Parental Discipline and Affection and Children's Prosocial Behavior: Genetic and Environmental Links

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The authors investigated genetic and environmental contributions to the relationships between children's (*N* = 9,319 twin pairs) prosocial behavior and parental positivity and negativity toward them. Children's prosocial behavior was rated by parents at ages 3, 4, and 7 and by teachers at age 7. At each age, parents described their feelings and discipline toward each twin. Parental positivity was indexed by positive feelings and positive, noncoercive discipline, and parental negativity was indexed by negative feelings and coercive, punitive discipline. Genetics and the environment both contributed to individual differences in prosocial behavior and in parenting. At all ages, parental positivity correlated positively, and parental negativity correlated negatively with prosocial behavior. Genetic factors largely mediated the negative correlation between prosocial behavior and parental negativity. Shared environmental effects contributed mainly to the positive relationship between prosocial behavior and parental positivity. This pattern was found both cross-sectionally and longitudinally. The findings point to the importance of children's characteristics and of the parent–child relationship in family processes.

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The importance of prosocial behavior, that is, behavior intended to benefit others (Eisenberg & Fabes, 1998), makes it a major socialization goal for many parents. The relationship between parenting and children's prosocial behavior has been studied extensively (e.g., Eisenberg & Fabes, 1998; Grusec, Davidov, & Lundell, 2002). Parents' warmth and their use of reasoning, induction, and autonomy support as opposed to power-assertive discipline are related to children's empathy and prosocial behavior (Clark & Ladd, 2000; Krevans & Gibbs, 1996). The obvious explanation for these findings is that positive parenting makes children more prosocial, for example, by providing a prosocial model for them. However, another possibility is that the correlational effects attributed to parenting may represent in part the reaction of parents' to children's partially genetically influenced prosocial behavior (i.e., an evocative gene–environment correlation; Plomin, DeFries, & Loehlin, 1977). For example, warm

parents may share with their children a genetic tendency for prosocial behavior. The purpose of this study is to investigate these possibilities. Specifically, we study the genetic and environmental contributions to the relationships between parenting and children's prosocial behavior using a twin design. The following section reviews the extensive literature showing phenotypic relationships between parenting and prosocial behavior, and the next section addresses the possibility of genetic mediation of these relationships.

Prosocial Behavior and Parenting: Phenotypic Relationships

The two main dimensions of parenting involve the affective response of parents toward their children (e.g., warmth) and the practices parents use when they attempt to influence their children's behaviors (e.g., control; Maccoby & Martin, 1983). The current study is interested in aspects of both these dimensions—in parental feelings (positive vs. negative) toward their children and in parental discipline practices (reasoning vs. harsh or indifferent parenting). Both parental feelings toward children and parents' discipline practices have been shown to predict prosocial behavior (e.g., Eisenberg & Fabes, 1998). The more positive the affect and discipline of the parent, the higher the prosocial behavior of the child; the more negative they are, the lower the child's prosocial behavior.

Parental positivity as expressed in disciplinary practices has been linked to prosocial behavior (e.g., Krevans & Gibbs, 1996). It has been suggested that disciplinary practices that involve reasoning increase children's awareness of the consequences of their

behavior and are more likely to promote adaptive behavior (e.g., Hoffman, 1970). When positive disciplinary practices such as reasoning are used (e.g., when parents tell children what the consequences of their behavior are), children pay more attention to parental messages, empathize with people in need, and actively process parental messages (Hoffman, 1970). On the other hand, negative disciplinary practices such as power-assertive or punitive discipline may reduce prosocial behavior because they induce compliance to imposed rules rather than internalization of moral standards and because the fear associated with punishment may interfere with learning (Hoffman, 1970; Staub, 1979).

Most evidence supports this hypothesis (see Eisenberg & Fabes, 1998, for a review). For example, parents' use of reasoning as opposed to power-assertive discipline was related to early adolescents' empathy and prosocial behavior (Krevans & Gibbs, 1996). Similarly, use of responsive rather than harsh parenting related positively to toddlers' empathy and cooperation (Whiteside-Mansell, Bradley, Tresch Owen, Randolph, & Cauce, 2003).

The affective behavior of parents toward their children has also been hypothesized to affect prosocial behavior. Warm, supportive parenting may increase prosocial behavior by providing a caring model for children and by increasing both children's willingness to attend to parental messages and their accuracy in detecting these messages (Hoffman, 1970; Knafo & Schwartz, 2003; Staub, 1979). Studies of parental effects found evidence that children's prosocial behavior and empathy relate positively to parental warmth (Eisenberg & Fabes, 1998; Zhou et al., 2002). For example, children who had a warm relationship with their parents, as rated by behavioral observation, were rated by their teachers as more prosocial (Clark & Ladd, 2000). In another study, parental negative feelings toward children related negatively to their prosocial behavior (Deater-Deckard, Dunn, O'Connor, Davies, & Golding, 2001).

In summary, the literature suggests that parental positive behavior toward children—both positive affect and discipline—relates positively to prosocial behavior. In contrast, parental negative affect and discipline toward children relate negatively to prosocial behavior. In the present study, we investigated the relationships between prosocial behavior and parental positivity and negativity.

On the basis of theories and past research, we expected relationships between prosocial behavior and parental affect and discipline. However, the possibility that child effects through gene–environment correlations cause some of these effects was also considered. This was, of course, based on the assumption that there are genetic effects on both parenting and prosocial behavior. We discuss the relevant evidence in the following section.

Genetic and Environmental Effects on Prosocial Behavior and Parenting

We approached the issue of genetic and environmental contributions to individual differences in prosocial behavior and parenting using the twin design. This design compares monozygotic (MZ) twins, who share all of their genes, with dizygotic (DZ) twins, who share on average half of their genes. Assuming that twins of both types share their environments (e.g., the family environment) to the same extent, higher similarity in MZ versus DZ twins indicates genetic influence. Similarity that is beyond this genetic effect can be attributed to the environment shared by twins, and any further differences between twins are ascribed to non-

shared environment or to measurement error (Plomin, DeFries, McClearn, & McGuffin, 2001).

Prosocial Behavior

The few studies that have addressed directly the issue of the origins of individual differences in children's prosocial behavior using a genetically informed design found both genetic and environmental (shared and nonshared) effects (Deater-Deckard et al., 2001; Scourfield, John, Martin, & McGuffin, 2004; Zahn-Waxler, Robinson, & Emde, 1992; Zahn-Waxler, Schiro, Robinson, Emde, & Schmitz, 2001). Heritability estimates (the proportion of variance, within a sample, in children's prosocial behavior that can be accounted for by genetics) ranged from .04 to .53 (.31 on average), depending on the age of the children and the measures used, but all these studies reported significant heritabilities at least for one age group. Similarly, shared environment estimates ranged from .00 to .49 (.23 on average), and nonshared environment estimates ranged from .17 to .80 (.46 on average).

We have studied the prosocial behavior of twins at ages 2, 3, 4, and 7 elsewhere (Knafo & Plomin, in press). As in past studies, we found both genetic and environmental effects. However, there were consistent age differences in the strength of the different influences. Using parental reports on children's prosocial behavior, we determined that shared environmental effects gradually decreased from .47 on average at age 2 to .03 at age 7, and genetic effects increased from .32 on average to .61. This trend was largely confirmed at age 7 on the basis of teacher ratings. In the current study, we used the same data on prosocial behavior at ages 3, 4, and 7. In our previous research, we focused on the use of longitudinal genetic analyses to study the extent to which genetic and environmental effects account for change and continuity in prosocial behavior. In the current research, we focused on the relationship with parenting.

Parenting

Genetically informative research designs that rely on children, such as our study of twins who are children, can investigate the extent to which children's genetic propensities affect their parents' behavior (McGuire, 2003). This is done by comparing the similarity in the parenting received by MZ and DZ twins (Plomin, 1994; Rowe, 1981). The complementary genetic design that relies on parents, such as twins who are parents, is not limited to genetic influences on parenting that reflect children's genetic propensities (Neiderhiser et al., 2004); there are far fewer studies of this type and the focus of our review is the former twins-as-children design. It is impossible to fully review this large literature here; reviews are available that show consistent genetic influence on parenting (e.g., McGuire, 2003; Plomin, 1994). For example, in a dozen studies of twins as children, higher MZ than DZ twin correlations indicate genetic influences on most parenting dimensions including affect and discipline (e.g., Elkins, McGue, & Iacono, 1997; Neiderhiser et al., 2004; Wade & Kendler, 2000), although some studies report no genetic influence on certain dimensions such as parental control (Deater-Deckard, 2000; Rowe, 1981). One study estimated, using children's reports of parents' behavior from 707 sibling and twin pairs, that genetic effects accounted for 49%–56% of the variance in parental positivity, shared environment effects

for 4%–20%, and nonshared environment (and error) for 38%–66% of the variance (Plomin, Reiss, Hetherington, & Howe, 1994). Estimates for parental negativity were 23%–40% for heritability, 14%–24% for shared environment effects, and 46%–53% for nonshared environment (and error; Plomin et al., 1994). Because these studies used twins-as-children genetic designs, they provided evidence for genetic influence on parenting that reflects genetic propensities of their children as well as providing evidence for environmental effects on parents' behaviors toward their children while controlling for the effects of the child's genetic propensities.

The Current Study

The evidence regarding the influence of children's genetic propensities on their parents' behavior suggests gene–environment correlation (Neiderhiser et al., 2004; Plomin et al., 1994). The most likely mechanism for this gene–environment correlation is that children's genetically influenced behavior evokes certain parental behaviors, although other types of gene–environment correlations have been described (Plomin et al., 1977; Scarr & McCartney, 1983). An example of evocative gene–environment correlation is that children who are relatively well adapted may have parents who respond with positive affection and who do not need to consider strict or harsh parenting.

Univariate analyses of genetic and environmental influences on the parenting addressed toward children are informative, but they only indirectly imply gene–environment correlation. In contrast, multivariate genetic analyses can assess the extent to which genetic factors mediate the correlation between specific measures of parenting and specific measures of children's behavior (Plomin, 1994). When applying this type of analysis to the case of parenting and children's prosocial behavior, researchers can use multivariate genetic analysis to investigate genetic mediation of the relationship between prosocial behavior and parenting (i.e., whether there is overlap between the genetic effects on prosocial behavior and parenting). If such genetic links are found, they would indicate that the child's genetically influenced prosocial behavior may have initiated a family process in which parents responded, for example, by becoming more affectionate and by engaging in increased positive parenting.

We used the twins-as-children design to investigate this issue. As described below, cross-twin/cross-trait variance–covariance matrices are used to study, in addition to the genetic and environmental contributions to parenting and prosocial behavior, the contribution of genes and the environment to the relationship between these two constructs. Capitalizing on twin differences in genetic resemblance, multivariate genetic analyses can assess the extent to which the relationship between parenting and prosocial behavior is mediated genetically. In addition to genetic mediation of the association between parenting and prosocial behavior, the same type of analysis can be used to estimate the extent to which the phenotypic relationship is mediated by shared and nonshared environmental effects. There is evidence from a single genetically informed study (Deater-Deckard et al., 2001) regarding the correlations between parental feelings and prosocial behavior. This study used the step-family sibling design, assuming that shared environment effects are common to siblings and half siblings living together and capitalizing on the lower genetic resemblance of half siblings (25% on average) as compared with full siblings

(50%). The negative relationship between maternal negative feelings toward children and children's prosocial behavior was mediated largely by environmental effects. The relationship between mothers' partners' feelings and children's prosocial behavior was mediated by genetic as well as environmental factors.

We extended this analysis to positive and negative parental discipline in addition to parental affect, using data from a sample of 9,319 pairs of twins. We investigated the genetic and environmental contributions to the relationships between these two parenting dimensions and children's prosocial behavior at ages 3, 4, and 7. The repeated assessments of parenting and prosocial behavior enabled analyses of the stability of the genetic and environmental contributions to the relationship between the two from early to middle childhood and of the relative contributions of genetics and the environment to the longitudinal relationships between parenting and prosocial behavior.

Finally, the role of gender in prosocial behavior has been established, with consistent evidence that prosocial behavior is more common among girls than among boys (Eisenberg & Fabes, 1998). Socialization agents have been found to promote a nurturing social role for girls more than for boys (Maccoby, 1998). Similarly, differences in the parenting girls and boys receive have been reported (Maccoby, 1998). For example, compared with girls, boys received more negative parenting such as power-assertive discipline (Maccoby, 1998). We expected to find similar findings in the current investigation. However, we hoped this study would go beyond the description of mean differences between boys and girls and explore sex differences with respect to the origins of individual differences.

Method

Participants

Families in this study were participants in the Twins Early Development Study (TEDS), a longitudinal study of development in which parents of all twins born in England and Wales during 1994–1996 were invited to participate (Trouton, Spinath, & Plomin, 2002). Assessments were made at 18 months and at 2, 3, 4, and 7 years. At the first assessment, 16,286 families were sent booklets to complete. Of these families, 13,601 (84%) provided data at the first assessment. From this initial sample, 763 twin pairs (5.6%) were excluded because there were extreme pregnancy or perinatal difficulties (e.g., gestation <32 weeks) or because one of the twins had an extreme medical condition (e.g., chromosomal abnormalities such as Down's syndrome and cerebral palsy). Twin zygosity was assessed through a parent questionnaire of physical similarity. This method has been shown to be over 95% accurate when compared with DNA testing (Price et al., 2000). A further 271 twin pairs for whom zygosity was not established were not included in the current sample, resulting in a final sample of 12,567 pairs.

The present sample included families providing data on children's prosocial behavior and on parent-rated parenting at one or more times during the ages 3, 4, and 7 assessments. Of the families participating in the age 7 assessments, 91% granted permission for us to contact the twins' teachers via postal questionnaire and provided accurate information about the teachers and schools. In 81% of these families, teacher reports were available for both twins.

Data for both twins' prosocial behavior were available for 5,521 pairs at age 3 and 7,245 pairs at age 4 (the 1996 cohort was not studied at age 3 but was included at age 4). At age 7 parent-rated data were available from 6,243 pairs and teacher-rated data from 4,632 pairs (see Table 1). Full data from both twins on all study variables at all ages were available from 3,400

Table 1
Means and Standard Deviations of Scores on the Prosocial Behavior and Parenting Scales

Zygosity	Prosocial behavior				Parental positivity			Parental negativity		
	Age 3	Age 4	Age 7 (parent report)	Age 7 (teacher report)	Age 3	Age 4	Age 7	Age 3	Age 4	Age 7
MZM										
M	14.54	10.51	7.90	6.69	−0.05	−0.05	−0.03	0.02	0.06	0.08
SD	3.60	2.66	1.85	2.47	0.81	0.77	1.01	0.85	0.86	0.86
n[a]	943	1,215	984	734	943	1,215	984	943	1,215	984
DZM										
M	14.48	10.41	7.89	6.80	−0.01	0.02	0.09	0.15	0.12	0.11
SD	3.68	2.70	1.91	2.44	0.81	0.82	0.99	0.87	0.86	0.88
n[a]	913	1,193	957	694	913	1,193	957	913	1,193	957
DZO boys										
M	14.64	10.41	8.07	6.57	0.02	0.01	0.06	0.09	0.14	0.11
SD	3.61	2.57	1.75	2.49	0.72	0.74	0.95	0.81	0.81	0.85
n[a]	887	1,156	1,063	792	887	1,156	1,063	887	1,156	1,063
MZF										
M	15.71	11.24	8.42	7.83	0.02	0.03	−0.01	−0.07	−0.07	−0.07
SD	3.47	2.59	1.65	2.10	0.75	0.75	1.01	0.84	0.84	0.89
n[a]	898	1,243	1,068	775	898	1,243	1,068	898	1,243	1,068
DZF										
M	15.64	11.42	8.58	7.93	0.01	−0.03	−0.12	−0.16	−0.16	−0.15
SD	3.48	2.55	1.55	2.10	0.78	0.78	1.09	0.84	0.85	0.89
n[a]	900	1,187	993	754	900	1,187	993	900	1,187	993
DZO girls										
M	15.36	11.14	8.52	7.73	−0.01	0.02	0.04	−0.01	−0.07	−0.09
SD	3.60	2.53	1.58	2.18	0.72	0.71	0.97	0.79	0.80	0.85
n[a]	1,026	1,332	1,212	905	1,026	1,332	1,212	1,026	1,332	1,212

Note. MZM = monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DZO = dizygotic opposite-sex twins.
[a] One twin per pair was selected randomly.

pairs. The overall final sample with parenting and prosocial behavior data available for one or more ages included 9,319 twin pairs: 1,505 monozygotic male, 1,540 dizygotic male, 1,685 monozygotic female, 1,582 dizygotic female, and 3,007 opposite-sex (DZO) twin pairs.

Family structure was stable across the years. Most children lived with both their parents (age 3, 84.0%; age 4, 84.7%; age 7, 85.0%). Small proportions of children lived with their mother and a new partner (age 3, 9.6%; age 4, 8.8%; age 7, 3.5%) or with the mother alone (age 3, 3.4%; age 4, 3.6%; age 7, 8.9%). Data were not available on family structure for 3% of families. Nearly all responding parents were mothers (e.g., 97% at age 7). Most (64%) twin pairs had the same teacher at age 7, and 36% had different teachers.

Attrition analyses revealed some small differences between families providing data at 18 months and those providing data at ages 3 and 4: Namely, families remaining in the study were slightly more likely to be in the top or second social class and to have a father who was employed than were those with missing data (Ronald, Eley, & Plomin, 2003). Despite attrition, analyses showed that the TEDS sample continues to be reasonably representative of parents of young children in the U.K. population. For example, U.K. 2001 census data (Office for National Statistics, 2003) suggest that 92% of U.K. mothers are White, and 92% of mothers in TEDS are White. In addition, 32% of mothers in the U.K. population have completed A-level examinations (Office for National Statistics, 2004), taken by pupils before going to university, as do 34% of TEDS mothers. TEDS is described in more detail elsewhere (Spinath, Ronald, Harlaar, Price, & Plomin, 2003; Trouton et al., 2002).

Further attrition analysis found no differences in parenting or prosocial behavior at age 3 between families who went on to participate at age 4 and those who did not. Children who remained until age 7 in the study were slightly more prosocial at age 4 than those whose families no longer participated, $t(5926) = 3.20$; $p < .01$, but this difference accounted for only 0.1% of the variance in prosocial behavior and emerged as significant because of the large sample size. No differences in parenting were found between families who participated at all ages versus those who participated at age 3 but not later.

Measures

A parent (usually the mother) of each twin pair completed the Revised Rutter Parent Scale for Preschool Children (RRPSPC; Hogg, Rutter, & Richman, 1997). The RRPSPC is based on the Preschool Behavior Questionnaire, which has been demonstrated to have good reliability (Behar & Stringfield, 1974). Parents responded to each item on the RRPSPC on a 3-point scale (0 = *not true*; 1 = *sometimes true;* 2 = *certainly true*). The Prosocial Behavior scale in the RRPSPC consisted of 11 items at age 3 (e.g., "often volunteers to help others"; "shares out treats with friends"). At age 4, the wording of some of these items was slightly altered so as to be consistent with the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), a 25-item questionnaire that was developed from the RRPSPC. In addition, some similarly phrased items (e.g., "will try to help someone who has been hurt"; "helps other children who are ill") were combined ("helpful if someone is hurt, upset, or feeling ill"), resulting in 8 items that were very similar to those used at earlier ages. Factor analysis of the Prosocial Behavior scale at both ages yielded a first factor on which all items loaded positively. The internal consistency of the scales was good (age 3, $\alpha = .80$; age 4, $\alpha = .73$).

At age 7, parents and teachers rated twins' behavior on the SDQ. The Prosocial Behavior scale contains five items, largely overlapping with the

RRPSPC items used in earlier ages (e.g., "kind to younger children"; "shares readily with other children"). The response scale was identical to the one used earlier. Factor analyses of the Prosocial Behavior scale for parents and for teachers yielded a first factor on which all items loaded positively. The internal consistency of the scales was good (parents, α = .84; teachers, α = .67).

Parental feelings. We assessed parental feelings when children were at ages 3 and 4, using a shortened version of the Parent Feelings Questionnaire (Deater-Deckard, 2000). The measure has seven items that parents rated on a 5-point scale (in which 1 = *definitely untrue* and 5 = *definitely true*) for the firstborn twin, including statements such as "sometimes I feel very impatient with him/her" and "I usually feel close to him/her." After answering about the firstborn twin, parents were then asked, "do you feel this way more or less with your second-born twin?" This was rated on a 5-point scale from 1 = *a lot more* to 5 = *a lot less* (Asbury, Dunn, Pike, & Plomin, 2003). Exploratory factor analysis of the scores for the firstborn twin revealed two slightly correlated (age 3, r = −.07; age 4, r = −.04) factors with eigenvalues greater than one, corresponding to Negative Feelngs (impatient, wishing the child would go away, angry, frustrated) and Positive Feelings (happy, amused, close). Item loadings ranged from .68 to .83 at age 3 and from .68 to .84 at age 4, and the scale scores for Positive Feelings and Negative Feelings were constructed from these groups of items in the following way. For firstborn twins, the sum of the respective items was standardized for the whole population to zero mean and unit variance. For second-born twins, this standardized sum was added to the standardized sum of the differential items indicating more or less parental feeling; this composite score was then standardized.

At age 7, slightly modified items were used to assess feelings toward the firstborn twin (e.g., "do you ever feel impatient with the ELDER twin?"; "do you feel close to the ELDER twin?"). The 7 items were rated on a 4-point scale (in which 1 = *never* and 4 = *often*) for the firstborn twin. After answering about the firstborn twin, parents were then asked, "do you feel this way more or less often with the YOUNGER twin?" rated on a 3-point scale ranging from 1 = *more* to 3 = *less*. Exploratory factor analysis of the scores for the firstborn twin revealed the same two slightly correlated (r = −.13) factors of Negative Feelings and Positive Feelings. Item loadings ranged from .64 to .83. Treatment of the second-born twin scores at age 7 was similar to that at ages 3 and 4.

Parental discipline. We assessed parental discipline using questionnaire items adapted from a semistructured interview (see Deater-Deckard, Dodge, Bates, & Pettit, 1998), at ages 3 and 4. Parents reported on their use of different discipline strategies with the child: smacking, shouting, reasoning, being firm and calm, making a joke of it, and asking someone else to deal with the situation. These six items were rated on a 5-point scale (in which 1 = *I rarely or never do this* to 5 = *I usually do this*), and, after answering for the firstborn twin, parents were asked, "do you do this more or less with your 2nd-born twin?" rated on a 5-point scale from 1 = *a lot more* to 5 = *a lot less* (Asbury et al., 2003). Three subscales of two items are derived from these questions for each of the twins: Negative Discipline (shout, smack), Positive Discipline (reasoning, being firm and calm), and Displacement (making a joke of it, asking someone else to deal with the situation). The Displacement items were not used in this study. Exploratory factor analysis of the scores for the firstborn twin revealed two slightly correlated (age 3, r = −.16; age 4, r = −.18) factors with eigenvalues greater than one, corresponding to parental Negative Discipline and Positive Discipline. Item loadings ranged from .80 to .87 at age 3 and from .81 to .88 at age 4. Scale scores were constructed in a similar way as for parental feelings. For firstborn twins, the sum of the respective items was standardized for the whole population to zero mean and unit variance. For second-born twins, this standardized sum was added to the standardized sum of the differential items indicating more or less parental feeling; this composite score was then standardized.

At age 7, a revised version of this measure was used. Four items indicated Negative Discipline: restraining or smacking, sending the child to his or her room or withdrawing privileges, shouting, and ignoring the child when he or she is misbehaving. The four items were rated on a 4-point scale (in which 1 = *never* and 4 = *often*) for the firstborn twin. After answering about the firstborn twin, parents were then asked, "do you do this more or less often with the YOUNGER twin?" rated on a 5-point scale ranging from 1 = *more* to 3 = *less*. The four items all loaded positively on a first factor in an exploratory factor analysis, with loadings ranging from .54 to .70. Treatment of the second-born twin scores at age 7 was similar to that at ages 3 and 4.

Computation of parental positivity and negativity scores. To reduce the number of scales used in this study, we performed an exploratory higher order factor analysis on the factor scores found at each age. At both age 3 and age 4, two clearly interpretable factors emerged following an oblique rotation. On the first factor, Negative Feelings and Negative Discipline both loaded positively (loadings of .83 or higher), and Positive Feelings and Positive Discipline had loadings smaller than .10 in absolute value. On the second factor, Positive Feelings and Positive Discipline both loaded positively (loadings of .74 or higher), whereas Negative Feelings and Negative Discipline had loadings smaller than .10 in absolute value. We labeled these factors, respectively, Parental Negativity and Parental Positivity. The higher order factor analysis at age 7 with the two negativity scales and the single positivity scale of parental Positive Feelings yielded a single factor, with positive loadings for the negativity scales and a negative loading for the Positive Feelings scale. To enable measure consistency across ages, at age 7 we retained the positivity versus negativity distinction. Cronbach's alphas for the negativity factors ranged from .77 to .78. Alphas for the positivity factors ranged from .46 to .57, attesting to the breadth of the factor. The correlations between negativity and positivity ranged from −.14 to −.16 (all ps < .01), indicating the two factors are largely distinct.

Analyses

Descriptive analyses. Descriptive analyses included mean comparison of prosocial behavior and parent-rated parenting scores across zygosity and sex. Age differences were not assessed, because of the change in prosocial scales across the years and because the nature of the parenting measures involved standardization within age. Concurrent and longitudinal correlations between prosocial behavior and parental positivity and negativity were calculated. In addition, we regressed prosocial behavior on the parenting measures, both concurrently and longitudinally. Finally, we ran a structural equation modeling analysis combining Prosocial Behavior and Parental Positivity and Parental Negativity at ages 3, 4, and 7 to test these relationships simultaneously.

Twin correlations for all five zygosity groups (male and female MZ and DZ and DZO pairs) were calculated for Prosocial Behavior and parent-rated parenting at each age and across ages. Although the major results of twin analyses can be gleaned from correlations, model fitting analyzes all of the data simultaneously, tests the fit of models, yields confidence intervals for parameter estimates, and compares alternative models (Plomin et al., 2001).

Model fitting analyses. We analyzed a trivariate genetic model, including Prosocial Behavior and the two parenting measures, separately at each age, using the variance–covariance matrices of the twin Prosocial Behavior scores and parent-rated parenting. The order of variables in the model was irrelevant for the results of interest in this study. Models were fit to variance–covariance matrices using the Mx program (Neale, Boker, Xie, & Maes, 1999), which calculated specific parameter estimates and their 95% confidence intervals. Figure 1 presents this model. Although the model was based on results from both twins within a pair, it was used to estimate the variance components and correlations across individuals (capitalizing on genetic differences between different types of pairs). Therefore, Figure 1 is simplified by presenting the relationships among variables for one of the twins only.
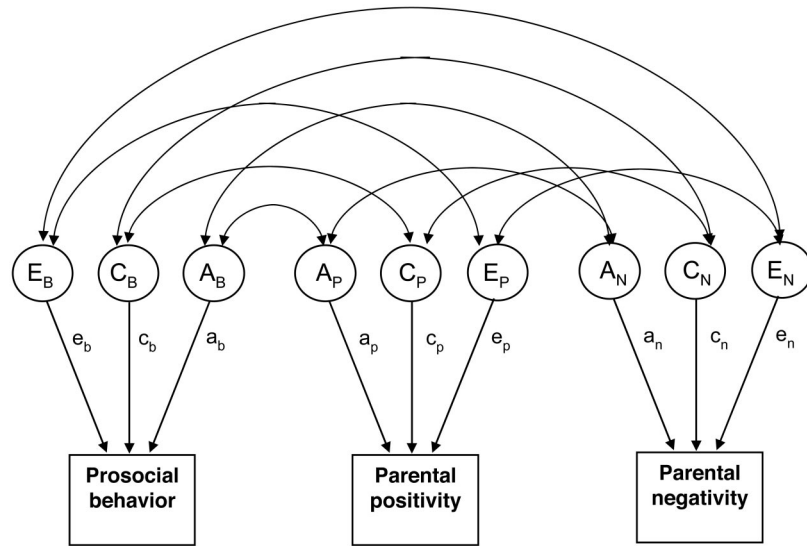
Figure 1. A trivariate genetic and environmental model linking twins' prosocial behavior and parental positivity and negativity toward them. Circles indicate latent variance components estimates, and rectangles indicate observed scores on prosocial behavior and parenting. A = heritability; C = shared environment; E = nonshared environment (and error); lowercase a, c, and e refer to coefficients of estimated influences of the factors A, C, and E; b = prosocial behavior; p = parental positivity; n = parental negativity.

For each of the variables separately, variance components were estimated for additive genetic influence (A), shared or common environment (C), and nonshared environment and error (E). The left part of the figure shows the ACE components that contributed to Prosocial Behavior (b). The middle part shows the influences on Parental Positivity (p), and the right part presents influences on Parental Negativity (n). Squaring the standardized genetic path coefficients yielded an estimate of the total additive genetic variance components (heritabilities) for Prosocial Behavior, Parental Positivity, and Parental Negativity (a_b^2, a_p^2, and a_n^2). Similarly, squaring the standardized environmental path coefficients yielded three estimates of shared environment variance components (c_b^2, c_p^2, and c_n^2) and three estimates of nonshared environment variance components (e_b^2, e_p^2, and e_n^2).

On the basis of their genetic relatedness, the A (additive genetic) factors correlated 1.0 and .5 for MZ and DZ twins, respectively. The C factors referred to the influence of shared rearing environments on twin resemblance. Because twins were reared in the same family, the correlation for shared environment was 1.0 for MZ and DZ twins. (This indicated the degree of overlap of the shared effects making children in the same family similar to each other—100% by definition.) Finally, the E factors reflected nonshared environmental variance and measurement error. These influences (which included any environmental effect that was not shared by twins, such as attending different classrooms) are unique to each member of a twin pair and therefore correlated 0 for all twins.

In addition to estimating the variance components for each variable separately, this correlated factors model (Neale & Maes, 1999) specifies correlated additive genetic, shared environmental, and nonshared environmental effects that influence both prosocial behavior and parenting. The extent that the MZ cross-trait (e.g., prosocial behavior and parental positivity) twin correlation exceeds the DZ cross-trait/cross-twin correlation indicates the degree of genetic overlap between the two traits weighted by the square roots of heritabilities of the two traits. This genetic contribution to the phenotypic correlation between the two traits includes the genetic correlation—for example, the double-headed arrow linking a_b and a_p in Figure 1—that indicates the extent to which the genetic influences on prosocial behavior overlap with the genetic influences on parental positiv-

ity regardless of the heritabilities of the two traits. Similarly, the double-headed arrow linking c_b and c_p indicates the extent to which the shared environmental influences on prosocial behavior overlap with those influencing parental positivity. Finally, the arrow linking e_b and e_p indicates the extent to which the nonshared environmental influences on prosocial behavior overlap with those influencing parental positivity and the degree of relationship between measurement errors for both variables.

The proportion of the phenotypic covariance between two variables attributed to genetic covariance between them is called bivariate heritability (Plomin & DeFries, 1979). It is the product of the genetic path coefficient influencing each of the variables and the genetic correlation between them, divided by the total phenotypic correlation between the variables. Bivariate shared and nonshared environmental contributions to variance and covariance between the two variables are estimated in a similar way. Together, bivariate heritability and shared and nonshared environmental effects sum to the total phenotypic correlation.

Sex-limitation models. Model fitting can also be used to assess sex differences in ACE parameters (Neale & Cardon, 1992). For same-sex twin pairs, the model assumes a genetic correlation of 1.0 for MZ twins and .5 for DZ twins and a correlation of 1.0 for shared environment influences for both MZ and DZ twins. For opposite-sex twins, the phenotypic variation is also a function of additive genetic variance and shared and nonshared environmental influences, but the genetic correlation between opposite-sex twin pairs may be less than .5 if there are qualitative sex-specific genetic effects (i.e., different genes operate on the behavior for each sex). Similarly, although opposite-sex twins are reared in the same family, they may have fewer shared environmental experiences than same-sex twins, and hence, the correlation for shared environment DZO twins may be less than 1.0. The sex-limitation model allows estimation of either a specific DZO genetic correlation (r_gO) or DZO shared environmental correlation (r_cO); however, both parameters cannot be estimated simultaneously. We therefore tested separately the assumptions that the genetic effects for DZO twins correlate .5 and that their shared environments correlate 1.0. These assumptions were tested simultaneously for prosocial behavior and the two parenting variables, with the trivariate model depicted in Figure 1.

We tested each of these assumptions and relaxed each of them in turn, using a series of four nested, reduced models for each age and at age 7 separately for teacher and parent ratings. The full, basic sex-limitation model starts with the assumptions of (a) qualitative sex differences as assessed by $r_g O$ smaller than .5 (or $r_c O$ smaller than 1.0), (b) quantitative sex differences in ACE parameters (e.g., differences in heritability), and (c) phenotypic variance differences between the sexes. This model was tested against models assuming shared environment effects correlate 1 for DZO twins as they do for MZ and DZ same-sex twins. First, it was tested against a *common effects* (quantitative sex differences) model in which ACE parameter estimates for boys and girls were allowed to differ but r_g for DZO twins was constrained to equal .5, as for same-sex DZ twins, which excludes qualitative sex-specific genetic effects. An alternative model tested whether shared environment effects could be assumed to correlate 1 between DZO twins. Second, the basic sex-limitation model was tested against a *scalar* model (phenotypic variance differences between the sexes), which allows phenotypic variances between boys and girls to differ but equates ACE parameters for boys and girls and constrains $r_g O$ to equal .5 and $r_c O$ to equal 1.0. Third it was tested against a *null* model (no sex differences) in which ACE parameters and phenotypic variances for boys and girls were constrained to be equal and $r_g O$ is constrained to equal .5 and $r_c O$ to equal 1.0. Because the alternative models were hierarchically related (i.e., one model is nested within the other), the relative fit of each alternative model was determined by the difference in chi-square between the two models, with degrees of freedom equal to the difference in degrees of freedom between the two models.

The overall fit of the model was evaluated using three indices. The chi-square statistic, in which degrees of freedom equal the number of observed effects minus the number of estimated parameters, indicates the fit of the full model with a low value indicating good fit. However, the chi-square statistic is inflated with large sample sizes. The other two indices—Akaike's information criterion (AIC $= \chi^2 - 2\ df$; Akaike, 1987) and the root-mean-square error of approximation (RMSEA)—give more interpretable estimates of fit for large samples, with lower values representing better fitting models.

Results

Average Differences

Table 1 presents the means and standard deviations of prosocial behavior and parenting scores at ages 3, 4, and 7 and the number of twin pairs for which data were available, separately for each zygosity group. Because twin scores are not independent of each other, for mean comparisons only the scores of one twin per pair, randomly chosen, were used for all variables at all ages. Age effects were not reported because the Prosocial Behavior scales were modified at each age and scores on the parenting variables were standardized for each age.

The mean scores on prosocial behavior at age 7 are comparable with norms reported from a large representative British sample using the SDQ Prosocial Behavior subscale (Meltzer, Gatward, Goodman, & Ford, 2000). Norms for parents' mean scores on prosocial behavior of children ages 5–10 are 8.90 for girls and 8.40 for boys. The results for TEDS twins at age 7, respectively, are 8.50 and 7.96. Teacher scores in the current study (girls, 7.80; boys, 6.67) are also very similar to those of the Meltzer et al. (2000) study (girls, 8.00; boys, 6.70). Similarly, mean prosocial behavior scores in the current study resembled those obtained from younger siblings of a subsample of TEDS twins (Koeppen-Schomerus, Spinath, & Plomin, 2003) at age 3 (girls, 15.91; boys, 14.57, vs. twin girls, 15.52; twin boys, 14.56) and at age 4 (girls,

11.41; boys, 10.54, vs. twin girls, 11.20; twin boys, 10.46), suggesting that prosocial behavior in twins does not differ from nontwin children.

A set of analyses of variance tested for sex and zygosity differences, separately for each variable at each age, using a 2 (male vs. female) × 3 (MZ, DZ, same-sex, and DZO) design. Girls scored higher than boys on prosocial behavior at all ages—age 3, $F(1, 5561) = 112.40$; age 4, $F(7, 7320) = 182.01$; age 7, parent rating, $F(1, 6271) = 163.11$; age 7, teacher rating, $F(1, 4648) = 287.75$; all $ps < .01$—with sex accounting for 2%–5.8% of the variance. At the younger ages, no zygosity effects were found. At age 7, small effects for zygosity were found, accounting for only 0.1% of the variance in parent ratings, $F(2, 6271) = 3.46, p < .05$, and teacher ratings, $F(2, 4648) = 3.46, p < .05$. No interactions were found between sex and zygosity in the mean level of prosocial behavior.

No main effects on parental positivity were found at age 3 or 4 for either sex or zygosity. At age 7, boys received more parental positivity than girls, $F(1, 6271) = 7.39, p < .01$, but this sex difference accounted for only 0.1% of the variance. At this age, MZ twins received somewhat more parental positivity in comparison with DZ twins, $F(2, 6271) = 3.47, p < .05$, but again this difference accounted for only 0.1% of the variance. In addition, there were two significant interactions between sex and zygosity in the mean level of parental positivity—age 3, $F(21, 5561) = 4.35, p < .05$; age 7, $F(2, 6271) = 7.75, p < .01$—but neither accounted for more than 0.2% of the variance.

Finally, sex differences were consistent for parental negativity, with boys getting higher levels of parental negativity: age 3, $F(1, 5561) = 55.52$; age 4, $F(1, 7320) = 107.33$; age 7, $F(1, 6271) = 84.02$; all $ps < .01$. These sex differences accounted for 1%–1.4% of the variance in parental negativity. At age 3, MZ twins received more parental negativity in comparison with DZ twins, $F(2, 5561) = 3.00, p < .05$, a difference accounting for only 0.1% of the variance. In addition, there were two significant interactions between sex and zygosity in the mean level of parental negativity: age 3, $F(2, 5561) = 10.76, p < .01$; age 4, $F(2, 7320) = 4.74, p < .01$. At both ages, DZ girls were the lowest in parental negativity, but DZ boys received relatively high levels of parental negativity. However, these effects were small, and the interactions accounted for only 0.1%–0.4% of the variance in parental negativity.

Prosocial Behavior and Parenting: Phenotypic Relationships

Table 2 presents the correlations among twins' scores on prosocial behavior and parent's twin-specific scores on parenting, in the different ages. Correlations are presented separately for each zygosity group. Cross-twin same-age/same-trait correlations are printed in boldface type and appear on the diagonals of each zygosity group. Same-twin/cross-trait or same-twin correlations appear below the diagonals, and cross-twin correlations above them.

The same-age/same-twin correlations between parenting and prosocial behavior are italicized in Table 2. They indicate the degree of correspondence between parenting and prosocial behavior. All of the relevant correlations were significant ($p < .05$).

Parental positivity correlated positively with parental reports on children's prosocial behavior at all ages, with correlations ranging from .15 to .24, and averaging .21 across ages and zygosity groups.

Table 2
Twin Correlations for Prosocial Behavior and Parenting at Different Ages

Variable and age	Prosocial behavior				Parental positivity			Parental negativity		
	Age 3	Age 4	Age 7[a]	Age 7[b]	Age 3	Age 4	Age 7	Age 3	Age 4	Age 7
Monozygotic male twins										
Prosocial behavior										
Age 3	**.71**	.51	.31	.09	.23	.20	.10	−.10	−.12	−.09
Age 4	.57	**.59**	.27	.10	.20	.16	.03	−.08	−.13	−.15
Age 7[a]	.35	.35	**.67**	.21	.21	.20	.12	−.10	−.13	−.16
Age 7[b]	.09	.11	.24	**.72**	.02	.02	.11	−.06	−.04	−.09
Parental positivity										
Age 3	*.22*	.18	.16	.00	**.77**	.34	.10	−.14	−.16	−.07
Age 4	.13	*.19*	.20	.04	.37	**.77**	.14	−.13	−.12	−.09
Age 7	.04	.05	*.19*	.09	.15	.15	**.68**	−.10	−.14	−.10
Parental negativity										
Age 3	−*.16*	−.15	−.07	−.09	−.17	−.15	−.08	**.76**	.51	.41
Age 4	−.14	−*.19*	−.11	−.06	−.11	−.10	−.16	.57	**.75**	.45
Age 7	−.13	−.17	−.24	−*.15*	−.10	−.11	−.17	.44	.51	**.73**
Dizygotic male twins										
Prosocial behavior										
Age 3	**.41**	.27	.14	.07	.17	.13	.04	−.02	−.06	−.06
Age 4	.56	**.29**	.12	−.03	.13	.14	.04	.02	−.05	−.03
Age 7[a]	.34	.43	**.35**	.04	.04	.10	.12	.05	−.04	−.10
Age 7[b]	.12	.13	.24	**.30**	.06	.04	.03	.00	−.01	−.08
Parental positivity										
Age 3	*.24*	.20	.11	.06	**.67**	.30	.21	−.14	−.14	−.16
Age 4	.21	*.20*	.15	.02	.42	**.61**	.15	−.12	−.11	−.10
Age 7	.06	.09	*.19*	*.06*	.26	.23	**.58**	−.01	−.05	−.05
Parental negativity										
Age 3	−*.11*	−.09	.02	−.10	−.10	−.12	−.08	**.48**	.34	.21
Age 4	−.14	−*.17*	−.04	−.12	−.10	−.13	−.10	.62	**.51**	.16
Age 7	−.15	−.18	−.22	−*.21*	−.12	−.12	−.18	.43	.46	**.46**
Monozygotic female twins										
Prosocial behavior										
Age 3	**.69**	.48	.34	.01	.21	.21	.11	−.13	−.13	−.09
Age 4	.55	**.61**	.33	.03	.20	.21	.12	−.12	−.10	−.11
Age 7[a]	.36	.39	**.60**	.13	.15	.16	.12	−.08	−.10	−.12
Age 7[b]	.06	.09	.19	**.68**	.02	.04	.06	−.03	−.07	−.10
Parental positivity										
Age 3	*.23*	.20	.14	.01	**.79**	.37	.17	−.15	−.12	−.10
Age 4	.23	*.22*	.15	.04	.44	**.78**	.16	−.20	−.12	−.15
Age 7	.13	.11	*.15*	.09	.26	.23	**.69**	−.07	−.04	−.02
Parental negativity										
Age 3	−*.18*	−.14	−.09	−.05	−.20	−.17	−.07	**.76**	.52	.42
Age 4	−.14	−*.18*	−.11	−.07	−.15	−.16	−.08	.56	**.76**	.40
Age 7	−.12	−.15	−.19	−*.13*	−.19	−.17	−.10	.39	.47	**.72**
Dizygotic female twins										
Prosocial behavior										
Age 3	**.53**	.37	.19	−.02	.19	.19	.11	−.07	−.07	−.04
Age 4	.58	**.36**	.13	.02	.23	.16	.10	−.06	−.06	−.01
Age 7[a]	.37	.42	**.24**	.01	.16	.14	.16	−.07	−.06	−.06
Age 7[b]	.07	.11	.13	**.42**	.07	.04	.09	−.01	−.01	.01
Parental positivity										
Age 3	*.20*	.22	.16	.05	**.68**	.36	.14	−.11	−.11	−.11
Age 4	.22	*.21*	.16	.06	.46	**.68**	.10	−.12	−.12	−.12
Age 7	.10	.09	*.19*	*.12*	.25	.21	**.61**	.04	.06	−.01
Parental negativity										
Age 3	−*.18*	−.14	−.09	−.05	−.15	−.13	−.02	**.51**	.33	.24
Age 4	−.16	−*.22*	−.10	−.08	−.17	−.14	−.05	.61	**.54**	.24
Age 7	−.14	−.13	−.18	−*.10*	−.12	−.12	−.13	.44	.50	**.48**
Dizygotic opposite-sex twins										
Prosocial behavior										
Age 3	**.49**	.31	.19	.02	.17	.19	.09	−.04	−.05	−.08
Age 4	.61	**.38**	.15	−.02	.10	.16	.06	.03	−.04	.00
Age 7[a]	.37	.42	**.33**	.01	.11	.11	.11	−.05	−.07	−.07

Table 2 (*continued*)

Variable and age	Prosocial behavior				Parental positivity			Parental negativity		
	Age 3	Age 4	Age 7[a]	Age 7[b]	Age 3	Age 4	Age 7	Age 3	Age 4	Age 7
				Dizygotic opposite-sex twins (*continued*)						
Prosocial behavior (*continued*)										
Age 7[b]	.09	.12	.18	**.32**	.00	.02	.04	−.01	−.02	−.04
Parental positivity										
Age 3	*.23*	.16	.13	.03	**.65**	.32	.12	−.12	−.11	−.16
Age 4	.24	*.21*	.14	.02	.44	**.65**	.14	−.16	−.10	−.14
Age 7	.09	.10	*.21*	*.06*	.18	.20	**.58**	−.07	−.04	−.09
Parental negativity										
Age 3	−*.17*	−.16	−.07	−.03	−.16	−.19	−.10	**.50**	.34	.17
Age 4	−.16	−*.23*	−.14	−.07	−.15	−.16	−.13	.67	**.49**	.19
Age 7	−.13	−.16	−*.21*	−*.11*	−.15	−.13	−.23	.38	.49	**.46**

Note. Same-age correlations between parenting variables and prosocial behavior are italicized. Cross-twin same-age same-trait correlations are printed in boldface type and appear on the diagonals. Within-twin correlations appear below the diagonals, and cross-twin correlations above them.
[a] Parent report.
[b] Teacher report.

This effect was replicated, although moderately, at age 7 for teacher reports on children's prosocial behavior, with correlations ranging from .06 to .12, and averaging .08 across zygosity groups.

Parental negativity correlated negatively with parental reports on children's prosocial behavior at all ages, with correlations ranging in size from −.11 to −.24 and averaging −.19 across ages and zygosity groups. This effect was again replicated at age 7 with teacher reports on children's prosocial behavior, with correlations ranging from −.10 to −.21 and averaging −.14 across zygosity groups.

The two parenting measures correlated significantly with each other (age 3, $r = -.16$; age 4, $r = -.14$; age 7, $r = -.16$; all $ps < .01$). Therefore, to establish the utility of studying separately the relationship between prosocial behavior and both variables, we ran multiple regression analyses with both parenting variables as predictors of prosocial behavior. The results appear in the upper panel of Table 3. At all ages, both parenting variables significantly predicted prosocial behavior independently of each other. Specifically, parental negativity related negatively to prosocial behavior and parental positivity related positively to prosocial behavior. Together, they accounted for 7% to 8% of the variance in prosocial behavior as reported by parents and 3% of the variance in teachers' reports of prosocial behavior.

Next, we examined the role of parental positivity and negativity in predicting long-term prosocial behavior. Parental positivity at age 3 correlated positively with prosocial behavior at age 4 ($r = .19$, $p < .01$), and positivity at age 4 correlated with prosocial behavior at age 7 as reported by parents ($r = .15$, $p < .01$) and to

Table 3
Regression of Prosocial Behavior on Parental Positivity and Negativity and Earlier Prosocial Behavior

Age	Predictor	df	B	SE B	β	t	R^2
		Concurrent relationships					
Age 3	Parental positivity	5518	0.95	0.06	.20	15.42**	.07
	Parental negativity	5518	−0.62	0.06	−.14	−10.96**	
Age 4	Parental positivity	7242	0.58	0.04	.17	14.97**	.08
	Parental negativity	7242	−0.65	0.04	−.21	−18.45**	
Age 7 (parent report)	Parental positivity	6240	0.23	0.02	.13	9.80**	.07
	Parental negativity	6240	−0.42	0.03	−.21	−15.30**	
Age 7 (teacher report)	Parental positivity	4629	0.09	0.04	.04	2.48*	.03
	Parental negativity	4629	−0.45	0.04	−.17	−10.70**	
		Longitudinal effects					
Age 4	Age 3 prosocial behavior	4538	0.41	0.01	.56	44.26**	.34
	Age 3 parental positivity	4538	0.20	0.04	.06	4.46**	
	Age 3 parental negativity	4538	−0.16	0.04	−.05	−3.98**	
Age 7 (parent report)	Age 4 prosocial behavior	4921	0.26	0.01	.39	29.00**	.17
	Age 4 parental positivity	4921	0.15	0.03	.07	4.96**	
	Age 4 parental negativity	4921	−0.06	0.03	−.03	−2.03*	
Age 7 (teacher report)	Age 4 prosocial behavior	3996	0.11	0.02	.12	7.55**	.02
	Age 4 parental negativity	3996	−0.21	0.05	−.08	−4.69**	

* $p < .05$. ** $p < .001$.

a lesser extent as reported by teachers ($r = .03$, $p < .05$). Similarly, parental negativity at age 3 correlated negatively with prosocial behavior at age 4 ($r = -.16$, $p < .01$), and negativity at age 4 predicted prosocial behavior at age 7 as reported by both parents ($r = -.13$, $p < .01$) and teachers ($r = -.10$, $p < .01$).

Finally, we tested whether these longitudinal effects held after earlier prosocial tendencies were accounted for. In other words, we wanted to see if the longitudinal relationships between parenting and prosocial behavior simply represent the longitudinal continuity of prosocial behavior or, of greater interest, if they represent potential effects of parenting over and above the effects of earlier behavior. The results appear in the bottom panel of Table 3. Parenting at age 3 significantly predicted prosocial behavior at age 4, over and above the longitudinal effect of prosocial behavior at age 3. In a similar fashion, parenting at age 4 significantly predicted prosocial behavior at age 7, over and above the longitudinal effect of prosocial behavior at age 4 (although for teacher reports only parental negativity had an additional longitudinal effect). As in the concurrent analyses, in the longitudinal analyses parental negativity related negatively to later prosocial behavior and parental positivity related positively to prosocial behavior. The meaning of this set of findings is that parenting may have an effect on change in prosocial behavior as children grow up.

We next used structural equation modeling (AMOS statistical package, Arbuckle, 1997) to examine a comprehensive model in which data on parenting and prosocial behavior (as rated by parents) from ages 3, 4, and 7 were examined together. Figure 2 presents the standardized coefficients (betas and correlations) among latent variables estimated in this analysis. Factor indicators and error terms were omitted to enhance clarity. All the indicators loaded significantly on their hypothesized latent factors ($p < .001$), with standardized loadings ranging from .25 to .75. We

permitted correlated errors between parallel indicators of the latent factors at different ages (e.g., smacking at age 3 and age 4) to allow longitudinal effects that were over and above the longitudinal relationships between the latent factors. Correlated errors between items indicating positivity and negativity in parental discipline were also allowed, because both sets of items referred to parental discipline. Similarly, we allowed correlated errors between items indicating positivity and negativity in parental affection. Dropping the correlated errors had little influence on the relationships between latent variables but substantially reduced model fit, as indicated by the chi-square difference test, $\Delta\chi^2(110, N = 3,400) = 6,743$, $p < .01$.

We first tested a model allowing all longitudinal relationships among latent variables. Chi-square difference tests showed that model fit was not worsened when the longitudinal correlations between positivity and negativity were dropped from the model, $\Delta\chi^2(6) = 5.98$, ns. The next test showed that dropping the nonsignificant effects of prosocial behavior on parenting did not reduce model fit, $\Delta\chi^2(6) = 8.71$, ns. Finally, dropping the longitudinal effects of negativity on prosocial behavior did not worsen fit, $\Delta\chi^2(3) = 2.18$, ns. Overall model fit was fair: The index of comparative fit (CFI) was .93, and the RMSEA was .027, $\chi^2(1410) = 4,541.46$; $p < .001$.

The results of this analysis take into account the concurrent relationships among variables when estimating longitudinal effects. There was substantial continuity both in parenting and in prosocial behavior, especially from age 3 to age 4. Prosocial behavior had negative relationships with parental negativity and positive relationships with parental positivity at all ages. Finally, parental positivity at age 3 had a longitudinal effect on prosocial behavior at both age 4 and age 7 over and above its concurrent relationships with prosocial behavior and continuity in prosocial
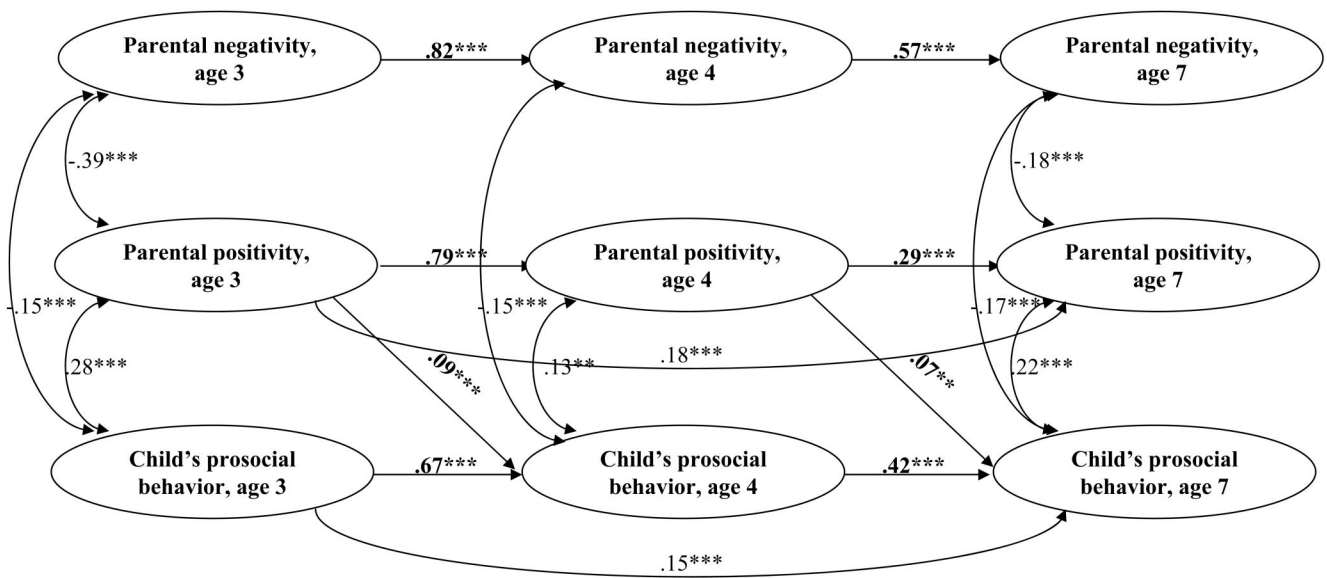

Figure 2. Relationships between prosocial behavior and parental positivity and negativity at ages 3, 4, and 7. Data are standardized coefficients (betas and correlations) among latent variables. Factor indicators and error terms are omitted to enhance clarity. Single-headed arrows represent standardized longitudinal regression coefficients. Double-headed arrows represent concurrent correlations between error terms of latent variables. **$p < .01$. ***$p < .001$.

behavior across these ages. On the other hand, prosocial behavior had no longitudinal effect on parenting. This pattern of results is compatible with an influence of parental positivity on prosocial behavior and not the other way around. In the results listed below and in Table 6, we examine whether these longitudinal effects are to be attributed to environmental or genetic effects.

We next tested the role of sex as a moderator of the relationships between prosocial behavior and parenting. We ran a multigroup structural equation analysis (Arbuckle, 1997) with the same model depicted in Figure 2 simultaneously for girls and boys. The relationships between variables in the model were all significant, as for the model in Figure 2. The path coefficients found for both sexes together were similar when the two sexes were studied separately. No significant differences were found between boys' and girls' coefficients, except for the prediction of parental positivity from age 3 to age 4, which was slightly higher for boys than for girls (respectively, $\beta = .92$ vs. $.78$, both $ps < .01$; difference between coefficients: $z = 2.71$, $p < .01$). Thus, sex does not moderate the phenotypic relationships reported in Figure 2.

Finally, because girls scored higher than boys on prosocial behavior and lower on parental negativity at all ages, as described above, we investigated the role of sex as a longitudinal predictor of prosocial behavior and parenting. We ran the same model depicted in Figure 2, this time with sex added as a predictor of prosocial behavior and parenting at all ages. The concurrent and longitudinal relationships between prosocial behavior and parenting did not substantially change. At age 3, sex (coded as $0 =$ boys; $1 =$ girls) positively predicted prosocial behavior ($\beta = .18$, $p < .01$) and negatively predicted parental negativity ($\beta = -.09$, $p < .01$), as was found in the mean comparisons. There was a weak yet significant relationship between sex and parental positivity as well ($\beta = .06$, $p < .05$). No longitudinal effects of sex on parenting were found beyond age 3. The sex differences in parental negativity at ages 4 and 7 were accounted for by earlier effects. However, sex did have an additional longitudinal effect on prosocial behavior, over and above effects at age 3 ($\beta = .08$, $p < .01$ at both age 4 and age 7), indicating an ongoing influence of sex on children's prosocial behavior.

Genetic and Environmental Effects on Prosocial Behavior and Parenting

To examine genetic and environmental influences on prosocial behavior and parenting, we began by comparing MZ and DZ twin correlations. Between-twin correlations at the same age and for the same trait, shown in boldface type in Table 2, indicate whether there is some degree of genetic influence on parenting or prosocial behavior and whether there are environmental effects. At all ages, and for prosocial behavior as well as both parenting variables, MZ correlations were larger than DZ correlations, indicating genetic influence. MZ correlations were less than 1.0, which suggested influence of nonshared environment and error of measurement. In most cases, DZ correlations were greater than half the MZ correlations, indicating shared environmental influence. The exceptions suggesting little shared environmental influence were prosocial behavior for boys at age 4, for both sexes at 7 (using parent reports), and boys at age 7 (using teacher reports).

Trivariate model fitting analyses were carried out on the prosocial scale and the two parenting variables, separately at each age

(and, at age 7, separately for teacher reports and parent reports). We used the Mx program (Neale, et al., 1999) to fit models to variance–covariance matrices and to calculate specific parameter estimates and their 95% confidence intervals. The focus of the current study was on the bivariate relationship between each of the parenting variables and children's prosocial behavior. These bivariate relationships can be derived from the trivariate model as described in the analysis section.

Sex-limitation models. Preliminary analyses tested whether shared environment effects correlated 1.0 for DZO twins as they did for MZ and DZ same-sex twins. At all ages, results showed that this assumption was valid, as constraining r_cO to be equal to 1 did not reduce model fit: age 3, $\chi^2(3) = 4.44$; age 4, $\chi^2(3) = 5.55$; age 7, parent report, $\chi^2(3) = 0.55$; age 7 teacher report, $\chi^2(3) = 0.72$; all *ns*. Similarly, constraining r_gO to equal .50 for DZO twins as for DZ same-sex twins did not result in worsening of model fit: age 3, $\chi^2(3) = 1.49$; age 4, $\chi^2(3) = 3.11$; age 7, parent report, $\chi^2(3) = 0.57$; age 7 teacher report, $\chi^2(3) = 0.75$; all *ns*. The full sex-limitation model, allowing for qualitative sex differences, was therefore rejected in favor of the next, common effects model. The common effects (allowing quantitative sex differences in ACE parameters but constraining r_g for DZO twins to .50 as with same-sex DZ twins and r_cO to equal 1 for all twins) was next tested against the scalar model (which equates ACE parameters for boys and girls and constrains r_gO to .50 but allows phenotypic variances between boys and girls to differ). At age 7, the scalar model proved to worsen the fit—parent report, $\chi^2(18) = 37.86$; teacher report, $\chi^2(18) = 43.42$; both $ps < .05$—and was rejected. We therefore estimated ACE parameters separately for boys and girls at age 7, keeping the common effects model for age 7. At the younger ages, equating ACE parameters for girls and boys did not worsen the fit: age 3, $\chi^2(18) = 16.27$; age 4, $\chi^2(18) = 29.48$; both *ns*. We therefore estimated ACE parameters jointly for boys and girls at these ages, preferring the scalar model.

The scalar model was compared with the null model (no sex differences) in which ACE parameters and phenotypic variances for boys and girls were constrained to be equal and r_gO was constrained to .50. Boys' and girls' phenotypic variances could not be equated in any of the analyses, as revealed by the comparison between the null model and the scalar model: age 3, $\chi^2(3) = 31.36$; age 4, $\chi^2(3) = 22.77$; age 7, parent report, $\chi^2(3) = 135.13$; age 7, teacher report, $\chi^2(3) = 99.46$, all $ps < .01$. Nevertheless, it can be seen from Table 1 that sex differences in standard deviations are quite small and detected as significant because of the statistical power of our large sample.

To sum up the different sex-limitation models, all model-fitting results that are presented in Tables 4 and 5 are based on a model that sets $r_gO = .50$ and $r_cO = 1$ for DZO twins as for DZ same-sex twins (no qualitative sex differences). The scalar model with no qualitative sex differences and equal ACE parameters for boys and girls was chosen at ages 3 and 4. We therefore report heritability and environmental estimates for these ages without distinguishing between girls and boys (no quantitative sex differences). The common effects model with no qualitative sex differences (but allowing sex differences in ACE parameters) was chosen at age 7 because it fit the data best at this age (therefore, results are presented separately for girls and boys at age 7, allowing quantitative sex differences).

Model fit was fair in all analyses, as indicated by the fit indices: age 3, $\chi^2(81, N = 5{,}521) = 117.37$, $p < .01$; AIC = -44.63; RMSEA = .012; age 4, $\chi^2(81, N = 7{,}245) = 186.43$, $p < .01$; AIC = 24.43; RMSEA = .022; age 7 with parent reports, $\chi^2(63, N = 6{,}243) = 118.95$, $p < .01$; AIC = -7.05; RMSEA = .017; age 7 with teacher reports, $\chi^2(63, N = 6{,}243) = 98.55$, $p < .01$; AIC = -27.45; RMSEA = .012.

Estimates of variance components. Table 4 presents in its upper panel the ACE components of variance (and 95% confidence intervals) in prosocial behavior at ages 3, 4, and 7. Both genetic and environmental factors contributed to prosocial behavior. However, heritability increased from age 3 to age 7, whereas shared environment effects dropped in magnitude throughout this period, as indicated by the nonoverlap of confidence intervals for the estimates at the different ages. At age 7, very small (.03–.05) effects for shared environment were found for prosocial behavior as reported by parents. For girls, there was a significant shared environment effect at age 7, using teacher reports. These results are similar to those reported elsewhere in univariate analyses of these data on prosocial behavior (Knafo & Plomin, in press).

The lower panels of Table 3 present the ACE components of variance in parenting at ages 3, 4, and 7. Again, both genetic and environmental factors contributed to parent-reported parenting. At all ages, all three components had substantial and significant influence on parenting. For parental positivity, heritability ranged from .19 to .30, shared environment ranged from .40 to .52, and nonshared environment and error accounted for an effect of .20 to .30. The results are remarkably similar for ages 3 and 4, with slight sex differences at age 7.

Regarding parental negativity, heritability was relatively high, ranging from .53 to .58. Shared environment effects were weaker (.18 to .24) when compared with genetic effects and with the shared environment effects on parental positivity. No apparent age or sex differences were found.

We ran the genetic analyses again, with families with full data at all ages ($n = 3{,}400$ pairs), and the results were virtually unchanged. For example, heritability was estimated at .30 for parental positivity at age 3 (.29 in Table 3). The estimates for shared environment effects (.48) and nonshared environment effects (.22) were also similar to the corresponding figures with the full sample (.51 and .20).

Genetic and Environmental Effects on the Prosocial Behavior—Parenting Relationship

Cross-twin/cross-trait correlations indicate whether there is genetic or environmental influence on the relationship between parenting and prosocial behavior. As shown in Table 2, the cross-twin/cross-trait correlations between prosocial behavior and parental positivity were not substantially larger for MZ twins than they were for DZ twins. Specifically, the MZ cross-twin correlation between prosocial behavior and parental positivity averaged .15, and the same DZ cross-twin correlation averaged .13. These results indicate that the phenotypic association between prosocial behavior and parental positivity is largely mediated by the shared environment.

The MZ cross-twin correlation between prosocial behavior and parental negativity averaged $-.12$ and was greater than the same DZ cross-twin correlation, which averaged $-.05$. This pattern of results indicates that the phenotypic association between prosocial behavior and parental negativity is largely mediated genetically. In addition, the DZ cross-trait/cross-twin correlation is about half the MZ correlation, indicating no shared environment effects are common to parental negativity and to prosocial behavior. Model fitting analyses decomposed these phenotypic correlations into their genetic and environmental components by analyzing the variance–covariance matrices for prosocial behavior and parenting.

Table 5 presents in its first column the phenotypic correlations between prosocial behavior and parental negativity and positivity, estimated across zygosity groups. Next in Table 5 are the relative contributions of bivariate heritability, bivariate shared environment effects, and bivariate nonshared environment to the phenotypic correlations between each parenting measure and prosocial

Table 4

Estimates of Variance Components (and 95% Confidence Intervals) Accounting for Individual Differences in Prosocial Behavior and Parental Negativity and Positivity at Ages 3, 4, and 7

Variable and age	Sex	Heritability	Shared environment	Nonshared environment
Prosocial behavior				
Age 3	Combined	.40 (.34–.45)	.29 (.25–.34)	.31 (.29–.33)
Age 4	Combined	.52 (.46–.58)	.09 (.04–.14)	.39 (.37–.42)
Age 7 (parent report)	Boys	.63 (.53–.70)	.05 (.00–.15)	.32 (.29–.35)
Age 7 (parent report)	Girls	.57 (.52–.62)	.03 (.01–.07)	.40 (.37–.43)
Age 7 (teacher report)	Boys	.71 (.68–.74)	.00 (.00–.02)	.29 (.26–.32)
Age 7 (teacher report)	Girls	.45 (.34–.57)	.23 (.12–.33)	.32 (.29–.36)
Parental positivity				
Age 3	Combined	.29 (.25–.33)	.51 (.48–.55)	.20 (.19–.21)
Age 4	Combined	.30 (.27–.34)	.50 (.46–.53)	.20 (.19–.21)
Age 7	Boys	.30 (.22–.37)	.40 (.35–.47)	.30 (.27–.33)
Age 7	Girls	.19 (.10–.27)	.52 (.44–.59)	.30 (.27–.32)
Parental negativity				
Age 3	Combined	.58 (.53–.63)	.20 (.15–.25)	.22 (.21–.24)
Age 4	Combined	.53 (.49–.58)	.24 (.19–.28)	.23 (.22–.25)
Age 7	Boys	.55 (.45–.64)	.18 (.10–.27)	.27 (.25–.30)
Age 7	Girls	.54 (.44–.62)	.19 (.11–.28)	.27 (.25–.29)

Table 5
Concurrent Phenotypic Correlations Between Prosocial Behavior and Parental Negativity and Positivity and Percentage of Correlation Mediated by Bivariate Genetic and Environmental Factors

| | | | Percentage of correlation accounted for by | | |
Age	Sex	Correlation—parenting and prosocial behavior	Bivariate heritability	Bivariate shared environment	Bivariate nonshared environment
		Prosocial behavior and parental positivity			
Age 3	Combined	.23	10	78	12
Age 4	Combined	.21	24	64	11
Age 7 (parent report)	Boys	.20	53	24	23
Age 7 (parent report)	Girls	.18	27	54	19
Age 7 (teacher report)	Boys	.09	88	0	12
Age 7 (teacher report)	Girls	.06	14	85	1
		Prosocial behavior and parental negativity			
Age 3	Combined	−.18	64	0	36
Age 4	Combined	−.21	68	0	32
Age 7 (parent report)	Boys	−.21	45	20	35
Age 7 (parent report)	Girls	−.20	40	24	36
Age 7 (teacher report)	Boys	−.14	77	0	23
Age 7 (teacher report)	Girls	−.12	81	0	19

behavior. Bivariate heritability estimates the extent to which the phenotypic correlation between parenting (parental feelings or discipline) and prosocial behavior are mediated genetically. Similarly, bivariate shared and nonshared environment effects estimate the contribution of the environment to the phenotypic correlation. These relative contributions are estimated and the contribution of each variance component to each of the variables is taken into account. (Note that in the case of teacher reports, the univariate shared environment influence on boys' prosocial behavior was estimated at zero. Therefore, the bivariate shared environment effects were also estimated at zero. In addition, at age 4 we fixed the bivariate shared environment effect between parental negativity and prosocial behavior to zero, because this negligible effect—accounting for 12% of the .21 correlation—contributed negatively to the correlation, indicating a positive effect of parental negativity on prosocial behavior, in a way that was impossible to interpret.)

Bivariate heritability, shared environment, and nonshared environment all mediate the correlations between parenting and prosocial behavior to some extent, but their relative contributions vary dramatically by parenting variable. Although bivariate heritability

accounted for 40% to 81% of the correlation between prosocial behavior and parental negativity (63% on average), it accounted on average for only 36% of the correlation between prosocial behavior and parental positivity (range = 10%–88%). On the other hand, bivariate shared environment effects accounted on average for 51% of the correlation between prosocial behavior and parental positivity (range = 0%–85%) and were especially important at ages 3 and 4 in which substantial contributions of the shared environment to prosocial behavior were found. In contrast, bivariate shared environment effects were almost irrelevant for the correlation between prosocial behavior and parental negativity. The bivariate nonshared environment accounted for a part of the correlation in almost all of the cases, although it was more important for the correlation between parental negativity and prosocial behavior (30% on average) than for the correlation between parental positivity and prosocial behavior (13% on average).

Does the finding that bivariate heritability is more important for the correlation between prosocial behavior and parental negativity than for positivity (and the opposite finding for bivariate shared environment) hold for longitudinal relationships as well? Table 6

Table 6
Longitudinal Phenotypic Correlations Between Prosocial Behavior and Parental Negativity and Positivity and Percentage of Correlation Mediated by Bivariate Genetic and Environmental Factors

| | | Percentage of correlation accounted for by | | |
Variable	Correlation—parenting and prosocial behavior	Bivariate heritability	Bivariate shared environment	Bivariate nonshared environment
Parental positivity at age 3, prosocial behavior at age 4	.19	7	89	4
Parental positivity at age 4, prosocial behavior at age 7	.15	40	60	0
Parental negativity at age 3, prosocial behavior at age 4	−.17	68	0	32
Parental negativity at age 4, prosocial behavior at age 7	−.10	51	0	49

Note. Data are based on parental reports of children's prosocial behavior. Results from girls and boys were combined for these analyses.

presents the longitudinal correlations between parent-rated proso-cial behavior and parenting and the relative contributions of bivari-ate heritability, bivariate shared environment effects, and bivari-ate nonshared environment to these correlations. The longitudinal relationship between parental negativity at age 3 and prosocial behavior at age 4 was accounted for mainly by bivariate heritabil-ity (68%), with the remaining correlation accounted for by biva-riate nonshared environment (32%). The longitudinal correlation between parental positivity at age 3 and prosocial behavior at age 4 was accounted for almost totally by bivariate shared environment effects (89%). Predicting from parenting at age 4 to prosocial behavior at age 7, the relationship between parental negativity and prosocial behavior was accounted for about equally by bivariate heritability (51%) and bivariate nonshared environment (49%). The longitudinal relationship between parental positivity at age 4 and prosocial behavior at age 7 was accounted about equally by bivariate heritability (40%) and bivariate shared environment (60%).

Discussion

Parental reports both of positive and negative parental feelings and of discipline related moderately to children's prosocial behav-ior as rated by the parents, regardless of children's age; similar results were found when prosocial behavior was assessed by children's teachers at age 7. Moreover, parenting predicted proso-cial behavior longitudinally, over and above earlier prosocial be-havior. Another consistent finding concerned the joint contribution of genetics and the environment to individual differences in proso-cial behavior as well as in parenting, although there were differ-ences in the relative contributions of these variance components at different ages. More central to the aims of this article, we exam-ined evidence about the etiology of the relationship between par-enting and prosocial behavior that can enhance our understanding of development.

The Relationship Between Parenting and Prosocial Behavior

There were consistent relationships between parenting and prosocial behavior. Using parental reports of prosocial behavior, we found correlations ranged around .20 for positivity and around $-.20$ for parental negativity. Smaller effects were found with teacher reports. That the effects are not very large reflects the fact that there are many other potential independent influences on prosocial behavior and parenting. The size of the phenotypic correlations between parenting and prosocial behavior in our study is similar to those found in other studies (e.g., Clark & Ladd, 2000; Deater-Deckard et al., 2001; Krevans & Gibbs, 1996).

Although the effects are modest, they are robust. Thus, a posi-tive correlation between prosocial behavior and parental positivity was found at all ages and for both the sexes (see Tables 2 and 5). Similarly, a negative correlation between prosocial behavior and parental negativity was found at all ages and for both the sexes. Moreover, when we used teacher ratings instead of parent ratings of prosocial behavior, it resulted in significant but weaker corre-lations in the same direction. These correlations were expected to be weaker because they involve different raters and because these correlations related behavior at school to parenting behavior in the

family (in a similar vein, the higher correlations for parent reports reflect the fact that they come from the same person). In addition, the relationships between parenting, especially parental positivity, and prosocial behavior held longitudinally, after controlling for past behavior, despite the modest internal consistency of the pos-itivity scales, which may have reduced the phenotypic relationship between this variable and prosocial behavior. This indicates that positive parenting may increase children's prosocial behavior over time.

Parental positivity was influenced mainly by environmental factors, mostly of the shared type, although 19%–30% of the variance was nonetheless accounted for by genetic factors. The meaning of this finding is that factors in the shared environment are more important for parental positivity than factors in the child's genetic disposition. Because the shared environment was important for this trait and because the shared environment effects of prosocial behavior and positivity correlated substantially, biva-riate shared environment effects accounted for most of the pheno-typic correlation between parental positivity and prosocial behav-ior. This contribution tended to become smaller with age, because shared environment effects on prosocial behavior become smaller as children grow up, as we discuss elsewhere (Knafo & Plomin, in press). The contributions, especially at the younger ages, indicate that parental positive discipline and affection that is shared by siblings may to some extent make them similar in the degree of prosocial behavior. This may indicate a familywide effect of parenting that is common to children within the same family and has similar effects on them.

This study provides evidence for the importance of parents' behavior to children's prosocial behavior that remains after con-trolling for developmental change and genetic effects. There was a modest contribution of parental positivity to positive change in prosocial behavior, as the phenotypic longitudinal analyses show. This is in line with previous longitudinal studies, in which parental positive affection predicted children's prosocial behavior toward siblings (Volling & Belsky, 1992) and empathy-related responding (Zhou et al., 2002), after controlling for children's earlier behavior. This study's genetic longitudinal analyses further show that this effect is mediated by shared environmental factors and much less so by genetic factors. Thus there is evidence for longitudinal parental influence that is independent of children's genetic tenden-cies. This last finding demonstrates the utility of genetic longitu-dinal designs in providing evidence for environmental effects.

Parental negativity provides a strikingly different pattern of results. First, individual differences in parental negativity were strongly influenced by genetic influences, accounting for over 50% of the variance. Evidence for especially high heritability for neg-ative parenting has been found in other studies. For example, genetic effects accounted for about 70% of the variance in parents' conflict and negativity in relation to their adolescent children (Neiderhiser et al., 2004). In another study, genetic effects were found for parental hostile-reactive behavior, but not for parental self-efficacy, perceived parental impact, or parental overprotection (Boivin et al., 2005). The reason for this may be that parental negativity is more conditional upon the child's (negative) behav-iors. When a child behaves in a disruptive manner (e.g., tantrums or refusal to share with a peer), he or she is highly likely to elicit negative behavior from parents (evocative gene–environment cor-relation). This is less likely to happen with regard to positive

parental behaviors in response to the child's positive behavior. We discuss the processes relevant to gene–environment correlations below.

Second, the links between parental negativity and prosocial behavior were mainly mediated by bivariate heritability, both concurrently (see Table 5) and longitudinally (see Table 6). This is true for both sexes and at all ages and for teacher reports of prosocial behavior as well as parent reports. In other words, behaviors characteristic of less prosocial children that are influenced by their genetic tendencies contribute to their parents' negative feelings and discipline toward them. This is probably a circular process, as children may then become increasingly less prosocial in response to their parents' negative feelings and discipline toward them. We note here that Deater-Deckard et al. (2001) did not find bivariate heritability to be important for the relationship between prosocial behavior and maternal negative feelings (but they did find a bivariate heritability effect for mothers' partners' behavior). Our results at least call for further investigation of this issue, especially because our results in the current study are stable across ages and across reporters of prosocial behavior.

Bivariate nonshared environment provided a consistent contribution to the parenting–prosocial behavior relationship. It accounted for 11%–23% of the positive correlation between parental positivity and prosocial behavior (but was not relevant for the correlation between parental positivity and girls' prosocial behavior as rated by teachers). It also accounted for 19%–36% of the negative correlation between parental negativity and prosocial behavior. The meaning of this finding is that the nonshared environment effects that make one twin less prosocial than the other overlap in part with the effects that make this twin receive relatively low levels of positive discipline and parental feelings and relatively high levels of parental negativity.

The role of parental differential treatment may be especially important here. The MZ twin differences design can provide evidence for this process, because MZ twins are genetically identical, and the differences between them cannot be explained by genetics and indicate nonshared environmental influences. In one study, the MZ twin who was higher than his or her cotwin in prosocial behavior received more positive parental feelings and less negative parental feelings, harsh parenting, and negative discipline (Deater-Deckard et al., 2001). A prior report from TEDS at age 4 found similar findings with parental discipline and parental feelings (Asbury et al., 2003). Thus, some of the phenotypic correlation between parenting and prosocial behavior is the result of child-specific effects, which make children within the same family different from each other.

The longitudinal bivariate nonshared environment effect between parental negativity and children's later prosocial behavior (see Table 6) suggests that parental differential treatment causes siblings to differentiate in their behavior over time. This is in line with findings by Caspi and colleagues (2004). In their study, MZ twin differences in maternal negative emotions at age 5 predicted twin differences in antisocial behavior at age 7, over and above predicted twin differences in antisocial behavior at age 5. Like the study by Caspi and colleagues (2004), the current study points to the emergence of a different relationship between parents and their children that is partly based on nonshared environmental differences and that accounts for differential development in later ages. However, by incorporating DZ as well as MZ twins in a full design, it is possible to estimate genetic as well as shared environmental mediation of relationships between parenting and children's prosocial behavior.

Family Processes Accounting for Genetic Mediation of the Relationship Between Parenting and Prosocial Behavior

How do genetic effects come to account for a substantial proportion of the relationship between parenting and prosocial behavior? There are three types of processes through which environmental factors such as parenting can mediate genetic effects, that is, gene–environment correlations: passive, reactive (or evocative), and active (Plomin et al., 1977). As an example of passive gene–environment correlations, consider the possibility that children's genetic tendencies affect their prosocial behavior, whereas parents' behavior is influenced by their own genetically influenced temperamental and personality characteristics (e.g., negative emotionality). Because parents' and children's genes are partially shared, their respective traits (prosocial behavior and parenting) should be correlated.

Evocative and active gene–environment correlations are especially suitable candidates for explaining genetic effects on parenting when the focus is on the child's genes (Neiderhiser et al., 2004). The role of children in affecting parenting and socialization is now an important part of our understanding of family processes. Family theories such as family systems theory (O'Connor, Hetherington, & Clingempeel, 1997) have emphasized the role of children as active agents within the family, who have their own wishes and goals and can influence their parents' behaviors. The notion of the child as a passive recipient of parental influences is no longer seriously considered (Grusec & Goodnow, 1994; Kochanska, 1995; Kuczynski, Marshall, & Schell, 1997).

Children as active agents in the family evoke parental responses in reaction to their behavior (Kochanska, Friesenborg, Lange, & Martel, 2004). A child's prosocial behavior may influence parental feelings by making parents fond of the child (alternatively, a child who is low on prosocial behavior, not willing to share, and not responding positively to the needs of others may increase negative feelings in parents). This idea is supported by the modest bivariate heritability effects found for parental positivity and prosocial behavior. Stronger evidence was found for negativity, as indicated by the large and consistent bivariate heritability effects between parental negativity and prosocial behavior. A prosocial child may evoke less parental negative discipline (e.g., by sharing more readily). Because individual differences in prosocial behavior are partially determined by genetic factors, as this study and others have shown (e.g., Scourfield et al., 2004), the effects of the child on parental behavior are partially the result of genetic propensities (concurrent effects on parental negativity may have long-term influence through the high stability in parental negativity). Thus, evocative gene–environment correlation may account for some of the bivariate heritability between parenting and prosocial behavior. The current findings are in line with a recent study on infant temperament. In that study, about half of the genetic variance in maternal hostile-reactive behaviors was associated with the genetic effects underlying child difficultness (Boivin et al., 2005).

Consider also active gene–environment correlations, in which children actively create environments that foster their genetic

propensities. A prosocial child may engage in behaviors or select environments that can influence parental behavior. For example, a child may like to listen to a story in which the main theme concerns social understanding and care that fits the child's prosocial tendencies. If parents read the story to the child, their behavior may also change to become more compassionate, resulting in more positive discipline. Because prosocial behavior is in part the result of genetic influences, the child's behavior can be considered as mediating the genetic effect. It has been suggested that active gene–environment correlation becomes more important in adolescence (Scarr & McCartney, 1983).

One important point to make about the contribution of children's genetically influenced behavior to the parenting–prosocial behavior relationship is that the effects attributed to prosocial behavior can reflect the effect of a broader set of child behaviors. It is possible that children's prosocial behavior indeed initiates a causal chain through which parental behaviors are changed. However, it is also possible that prosocial behavior is just one of a set of several adaptive behaviors that increase parental positive feelings and discipline and reduce parental negativity. A multivariate model that includes other measures of adaptive or maladaptive child behaviors can establish whether the gene–environment correlation is due to prosocial behavior itself or is the result of a set of several behaviors. Another path for future research is to try to differentiate between parental behaviors that are in response to children's behavior (e.g., praise) and those that are stable across situations (e.g., overall warmth). The parenting measures in the current study did not distinguish between these two aspects of parenting, but studies doing so might try to distinguish between passive gene–environment correlation and the evocative and active types of gene–environment correlation. The latter types are more likely to emerge when measures of parenting include behaviors that are relatively conditional on the child's behavior.

Sex differences can exemplify one kind of child effect, although it cannot be assumed that such effects are genetic in origin and the twin design cannot determine whether these sex effects are due to genetic or environmental sources. In our study, children's sex had an effect on their prosocial behavior and on parents' negativity toward them. Thus, at least some of the phenotypic relationship between parenting and prosocial behavior is due to sex effects (although the analyses have shown that the effects exists for each of the sexes separately as well).

Strengths and Limitations

The use of a very large community sample is a methodological strength of this study. It enabled detecting the modest phenotypic correlations between parenting and teacher-rated prosocial behavior, separately by sex, and assessing the genetic and environmental contributions to these modest phenotypic correlations. The assessment of prosocial behavior and parenting at three different ages is another advantage. Most of the central findings were replicated at ages 3, 4, and 7, particularly the important and consistent contributions of genetics and the environment to the phenotypic correlation. This replication suggests that the findings are robust and are relevant for both early and middle childhood. We could not compare mean scores on our parenting or prosocial measures, as these have changed across the different ages, mainly in order to adapt the items to the age they were measured in. Similarly, longitudinal

relationships across variables may have been influenced by age differences in measures. Nevertheless, the consistency in concurrent and longitudinal phenotypic correlations between prosocial behavior and parenting attest to the validity of using the modified measures across the ages in our study.

Because the same parent rated both twins' prosocial behavior, an increased between-twin similarity might have occurred for both MZ and DZ twins, resulting in an inflated shared environment estimate. The modest shared environment effects on prosocial behavior beyond age 3 suggest this may not be the case. The parenting measures were specifically designed to allow parents to express child-specific feelings and describe child-specific discipline practices. The shared environment effects on parenting may therefore represent the actual similarity in how parents treat both their children. Nevertheless, there is still a possibility that the correlation between parental ratings of prosocial behavior and parenting are inflated because they come from the same source. This could have increased the bivariate shared environment effects, but the latter effects are not very large.

The use of teacher data on prosocial behavior at age 7 further reduced the possibility of rater bias. The findings of bivariate heritability (for parental negativity) and nonshared environment (for both parenting variables) were replicated across parents and teachers, thus increasing our confidence in the importance of bivariate heritability and nonshared environment for the parenting-prosocial behavior relationship. There was less consistency for bivariate shared environment at age 7. The sex differences in bivariate shared-environment effects on the relationship between parental positivity and teacher-reported prosocial behavior were due to the fact that univariate shared environment effects on the latter were found only for girls (see Knafo & Plomin, in press).

Because most of the parents who participated in this study were mothers, an important step for future research would be to establish whether similar findings are obtained with fathers' parenting. At least for parental negativity, there is evidence that fathers' behaviors and mothers' behaviors are influenced by similar proportions of genetic and environmental effects (Neiderhiser et al., 2004).

Finally, there is evidence that questionnaire measures yield higher heritability estimates when compared with observational measures (Deater-Deckard, 2000). There are several potential explanations for this phenomenon (see Deater-Deckard, 2000, for an in-depth discussion), one of which is that observers typically see a small portion of children's behaviors, whereas parents or teachers are exposed to a wider range of behaviors that are more likely to include genetically influenced behaviors. There is evidence that behavior consistent across contexts, as parents observe in an accumulative way, is more heritable than behavior specific to a situation, which is accessible to observers (McGuire, 2003; Towers, Spotts, & Neiderhiser, 2002). This consideration calls for studies including multimethod assessment of children's prosocial behavior and parenting.

Our study has focused on gene–environment correlations. In addition to these effects, gene–environment interactions should be considered, although these were not in the focus of the current study. One such interaction was exemplified with girls' prosocial behavior in this study. Teachers observed children at schools, and parents observed children at home and in other settings but much less so at school. The different heritability estimates for girls (57%

with parent reports, 45% with teacher reports) may indicate that girls' genotypes have different relative influences in the two environments (or that the shared environment has more impact at schools). We discuss this issue more fully elsewhere (Knafo & Plomin, in press). Gene–environment interactions are an important path for future research, including molecular genetic as well as quantitative genetic studies. For example, the effects of the *MAOA* gene on young adults' aggression vary by the extent of pathogenic experiences in childhood (Caspi et al., 2002). However, it has recently been argued that gene–environment correlation will prove to be more important than gene–environment interaction (Plomin & Palfrey-Davis, in press).

This study has used a combination of the longitudinal method and the twin method to address some complex issues relevant to family socialization and child development. The current design, using a large sample with multiple measurement occasions, is powerful and allowed us to study the modest correlations between parenting and prosocial behavior and understand the sources for these correlations. Other research designs can provide additional valuable information. For example, adoption studies can also be helpful in distinguishing the different kinds of gene–environment correlations, as the correlation between adoptive parents' parenting and the genetic tendencies of their children can be the result of active or evocative gene–environment correlations, but not of a passive correlation (see O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). Ultimately, issues of gene–environment correlation can best be resolved when specific genes are identified that are responsible for the heritability of children's prosocial behavior and for the heritability of parenting, especially negative parenting. The present results suggest that some of the same genes will be associated with prosocial behavior and parenting.

Conclusions

This study addressed the relationship between parenting and children's prosocial behavior from early to middle childhood. Parents who have positive feelings toward their children and provide positive, noncoercive discipline have children who are relatively more prosocial. The opposite is true for parents high in negativity toward their children. The nature of the relationship includes both environmental and genetic factors, demonstrating the complexity of family influences. Our findings point to the important role of children's characteristics in family processes.

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