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SPECIAL SECTION ARTICLE

Heritability of children's prosocial behavior and differential susceptibility to parenting by variation in the dopamine receptor D4 gene

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Abstract

Theoretical considerations and new empirical evidence suggest that children's development cannot simply be explained by either genes or environment but that their interaction is important to understanding child behavior. In particular, a genetic polymorphism, the exon III repeat region of the dopamine receptor D4, has been the focus of interest regarding differential susceptibility to parental influence. To study environmental and genetic influences on children's prosocial behavior, 168 twin pairs (mean age = 44 months) participated in an experiment that assessed prosocial behavior via three measures: compliant prosocial behavior elicited in response to social requests, self-initiated prosocial behavior enacted voluntarily, and mothers' rating of children's behavior. Genetic effects accounted for 34% to 53% of the variance in prosocial behavior. The rest of the variance was accounted for by nonshared environment and error. Parenting measures of maternal positivity, negativity, and unexplained punishment did not correlate significantly with children's prosocial behavior. However, when parenting was stratified by presence or absence of the child's dopamine receptor D4 7-repeat allele in an overlapping sample of 167 children to model differential susceptibility to parental influence, a richer picture emerged. Positive parenting related meaningfully to mother-rated prosocial behavior, and unexplained punishment related positively to self-initiated prosocial behavior, but only among children carrying the 7-repeat allele. The findings demonstrate that a molecular genetic strategy, based on genotyping of common polymorphisms and combined with a classic twin approach, provides a richer description of how genes and environment interact to shape children's behavior, and allows for the identification of differential sensitivity to parental influence.

Theoretical considerations and new empirical evidence suggest that children's development cannot simply be explained by either biological or contextual factors but that their interaction is important to understanding contributions to child behavior (Ellis & Boyce, 2008). There is growing evidence for the importance of Gene × Environment interactions (G × E), in which the relationship between child outcomes (such as cognitive abilities, aggressive tendencies, and externalizing behaviors) and environmental factors (such as socialization patterns, parental abuse, and socioeconomic status) is moderated by genetic factors (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Caspi et al., 2002). In particular, a genetic polymorphism in the exon III repeat region of the dopamine receptor D4 (*DRD4*) gene has been the focus of interest regarding differential susceptibility to parental influence (Bakermans-Kranenburg & van IJzendoorn, 2007).

Research on the development of prosocial behavior has largely focused on parenting (for reviews, see Eisenberg, Fabes, & Spinrad, 2006; Hastings, Utendale, & Sullivan, 2007) and to a lesser extent on genetic influences on prosociality (reviewed by Knafo & Israel, 2009). The current study will address the joint contribution of genetics and the parenting environment to children's prosocial development. Following a brief review of the evidence for the effects of children's genes and the parenting they receive on prosocial development, we will address the issue of G × E, in particular, the topic of genetically based differential susceptibility to parenting. We will then provide evidence from a twin sample for the genetic and environmental influences on observed and mother-rated prosocial behavior. Turning to the core question of the current investigation, we provide evidence for genetic differential susceptibility to parenting with regard to prosocial behavior, focusing on the exon III repeat region of *DRD4* (*DRD4-III*).

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Varieties of Prosocial Behavior

Prosocial behavior is defined here as voluntary behavior enacted with the intent of benefiting others (e.g., Eisenberg et al., 2006). Exemplary prosocial behaviors include sharing personal resources, providing instrumental help, and supporting others emotionally in times of distress (Eisenberg et al., 2006). A related construct is empathy, a vicarious response to others' affective states (e.g., Hoffman, 1988). Empathy has been shown to relate positively to prosocial behavior (e.g., Van Lange, 2008) and arguably serves as the motivational basis for many prosocial behaviors (Batson, 2009; de Waal, 2008). We therefore rely on the literature on empathy in addition to that of prosocial behavior.

In this study we asked mothers to rate children's overall prosocial behavior and observed children's behaviors of helping, sharing, and comforting in an experimental setting. We also made an important distinction between compliant prosocial behavior (following a specific verbal or nonverbal social request) and self-initiated prosocial behavior (performed without an explicit request; Eisenberg, Cameron, & Tryon, 1984). Self-initiated and compliant prosocial behavior have been shown to intercorrelate only weakly or not at all (Eisenberg et al., 1984). The self-initiated/compliant distinction is relevant to age differences in prosocial behavior (Eisenberg, Wolchik, Goldberg, & Engel, 1992; Zarbatany, Hartmann, & Gelfand, 1985) and to children's affective knowledge (Knafo, Steinberg, & Goldner, in press). The distinction is also meaningful because it refers to the underlying motivation behind prosocial behavior; a higher level of self-initiated (but not compliant) prosocial behavior has been suggested to require a conviction in justice and/or caring for the well-being of other individuals (Bar-Tal, 1982). In addition, children's moral judgment was found to be related to spontaneous high-cost prosocial behavior but not to compliant prosocial behavior (Eisenberg, Pasternack, Cameron, & Tryon, 1984). It is therefore imperative to study both kinds of prosocial behavior.

Research has shown how parenting techniques can have selective effects on concomitant child prosocial behaviors (Grusec, 1991). For example, parents' reinforcement of prosocial behavior following a request, but not of spontaneous prosocial behavior, related negatively to children's compliant prosocial behavior (Eisenberg et al., 1992). However, no study has addressed separately the genetic influences on the two kinds of prosocial behavior. This study will be the first to estimate the genetic and environmental contributions to self-initiated and compliant prosocial behavior, as well as the role of $G \times E$ in the development of the two types of prosociality.

Genetic Influences on Prosocial Behavior

Most studies of genetic and environmental contributions to individual differences in prosocial behavior have used the twin design, which compares monozygotic (MZ) twins, who share virtually all of their genetic sequence, with dizygotic (DZ) twins, who share on average half of their genes (e.g., Gregory, Light-Häusermann, Rijsdijk, & Eley, 2009; Hur & Rushton 2007). The twin method uses this genetic difference in conjunction with the equal environments assumption, that MZ and DZ twins growing up in their biological families are equal in terms of how similar their environments are, in which case greater similarity of MZ twins indicates genetic influence (*heritability*). Similarity beyond this genetic effect is attributed to the environment the twins share (*shared environment* effect), and any further differences between the twins are ascribed to *nonshared environment* and measurement error (see Plomin, DeFries, McClearn, & McGuffin, 2001).

Twin studies show a genetic effect on children's prosocial behavior and empathy (Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008; Scourfield, John, Martin, & McGuffin, 2004; for an exception, see van IJzendoorn, Bakermans-Kranenburg, Pannebakker, & Out, 2010). Genetic effects increase in importance, whereas shared environment effects decrease in importance throughout childhood, and the rest of the variance is accounted for by the nonshared environment and error (for a review, see Knafo & Israel, 2009). It is important to note that nonshared environmental effects, which have been shown to increase in importance in early childhood (Knafo & Plomin, 2006b), may indicate the presence of within-family differences in susceptibility to parental influences (Belsky, Hsieh, & Crnic, 1998; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011 [this issue]; Kan, Ploeger, Raijmakers, Dolari, & van der Maas, 2010), a topic we address below.

Only a handful of studies have addressed the genetic influences on prosocial behavior with measured genetic polymorphisms. Single nucleotide polymorphisms in the oxytocin receptor gene are associated with donation of money to an unknown stranger in the dictator game (Israel et al., 2009) as is the length of the promoter region of the gene coding for the arginine vasopressin receptor (Knafo, Israel, et al., 2008). A functional polymorphism on the *DRD4* gene, the variable number tandem repeat on the third exon (*DRD4-III*), has also been related to young women's self-reported selflessness, a form of extreme prosociality in which one's interests are effaced in favor of others (Bachner-Melman, Gritsenko, Nemanov, Zhoar, & Ebstein, 2005) and to preschool-age twins' sharing with each other (DiLalla, Elam, & Smolen, 2009). Although our main focus was to test the interaction of parenting with this polymorphism with regard to children's prosocial behavior, our design enabled a conceptual replication test for this finding with children's observed and mother-reported prosocial behavior. Below we provide more details about this polymorphism.

Parenting and Prosocial Behavior

Many environmental forces, such as schools, peer, and the media (Eisenberg et al., 2006), may influence prosocial behavior, as indicated by the environmental estimates derived from twin studies. The focus of the current study, as well as most of the environmental research on individual differences in prosociality (Eisenberg et al., 2006; Hastings et al., 2007; Staub, 1979), is on differences in parental attitudes, affective

responses, and behaviors toward their children, often generally referred to as parenting.

In an influential review, Eisenberg et al. (2006) surveyed the extensive literature on the relationship between prosocial behavior and a variety of parenting attitudes and disciplinary practices. The bottom line of this vast literature is that the positive aspects of parenting, such as induction (a reasoning practice that can increase children's awareness of the consequences of their behavior to others), warmth and support toward children (providing a caring model for children, and increasing children's willingness to attend to parental messages), and autonomy support (focusing on the child's needs and abilities rather than imposing rules and directives) are related to children's empathy and prosocial behavior, whereas the opposite is true for power-assertive and negative discipline (Clark & Ladd, 2000; Hoffman, 1970; Krevans & Gibbs, 1996; Staub, 1979; Whiteside-Mansell, Bradley, Tresch Owen, Randolph, & Cauce, 2003). In a longitudinal twin study of parental positivity and negativity toward children, these effects were replicated with regard to parent-rated and teacher-rated prosocial behavior, with parental positivity having a longitudinal positive effect on change in prosociality over and above earlier prosociality (Knafo & Plomin, 2006a).

Note, however, that Eisenberg et al. (2006) also reviewed a number of null findings in which parenting did not relate to prosocial behavior. These exceptions typically indicated that the effects noted by prior studies are less relevant at certain ages, for one of the sexes, or when a particular measure of prosocial behavior is used. It is therefore important to consider moderating factors affecting the parenting–prosociality relationship. One such factor, children's genetics as a basis for differential susceptibility to parenting, is discussed in the next section. Another important factor concerns the context of the prosocial behavior performed, for example, whether it is performed following a request (Grusec, 1991).

In a recent review of parenting across socialization and parent–child interaction domains, Grusec and Davidov (2010) show strong evidence that parents may be successful in one domain of interaction but not in another, because successful parenting in each domain of interaction requires parents to show different abilities and invest different resources in their interaction with children. It is therefore important to study parenting in relation to different aspects of prosocial behavior. In the current study we used three measures of prosociality: overall mother-rated prosocial behavior, referring mainly to behavior in the peer group; compliant prosocial behavior, in reaction to an experimenter's request; and self-initiated prosocial behavior toward an experimenter without a preceding request.

In addition to overall parental positivity and negativity, one particular aspect of parenting we studied was the role of punishment. Eisenberg et al. (2006) reported in their review that parental punishment was either unrelated or negatively related to prosocial behavior, suggesting a modest negative relationship between punishment and prosociality. However, punishment could relate positively to empathy if it was accompanied by high levels of inductive discipline (Miller, Eisenberg, Fabes, Shell, & Gular, 1989). In the current study we operationalized punishment as a punitive behavior performed without explanation of the reason for the punishment. Thus, one could expect unexplained punishment to relate negatively to prosocial behavior. In contrast, avoidance of punishment is a powerful motive that can increase prosocial behavior when children can expect that such behavior would reduce the likelihood of punishment (Eisenberg et al., 2006; Hartmann et al., 1976). In the following section we propose that some children may be especially sensitive to unexplained punishment, and could react more strongly to punishment by developing a tendency to react prosocially to avoid punishment.

Gene × Parenting Interactions

Developmental research increasingly recognizes that the effects of parenting are dependent on children's tendencies (e.g., Rubin, Burgess, & Hastings, 2002). For example, in a study of children's internalization of mothers' demand not to touch desired toys in mothers' absence, Kochanska (1995) found that maternal discipline deemphasizing power related positively to internalization, but only among fearful/anxious children, whereas for other children a sense of secure attachment predicted internalization. In another study, adolescents' prosocial behavior was not predicted by parental support, but by the interaction of adolescents' temperament and parents' support (Carlo, Roesch, & Melby, 1998). As temperament is substantially heritable (Benish-Weisman, Steinberg, & Knafo, 2011; Plomin et al., 2001; Saudino, Plomin, & DeFries, 1996), these interactions between children's and adolescents' temperament and the parenting they receive suggest the need to look for genetic moderators of the relationship between parenting and prosocial behavior.

Although the theoretical importance of G × E has been known for a long time (Plomin, DeFries, & Loehlin, 1977), strong empirical evidence in psychological research has emerged only in the last decade (e.g., Caspi et al., 2002). In our study (Knafo & Israel, 2009) we approached this issue by comparing the genetic contributions to individual differences across different levels of a measured environmental variable (e.g., Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Changes in heritability across levels of the environmental variable suggest that the magnitude of genetic effects is influenced by the environment. With regard to prosociality, we focused on the presence of other siblings as the moderating environmental variable and found that in families in which twins were the only children an additive genetic effect of 0.23 and a nonshared environment effect of 0.77 accounted for individual differences in children's mother-reported prosocial behavior. In contrast, in families with additional siblings, a larger nonadditive genetic effect of 0.64 to 0.72 was found, illustrating how genetic and environmental factors interact to affect prosocial behavior (Knafo & Israel, 2009).

A more powerful approach to G × E compares the association between a phenotype and a measured environmental variable across different measured genetic profiles (Caspi et al.,

2002). Based on Belsky's (1997) evolutionary reasoning that it would be adaptive for children to differ in their susceptibility to environmental influences (see also Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011 [this issue]), the idea that some children are genetically more susceptible to parental influence has been proposed. Belsky's reasoning suggests that even within the same family, children might differ genetically in their susceptibility to rearing experiences (Belsky, 1997), although this within-family effect has not been tested previously in G × E studies.

Several polymorphisms involved in the serotonergic and dopaminergic systems have been shown to underlie differential susceptibility to parental influence (Belsky & Pluess, 2009b; Belsky et al., 2007; Propper & Moore, 2006). The dopaminergic system is a particularly appealing candidate for differential susceptibility because it plays a critical role in the "stamping-in" of motivational importance to environmental stimuli (Wise, 2004). This association building likely translates to the parenting domain as well, where the effectiveness of maternal influences on motivation, learning, and reinforcement are fundamentally dependent on dopamine-based mesolimbic reward pathways. Genetic variability in dopaminergic genes can serve as a modulatory mechanism for limiting the sensitivity of maternal influences on developmental outcome. This is particularly relevant for the development of social behavior, where the dopaminergic system plays a critical role (Insel, 2003).

To date, the candidate gene showing the most promise for differential susceptibility is the *DRD4-III* polymorphism, a variable number tandem repeat ranging from 2 to 10 repeats of a 48 base pair sequence; the 2, 4, and 7 repeats are the most common (Ashgari et al., 1995). As noted by David and Munafo (2008), the 7-repeat allele is associated with decreased ligand binding, lower levels of gene expression in vitro, and attenuated cyclic AMP when dopamine is bound to the receptor, resulting in a suboptimal response to dopamine and lower dopaminergic tone. In addition, the 7-repeat allele has been associated with the human temperament of novelty seeking (Benjamin et al., 1996; Ebstein et al., 1996). Although later studies did not find such significant associations with novelty seeking (Canli, 2008; Munafò, Yalcin, Willis-Owen, & Flint, 2008), evidence suggests that the relationship is more consistent with younger samples (Dillala et al., 2009; Laucht, Becker, & Schmidt, 2006). In addition, boys carrying the 7-repeat allele exhibit greater rates than carriers of other alleles in visual exploratory behavior, such as response decrement over repeated stimulation, in early infancy (Laucht et al., 2006). A study of Hungarian infants showed that children with the 7-repeat allele (7-present) were less likely to develop secure attachment (Gervai et al., 2005). Together with the visual exploratory behavior and novelty seeking findings, a behavioral pattern emerges for 7-present children in which they are more alert, less secure, and possibly constantly scanning the environmental contingencies they face.

Lower dopaminergic tone of 7-present individuals may also modulate sensitivity to reward and increase reward dependence, supporting the hypothesis that 7-present children differ from other children attentionally and motivationally; along these lines van IJzendoorn and Bakermans-Kranenburg (2006) successfully predicted that 7-present infants would show a stronger association between maternal unresolved loss or trauma and infant attachment disorganization.

Since then, a myriad of studies have provided evidence for *DRD4-III* as a polymorphism related to susceptibility to parental influence. Bakermans-Kranenburg and van IJzendoorn (2006) reported a sixfold increase in externalizing behaviors in 7-present children exposed to parental insensitive care, in comparison with children without this combination of risk factors. An American study also found an interaction between parental sensitivity and longer versions (≥6 repeats) of child *DRD4-III* genotype in predicting children's externalizing behaviors (DiLalla et al., 2009). A powerful demonstration of the interaction between parenting and the *DRD4-III* polymorphism was provided by Bakermans-Kranenburg, van IJzendoorn, Pijlman, Mesman, and Juffer (2008). In a randomized controlled trial of Dutch 1- to 3-year-old children with high levels of externalizing behavior, a video-feedback intervention was performed to promote positive parenting and sensitive discipline. For 7-present children, the intervention was effective in decreasing externalizing behavior, whereas for children without the *DRD4-III* 7-repeat allele (7-absent) the intervention was not effective. In addition, intervention effects were largest in 7-present children whose parents showed the largest increase in the use of positive discipline.

Sheese, Voelker, Rothbart, and Posner (2007) observed an interaction between *DRD4* genotype and the influence of parenting quality on an aggregate measure of child sensation seeking. High-quality parenting predicted reduced levels of activity level, impulsivity, and high-intensity pleasure, but only in the presence of the 7-repeat allele. In 7-absent children, parenting quality did not influence sensation seeking. In interpreting their results, Sheese et al. posited an evolutionary accounting for the increased sensitivity of the 7-repeat allele on parenting style, noting that it could allow caregivers more latitude in influencing child temperament, allowing for greater adaptability to the social environment.

Differential susceptibility to parental influence implies not only the presence of G × E, but also a particular pattern of the interaction (Boyce et al., 1995). In this pattern of interaction, the more susceptible children are not only affected negatively by inappropriate, insensitive, or incompetent parenting, but also benefit from having the opposite pattern of parenting, to the extent of being better adjusted than low-susceptibility children with competent parents (Bakermans-Kranenburg & van IJzendoorn, 2007; Belsky et al., 2007; Belsky & Pluess, 2009a; Boyce & Ellis, 2005). However, note that all of these studies had children's malfunctioning (externalizing or disorganized attachment) as their phenotype of interest. More direct evidence for the positive outcomes for susceptible children in favorable parenting environments is needed.

Although one study (DiLalla et al., 2009) found no significant parenting by *DRD4-III* interaction in predicting twin

children's sharing in a play session with their parents and their co-twins, it is important to study the G × E of prosocial behavior and parenting in additional contexts. For example, in a recent study, an interaction was found in which children's representations of secure attachment to their mothers (possibly an indicator of quality of parenting) were positively associated with donations to UNICEF, but only among carriers of the *DRD4-III* 7-repeat allele (Bakermans-Kranenburg & van IJzendoorn, 2009). Finally, in a small twin sample (partially overlapping with the one reported in the present investigation), mother-reported negativity toward their child correlated negatively with children's empathic concern toward an examiner's simulated pain, again only among 7-present children (Knafo & Uzefovsky, in press).

Based on these considerations, we hypothesized that the relationships between parenting and prosocial behaviors would be stronger among 7-present children than among 7-absent children. Particularly, we expected parental positivity to relate positively, and parental negativity to relate negatively, to children's prosocial behavior mainly when children were carriers of the 7-repeat allele.

However, we do not propose a directional hypothesis with regard to maternal unexplained punishment. As there is some evidence for the negative relationship between punitive parenting and prosocial behavior in children, one possibility is that 7-present children would be less prosocial when they have punitive mothers. However, another possibility should be considered. Typically, mothers are more likely to punish children's noncompliance with requests for prosocial behavior, than they are to punish failure to behave prosocially without a direct request (Grusec, 1991). This pattern of contingent punishment signals to children the conditions in which failure to behave prosocially is likely to elicit punishment. The case for unexplained punishment is more complex, because when mothers do not explain why a punishment is given, children cannot easily determine the appropriate conditions for prosocial behavior. The 7-present children, who are more alert and open to experiences than other children, may be on the lookout for maternal punishment more than their 7-absent peers. Being relatively sensitive to rewards, when their mothers are high in unexplained punishment, they may play it safe and choose to behave prosocially when in doubt about the appropriateness of such behavior, in order to avoid potential punishment.

Establishing the case for differential genetic susceptibility to parenting involves more than a finding of a gene by parenting interaction, as discussed by Belsky et al. (2007). The idea that susceptible children are not only more vulnerable to the adverse effects of negative parenting (diathesis–stress), but also more susceptible to the positive aspects of parenting (referred to as *plasticity* by Belsky & Pluess, 2009a) means that a specific interaction pattern is indicative of differential susceptibility: for children with the genotype characterized by low susceptibility (7-absent), parental influence on prosocial behavior should be more restricted. In contrast, children with the 7-present genotype, indicative of differential susceptibility, should demonstrate greater relationships between parenting and prosocial behavior, including *both* higher and lower prosocial behaviors, dependent on mothers' parenting.

Method

Participants

Families in this study were participants in the Longitudinal Israeli Study of Twins, whose focus is on children's social development as influenced by genetics, abilities, and socialization (Knafo, 2006). All Hebrew-speaking families identified as having twins during 2004 and 2005 by The Israeli Ministry of the Interior were contacted with mail surveys regarding children's development close to the twins' third birthday. The questionnaire included information on twins' zygosity and their behavior problems, as well as additional information described by Knafo (2006).

Sample 1. At 3.5 years of age, families in which mothers answered the questionnaire mailed at twins' third birthday and identified as living in the Greater Jerusalem area were invited to the lab to participate in an experimental session in which children's prosocial behavior, empathy, and other variables were assessed (Knafo et al., 2009).

Twin zygosity was assessed through a parent questionnaire of physical similarity, which has been shown to be 95% accurate when compared to DNA testing (Price et al., 2000). In the current study, DNA results from a partial sample were in 95% agreement with assessments made using mothers' reports. In families where zygosity was uncertain from the questionnaires, DNA was used to identify zygosity. When DNA information was not available, zygosity was estimated from videos of the twins (the video assessment was in 94% agreement with DNA results). The final sample included 45 MZ, 70 DZ same-sex, and 53 opposite-sex twin pairs.

Sample 2. This sample included all children from Sample 1 for which DNA was available. (DNA was not available when parents or children did not agree to give samples and in rare cases when the quality of the sample did not enable analysis.) This included 184 individual twins. In addition, a small sample of 19 same-sex sibling pairs (DNA was available from 27 children) participated. This subsample (Knafo et al., in press) was reached through ads in places such as kindergartens, and preliminary analyses showed it did not differ from the twin sample on the study variables. After dropping families in which mothers did not provide parenting data, the final sample included 36 children from 19 MZ twin pairs, 104 children from 53 DZ twin pairs, and 27 children from 19 singleton sibling pairs ($N = 167$ children).

Procedure

Around age 3.5 years (mean age = 43.81 months, $SD = 3.27$ months), families (the mother and the two children, sometimes accompanied by additional family members) came to

the lab, where they met two female experimenters. Visits were scheduled at a time when mothers estimated children were likely to be at their best. Most visits were completed in less than 2 hr. In the visit, each child was asked to enter a testing room with one of the experimenters, where assessments of children's prosocial behavior and general cognitive abilities were made. Prosociality was assessed by experimenters presenting children with six situations (described below) designed to potentially elicit self-initiated or compliant prosocial responses of help, sharing, and providing emotional support (see Knafo et al., in press). While children were with the experimenters, mothers filled questionnaires providing demographic, zygosity (when relevant), and birth information about the children, as well as describing their own parenting toward each child and the child's behavioral characteristics.

Self-initiated prosocial behavior. This behavior was examined in three situations.

1. *Helping:* The experimenter "accidentally" knocks a pencil box on the floor. She says, "Oops," continues writing for 20 s, and retrieves the pencils for 30 s (Iannotti, 1985). Prosocial behavior is coded if the child helps picking the pencils spontaneously, without being requested to do so and before the experimenter starts picking the pencils up.
2. *Providing emotional support:* The experimenter pretends to have hurt her knee while getting up. She vocally expresses moderate pain for 30 s, a behavior that subsides over another 30 s (Zahn-Waxler, Schiro, Robinson, Emde, & Schmitz, 2001). Prosocial behavior is coded if the child helps or comforts the experimenter.
3. *Sharing:* The experimenter gives the child and herself a pack of the popular "Bamba" snack, then expresses surprise and disappointment for having in her own pack only 3 (instead of about 20) "Bambas," her favorite treat, but never asks the child to share with her (adapted from Yarrow et al., 1976). Prosocial behavior is coded if the child shares at least one of the Bambas.

Compliant prosocial behavior. This behavior was examined in three situations.

1. *Helping:* The experimenter pretends to have lost a finger doll, which she has actually hidden earlier in a predetermined location requiring the child to actively look for the doll in order to find it. Experimenter asks the child: "Have you seen Sousou? Can you help me find it?" the request is repeated without pressure in different ways up to three times. Prosocial behavior is coded if the child actively looks for the doll.
2. *Providing emotional support:* The experimenter holds a finger doll, which has been previously lost, and pretends that the doll is now "very sad and wants to cry." She then asks the child, "Can you help the doll feel better?" and "Do you want to comfort the doll?" Prosocial behavior is coded if the child behaves in a comforting manner toward the doll (e.g., caressing, soothing voices).
3. *Sharing:* This is a child version of the Dictator Game (similar to the one used by Benenson, Pascoe, & Radmore, 2007). The experimenter gives the child an envelope with six attractive sticker sheets in it as a gift. The experimenter asks, "Would you like to give any stickers to a child you don't know, who has not gotten any stickers?" The experimenter makes it clear the stickers belong to the participating child, and the decision to donate is up to the child only. Children place the donated sticker sheets in a closed envelope and return them to the experimenter. Prosocial behavior is coded if the child shares at least one of the sticker sheets.

Prosocial responses across the six tasks were counted to create two prosociality measures: compliant and self-initiated. Note that the responses of children in most situations could be rated on more than one dimension (e.g., in the sticker task both the presence of sharing and the quantity shared were coded). However, in order to increase comparability across tasks, we treat them here as dichotomous variables, where performance of a prosocial behavior is coded 1, and the lack of it is coded 0. The measures of compliant and self-initiated prosociality had a possible range of 0–3. Another study reports that interrater agreements ranged from 90% to 100% for each of the six prosocial behaviors (Knafo et al., in press).

Mother-rated prosocial behavior. In addition, mothers rated their children's prosocial behavior with the Strengths and Difficulties Questionnaire (Goodman, 1997), a 25-item questionnaire asking parents to indicate whether various symptoms and behaviors are "Not true," "Somewhat true," or "Certainly true" of their child. Indicative prosocial behavior items included "Kind to younger children" and "Shares readily with other children." Items did not refer to the source of the prosocial behavior (compliant or self-initiated).

Parenting. Mothers described parenting toward each child using the Robinson, Mandleco, Olsen, and Hart (1995) scales, assessing warmth, induction and reasoning, autonomy support, punishment, verbal hostility, and physical coercion. A research review of a wide variety of parenting scales described this measure as good in terms of breadth, reliability, and validity (Locke & Prinz, 2002). In addition, mothers described their behavior toward each twin using a scale assessing love withdrawal (adapted from Knafo & Schwartz, 2003).

Because we were interested in how children with different alleles of the *DRD4-III* polymorphism reacted specifically to punishment behavior, we retained the unexplained punishment scale independent of the other parenting scales. To reduce the number of variables, all other scales were subject to a factor analysis in which two components emerged, accounting together for 56% of the variance. Following an oblimin rotation, two factors emerged, with warmth, induction and reasoning, and autonomy support all loading positively (.61 or higher) on a factor we termed *maternal positivity.* Verbal hostility, physical coercion, and love withdrawal, all loaded positively (.51 or higher) on a second factor we termed *maternal negativity.* The two factors

correlated moderately negatively ($r = -.22, p < .01$), indicating they were related but independent aspects of parenting.

DRD4-III polymorphism. DNA was extracted with a Master Pure kit (Epicentre, Madison WI). Polymerase chain reaction (PCR) amplification was carried out using a Reddy Mix kit (AB Gene, Surrey, UK). The exon III repeat region of the *DRD4* receptor was characterized using the PCR amplification procedure with the following primers: F5′-TTCCTACC CTGCCCGCTCATGCTGCTGCTCATCTGG-3′ and R5′-A CCACCACCGGCAGGACCCTCATGGCCTTGCGCTC-3′. PCR reactions were performed using 5 μl of Master Mix (Thermo scientific), 2 μl of primers (0.5 μ*M*), 0.6 μl of Mg/Cl_2 (2.5 m*M*), 0.4 μl of dimethylsulfoxide 5%, and 1 μl of water for a 9 μl total volume; an additional 1 μl of genomic DNA was added to the mixture. All PCR reactions were employed on a Biometra T1 Thermocycler (Biometra, Güttingem, Germany). PCR reaction conditions were as follows: preheating step at 94.0°C for 5 min, 34 cycles of denaturation at 94.0°C for 30 s, reannealing at 55°C for 30 s, and extension at 72°C for 90 s. The reaction proceeded was then held at 72°C for 5 min. The reaction mixture was then electrophoresed on a 3% agarose gel (AMRESCO) with ethidium bromide to screen for genotypes.

Table 1 provides the genotype distribution of the polymorphism in our sample. Because we were particularly interested in comparing children who carried the 7-repeat allele to other children, we divided children to two groups based on the presence or absence of the 7-repeat allele of the *DRD4-III* polymorphism. A third of the children ($N = 71$) were carriers of the 7-repeat allele (with 6 of them having two copies of the allele). Of the remaining 140 7-absent children, all but 2 had at least one copy of the 4-repeat allele.

Results

Descriptive statistics

Self-initiated and compliant prosocial behavior did not intercorrelate, as has happened in past studies (Eisenberg et al., 1984), indicating that we were measuring substantially different facets of children's prosociality. No linear correlation was found between mother-rated and observed prosocial behavior, but highly prosocial children (i.e., children who behaved prosocially in at least five of the six observational prosocial behavior tasks) were rated by their mothers as more prosocial ($M = 1.63$, $SD = 0.30$) than other children ($M = 1.46$, $SD = 0.41$), Wald χ^2 (1) = 5.66, $p = .017$, $D = 0.40$.

Table 2 presents the means and standard deviations of all study variables for children with the 7-repeat allele of the *DRD4-III* polymorphism present or absent. An important finding concerns compliant and self-initiated prosocial behavior. On average, children performed 0.53 self-initiated prosocial behaviors out of the possible three, while they performed 2.19 compliant prosocial behaviors. This difference was significant as indicated by the McNemar–Bowker test, χ^2 (6) = 53.37, $p < .001$. Girls performed, on average, more compliant prosocial behaviors than boys ($M = 2.33$, $SD = 0.70$ vs. $M = 1.97$, $SD = 0.94$), a significant difference as indicated by an independence test for ordinal variables, χ^2 (1) = 3.89, $p < .05$. This replicates past findings of higher prosociality in girls (Eisenberg et al., 2006). No significant sex differences were observed for self-initiated or mother-reported prosocial behaviors, or for the parenting variables.

Genetic and environmental influences on prosocial behavior

To examine genetic and environmental influences on prosocial behavior, we first compared twin correlations obtained within MZ and DZ pairs in Sample 1 (Table 3). A positive correlation among MZ twins that was about twice as large as that of the DZ twins suggested a genetic influence with no shared environmental effect for compliant prosocial behavior. In contrast, for self-initiated prosocial behavior, whereas MZ twins correlated positively, same-sex DZ twins did not correlate at all (although a small positive correlation was found between brothers and sisters in mixed-sex twin dyads). This pattern of result is indicative of a nonadditive ge-

Table 1. *Distribution of the DRD4-III polymorphism and assignment to groups*

Genotype	Frequency	Relative Frequency	Group
2, 2	2	0.9%	7-Absent
2, 4	15	7.1%	7-Absent
3, 4	11	5.2%	7-Absent
4, 4	102	48.3%	7-Absent
4, 5	10	4.7%	7-Absent
2, 7	2	0.9%	7-Present
3, 7	1	0.5%	7-Present
4, 7	55	26.1%	7-Present
5, 7	7	3.3%	7-Present
7, 7	6	2.8%	7-Present

Note: DRD4-III, the exon III repeat region of the dopamine receptor D4 gene; 7-Absent, 7-Present, 7-repeat allele is absent or present.

Table 2. *Means and standard deviations of study variables by DRD4-III genotype*

	7-Absent		7-Present	
	M	*SD*	*M*	*SD*
Prosocial behavior				
Self-initiated	0.64	0.85	0.53	0.93
Compliant	2.01	0.90	2.35	0.73
Mother rated	1.42	0.46	1.48	0.43
Parenting				
Maternal positivity	−0.08	1.05	−0.16	0.86
Maternal negativity	0.11	1.17	0.03	1.01
Unexplained punishment	2.13	0.65	2.01	0.60

Note: DRD4-III, the exon III repeat region of the dopamine receptor D4 gene; 7-Absent, 7-Present, 7-repeat allele is absent or present.

Table 3. *Twin correlations and genetic and environmental variance component estimates in prosocial behavior*

	Twin Correlations				Variance Component Estimates	
Prosocial Behavior Aspect	MZ	DZ	DZ (Same Sex)	DZ (Diff. Sex)	Genetic Influence	Nonshar. Environ. and Error
Self-initiated	.46**	.06	.01	.14	.43 (.17–.62)	.57 (.38–.83)
Compliant	.35*	.16	.11	.23	.34 (.07–.57)	.66 (.44–.93)
Mother rated	.58**	.13	.14	.11	.53 (.27–.70)	.47 (.30–.73)

Note: DZ, dizygotic; MZ, monozygotic. The values in parentheses are 95% confidence intervals.
*$p < .05$. **$p < .01$.

netic effect (Plomin et al., 2001). A similar pattern was found for mother-rated prosocial behavior.

As a more direct test of genetic and environmental effects, we conducted model fitting in the Mx structural equation modeling software (Neale, Boker, Xie, & Maes, 1999). The differences in correlations between same-sex and opposite-sex DZ dyads indicated a need for testing sex-limitation models, which could not be performed with our sample size. We therefore estimated genetic and environmental effects only with same-sex dyads; estimates were very similar when opposite-sex DZ twins were added. For all measures of prosocial behavior, it was possible to drop the shared environment component (estimated at 0) from the model without affecting model fit. Table 3 provides the estimates of genetic and environmental influences (with 95% confidence intervals) for all prosocial behavior variables.

Significant genetic effects were found, as hypothesized, accounting for 34% to 53% of the variance in the different prosocial behavior variables. Model fit for compliant prosocial behavior was good, as indicated by several fit indices: χ^2 (4) = 4.95, *ns*, Akaike information criterion (AIC) = −3.05, root mean square error of approximation (RMSEA) = 0.055. Regarding self-initiated prosocial behavior, in a model including both an additive and a nonadditive genetic effect the additive genetic effect was estimated at zero and could be dropped without affecting model fit. The model estimating a nonadditive genetic effect had an excellent fit to the data, χ^2 (4) = 0.92, *ns*, AIC = −7.08, RMSEA = 0.00, better than the fit of the additive model as judged by higher AIC in the latter (−5.027). Similarly, for mother-rated prosocial behavior, the additive genetic effect was estimated at 0.09 and could be dropped without affecting model fit, $\Delta\chi^2$ (1) = 0.02, *ns*. The model estimating a nonadditive genetic effect had a good fit to the data, χ^2 (4) = 5.06, *ns*; AIC = −2.94, RMSEA = 0.06, better than the fit of the additive model as judged by higher AIC in the latter (−2.42).

The DRD4 polymorphism and prosocial behavior

To test for a specific genetic effect on prosocial behavior, we compared carriers of the *DRD4-III* 7-repeat allele to 7-absent children in their degree of prosocial behavior. Population-based association tests checked for differences in study variables based on the presence or absence of the 7-repeat allele (see Table 2). No significant association was observed between any of the prosocial behaviors and the *DRD4* genotype. Mean comparisons also showed no significant differences in parenting based on the presence or absence of the 7-repeat allele.

Because we were interested in the relationship between prosocial behavior and parenting for 7-present and 7-absent children, we compared the variances for all study variables in the two groups (standard deviations are presented in Table 2). There were no significant differences in the variances between 7-present and 7-absent children.

A Gene × Parenting interaction affecting prosocial behavior

No significant relationship was found between parental positivity, negativity, or unexplained punishment, and any of the three measures of prosocial behavior, looking across 7-present and 7-absent children; but our focus was on the interaction between the *DRD4* polymorphism and parenting. We next compared the relationships between parenting and children's prosocial behavior in the presence and absence of the *DRD4* 7-repeat allele (Table 4). Because in many of the fam-

Table 4. *Correlations between parenting and prosocial behavior*

	Maternal		
Prosocial Behavior	Positivity	Negativity	Unexplained Punishment
DRD4-III 7-absent			
Self-initiated	.08	.09	−.04
Compliant	.09	−.03	−.21
Mother rated	.04	−.04	−.12
DRD4-III 7-present			
Self-initiated	.03	.14	.32**
Compliant	−.20	−.20	.07
Mother rated	.34**	−.23*	−.12

Note: Correlations are based on data from 167 children from 91 twin and sibling pairs using robust variance estimators accounting for nonindependence of siblings. *DRD4-III*, the exon III repeat region of the dopamine receptor D4 gene; 7-absent, 7-present, 7-repeat allele is absent or present.
*$p < .05$ (one tailed). **$p < .05$.

ilies two children participated, we employed robust estimates, the so-called Huber–White sandwich, with prosocial behavior regressed onto parenting style and twin pairs clustered by family. Among 7-absent children, no significant relationship was found between parenting and prosocial behavior. In contrast, among 7-present children, mother-rated prosocial behavior correlated positively with maternal positivity, and negatively with maternal negativity. In addition, a positive relationship was found between maternal punishment and self-initiated prosocial behavior, again only among 7-present children.

As a formal test of the hypothesis that the presence of the *DRD4-III* 7-repeat allele moderates the association between parenting and prosocial behavior, we predicted children's prosocial behavior with parenting, their *DRD4-III* polymorphism (where 7-present was coded 1 and 7-absent 0), and the interaction term of these two variables. We did this for the three cases in which a correlation was found for 7-present children but not for 7-absent children. Generalized estimating equations with robust covariance estimators were used, accounting for independence between twins coming from the same families.

Neither maternal positivity ($B = 0.019$), Wald χ^2 (1) = 0.24, *ns*, nor the *DRD4-III* polymorphism ($B = 0.089$), Wald χ^2 (1) = 1.69, *ns*, predicted mother-rated prosocial behavior, yet their interaction was a significant predictor ($B = 0.158$), Wald χ^2 (1) = 5.58, $p = .018$. However, the interaction between maternal negativity and *DRD4-III* did not reach significance relating to mother-rated prosocial behavior ($B = -0.10$), Wald χ^2 (1) = 3.47, $p = .062$. Finally, although there were no main effects of maternal punishment ($B = -0.06$), Wald χ^2 (1) = 0.13, *ns*, or the *DRD4-III* polymorphism ($B = -0.025$), Wald χ^2 (1) = 0.27, *ns*, on self-initiated prosocial behavior, the interaction of the two variables predicted self-initiated prosocial behavior ($B = 0.55$), Wald χ^2 (1) = 4.22, $p < .05$.

To better understand the G × E interaction, which can take many forms, we examined the relationship of parenting with children's behavior separately for children who differ in their genotype. Figure 1 presents the mean mother-rated prosocial behavior based on the presence of the *DRD4-III* 7-repeat allele and on maternal positivity. For convenience of presentation, positivity was divided into three levels: low, moderate, and high. The interaction pattern indicated a differential susceptibility to parenting as described by Belsky et al. (2007): there was no clear relationship between maternal positivity and children's mother-rated prosocial behavior levels among 7-absent children. In contrast, among 7-present children, a linear relationship was observed, in which prosocial behavior increased with levels of maternal positivity, and in which 7-present children with the most positive mothers were the most prosocial, whereas 7-present children with the least positive mothers were the least prosocial.

A similar pattern was found for the other significant interaction. Figure 2 presents the proportion of children who performed at least one self-initiated prosocial behavior based on

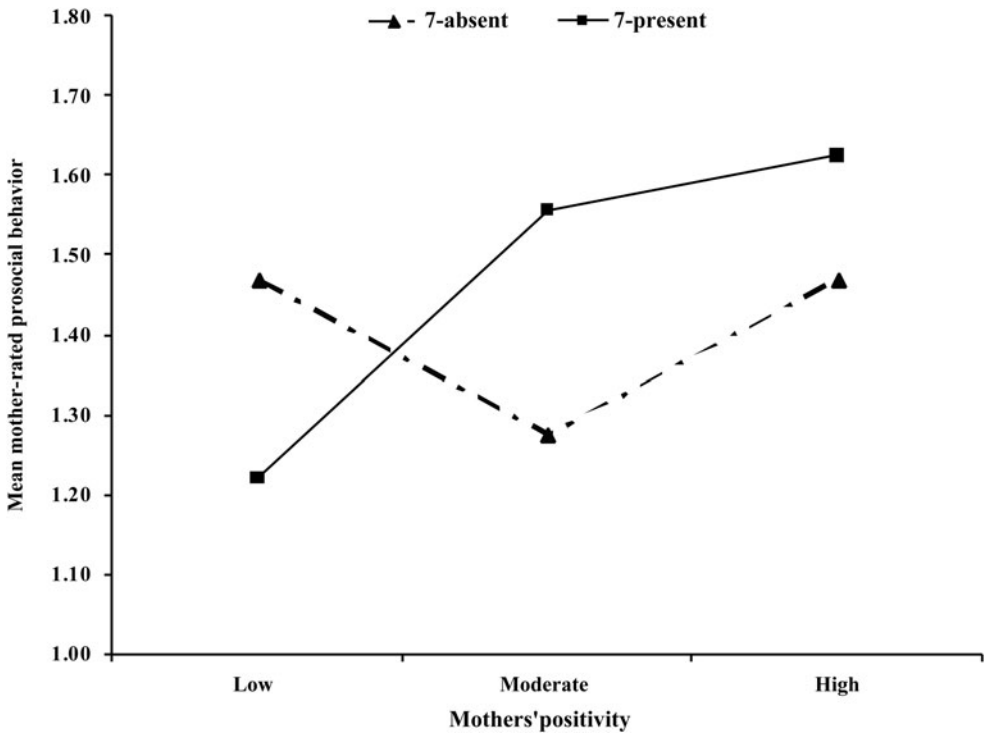

Figure 1. Mean mother-rated prosocial behavior based on the presence of the dopamine receptor D4 exon III (*DRD4-III*) 7-repeat allele and on maternal positivity; 7-present, subjects with the 7-repeat allele present; 7-absent, subjects with the 7-repeat allele absent.

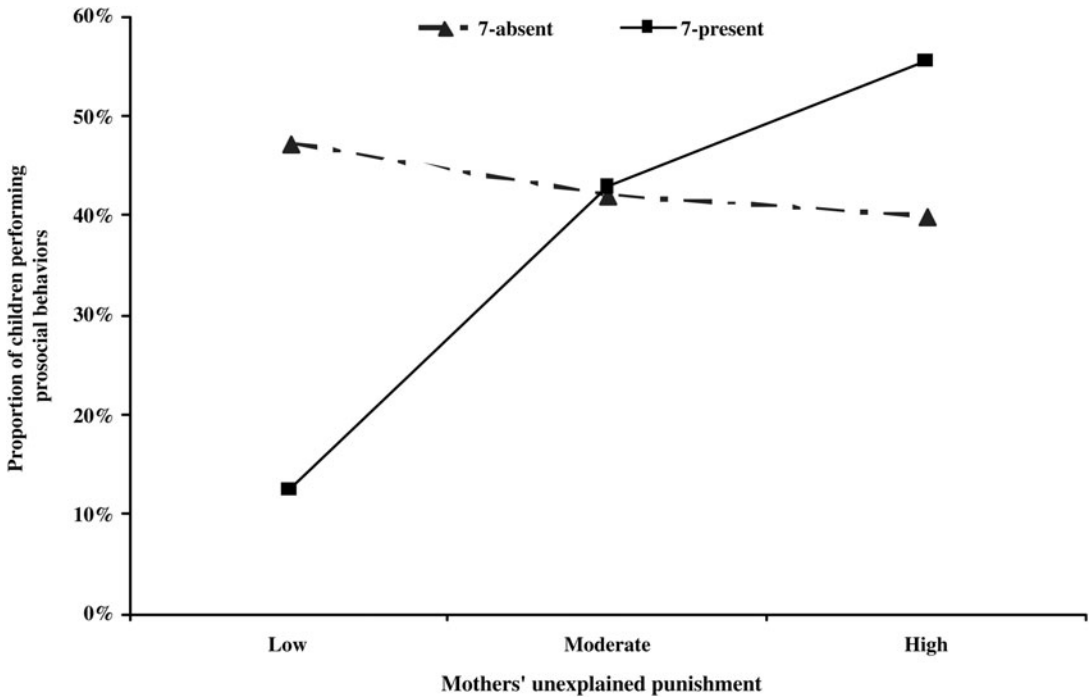

Figure 2. The proportion of children performing at least one self-initiated prosocial behavior based on the presence of the dopamine receptor D4 exon III (*DRD4-III*) 7-repeat allele and on maternal unexplained punishment.

the presence of the *DRD4-III* 7-repeat allele and on maternal unexplained punishment, which was again divided into three levels: low, moderate, and high. Again, children's levels of prosocial behavior did not vary by maternal punishment levels among 7-absent children, χ^2 (1) = 0.66 *ns.* In contrast, among 7-present children, a linear relationship was observed, χ^2 (1) = 5.17, $p = .05$, in which self-initiated prosocial behavior was four times more frequent among children of punishing mothers than among children of mothers low in punishment. As we did not specifically expect a *positive* relationship between punishment and prosocial behavior among 7-present children, it is important to note that splitting the sample randomly to include one child per pair resulted in a positive relationship in both subsamples ($r = .31$ and .34, both $p < .05$). As was the case with mother-reported prosocial behavior, 7-present children included both the most prosocial and the least prosocial, depending on mothers' degree of unexplained punishment, a pattern compatible with a differential susceptibility to parenting.

Perhaps the strongest test of the Gene × Parenting interaction would be to compare siblings with different genotypes with regard to how parenting affected these children. The use of DZ and sib pairs discordant for the polymorphism under investigation allows for the matching of socioeconomic status, ethnicity, and other important variables that vary across families, while testing in a more controlled manner for genetic association and interaction. There were only 14 sibling and twin pairs in which one was a carrier of the 7-repeat allele, whereas the other was not. Therefore, we view the results as illustrative. The correlations between the parenting toward the two siblings were all very high ($r > .75, p < .001$). Nevertheless, the association of parenting with prosocial behavior was quite different for the two siblings, as shown in Table 5. Again, in the absence of the 7-repeat allele, no significant relationship was found between parenting and prosocial behavior. In contrast, among the offspring of the same mothers who were carriers of the 7-repeat allele, a positive re-

Table 5. *Correlations between parenting and prosocial behavior in sibling/dizygotic twin pairs according to DRD4-III 7-absent or 7-present*

	Maternal		
Prosocial Behavior	Positivity	Negativity	Unexplained Punishment
DRD4-III 7-absent sibling/twin			
Self-initiated	.15	.13	−.20
Compliant	−.32	.31	.23
Mother rated	−.09	−.14	.30
DRD4-III 7-present sibling/twin			
Self-initiated	−.10	.27	.34
Compliant	.05	−.41	.15
Mother rated	.57*	−.49	−.04

Note: Correlations for self-initiated and compliant prosocial behavior are Kendall tau correlations. Correlations for mother-rated prosocial behavior are Pearson correlations. *DRD4-III*, the exon III repeat region of the dopamine receptor D4 gene; 7-absent, 7-present, 7-repeat allele is absent or present.
*$p < .05$.

lationship was found between maternal positivity and mother-rated prosocial behavior ($r = .57$, $p < .05$). A correlation comparison test (DeCoster, 2007), showed that the correlations for the two siblings differed significantly ($Z = 1.75$, $p < .05$, one tailed). In addition, self-initiated prosocial behavior correlated positively, although insignificantly, with unexplained punishment, again only for their 7-present children.

Discussion

The field of child development has focused great efforts on the classic *Nature OR Nurture* question. The results of our twin study point clearly to the importance of both genetic and environmental factors in explaining individual differences in children's prosociality. Important breakthroughs have shown progress from merely parsing genetic and environmental contributions to showing that genetic and environmental influences are interrelated (gene–environment correlations; e.g., Burt, 2008; Jaffee & Price, 2007) and that genetic and environmental influences often work in a nonadditive manner to affect developmental outcomes (gene–environment interactions; e.g., Bakermans-Kranenburg & van IJzendoorn, 2006; Caspi et al., 2002). Thus, the question has been rephrased to focus on *Nature AND Nurture* (Plomin, 1994). It is important to emphasize the joint contributions of children's biology and their environment, understanding that genes and contexts are in continuous, dynamic interplay over time (Ellis & Boyce, 2008). Our results provide further evidence for the importance of considering the joint contribution of genes and the (parental) environment to children's development.

Genetic influences on prosocial behavior

We used the twin design to examine the magnitude of genetic and environmental contributions to observed and mother-reported prosociality. Out study joins the young but growing literature showing that genetic factors contribute to children's and adolescents' prosocial behavior (Gregory et al., 2009; Hur & Rushton, 2007; Knafo & Israel, 2009; Knafo & Plomin, 2006b; Scourfield et al., 2004). Except for three studies of observed empathy (Knafo et al., 2009; Volbrecht, Lemery-Chalfant, Aksan, Zahn-Waxler, & Goldsmith, 2007; Zahn-Waxler et al., 2001) and one study (van IJzendoorn et al., 2010) that found no heritability for children's donations to UNICEF, all of the other studies were questionnaire based. Similarly, only one set of studies provided evidence for the heritability of adults' observed prosociality (Cesarini, Dawes, Johanhesson, Lichtenstein, & Wallace, 2009). The finding of substantial genetic effects on a set of mother-reported as well as observed prosocial measures is an important contribution of our study.

Although all three measures of prosociality showed substantial heritability, they showed different patterns of genetic influence. Heritability for compliant prosocial behavior was modest, accounting for a third of the variance, and indicated an additive genetic influence. In contrast, genetic influences on self-initiated and mother-reported prosocial behavior were stronger, and represented nonadditive effects. In addition, the observed prosociality measures did not correlate with each other. It is therefore important to view prosociality as a complex, multifaceted construct (Eisenberg et al., 2006). Developmental processes accounting for compliant and self-initiated prosocial behavior may be quite different (Eisenberg, 2004). For example, children's low affective knowledge predicts lower levels of self-initiated but not compliant prosocial behavior (Knafo et al., in press). In addition, the genes partially responsible for variations in compliant prosocial behavior may be different from those accounting for individual differences in self-initiated prosociality. This may explain why the *DRD4-III* polymorphism, shown in past studies to relate to self-reported selflessness and to sharing in a triadic family play session (Bachner-Melman et al., 2005; DiLalla et al., 2009) did not relate to prosocial behavior that was measured very differently in the current study. Multitrait–multimethod genetic studies of prosocial behavior are needed.

Environmental influences on prosocial behavior

The twin analyses, in addition to pointing out the importance of genetics, suggested that about half of the variance in prosociality is accounted for by environmental factors. A review of the literature (Knafo & Israel, 2009) has shown that in adult studies the environmental influence on prosocial behavior is of the nonshared kind (e.g., Gillespie, Cloninger, Heath, & Martin, 2003) and that from early childhood to adolescence environmental effects that initially are partially of the shared kind, making children growing up in the same family similar to each other become steadily nonshared. That conclusion was based mainly on studies from Western, Anglophone countries. Our Israeli data show no shared environment effects at age 3 (Knafo & Israel, 2009) and in this study at 3.5 years. This may indicate a cross-cultural effect (e.g., because of the structure of the preschool system in Israel and elsewhere) on genetic and environmental processes (Knafo & Israel, 2009).

It is tempting to equate the lack of shared environment factors with the lack of direct relationships between prosociality and parenting. However, we should first note that in the twin design, shared environmental effects represent the joint contribution of a wide set of variables, not only that of parenting (e.g., neighborhood effects). Moreover, the effect of parenting could well be of the nonshared kind. In a study focusing on differences in 4-year-old MZ twins' behavior and in the parenting they receive, Asbury, Dunn, Pike, and Plomin (2003) showed that twins receiving more negative parental discipline and affection were less likely than their co-twin to engage in prosocial behavior. As these MZ twins were not only genetically identical, but were also raised together, differences between them could only indicate nonshared environmental influences (and error). Because parental differential treatment has a longitudinal effect on children over and above their own behavioral differences (Caspi et al., 2004), differential treatment is an important venue for studying the effect of parenting. We are currently collecting longitudinal twin data to

address this issue. Finally, the relationships between prosociality and parenting can also be seen as moderated by a G × E, as we have demonstrated in the current study.

DRD4-III and susceptibility to parenting

Although parenting did not directly relate to prosociality in this study, a fuller picture of prosocial development is reflected in the interaction between genetics and the environment. This is the first study, to the best of our knowledge, to report a significant Gene × Parenting interaction predicting prosocial behavior. Our finding of no relationship between prosocial behavior and parenting in the 7-absent children was accompanied by meaningful associations among the 7-present children. It is important that all three methods applied to investigate the Gene × Parenting interaction, linear regression, comparisons of the associations between prosociality and parenting in the two genotype groups, and intrafamily comparisons of discordant genotypes provide similar directional results, attesting to the robustness of the interaction.

The differences between siblings in how their mothers' parenting relates to their prosociality, although based on a small sample, provide strong evidence for the differential role of parenting depending on children's genotype. These differences illustrate Belsky's (1997) evolutionary notion of within-family differential susceptibility, which may have evolved to enable children of the same parents to develop different enough phenotypes, conferring to at least some of the offspring an advantage as environmental constraints change.

This is the first study to show that *DRD4-III* is associated with susceptibility to parenting with regards to a positive trait (prosociality). As Boyce et al. (2005) note, differential susceptibility goes beyond vulnerability (in which susceptible children are more negatively affected by the environment), but pertains also to the better aspects of development, in which susceptible children benefit more than other children from positive environments (Belsky et al., 2007; Boyce & Ellis, 2005). The results for parental positivity and mother-rated prosocial behavior exemplify this pattern. Other research on externalizing behavior and disorganized attachment (Bakermans-Kranenburg et al., 2008; DiLalla et al., 2009; van IJzendoorn & Bakermans-Kranenburg, 2006) showed a similar pattern with less adjusted aspects of development. The converging evidence makes a strong case for *DRD4-III* as a differential susceptibility gene. Nevertheless, note that differential susceptibility can be accounted for by multiple genes, which have been proposed to have cumulative effects (Belsky & Pluess, 2009b). Gene–Gene interactions and epigenetic changes may also account for susceptibility.

Despite the growing evidence for the role of certain genes as related to differential susceptibility, much research is needed to understand the underlying processes behind this interaction. For example, our 7-present children whose mothers were low in unexplained punishment were the least likely to initiate prosocial behaviors toward the experimenter. Recalling the role of dopaminergic pathways in reward processes, 7-present children may be more sensitive to rewards. Our results may indicate that only when exposed to disciplinary efforts these children are likely to behave prosocially without being asked to. In contrast, 7-present children whose mothers were high in unexplained punishment were the most likely to initiate prosocial behaviors toward the experimenter. This could indicate that 7-present children, being more reward sensitive, try to avoid punishment as much as possible. Being raised by a mother with a strong tendency to punish without explanation, they (more than other children) learn to react when their help is needed. Another possibility is that the 7-present children, who are more alert and tend to explore their environments, are more likely to detect the need to behave prosocially and therefore more likely to initiate prosocial behaviors depending on their mothers' parenting. Thus, the role of socialization in self-initiated prosocial behavior may be very different among 7-present and 7-absent children.

Although spontaneous prosocial behavior is typically considered as reflecting an intrinsically oriented altruistic motivation (Eisenberg et al., 1984), the case of self-initiated prosocial behaviors shown by 7-present children with highly punitive mothers may actually represent a different motivation, perhaps that of ingratiation, avoidance of punishment, or pathogenic guilt. Oakley, Knafo, and McGrath (in press) described a small selected group of 3-year-old children high on prosocial behavior, but very low on self-actualizing behavior, such as enjoying success and autonomous persistence in pursuit of goals. These children were more sociable and exhibited lower levels of conduct problems than their peers; however, they were higher in emotional internalization symptoms, perhaps showing what may be an early form of *pathological altruism* (Oakley, Knafo, Madhavan, & Wilson, in press). One pathway toward the development of pathological altruism involves empathy-based pathogenic guilt. This process may be more likely in families of depressed or highly critical parents, who base socialization of children's empathy on anxiety and inappropriate blame and responsibility, a process that can have detrimental implications to children's self-worth (Zahn-Waxler & Van Hulle, in press). In sum, some cases of prosocial behavior may not reflect healthy empathic or altruistic development, as can be the case with the 7-present children of highly punitive mothers.

It is important to study development at different levels (Cicchetti & Dawson, 2002). Although the effects of the *DRD4-III* polymorphism at the cellular level have been documented (Asghari et al., 1995), studies about how variations in *DRD4-III* influence brain activation are ongoing (Durston et al., 2005; Fan, Fossella, Sommer, Wu, & Posner, 2003; McCLernon, Hutchison, Rose, & Kozink, 2007) and will provide additional insights into the processes involved. Finally, the temperamental factors at the behavioral, cognitive, and affective levels that account for this differential susceptibility are still largely unknown. More research into the core endophenotypes, such as the brain processes as well as attentional and behavioral factors associated with differential susceptibility, is needed (Ellis et al., 2011 [this issue]).

Strengths, limitations, and future directions

Our study has several notable methodological strengths. First, the twin study included a quality birth-records-based sample. Second, we went beyond questionnaire reports to testing children's prosociality in the lab, with each twin tested and coded by separate research assistants. Third, our use of a multimethod approach to prosociality enabled the demonstration of the complexity of the findings, depending on type of prosociality, and the use of very different measures (DNA, observation, and maternal questionnaires) reduced the risk of shared method variance.

However, the sample (168 pairs) was relatively small for a twin study, because of the need to use experimental measures. Because MZ correlations were more than twice larger than the DZ correlations, no shared environmental effects needed to be tested, and we had sufficient power to attribute the familial twin similarity in prosociality to genetic factors. In addition, for mother-reported prosociality, estimates for genetic and environmental factors were very similar to those obtained with a larger sample of 491 pairs at age 3 (Knafo & Israel, 2009). Nevertheless, Sample 2 was quite small, and we would not be able to detect smaller significant interactions. The within-family comparisons corroborated our findings, but we note that these results were based on 14 sibling pairs only. The use of mothers' self-reported parenting ratings is another limitation that we currently address in an ongoing study of parenting observed in mother–child play situations. Nevertheless, the value of mother reports is exemplified by their correlations with observed prosocial behavior.

Finally, our genetic findings point out the need for additional molecular genetic studies of prosocial behavior, applied to different aspects of prosociality. There is promising evidence for the role of the oxytocin receptor (*OXTR*) and vasopressin receptor (*AVPR1a*) genes in prosocial behavior (reviewed by Israel et al., 2008), but other polymorphisms may be important as well. Similarly, the role of gene–environment correlations, in which environmental effects are correlated with children's genotype, should not be ignored (Jaffee & Price, 2007). For example, Knafo and Plomin (2006a) reported that concurrent and longitudinal associations between prosocial behavior and parenting, especially parental negativity, indicated gene–environment correlation processes. A fuller understanding of gene–environmental interactions should involve the study of epigenetic processes in which environmental processes interact with the way genes are expressed through development (McGowan et al., 2009). These are just a few of the future avenues for research highlighted by the current investigation.

Conclusions

This study provides evidence for the genetic and environmental contributions to different aspects of children's prosocial behavior, as well as the interplay between children's *DRD4-III* genotype and mothers' parenting. Our findings provide proof of principle that a molecular genetic strategy, based on genotyping of common polymorphisms and combined with a classic twin approach, provides a fuller description than past studies of how genes and environment interact to shape children's behavior, especially differential sensitivity to parenting. The finding that the role of parenting depends upon children's genotype tells us not only about parents' limits of influence but also about children's increasingly appreciated role in family processes.

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