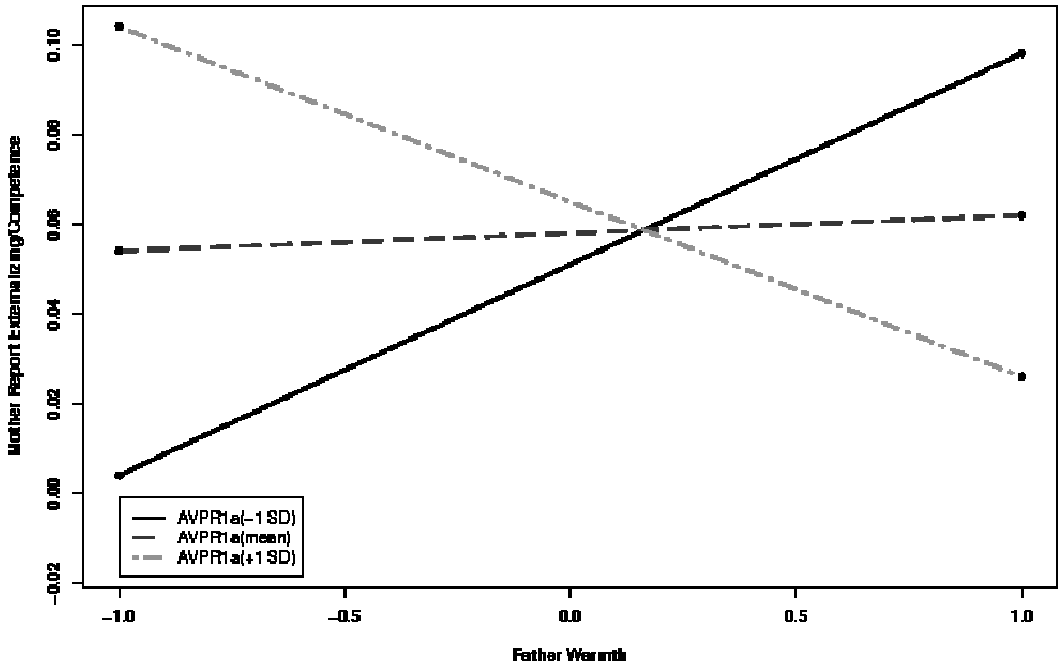


Figure 13. Significant longitudinal moderation of father warmth in middle childhood by vasopressin (AVPR1a; additively coded) on change in mother report of externalizing/competence from Time 2 to Time 3.

APPENDIX A

CHILD-REARING PRACTICES REPORT

This first set of questions asks about your attitudes on childrearing and how you plan to raise your twins. Please respond with the number that best reflects your degree of agreement or disagreement to each statement. Refer to Card A for the response choices. The card should read:

1	2	3	4	5	6
STRONGLY DISAGREE	MODERATELY DISAGREE	SLIGHTLY DISAGREE	SLIGHTLY AGREE	MODERATELY AGREE	STRONGLY AGREE

Encouraging Independence

1. I respect my twins' feelings and opinions and encourage the twins to express them:
2. When my twins get into trouble, I will expect them to handle the problem mostly by themselves:
8. I take into account my twins' preference in making plans for the family:
10. I try to let my twins make many decisions for themselves:
17. I intend to give my twins a good many duties and family responsibilities:
30. I intend to teach my twins that they are responsible for what happens to them:
33. I encourage my twins to be independent of me:

Open expression of affect

3. I feel children should be given comfort and understanding when they are scared or upset:
6. I express affection by hugging, kissing, and holding my twins:
14. I am easy going and relaxed with my twins:
16. I joke and play with my twins:
18. My twins and I have warm, intimate times together:
27. When I am angry with my twins, I let them know it:

Encouraging openness of expression

7. I try to encourage my twins to wonder and think about life:
9. I feel children should have time to think, daydream, and even loaf sometimes:
20. I encourage my twins to be curious, to explore, and to question things:
24. I intend to encourage my twins to talk about their troubles:

Rational guiding of child

15. I try to talk it over and reason with my twins when they misbehave:
22. I believe in praising children when they are good and think it gets better results than punishing them when they are bad:
23. I make sure that my twins know that I appreciate what they try or accomplish:

APPENDIX B

BRIEF INFANT-TODDLER SOCIAL AND EMOTIONAL ASSESSMENT

Instructions: This questionnaire contains statements about 1- to 3-year-old children. Many statements describe normal feelings and behaviors, but some describe things that can be problems. Some may seem too young or too old for your child. Please do your best to answer
every question.

For each statement, please circle the answer that best describes your child in the LAST MONTH.

0 = not true/rarely 1 = somewhat true/sometimes 2 = very true/often

Externalizing:
7. Cries or tantrums until s/he is exhausted
23. Often gets very upset
27. Hits, shoves, kicks, or bites children (not including brother/sister)
28. Is destructive. Breaks or ruins things on purpose
30. Hits, bites or kicks you (or other parent)
33. Purposefully tries to hurt you (or other parent)

Competence
1. Shows pleasure when s/he succeeds (for example, claps for self)
5. Follows rules
19. Plays well with other children (not including brother/sister)
20. Can pay attention for a long time
22. Tries to help when someone is hurt. For example, gives a toy
25. Imitates playful sounds when you ask him/her to
31. Hugs or feeds dolls or stuffed animals

APPENDIX C

MACARTHUR HEALTH AND BEHAVIOR QUESTIONNAIRE

Please read the following list of behaviors that some children exhibit during middle childhood. For each behavior, please respond with the statement that best describes how much the behavior applies to your child thinking about *the past 6 months*.

Rarely applies	Applies somewhat	Certainly applies
0	1	2

Externalizing

6. Steals; takes things that don't belong to her/him
14. Lies or cheats
23. Vandalizes
28. Sets fires
33. Cruel to animals
39. Physically attacks people
46. Threatens people
53. Destroys her/his own things
59. Destroys things belonging to her/his family or other children
67. Disobedient at school
73. Cruel, bullies or mean to others
80. Uses a weapon when fighting
3. Has temper tantrums or hot temper
11. Argues a lot with adults
12. Argues a lot with peers
21. Defiant, talks back to adults
37. Blames others for her/his own mistakes
44. Is easily annoyed by others
50. Angry and resentful
57. Gets back at people
64. Swears or uses obscene language
19. Taunts and teases other children
31. Does things that annoy others
70. Kicks, bites or hits other children
78. Gets in many fights

Competence

1. If there is a quarrel or dispute, s/he will try to stop it
2. Offers to share materials or tools being used in a task
3. Will invite bystanders to join in a game
4. Will try to help someone who has been hurt
5. Apologizes spontaneously after a misdemeanor
6. Shares candies and extra food

7. Is considerate of others' feelings
8. Stops talking quickly when asked to
9. Spontaneously helps to pick up objects someone has dropped
10. Takes the opportunity to praise the work of less able children
11. Shows sympathy to someone who has made a mistake
12. Offers to help other children who are having difficulty with a task
13. Helps other children who are feeling sick
14. Can work easily in a small peer group
15. Comforts a child who is crying or upset
16. Is efficient in carrying out regular tasks, such as helping with household chores
17. Settles down to work quickly
18. Will clap or smile if someone else does something well
19. Volunteers to help clean up a mess someone else has made
20. Tries to be fair in games

For each of the following questions, please choose the statement that best characterizes your child's relationships with others *during the past six months*.

Not well at all Constant Problems	Not too well frequent problems	Pretty well occasional problems	Quite well hardly any problems	Very well no problems
0	1	2	3	4

Competence

1. During the past six months, how well has s/he gotten along with children outside of your family, such as friends and classmates?
2. During the *past six months*, how well has *s/he* gotten along with *her/his* teacher(s) at school?
3. During the past six months, how well has s/he gotten along with family members?