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Prosocial Behavior From Early to Middle Childhood: Genetic and Environmental Influences on Stability and Change

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Prosocial behavior is important for the functioning of society. This study investigates the extent to which environment shared by family members, nonshared environment, and genetics account for children's prosocial behavior. The prosocial behavior of twins (9,424 pairs) was rated by their parents at the ages of 2, 3, 4, and 7 and by their teachers at age 7. For parent ratings, shared environmental effects decreased from .47 on average at age 2 to .03 at age 7, and genetic effects increased from .32 on average to .61. The finding of weak shared environmental effects and large heritability at age 7 was largely confirmed through the use of teacher ratings. Using longitudinal genetic analyses, the authors conclude that genetic effects account for both change and continuity in prosocial behavior and nonshared environment contributes mainly to change.

Keywords: prosocial behavior, development, genetics, TEDS

One of the most important aspects of humans, distinguishing us from other species, is the degree of helping, cooperation, and altruism among people (Fehr & Fischbacher, 2003). *Prosocial behavior*, that is, behavior intended to benefit others (Eisenberg & Fabes, 1998), is often considered as the basis of human relationships (Staub, 1979). Prosocial children are relatively well-adjusted and have better peer relationships than do children low in prosocial behavior (e.g., Clark & Ladd, 2000). The growing interest in positive human behavior is manifested in a call by Seligman and Csikszentmihalyi (2000) to study its antecedents. This study addresses the etiology of one positive aspect of human behavior, prosocial behavior. Using data from 9,424 pairs of twins, we study the genetic and environmental sources of individual differences in the development of prosocial behavior at ages 2, 3, 4, and 7.

Possible environmental sources of individual differences in prosocial behavior have often been considered (Grusec, Davidov, & Lundell, 2002; Staub, 1979). Most of the studies focused on parental influences on children's prosocial behavior (Eisenberg & Fabes, 1998). However, there is some evidence that, under certain conditions, peers and schools also affect children's degree of prosocial behavior (see review by Eisenberg & Fabes, 1998). In addition, television programs designed to increase children's prosocial behavior and attitudes have been shown to have at least short-term success (Calvert & Kotler, 2003; Cole et al., 2003).

Studies of parental effects found evidence that prosocial behavior in children relates positively to parental warmth and is enhanced by parental modeling of helping behavior (Eisenberg & Fabes, 1998). For example, parents' use of inductive discipline (explaining to children the consequences of their behavior) as opposed to power-assertive discipline has been related to early adolescents' empathy and prosocial behavior (Krevans & Gibbs, 1996). In another study, 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 addition to broad parenting styles, parents provide for children their first socialization system, and parent's actions regarding prosocial behavior have been documented to relate to children's behaviors. For example, mothers of 6- to 11-year-olds who felt comfortable about using rewards for increasing children's prosocial behavior reported their children to be relatively low on prosocial behavior (Fabes, Fultz, Eisenberg, May-Plumlee, & Christopher, 1989). For these children, rewards for helping undermined subsequent prosocial behavior (Fabes et al., 1989). In contrast, there is evidence that assignment of routine household work to children relates to concern for others (Grusec, Goodnow, & Cohen, 1996).

Thus, there is compelling evidence for environmental, particularly familial, effects on prosocial behavior. In addition, there is evidence for genetic influences, as we discuss below. This study uses a genetically informative design to disentangle environmental and genetic effects on prosocial behavior. We approach the issue of genetic and environmental contributions to individual differences in prosocial behavior by 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. The twin method uses this genetic difference in conjunction with the equal environments assumption, which assumes that MZ and DZ twins growing up in the same families are equal in terms of how similar the environments of the twins are, in which case greater similarity of MZ twins versus DZ twins indicates genetic influence. Similarity beyond this genetic effect is attributed to the

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environment twins share, and any further differences between twins are ascribed to nonshared environment or to measurement error (Plomin, DeFries, McClearn, & McGuffin, 2001). There is evidence suggesting that the equal environments assumption is reasonable, although it suggests caution in overinterpreting the precision of twin analyses (Plomin et al., 2001).

Evidence for Genetic and Environmental Influences on Prosocial Behavior

Few studies have addressed directly the issue of the origins of individual differences in prosocial behavior or relevant personality traits by using a genetically informed design that allows differentiation between genetic and environmental influences. Nearly all of the relevant studies used the twin design, with various methods, definitions of prosocial behaviors and traits, and age groups. Because we are interested in the developmental patterns of genetic and environmental effects, we review this literature according to the age of the samples studied, beginning with the oldest samples.

Adult and Adolescent Studies

One twin study involved 230 adult (ages 42 to 57) male twin pairs, who described themselves by using an "empathic responsiveness" (e.g., generous, emotional, kind) scale (Matthews, Batson, Horn, & Rosenman, 1981). MZ twins' scores were more similar than those of DZ twins, and heritability estimates were between .42 and .72. The twin correlations indicated that all residual variance comes from nonshared environment effects. A study that used a similar measure with self-reports by high school seniors ($N = 839$ pairs), yielded moderate significant heritability (.28) and no shared environment effects (Davis, Luce, & Kraus, 1994). Similar results of moderate (.27) heritability and no shared environmental effects were obtained for adult twins' (aged over 50; $N = 1{,}024$ pairs) self-reported cooperativeness (Gillespie, Cloninger, Heath, & Martin, 2003).

A fourth study assessed 475 twin pairs aged 19 to 60. Self-reports on three scales relevant to prosocial behavior (Altruism, Empathy, and Nurturance) all yielded substantial heritability coefficients (.38 to .72) for both men and women (Rushton, Fulker, Neale, Nias, & Eysenck, 1986). The only shared environment effect found was for men's empathy (.23). Thus, results from four twin studies that used different methods converge on the finding that, at least in adolescence and adulthood, genetics influence prosocial traits, as does the environment, but these environmental influences are of the nonshared type, meaning they make twins growing up in the same family dissimilar to each other rather than similar.

One adult study, however, deviated from this conclusion (Krueger, Hicks, & McGue, 2001). Twins (aged 33 on average) were enrolled in the Minnesota Twin Registry ($N = 213$ pairs). The Self-Report Altruism Scale (Rushton, Chrisjohn, & Fekken, 1981) was adapted to include questions about altruistic behavior toward friends and acquaintances in addition to actions in the benefit of strangers and organizations, which were assessed by the original scale. This study reported a moderate effect of shared environment on altruism (.35) and little genetic effect, as the MZ correlation (.38) was similar to the DZ correlation (.31).

In summary, although four out of five studies indicated moderate genetic influence and little shared environmental influence, a fifth study yielded opposite results, perhaps because of the different measure used. That is, the difference between the two studies that used the Self-Report Altruism Scale (Krueger et al., 2001; Rushton et al., 1986) could result from the adaptation of the scale for the purposes of the later study. Another possibility is that the difference is merely sampling error because both of these studies are relatively small in relation to the exorbitant demands for statistical power in twin studies.

Child Studies

Overall, most of the studies in childhood have found both genetic and shared environmental effects. For example, one study used the step-family sibling design, which assumes that shared environment effects are common to siblings and half siblings living together and capitalizes on the lower genetic resemblance of half siblings (25% on average) as compared with full siblings (50%) (Deater-Deckard, Dunn, O'Connor, Davies, & Golding, 2001). Through the use of maternal reports on the prosocial scale of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), this study reported modest heritability (.15) and modest shared environment effects (.18).

Some child studies assessed prosocial behavior within a twin dyad. For example, in a study of 390 twin pairs, mothers were interviewed regarding cooperation between their twins, aged 3 to 8 (Lemery & Goldsmith, 2001). Shared environment accounted for most of the variance in the cooperation between twins (.61), and the rest of the variance was mostly due to nonshared environment. A particularly important study of prosocial behavior involved 246 twin pairs from the age of 14 months through 20 and 24 months on to 36 months (Zahn-Waxler, Robinson, & Emde, 1992; Zahn-Waxler, Schiro, Robinson, Emde, & Schmitz, 2001). One of the measures included maternal reports, referring mainly to prosocial behavior between the twins. Similar to the study just described (Lemery & Goldsmith, 2001), prosocial behavior between the twins yielded substantial shared environment estimates, ranging from .59 to .80. Only at 14 months was there a significant heritability estimate, accounting for 28% of the variance (Zahn-Waxler et al., 2001). These two studies might be seen as providing evidence that shared environment may be relatively important for intertwin cooperation, perhaps because parents might monitor (and influence) within-family events more closely than they can affect children's behaviors in other settings. Alternatively, the shared environment estimates might actually reflect an effect of mutuality, with twins responding to each other's prosocial behavior with further cooperation, regardless of whether they are identical or fraternal. In this case, then, the twins are each other's environment in relation to their cooperative behavior.

An important feature of the Zahn-Waxler et al. (1992) study is its inclusion of experimental measures of prosocial behaviors. At 14, 20, 24, and 36 months, twins were observed in a situation in which their mother or the experimenter was ostensibly hurt, and the twins' reactions were coded into five categories: prosocial acts (trying to help or comfort the victim), concern for the victim, self-distress over the victim's suffering, indifference toward the distress, and "hypothesis testing" (attempting to comprehend the victim's distress). Two of the response categories, indifference toward the distress and "hypothesis testing", showed some shared environment influences (.14 to .27) but only at 20 and 24 months. No other shared environment influences were found. Genetic ef-

fects at most ages were found for prosocial acts, concern for the victim, and hypothesis testing, although these estimates varied from .10 to .42. A fourth category, indifference, showed genetic effects only at 14 and 36 months, and the remaining category, self distress over the victim's suffering, was accounted for mainly by nonshared environment.

These results point to the importance of age in the relative influences of genetics and the environment on prosocial behavior. The age range in the Zahn-Waxler study (14 to 36 months) is characterized by especially rapid cognitive and emotional changes, which might contribute to the seemingly inconsistent findings (e.g., shared environment effects only at 20 and 24 months. Our study addresses a wider age range—ages 2, 3, 4, and 7, which may facilitate finding more systematic developmental patterns.

Relative Genetic and Environmental Contributions to Prosocial Behavior at Different Ages

Research has shown that shared environment influence for some traits, especially cognitive abilities, decreases with age from infancy and early childhood to adolescence and adulthood (McGue, Bouchard, Iacono, & Lykken, 1993; Plomin et al., 2001). If this pattern can be generalized to prosocial behavior, then we would expect a decline throughout the years in the importance of shared environment in predicting prosocial behavior.

Despite some inconsistencies, the literature reviewed above has suggested that shared environmental effects on prosocial behavior become less important from childhood to adolescence and adulthood. Most adult studies reported very little shared environment effects. In contrast, childhood studies have generally indicated some degree of shared environment effects. One study of prosocial behavior in 619 twin pairs supported this pattern (Scourfield, John, Martin, & McGuffin, 2004). Through the use of teacher reports on the SDQ, this study found shared environment effects in their child (5–10) subsample but none in the adolescent (11–16) subsample (.30 vs. .00). Similar (though nonsignificant) differences were found with parent reports of twins' prosocial behavior (Scourfield et al., 2004).

Estimates of the three variance components, heritability, shared environment, and nonshared environment, are interdependent because they are expressed as proportions of total phenotypic variance. That is, the possibility of decline in shared environment effects entails an increase in either genetic effects or nonshared environment effects or both. Research typically shows an increase in genetic influence throughout the years (Plomin et al., 2001). This may apply to prosocial behavior too. Although evidence from the only other longitudinal twin study of prosocial behavior is inconclusive (Zahn-Waxler et al., 2001), this idea is supported when data from age 24 months are compared with data from age 36 months—the first two age groups studied in the current study. In each of the five experimental observations, heritability increased from age 2 (average heritability of .13) to age 3 (.27). Only for maternal reports was there a drop in heritability in this period (from .17 to 0.00). Similarly, Scourfield et al. (2004) reported lower heritability in their child subsample than in the adolescent subsample (.46 vs. .87).

Regarding the effects of nonshared environment at different ages, it is impossible to compare different studies as this estimate also includes measurement error, which is likely to vary when using different methods. Nevertheless, it is interesting to consider this issue, as environmental influences are usually more nonshared than shared. Especially at the ages studied here, new environments are added all the time to children's lives, and an increasingly large proportion of these environments are not shared by twins. For example, as children grow up, more of them are likely to attend daycare and to be exposed to new socialization forces as well as to new peers. The likelihood of twins sharing their friends when they are in daycare is lower than when they are not because more peers are available. Other influences such as books and television may become increasingly nonshared as the children grow up. Thus, it would not be surprising to find that as the shared environment decreases, the nonshared environmental influences increase in importance.

The Development of Genetic and Environmental Influences on Prosocial Behavior

Thus far we have discussed the relative influences of genetic and the environment at different ages. However, cross-sectional age differences in these relative influences tell only part of the story. For example, a rise in the importance of the nonshared environment can be the result of either new nonshared environmental influences or of the same environments exerting stronger influences as children mature. Similarly, an increase in heritability may be the result of new genetic effects emerging in later ages or of the same effects having stronger influence as children grow up (Plomin et al., 1993). Only longitudinal data can address issues of change and stability in the genetic and environmental origins of individual differences.

Genetic effects should not be equated with stability, although the genetic composition of individuals is, of course, fixed at conception. Just as balding is influenced by genes that have their effect only in adulthood, so other genetic influences may be relevant at early ages and then wane in importance, or, alternatively, new genetic influences may emerge as children mature. For example, because prosocial behavior entails cognitive tasks such as perspective taking and identifying others' need for help (Pearl, 1985; Roberts & Strayer, 1996), as children grow up new genetic effects relevant to the maturation of the brain may indirectly affect the extent of prosocial behavior. In the study by Zahn-Waxler et al. (2001), genetic effects were partially responsible for continuity (Plomin et al., 1993). However, new genetic effects emerged at different ages, accounting for change as well. We expect this pattern to be found in this study as well.

As is the case with genetic effects, environmental influences, even those shared by family members, can contribute to change as well as to continuity. Parents' attitudes and socialization practices change as their children develop (cf. Grusec et al., 1996), as do other factors (e.g., change in daycare, addition of a younger sibling), which may lead to new shared environmental effects as children grow up. There is evidence for new shared environment effects emerging in the 2nd and 3rd year of children's lives (Zahn-Waxler et al., 2001). However, if shared environment becomes less important throughout the years, new shared environmental effects are less likely to be found than new genetic effects.

Finally, although nonshared environmental influences might seem most likely to contribute to change, they could also contribute to continuity. For example, if one of the twins (but not the other) has a best friend who is particularly prosocial, then that twin may become consistently more prosocial than the cotwin. How-

ever, data from Zahn-Waxler et al. (2001) have suggested that nonshared environment contributes to change rather than to continuity. In their study, nonshared environment factors were generated at younger ages, but there was no longitudinal effect of these factors on later ages. Rather, new nonshared environment factors were generated at each age (Zahn-Waxler et al., 2001).

The Current Study

In this study, we investigate the genetic and environmental sources of prosocial behavior in a longitudinal study at ages 2, 3, 4, and 7, with data from a large twin sample. The prosocial behavior of twins was rated by their parents and also at age 7 by their teachers. We compare the relative contribution of genetics, shared environment, and nonshared environment with prosocial behavior at the different ages to determine whether the increase in the importance of genetics and decrease in shared environment effects over the years will be found for prosocial behavior. Moreover, we examine the role of genetics and environment in the change and stability in prosocial behavior. Specifically, we use longitudinal genetic analyses to ask whether the genetic and environmental effects present at age 2 continue to affect behavior in later ages or whether new influences emerge.

Method

Participants

Participants in this study were members of 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. Assessments were made at 18 months, 2, 3, 4, and 7 years of age. 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 if there were extreme pregnancy or perinatal difficulties (e.g., gestation < 32 weeks) or if either 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, which 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 at one or more times during the 2-, 3-, 4-, and 7-year-old 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 was available at ages 2 (n = 5,681 pairs), 3 (n = 5,639 pairs), and 4 (n = 7,430 pairs; the 1996 cohort was not studied at ages 2 and 3). At age 7, parent-rated data were available from 6,271 pairs, and teacher-rated data were available from 5,064 pairs (see Table 1). Most twins (64%) had the same teachers. The overall final sample included 9,424 twin pairs: 1,501 monozygotic male (MZM), 1,565 dizygotic male (DZM), 1,711 monozygotic female (MZF), 1,580 dizygotic female (DZF), and 3,067 opposite-sex (DZO) twin pairs.

Attrition analyses reveal some small differences between families providing data at 18 months and those providing data at ages 2, 3, 4 and 7; namely, families remaining in the study were slightly more likely to be White, in the top or second social class, and with the father employed, than were those with missing data (Ronald, Eley, & Plomin, 2003). Despite attrition, analyses show that the TEDS sample continues to be reasonably representative of parents of young children in the U.K. population. For example, U.K. census data 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 A-level examinations, taken by pupils before going to university, as do 34% of TEDS mothers. TEDS is described in more detail elsewhere (Spinath et al., 2003; Trouton, Spinath, & Plomin, 2002).

Measures

Parents of the twin pairs 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). It has been validated in relation to neurological, cognitive, and psychiatric assessments (e.g., Goodman, 1994). 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 subscale in the RRPSPC consists of 11 items at ages 2 and 3 (e.g., "Often volunteers to help others" and "Shares treats with friends"). At 4 years, 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" and "Helps other children who are ill") were combined ("Helpful if someone is

Table 1
Means and Standard Deviations of Scores on the Prosocial Behavior Subscales of the Revised Rutter Parent Scale for Preschool Children

	Age 2[a]			Age 3[a]			Age 4[b]			Age 7 (parent report)[c]			Age 7 (teacher report)[c]		
Twin pair	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
MZM	902	12.40	4.14	900	14.61	3.62	1,184	10.42	2.56	1,063	8.07	1.75	865	6.57	2.49
DZM	989	12.49	3.94	956	14.53	3.61	1,239	10.51	2.66	983	7.90	1.85	819	6.68	2.46
DZO (boys)	946	12.72	4.09	920	14.48	3.67	1,210	10.40	2.70	955	7.89	1.91	750	6.77	2.43
MZF	1,048	13.34	4.10	1,034	15.36	3.59	1,361	11.16	2.53	1,214	8.52	1.58	980	7.72	2.17
DZF	915	13.84	3.90	913	15.71	3.47	1,275	11.23	2.58	1,065	8.41	1.66	839	7.80	2.10
DZO (girls)	931	13.48	4.04	918	15.65	3.48	1,219	11.42	2.56	991	8.57	1.55	811	7.90	2.13

Note. MZM = monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DZO = dizygotic opposite-sex twins.
[a] Score range: 0–22. [b] Score range: 0–16. [c] Score range: 0–10.

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 subscale at all ages yielded a first factor on which all items loaded positively (at least .40). At age 2, the first factor accounted for 36% of the variance (35% at age 3), and a secondary factor accounted for 10% (9% at age 3). The secondary factor at age 2 did not load on the same items as the secondary factor at age 3, and both did not have a clear interpretation. At age 4, only one factor emerged, accounting for 35% of the variance. The internal consistency of the scales was good (age 2: $\alpha = .82$; age 3: $\alpha = .80$; age 4: $\alpha = .73$). The 1-year longitudinal correlation for these scales was .56 from age 2 to age 3 and .59 from age 3 to age 4.

At age 7, parents and teachers rated twins' behavior on the SDQ. The Prosocial Behavior subscale contains five items, largely overlapping with the RRPSPC items used in earlier ages (e.g., "Kind to younger children" and "Shares readily with other children"). The response scale was identical to the one used earlier. The SDQ is a widely used measure of children and adolescent adjustment and has been validated and translated into over 40 languages (Woerner et al., 2004). The SDQ as rated by parents and teachers proved to be valid and well suited for screening purposes, longitudinal monitoring of therapeutic effects, and scientific research purposes (e.g., Becker, Woerner, Hasselhorn, Banaschewski, & Rothenberger, 2004; Mathai, Anderson, & Bourne, 2004). Factor analyses of the Prosocial Behavior subscale for parents and teachers yielded a single factor on which all items loaded positively (at least .58), accounting for 43% (parent report) and 61% (teacher report) of the variance. Parent and teacher ratings correlated moderately positively ($r = .22$, $p < .001$). The internal consistency of the scales was good (parents: $\alpha = .84$; teachers: $\alpha = .67$).

Analyses

Descriptive analyses included mean comparison of Prosocial Behavior scores across zygosity and sex. Age differences were assessed only from age 2 to age 3 because scales were modified in later ages. In addition, twin intraclass correlations for all five zygosity groups (male and female MZ and DZ and opposite-sex DZ pairs) were calculated within individual twins across ages and between twins 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).

Cross-sectional model-fitting analyses. For each of the ages separately, the variance components of additive genetic influences (A), shared or common environment (C), and nonshared environment and error (E) were estimated with the variance–covariance matrices of the twin ratings on prosocial behavior. In each of these cross-sectional analyses, we used all of the twin pairs for whom valid data were available at a certain age to maximize the number of twin pairs at each age. A sex-limitation model (Neale & Cardon, 1992) was fit to observed covariance matrices at each age and separately for parent ratings and teacher ratings at age 7 with Mx structural equation modeling software (Neale, Boker, Xie, & Maes, 1999).

The model uses data from all zygosity groups to estimate genetic and environmental variance components and to evaluate sex differences in genetic and environmental contributions to variation in prosocial behavior. Within same-sex twin pairs, the model is a standard univariate twin model. On the basis of the degree of genetic relatedness, the A (additive genetic) factors correlate at 1.0 and .50 for MZ and DZ twins, respectively. The C factors refer to the influence of shared environments on twin resemblance. Because twins grew up in the same family, the correlation for shared environment is 1.0 for MZ and DZ twins. Finally, the E factors reflect nonshared environmental variance and measurement error. These influences are unique to each member of a twin pair and therefore correlate at 0.00 for all twins. The model allows A, C, and E to be estimated separately for each sex.

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 $<.50$ if there are qualitative sex-specific genetic effects (i.e., different genes operate on the behavior for each sex). Similarly, although opposite-sex twins grow up 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 1.00. 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 at .50 and that their shared environments correlate 1.00.

The full sex-limitation models estimating seven parameters (i.e., A, C, and E for boys and for girls and either r_go or r_co) can be compared with reduced models to test for sex effects. This full sex-limitation model permits quantitative sex differences in A–C–E parameters, qualitative sex differences as assessed by r_go (or r_co), and phenotypic variance differences between the sexes. We tested the fit of this full sex limitation against a series of three nested reduced models for each age and separately for teacher and parent ratings at age 7:

1. A *common effects* (quantitative sex differences) model in which A–C–E parameter estimates for boys and girls were allowed to differ, but r_g for opposite-sex DZ twins was constrained to be the same as r_g for same-sex DZ twins, which excludes qualitative sex-specific genetic effects. An alternative model tested whether shared environment could be assumed to correlate at 1.00 between DZO twins.

2. A *scalar* model (phenotypic variance differences between the sexes) allows phenotypic variances between boys and girls to differ but equates A–C–E parameters for boys and girls and constrains r_go to be the same as r_g same sex.

3. A *null* model (no sex differences) is one in which A–C–E parameters and phenotypic variances for boys and girls are constrained to be equal, and r_go is constrained to be the same as r_g for same-sex DZ twins.

Because these alternative models are hierarchically related (i.e., one model is nested within the other), the relative fit of each alternative model is 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.

Longitudinal model-fitting analyses. In addition to the cross-sectional analyses, we conducted longitudinal analyses. The Cholesky decomposition method uses within-twin and between-twin multivariate variance–covariance matrices in order to decompose the variance within and between ages into a set of genetic, shared environmental, and nonshared environmental factors. In other words, the model can be applied to longitudinal data to identify effects that are stable across the years, effects that exist early on but are inconsequential in following years, and new effects.

The model assumes a temporal relationship between the different variables, in this case, prosocial behavior at different ages. Figure 1 illustrates this model as a path diagram that shows the partitioning of the variance of prosocial behavior at each age (the rectangles in Figure 1) partitioned into three components of variance at each age and covariance across ages: additive genetic (A), common or shared environment (C), and nonshared environment plus error (E). For example, prosocial behavior at age 2 is affected by genetic, shared environmental, and nonshared environmental factors unique to this age group because it is the first in the temporal order (although age 2 effects can represent influences accumulated throughout earlier development). The Prosocial Behavior scores at ages 3, 4, and 7 all load on the genetic and environmental factors generated at age 2. Similarly, scores at ages 4 and 7 also load on the factors generated at age 3 and so on for later ages. The extent to which scores at later and younger ages load on the same factors indicates continuity. For example, if the genetic effects at

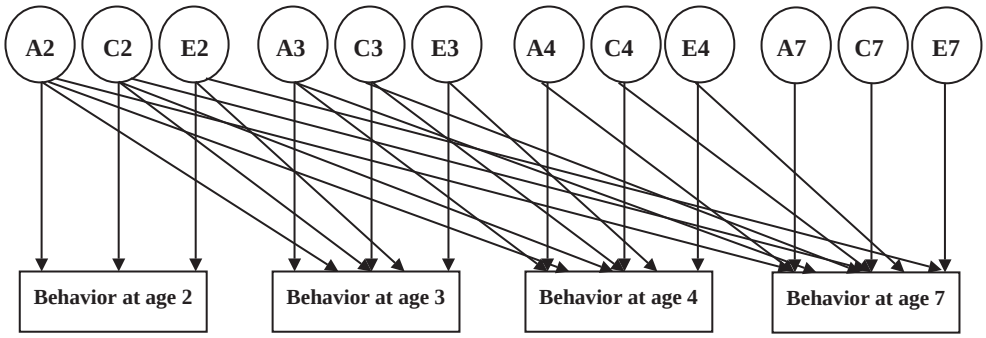

Figure 1. Theoretical model of Cholesky decomposition of variance across ages. Circles indicate variance components estimates, and rectangles indicate observed scores on prosocial behavior. A = heritability; C = shared environment; E = nonshared environment (and error). The number in each circle represents the age to which the variance component is attributed.

age 2 and those at age 3 have substantial loadings on the same genetic factor, then this suggests the contribution of genetics to stability. The extent to which scores at later ages do not load on the same factors as those at younger ages indicates change.

As with the cross-sectional analyses, we apply the principles of the sex-limitation model to the longitudinal analyses. In the longitudinal analyses, we used only twin pairs for whom valid parent data were available at all ages.

Results

Average Differences

Table 1 presents the means and standard deviations of Prosocial Behavior scores at ages 2, 3, 4, and 7 and separately for each zygosity group. Because twin scores are not independent of each other, for these analyses only the scores of one twin per pair, randomly chosen, were used. Age effects could only be investigated from age 2 to age 3 because Prosocial Behavior scales were modified in later ages. A repeated measures three-way ANOVA (Age × Sex × Zygosity) was conducted. An increase in prosocial behavior from age 2 ($M = 13.09$, $SD = 4.09$) to age 3 ($M = 15.06$, $SD = 3.62$) accounted for 23.9% of the variance in prosocial behavior scores, $F(1, 4589) = 1437.31$, $p < .01$. No Age × Sex × Zygosity interactions were found.

Girls scored higher than boys on prosocial behavior at all ages (ages 2–3: $F[1, 4589] = 112.70$; age 4: $F[1, 7482] = 190.30$; age 7: parent rating, $F[1, 6265] = 159.87$; age 7: teacher rating, $F[1, 5058] = 305.48$; all ps $< .01$), and sex accounted for 2.4–5.7% 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, $F(2, 6265) = 3.61$, $p < .05$, with MZ twins scoring slightly higher ($M = 8.31$, $SD = 1.68$) than DZ same-sex twins ($M = 8.16$, $SD = 1.77$) and DZ opposite-sex twins scoring between the two same-sex groups ($M = 8.24$, $SD = 1.77$). No Sex × Zygosity interactions were found in the mean level of prosocial behavior. It should be noted that most of the mean differences found in the extent of prosocial behavior were small with regard to the variance explained and were significant because of the size of our sample.

Cross-Sectional Analyses of Genetic and Environmental Effects on Parent-Rated Prosocial Behavior

To examine genetic and environmental influences on prosocial behavior at different ages, we began by comparing twin correlations obtained within MZ and DZ pairs. Table 2 presents the intraclass correlations between twins' scores on parent-rated prosocial behavior for all zygosity groups, within individual twins across the years and between twins at each age and across ages. For the cross-sectional analyses, between-twin correlations at the same age are relevant and are shown in boldface type in Table 2. The cross-age correlations are discussed later.

At all ages MZ correlations were larger than DZ correlations, indicating genetic influence. MZ correlations were less than 1.00, which suggests the influence of nonshared environment and error of measurement. In most cases (except for boys at age 4 and both sexes at age 7), DZ correlations were greater than half the MZ correlations, indicating shared environmental influence. For both boys and girls, genetic influence appears to increase across age in that the difference in correlations between MZ and DZ twins increases: for boys from 2 to 3 years of age and for girls from 3 to 4 to 7 years of age. Shared environmental influence tends to decrease, as seen in the general age decline in MZ correlations. The pattern of MZ and DZ correlations was generally similar for boys and girls, suggesting no quantitative genetic or environmental sex differences. Finally, the DZO correlations roughly equaled those of same-sex DZ twins, indicating no qualitative genetic or environmental sex differences, a hypothesis that is tested below in model-fitting analyses.

Cross-sectional genetic model-fitting analyses were then carried out on the Prosocial Behavior subscale separately for each age in order to divide the phenotypic variance into the three genetic and environmental components. Models were fit to variance–covariance matrices with the Mx program (Neale et al., 1999), which calculates specific parameter estimates and their 95% confidence intervals.

Sex limitation models. Preliminary analyses tested whether shared environment effects correlate for DZO twins as they do for MZ and DZ same-sex twins. In all ages, results showed that this assumption was valid for prosocial behavior. Similarly, constraining r_go to equal .50 for DZO twins, as for DZ same-sex twins, did

Table 2
Intraclass Correlations for Parent-Reported Prosocial Behavior in Twins at Different Ages

Age and twin	1	2	3	4	5	6	7	8
				MZM twins				
1. Age 2, Twin 1	—							
2. Age 3, Twin 1	.55	—						
3. Age 4, Twin 1	.37	.55	—					
4. Age 7, Twin 1	.21	.29	.34	—				
5. Age 2, Twin 2	**.81**	.49	.31	.18	—			
6. Age 3, Twin 2	.55	**.69**	.43	.28	.55	—		
7. Age 4, Twin 2	.36	.48	**.58**	.31	.37	.56	—	
8. Age 7, Twin 2	.20	.26	.26	**.66**	.18	.30	.33	—
				DZM twins				
1. Age 2, Twin 1	—							
2. Age 3, Twin 1	.50	—						
3. Age 4, Twin 1	.30	.55	—					
4. Age 7, Twin 1	.16	.24	.39	—				
5. Age 2, Twin 2	**.60**	.36	.16	.10	—			
6. Age 3, Twin 2	.34	**.42**	.21	.08	.57	—		
7. Age 4, Twin 2	.26	.24	**.29**	.16	.40	.52	—	
8. Age 7, Twin 2	.12	.11	.12	**.38**	.19	.26	.37	—
				MZF twins				
1. Age 2, Twin 1	—							
2. Age 3, Twin 1	.56	—						
3. Age 4, Twin 1	.40	.55	—					
4. Age 7, Twin 1	.15	.27	.36	—				
5. Age 2, Twin 2	.77	.48	.33	.14	—			
6. Age 3, Twin 2	.53	**.69**	.42	.19	.58	—		
7. Age 4, Twin 2	.39	.46	**.61**	.29	.40	.55	—	
8. Age 7, Twin 2	.15	.25	.30	**.61**	.16	.22	.32	—
				DZF twins				
1. Age 2, Twin 1	—							
2. Age 3, Twin 1	.54	—						
3. Age 4, Twin 1	.32	.55	—					
4. Age 7, Twin 1	.15	.29	.38	—				
5. Age 2, Twin 2	**.62**	.41	.25	.11	—			
6. Age 3, Twin 2	.39	**.53**	.35	.16	.56	—		
7. Age 4, Twin 2	.25	.34	**.35**	.19	.39	.57	—	
8. Age 7, Twin 2	.10	.15	.13	**.27**	.13	.24	.36	—
				DZO twins				
1. Age 2, Twin 1	—							
2. Age 3, Twin 1	.55	—						
3. Age 4, Twin 1	.41	.55	—					
4. Age 7, Twin 1	.17	.27	.38	—				
5. Age 2, Twin 2	**.60**	.39	.25	.08	—			
6. Age 3, Twin 2	.36	**.47**	.25	.09	.57	—		
7. Age 4, Twin 2	.25	.29	**.34**	.11	.42	.55	—	
8. Age 7, Twin 2	.10	.14	.10	**.27**	.20	.28	.40	—

Note. Between-twin same-age correlations are in bold. Twins were randomly assigned as either Twin 1 or Twin 2. MZM = monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DZO = dizygotic opposite-sex twins.

not result in worsening of model fit (common effects model). On the basis of these analyses, the following models assumed that there are no qualitative sex differences in twin similarity (common effects model), meaning that DZO twins share 50% of the genetic effects and all of the shared environment effects as same-sex DZ twins do. (This assumption is independent of the significant mean differences between boys and girls as indicated above.)

We next tested the scalar model, which allows phenotypic variances between boys and girls to differ but equates A–C–E parameters for boys and girls and constrains r_go to .50. At ages 2, 3, and 7, this model proved to worsen the fit (age 2: $\chi^2[2, N = 5681] = 9.33$; age 3: $\chi^2[2, N = 5639] = 8.67$; both ps $< .05$; age 7: $\chi^2[2, N = 7430] = 15.99$, $p < .01$), and was therefore rejected. In contrast, at age 4 the scalar model did not fare worse than the

common effects model, $\chi^2(2.\ N = 6271) = 1.51$, *ns*. Therefore, it cannot be assumed that variance component estimates at ages 2, 3, and 7 (but not at age 4) are identical for boys and girls.

The variances of boys' and girls' prosocial behavior could be equated at age 2, as revealed by the comparison with the scalar model, $\chi^2(1, N = 5681) = 0.00$, *ns*. However, the variances at later ages could not be equated (age 3: $\chi^2[1, N = 5639] = 11.06$; age 4: $\chi^2[1, N = 7430] = 14.95$; age 7: $\chi^2[1, N = 6271] = 126.74$; all $ps < .01$). However, it can be seen from Table 1 that differences in standard deviations between boys and girls are small (age 3: 3.65 vs. 3.54; age 4: 2.64 vs. 2.56; age 7: 1.85 vs. 1.58, respectively) and detected as significant because of the statistical power of our large sample.

Estimates of variance components. Table 3 presents the A, C, and E components of variance in prosocial behavior at ages 2, 3, 4, and 7. Results are presented separately for boys and for girls in order to allow comparison across the years, although estimates at age 4 did not differ significantly across the sexes, as noted. The common effects model fit the data well at age 2, $\chi^2[9, N = 5681] = 8.44$, *ns*; Akaike's information criterion (AIC) = −9.56; root–mean-square error of approximation (RMSEA) = .00. A satisfactory fit was also obtained at age 3, $\chi^2(9, N = 5639) = 11.14$, *ns*, (AIC = −6.86, RMSEA = .003); at age 4, $\chi^2(9, N = 7430) = 19.77$, $p < .05$, (AIC = 1.77, RMSEA = .012); and at age 7 for parent ratings, $\chi^2(9, N = 6271) = 22.96$, $p < .01$, (AIC = 4.96, RMSEA = .018).

Age 2 showed moderate and significant heritability for both girls and boys. Shared environmental influences were found to be more substantial accounting for about half of the variance. Finally, nonshared environment plus error of measurement accounted for at least 20% of the variance for both sexes.

Age 3 showed an increase (though a nonsignificant one) in heritability for both sexes. For boys, this was accompanied by a sharp drop from 43% to 23% in the influence of shared environmental factors. This is a significant drop, as judged by the nonoverlap of confidence intervals. For girls, shared environmental influence also declined from 50% to 38%, but the difference was not significant. Finally, the effect of nonshared environment increased significantly from age 2 to age 3.

Age 4 showed an increasingly high heritability, with heritability significantly higher for girls at age 4 (51%) than at age 2 (26%) and a similar significant difference for boys (age 4: 52%; age 2: 37%). Shared environment effects showed a sharp, significant drop for both sexes from age 3 to age 4. The increase in the impact of nonshared environment continued at this age, with a significant increase from 30% at age 3 to 40% at age 4 for boys and from 31% at age 3 to 38% at age 4 for girls.

Age 7 was characterized by the highest heritability of 60–62%, though heritability was not significantly higher than at age 4. Shared environment effects were the lowest in this age and were no longer significant (0–6%). As was the case since age 3, nonshared environment accounted for at least 30% of the variance, though the increased heritability was manifest at this age with a significantly reduced nonshared environment effect for boys. At age 7, nonshared environment effects were slightly but significantly lower for boys than for girls (31% vs. 39%).

Table 3
Estimates of Variance Components (and 95% Confidence Intervals) at Ages 2, 3, 4, and 7 (Cross-Sectional Analyses)

Age and informant	Heritability	Shared environment	Nonshared environment
	Boys		
Age 2	.37 (.31–.43)	.43 (.38–.49)	.20 (.18–.22)
Age 3	.47 (.40–.54)	.23 (.17–.29)	.30 (.27–.33)
Age 4	.52 (.44–.59)	.08 (.03–.14)	.40 (.37–.44)
Age 7 (parent report)	.62 (.51–.71)	.06 (.00–.16)	.31 (.29–.34)
Age 7 (teacher report)	.72 (.66–.74)	.00 (.00–.05)	.28 (.26–.31)
	Girls		
Age 2	.26 (.19–.34)	.50 (.43–.57)	.24 (.22–.26)
Age 3	.30 (.22–.39)	.38 (.30–.46)	.31 (.29–.34)
Age 4	.51 (.42–.59)	.11 (.04–.18)	.38 (.36–.42)
Age 7 (parent report)	.60 (.55–.64)	.00 (.00–.04)	.39 (.36–.43)
Age 7 (teacher report)	.51 (.39–.62)	.17 (.06–.27)	.32 (.29–.35)

Note. Analyses were performed separately at each age. Numbers in parentheses indicate confidence intervals. A report on results for the entire Strengths and Difficulties Questionnaire on a partial sample included some of the 7-years data (Saudino, Ronald, & Plomin, 2003).

Analysis of Genetic and Environmental Effects on Teacher-Rated Prosocial Behavior

Teacher reports at age 7 could be used to corroborate our findings of decreasing shared environmental influence and increased genetic influence obtained with parent reports. Twin correlations on Prosocial Behavior scores at age 7, as reported by teachers, were higher for MZ twins (MZM = .73; MZF = .68) than for DZ twins (DZM = .30; DZF = .43; DZO = .30), indicating substantial genetic influence on prosocial behavior. The relatively high DZF correlation indicates shared environment effects for girls.

As with parent reports, the common effects model fared better than other models, resulting in a good fit, $\chi^2(9, N = 5064) = 6.98$, *ns*, (AIC = −11.02, RMSEA = .001). Constraining r_go to be equal to .50 for DZO twins did not worsen model fit, but equating ACE parameters for boys and girls (the scalar model) resulted in worse model fit, $\chi^2(2, N = 5064) = 22.14$, $p < .01$. A model equating boys' and girls' variances was rejected, $\chi^2(1, N = 5064) = 87.53$, $p < .01$.

Teacher reports showed high heritability levels, not significantly different than those obtained with parent reports. Heritability for boys was .72, and for girls it was .51 (see Table 3). These estimates support the results of high heritability at age 7 obtained with parent reports, although girls' heritability at age 7 as indicated by teacher report was not higher than that estimated at age 4 with parent reports. Similarly, as with parent reports, the degree of nonshared environment was substantial at this age with teacher reports. The only difference between parent and teacher reports was that shared environment effects for girls were significant, although modest (teacher reports: .17), whereas there was no shared environment effect detected by parent reports. For boys, shared environment at age 7 was estimated at .00 with teacher reports, which was similar to parent reports.

Longitudinal Continuity and Change in Parent-Rated Prosocial Behavior

Table 2 also presents the longitudinal correlations for individual scores on Prosocial Behavior separately for each zygosity group. The 1-year (ages 2–3 and ages 3–4) longitudinal correlations were in the range of .50 to .58. Although this degree of continuity is substantial, considerable individual change occurs from year to year. The 2-year (ages 2–4) within-twin longitudinal correlations are naturally lower, ranging from .30 to .42. The 3-year (ages 4–7) within-twin longitudinal correlations are similar in size, ranging from .32 to .40. The correlations from age 2 to age 7 are the lowest but still represent some stability, ranging from .13 to .21.

As expected, the within-twin longitudinal correlations of MZ twins and DZ twins were similar: The average 1-year correlation for MZ twins was .56, and for DZ twins it was .55. Similarly, the 5-year correlation averaged .18 for MZ and .17 for DZ twins. However, if genes contribute to stability in prosocial behavior, then the cross-twin longitudinal correlations for MZ twins should be higher than those for DZ twins. As would be expected by a genetic model of continuity, the average 1-year cross-twin correlation for MZ twins (.48) was substantially higher than that for DZ twins (.33). The MZ cross-twin correlation of .48 indicates that MZ twins are nearly as similar to their cotwins 1 year later as they are to themselves (.56). Below we use longitudinal model fitting to estimate genetic and environmental parameters of change and stability.

Longitudinal Analyses of Change and Continuity in Genetic and Environmental Effects on Parent-Rated Prosocial Behavior

Within-twin and between-twin variance–covariance matrices of prosocial behavior scores across ages 2, 3, 4, and 7 were next analyzed by using Cholesky decomposition (see *Method* section). This analysis included 2,901 pairs for which full data at all ages were available. At age 7, parent reports were used. As with the cross-sectional case, it was possible to constrain r_go to .50 for DZO twins without worsening model fit. The common effects model fit the data well, $\chi^2(119, N = 2901) = 160.86$, $p < .01$, (AIC = −77.14, RMSEA = .024). To increase model parsimony, paths whose coefficients were estimated as .00 were dropped. These were the shared environment effects from ages 3 and 4 to age 7 and the nonshared environment effects from ages 2 and 3 to age 7. Dropping these paths did not significantly affect fit, $\chi^2(8, N = 2901) = 12.80$, *ns*. However, applying the scalar model worsened model fit, $\chi^2(26, N = 2901) = 140.26$, $p < .01$. Therefore, separate results are presented for girls and boys.

Figures 2 and 3 present the results from the Cholesky decomposition for boys and girls, respectively. To simplify the figures, the genetic, shared environment, and nonshared environment components are presented in Panels A, B, and C, respectively. It should be noted, though, that the three parts of both figures represent a single analysis. The figures also provide 95% confidence intervals for all the variance component estimates we report below.

Allowing for rounding error, the squared paths shown leading to the score on Prosocial Behavior at each age, summed across the A, C, and E components, account for 100% of the variance. For example, for boys' prosocial behavior at age 2 (Figure 2), the A, C and E parameter estimates are .38, .44, and .18, respectively, which sum to 1.00. In addition, the sum of all the squared genetic paths shown leading to the score on Prosocial Behavior at each age is roughly equal to the genetic influence derived above from cross-sectional analyses. For example, the above estimate of .47 for A for boys at 3 years (Table 3) corresponds to the sum of the squares of the paths leading to scores at age 3 and from the genetic factor at ages 2 (.17) and 3 (.24). The same is true for shared environment and nonshared environment estimates. The numbers in the figures are not identical to those reported in Table 3 because the longitudinal analysis used only twin pairs for whom data were available at all ages.

Genetic effects. As seen in Panel A of Figures 2 and 3, respectively, the genetic effects at age 2 were carried on, at least partially, to later ages. For boys, the genetic effects of .38 at age 2 years dropped in importance to .17 at age 3, to .13 at age 4, and to .08 at age 7. Thus, genetics accounted for some of the stability in children's prosocial behavior. However, genetics also accounted for change. The data suggest that the increase in heritability noted above comes from new genetic effects. The new genetic effect at age 3 accounted for 24% of the variance at age 3 and carried on to explain 15% of the variance at age 4 and 13% at age 7. The new genetic effect at age 4 (16%) contributed significantly at age 7 (4%). At age 7, there is evidence for further new genetic effects,

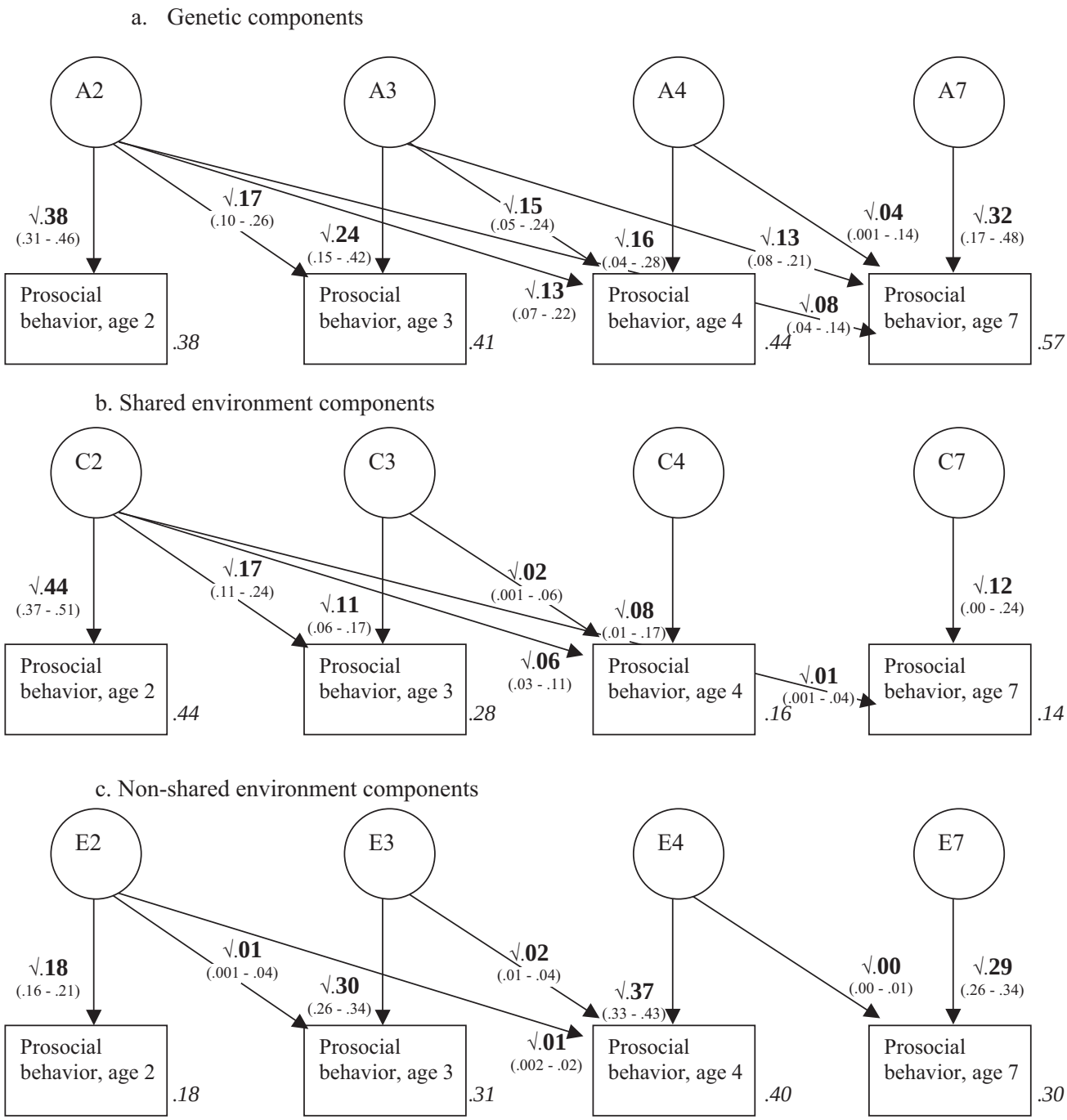

Figure 2. Cholesky decomposition of variance components of prosocial behavior at ages 2, 3, 4, and 7 (boys). Circles indicate variance components estimates, and rectangles indicate observed scores on prosocial behavior. A = heritability; C = shared environment; E = nonshared environment (and error). The number in each circle represents the age to which the variance component is attributed. Numbers in parentheses are 95% confidence intervals. Numbers beside the squares represent the percentage of variance accounted for by the variance component at that age.

possibly the result of accumulation of genetic influences in the 3 years elapsing from age 4 to age 7. This effect accounted for 32% of the variance, more than half of the genetic influence at age 7.

The longitudinal analysis for girls also showed both continuity and change of genetic effects. A modest genetic effect from age 2 was carried over to age 3 and then to age 4. At age 3 there was some (11%) additional genetic effect, which had further influence at age 4 (19%), and accounted for 28% of the variance at age 7. There was evidence for new genetic effects at age 7 (14%), which accounted for the increase in heritability at this age as described above.

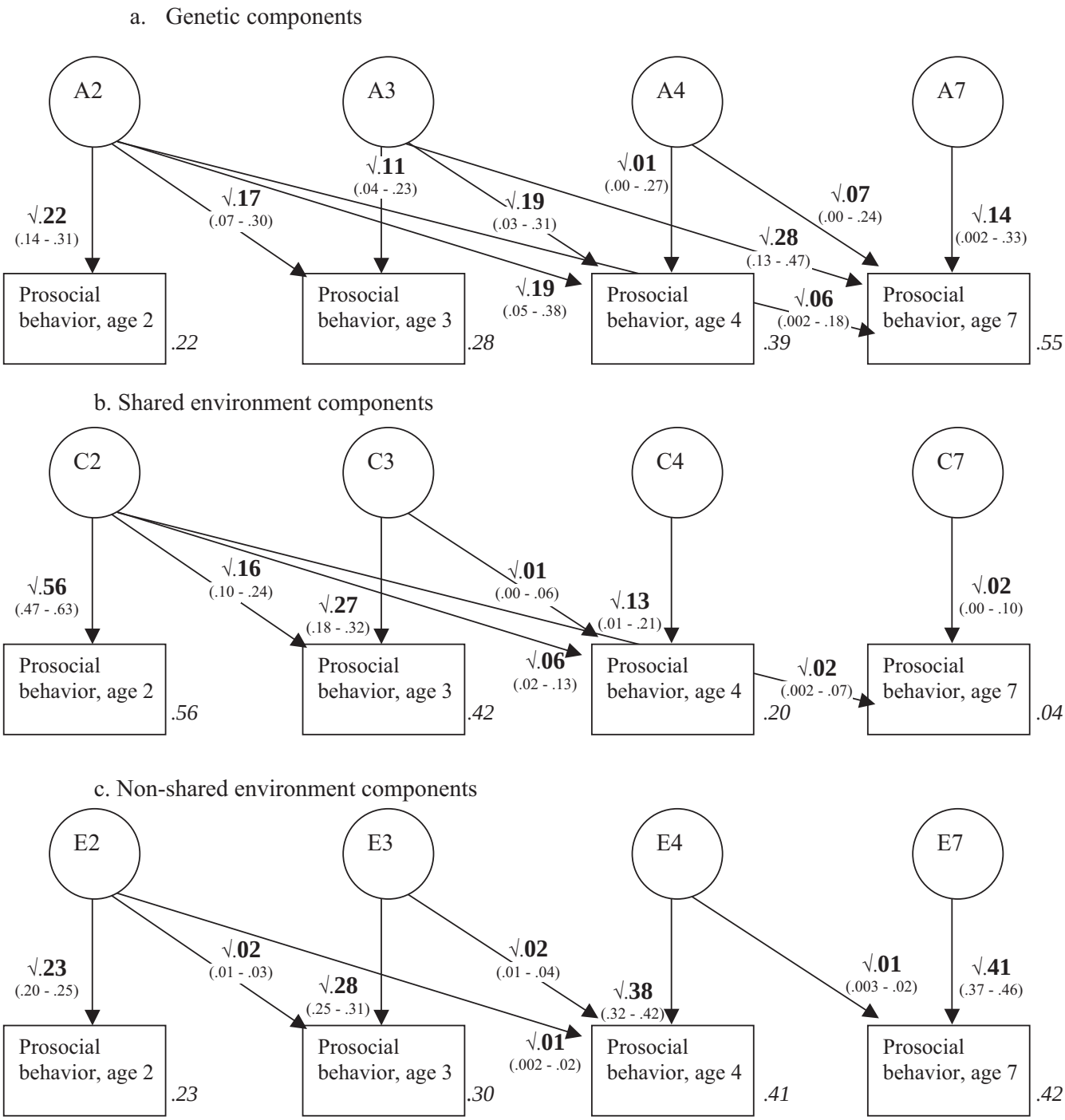

Figure 3. Cholesky decomposition of variance components of prosocial behavior at ages 2, 3, 4, and 7 (girls). Circles indicate variance components estimates, and rectangles indicate observed scores on prosocial behavior. A = heritability; C = shared environment; E = nonshared environment (and error). The number in each circle represents the age to which the variance component is attributed. Numbers in parentheses are 95% confidence intervals. Numbers beside the squares represent the percentage of variance accounted for by the variance component at that age.

Shared environmental effects. There was a substantial effect of shared environment at age 2 for boys (.44) and for girls (.56). This effect was carried over but waned in importance at later years, decreasing to .17 (.16) at age 3, .06 at age 4, and to .01 (.02) at age 7. For both sexes, there was evidence for new effects at age 3 (boys: .11; girls: .27) and at age 4 (boys: .08; girls: .13). These effects were increasingly small in size, and at age 7 no new significant effects were found. Thus, shared environment accounted for both change and stability, but both these effects became weaker as the children grew up.

Nonshared environmental effects. For both boys and girls, a similar pattern was found. Significant, new nonshared environmental (and error) effects were found at each age, generally increasing in size, with minimal (yet significant, considering the study's sample size) carry-over from age 2 to age 3 and then to age 4. Nonshared environment effects (in addition to the effects of measurement error) were responsible, therefore, mainly for change.

Discussion

This large twin study of prosocial behavior contributes importantly to understanding the origins of individual differences in prosocial development. A consistent pattern of results emerged, in which genetics and nonshared environment effects become increasingly important and shared environment effects decreased in importance as children grew up and moved from early childhood to middle childhood. Results from teacher reports at age 7 largely supported this conclusion drawn from parent reports at age 2, 3, 4 and 7. Genetics, and, to a lesser extent, shared environment accounted for both change and stability. Nonshared environment accounted for a substantial proportion of the variance and was largely responsible for change. Although at most ages it was not possible to fully equate genetic and environmental parameter estimates for boys and girls, the results for the two sexes are strikingly similar, except for the replication of the usual finding of a mean difference favoring girls (Eisenberg & Fabes, 1998; Warden & Mackinnon, 2003).

Heritability

That genetic effects account for a significant proportion of prosocial behavior comes as no surprise, as heritability explains a significant proportion of individual differences in many other traits (Plomin et al., 2001). Indeed, of all the studies of prosocial behavior we reviewed above only one (Krueger et al., 2001) found no significant genetic effects. This may have been due to a different operationalization of prosocial behavior.

The steady increase in heritability from 26–37% at age 2 to 51–72% at age 7 resembles findings for other traits, particularly cognitive abilities (McGue et al., 1993). This result may seem counterintuitive, as individuals' genes (i.e., the sequence of nucleotide bases of DNA, not the expression of genes) are present at conception, whereas their families and other socialization agents exert continuous influence on them, which might have steadily reduced genetic effects. However, children are not passive agents in the socialization process (Kuczynski, Marshall, & Schell, 1997). The influence of mothers on their children depends on the children's temperament (Kochanska, 1995). Such evocative and active gene–environment correlations, in which children's genes operate through their behavior on the environment they receive (Plomin, DeFries, & Loehlin, 1977), are more likely as children grow up (Scarr & McCartney, 1983). With the design of the current study, such gene–environment correlations end up as part of the heritability estimate and may explain some of the increasingly large genetic effect across ages.

In another report from the same sample studied here, we partially addressed the issue of gene–environment correlations (Knafo & Plomin, 2006). At ages 3, 4, and 7, parents' positivity and negativity in discipline and affection correlated moderately with children's prosocial behavior (parent reports: average *r*s = .21 and −.20, respectively; teacher reports: *r*s = .08 and −.13, respectively). We investigated genetic and environmental contributions to these relationships. Genetics, shared environment, and nonshared environment all mediated the correlations between parenting and prosocial behavior to some extent, but their relative contributions varied by age, sex, and parenting variable. For example, regarding prosocial behavior as rated by parents, genetic mediation accounted for 10% to 68% of the phenotypic correlation between parenting and prosocial behavior. Shared environment effects accounted for 0% to 78% of the correlations, and nonshared environment effects accounted for 11% to 36%. Genetic factors mainly mediated the negative correlation between prosocial behavior and parental negativity, and shared environmental effects contributed mainly to the positive relationship between prosocial behavior and parental positivity (Knafo & Plomin, 2006). Thus, gene–environment correlations account for some of the relationship between parenting and prosocial behavior, but because the relationships with the parenting variables available were moderate, their contribution to overall individual differences in prosocial behavior is modest (Knafo & Plomin, 2006). Clearly, behavioral genetic studies of prosocial behavior would benefit from incorporating more information on the socialization practices of parents.

Another question arising from the results concerns the role of genes in stability and change. As genes are present at birth, it is not surprising that they contribute to stability. However, genes also contribute to change (Plomin et al., 1993). For boys at age 7, most of the genetic effect (32% out of 57%) was due to new influences not present at age 4. For girls too, a substantial part of the genetic effect at age 7 was new (14% of 55%). What are these new genetic effects? One set of plausible candidates are genes influencing cognitive abilities because the ability to take the perspective of others and the ability to consider the benefit of others rather than hedonistic views are relevant to change in prosocial behavior (Eisenberg & Fabes, 1998). We are currently investigating the role of cognitive development in conjunction with prosocial behavior within a genetically informative design. Because we have twin data on both cognitive abilities and prosocial behavior at different ages, it is possible to investigate the contributions of genetics (or environment) in one domain to the development in the other domain in a later age. For example, if the genetic factor of cognitive abilities at age 2 predicts prosocial behavior at age 3 over and above the genetic factor of prosocial behavior at age 2, then this would indicate that genetic effects on abilities account for prosocial development.

Although the evidence for genetic effects on children's prosocial behavior is strong, especially at age 7, progress in identifying the specific genes responsible for such genetic effects has been slower than expected (Plomin, DeFries, Craig, & McGuffin, 2003). It is generally accepted that this slow progress is due to the likelihood that many more genes of much smaller effect than expected are responsible for genetic influence on complex traits (Cardon & Bell, 2001), consistent with the idea of quantitative trait loci (Plomin, Owen, & McGuffin, 1994).

One molecular genetic study of prosocial behavior has been reported (Comings et al., 2000). The results suggested that 17 of 59 genes examined were associated with individual differences in cooperativeness in a sample of 204 men, each accounting for less than 5% of the variance. Because molecular genetic results typically involve small effects, and are often not replicated in subse-

quent studies (Cardon & Bell, 2001), more research is needed to reach any conclusions regarding the genes affecting prosocial behavior. In addition, the results of the current study suggest that new genetic effects emerge as children grow up, therefore some of the true effects found for adults may not replicate with the prosocial behavior of children.

Shared Environment

The fact that adult studies show little shared environment influence on prosocial behavior (e.g., Rushton et al., 1986), whereas studies with children do show moderate to large shared environment effects (e.g., Lemery & Goldsmith, 2002), may be explained by our findings. Shared environment effects steadily drop in magnitude as children grow up. At age 7 they tend to be negligible. This again is in line with research on other traits, showing a decrease in shared environment effects, especially for cognitive abilities (Plomin et al., 2001). There is evidence that shared environment effects contribute to both stability and change, but new effects are increasingly weaker.

One reason for the reduction in shared environment is that as children grow up they come to meet increasingly different social environments. For example, in 1998, 79% of 4-year-olds but only 35% of 3-year-olds in England attended daycare (Great Britain Department for Education and Employment, 1998). Whereas for a 2-year-old most of the peer interaction can be with the cotwin or other siblings, by age 4, and even more so at age 7, siblings encounter other children and do not fully share their cotwin's social networks. This increase in children's variety of social environments may be one reason for the decline in shared environment effects. Moreover, parents may find it relatively easy to influence their children's prosocial behavior in the context of the home, as demonstrated by the large shared environment effects found for twin cooperation in early childhood (e.g., Lemery & Goldsmith, 2002). This may be the reason for the especially high shared environment effect at age 2. However, as children grow up, this prosocial behavior is not limited to their cotwins but can occur with any other child. Moreover, parental influence may become weaker.

Only in one case did we find a shared environment effect at age 7. Using teacher reports, we estimated this effect at .17 for girls and at .00 for boys. Results from teacher and parent reports were essentially identical for boys and quite similar for girls too (substantial heritability, substantial nonshared environment effect, and lower shared environment effect). However, the case of teacher reports on girls demonstrates the importance of studying genetic and environmental effects in different contexts (a similar finding, of shared environment effects with teacher reports but not with parent reports, was reported by Scourfield et al., 2004, without distinguishing between girls and boys). It is possible that teachers are more sensitive than are parents to some aspects of prosocial behavior and that their reports detect influences common to both twins. It is also possible that children's behavioral tendencies are different at school and at home and that shared environmental effects affect girls' behaviors more at school than at home. An alternative explanation is that in the case of girls the shared environment effect actually represents effects of the school that are shared between both twins. That this occurs only for girls might be attributed to the stronger socialization pressures toward nurturance and communion that are exerted on girls (e.g., Maccoby, 1998), as might also be the case with girls' higher shared environment effect at age 3 as rated by parents.

A future direction for research is to begin to identify factors that affect siblings' prosocial behavior similarly in infancy but become less important as shared environmental influences wane in early childhood. The decrease in shared environment effects may indicate that the finding of parental characteristics relating to children's prosocial behavior (e.g., Fabes et al., 1989; Krevans & Gibbs, 1996) reflects in part a correlation between children's genetically based characteristics and their parents' behavior (Eisenberg & Fabes, 1998). This notion points to the importance of child characteristics in the development of prosocial behavior. Because any two siblings who are not identical twins have different genetic propensities, they affect their parents differently than do MZ twins, create their own family niches, and therefore develop differently (Plomin, 1994).

Nonshared Environment

The results suggest a consistent pattern of developmental increases in nonshared environment effects on children's prosocial behavior. The increase in nonshared environment effects from age 2 to age 4 might be attributed to increase in measurement error, for the E estimate, by definition, includes an error component. However, the longitudinal correlations are not affected by the presumed increasing measurement error. If error had increased throughout the years, longitudinal correlations would have become smaller as children grew up. In the current study, the average correlation from age 2 to age 3 is identical to the one from age 3 to age 4 (.55). Moreover, the 2-year average correlation from age 2 to age 4 (.38) is roughly equal to the average 3-year correlation from age 4 to age 7 (.36), despite the new measure of prosocial behavior used at age 7 (Table 2). Increase in error, therefore, is unlikely to account for the increase in nonshared environment estimates.

The sources for this increase in nonshared environment effects are likely to be found, as noted above, in the increasingly dissimilar environments children encounter as they grow up and enter new social environments such as daycare. In addition, differential exposure to other socialization agents such as the media and various life events could affect children's prosocial behavior and make them increasingly dissimilar from each other.

It should be noted that even on children as young as age 2 nonshared environment has substantial influence. Thus, even when children are very young, and under the influence of their parents to a large degree, they are not rated as identical in their prosocial behavior by their parents. In addition to reflecting measurement error, the fact that identical twins do not correlate 1.00, points to the importance of the environment in shaping prosocial behavior.

The longitudinal analysis indicates that nonshared environment introduced new effects in every age. This pattern of nonshared environment, accounting mainly for change, has been found with other traits (Plomin et al., 2001). Because children's social networks change in time, and because extrafamilial influences, such as those of the media, may also change rapidly, the nonshared environment effects found in one age do not account for most of the effects in other ages. However, there were some small yet significant longitudinal nonshared environment effects in the ages of 2, 3, and 4 (Figures 2–3). These effects accounted for 1–2% of the variance and might reflect stable differences in the environ-

ments children encounter, such as parental differential treatment (cf. Caspi et al., 2004).

A prior report from TEDS sheds light on some of the nonshared environmental effects on children's prosocial behavior. Asbury, Dunn, Pike, and Plomin (2003) studied differences in prosocial behavior (and other behaviors) within MZ twin pairs at age 4. Because MZ twins are genetically identical, differences between them cannot be explained by genetics and indicate nonshared environmental influences (and error). Twins who received more harsh parental discipline and more negative parental feelings were less likely than their cotwin to engage in prosocial behavior (Asbury et al., 2003). It could be argued that identical twins' nongenetic differences in behavior account for parental differences in parenting rather than the other way around. However, longitudinal analyses show that parental differential treatment has an effect on children over and above their own behavioral differences, at least when it comes to children's antisocial behavior (Caspi et al., 2004). These results point to the importance of parenting, in this case, as a nonshared environment factor rather than a shared environment factor.

Strengths, Limitations, and Future Directions

The methodological strengths of this study include a very large community sample of children. The sample size provided power to detect effects as small as the longitudinal nonshared environment effects that accounted for only 1–2% of the variance (and whose practical importance might be small). The time span of this study, with children assessed near their 2nd, 3rd, 4th, and 7th birthdays, enabled us (a) to follow the development of prosocial behavior at four ages in an important period in which many social and cognitive changes occur and (b) to investigate the sources of change and continuity as children move from early childhood to middle childhood.

The use of parent ratings is a limitation. Because the same parent rated both twins, an increase in between-twin similarity might occur for both MZ and DZ twins, resulting in an inflated shared environment estimate. This did not happen in this case, as shared environment effects were not very large beyond age 3. Other biases resulting from the use of parent reports are also possible, such as twin contrast in parent reports or an inflated report of MZ similarity as compared with DZ similarity. However, we used teacher data to increase the validity of the findings, and at age 7 the pattern of strong heritability, substantial nonshared environment, and low shared environment was replicated with teacher data. This attests to the robustness of the findings but also demonstrates the importance of studying behavior in different contexts, as the results for girls were not identical for teacher and parent reports.

Another indication of the robustness of the findings is that limiting the longitudinal analyses to children with complete data yielded estimates of variance components in the different ages that were very similar to the estimates obtained with the full sample at each separate age.

The use of different scales to measure prosocial behavior at different ages makes it difficult to apply alternative longitudinal models such as latent growth modeling (Finkel, Reynolds, McArdle, Gatz, & Pedersen, 2003; McArdle & Hamagami, 2003). The meaning of age-to-age change is difficult to interpret when different measures are used at different ages. Thus, the advantages of latent growth modeling could not be achieved in this study.

The advantage of the use of parent and teacher reports is in the accumulated knowledge parents and teachers have of their children. However, although parent and teacher reports yield similar results, they are both limited as measures of prosocial behavior because they tap behaviors that adults can observe and remember and that they are willing to report. This is manifest in the modest correlation between parent and teacher reports. More objective measures such as observation or experimental manipulation are important, but their validity is also limited and the typical correlation between any two experimental measures of prosocial behavior rarely exceeds .30 (e.g., Rushton, 1984). Another disadvantage of observations or experimental manipulations is the huge costs associated with conducting them with the large sample size required for twin studies. Nevertheless, it would of course be desirable, when possible, to collect multivariate data, by using observations, experimental manipulations, parent, teacher, and self-reports.

The finding of increased heritability and reduced shared environment effects throughout early to middle childhood corresponds with past studies indicating heritability and no shared environment effects in adulthood (e.g., Rushton et al., 1986). However, more research on adolescents is needed before we can conclude that the situation remains stable from age 7 to adulthood. Genetics and the environment contribute to change, which means that later in life new genetic or environmental effects may emerge. As children enter adolescence, they are likely to undergo an identity formation process, in which they will reevaluate their values, attitudes, and behaviors (e.g., Knafo & Schwartz, 2004; Waterman, 1999). Because moral development is important to prosocial behavior (Miller, Eisenberg, Fabes, & Shell, 1996), and because in adulthood prosocial behavior is often anchored in prosocial values (Knafo & Sagiv, 2004; Schwartz, 1996), a further shift in the pattern of genetic and environmental influences on prosocial behavior may occur in adolescence. We are planning a study in which early adolescents' values would be assessed in order to investigate the development of prosocial behavior in conjunction with values development through the use of a longitudinal genetic design.

The use of twin data may also be a limitation. It is possible that because twins grow up together, the prosocial behavior in a pair is more positively correlated, as mutual support and cooperation, or the lack of these, can affect the similarity of twin pairs' prosocial behavior. If this special twin environment effect had occurred in our study, the result would have been an increased estimate for shared environment, whereas we found little evidence for shared environment after age 3. Nevertheless, in order to overcome this potential problem, we are also currently studying younger singleton siblings of twins for future analyses.

Finally, heritability estimates obtained at different ages, four in this study, shed light on important developmental processes. Similarly, longitudinal effects obtained from the longitudinal genetic analyses provide a compelling case for the role of genetics and the environment in the development of prosocial behavior. As Greenberg (2005) and Partridge (2005) have argued, knowing the heritability estimate for a trait provides only a partial answer to the question of how individuals develop and change. It is also important to pinpoint the processes underlying genetic changes. For example, studies involving DNA data and a repeated assessment of environmental causes are needed to more fully understand

genetic–environmental interactions (e.g., epigenetic processes) and correlations (e.g., child–parent reciprocal behavioral influences). In a separate report, we have provided an example of a longitudinal investigation of mutual child–parent influences within a genetic design (Knafo & Plomin, 2006).

Conclusions

This study addressed the development of prosocial behavior in children from late infancy to middle childhood. The evidence for the importance of both genetics and environment is strong, although the size of their effects varied and the nature of environmental effects gradually changed from those shared by twins to those not shared by them. The contributions of genetics and the environment to change and stability in prosocial behavior demonstrate the importance of longitudinal, genetically informed studies to enhance our understanding of the development of positive human behavior.

References

Asbury, K., Dunn, J. F., Pike, A., & Plomin, R. (2003). Nonshared environmental influences on individual differences in early behavioral development: A monozygotic twin differences study. *Child Development, 74,* 933–943.

Becker, A., Woerner, W., Hasselhorn, M., Banaschewski, T., & Rothenberger, A. (2004). Validation of the parent and teacher SDQ in a clinical sample. *European Child and Adolescent Psychiatry, 13*(Suppl.2), II11–II16.

Behar, L., & Stringfield, S. (1974). A behavior rating scale for the preschool child. *Developmental Psychology, 33,* 3–66.

Calvert, S. L., & Kotler, J. A. (2003). The Children's Television Act: Can media policy make a difference? *Journal of Applied Developmental Psychology, 24,* 375–380.

Cardon, L. R., & Bell, J. (2001). Association study designs for complex diseases. *Nature Genetics, 2,* 91–99.

Caspi, A., Moffitt, T. E., Morgan, J., Rutter, M., Taylor, A., Arseneault, L., et al. (2004). Maternal expressed emotion predicts children's antisocial behavior problems: Using monozygotic-twin differences to identify environmental effects on behavioral development. *Developmental Psychology, 40,* 149–161.

Clark, K. E., & Ladd, G. W. (2000). Connectedness and autonomy support in parent–child relationships: Links to children's socioemotional orientation and peer relationships. *Developmental Psychology, 36,* 485–498.

Cole, C. F., Arafat, C., Tidhar, C., Tafesh, W. Z., Fox, N. A., Killen, M., et al. (2003). The educational impact of Rechov Sumsum/Shara'a Simsim: A Sesame Street television series to promote respect and understanding among children living in Israel, the West Bank, and Gaza. *International Journal of Behavioral Development, 27,* 409–422.

Comings, D., Gade-Andavolu, R., Gonzalez, N., Wu, S., Muhleman, D., Blake, H., et al. (2000). A multivariate analysis of 59 candidate genes in personality traits: The temperament and character inventory. *Clinical Genetics, 58,* 375–385.

Davis, M. H., Luce, C., & Kraus, S. J. (1994). The heritability of characteristics associated with dispositional empathy. *Journal of Personality, 62,* 369–391.

Deater-Deckard, K., Dunn, J., O'Connor, T. G., Davies, L., & Golding, J. (2001). Using the stepfamily genetic design to examine gene-environment processes in child and family functioning. *Marriage and Family Review, 33,* 131–156.

Eisenberg, N., & Fabes, R. A. (1998). Prosocial development. In N. Eisenberg & W. Damon (Eds.), *Handbook of Child Psychology, Vol. 4: Social, emotional and personality development* (5th ed., pp. 701–778). New York: Wiley.

Fabes, R. A., Fultz, J., Eisenberg, N., & May-Plumlee, T. (1989). Effects of rewards on children's prosocial motivation: A socialization study. *Developmental Psychology, 25,* 509–515.

Fehr, E., & Fischbacher, U. (2003, October 23). The nature of human altruism. *Nature, 425,* 785–791.

Finkel, D., Reynolds, C. A., McArdle, J. J., Gatz, M., & Pedersen, N. L. (2003). Latent growth curve analyses of accelerating decline in cognitive abilities in late adulthood. *Developmental Psychology, 39,* 535–550.

Gillespie, N. A., Cloninger, C. R., Heath, A. C., & Martin, N. G. (2003). The genetic and environmental relationship between Cloninger's dimensions of temperament and character. *Personality and Individual Differences, 35,* 1931–1946.

Goodman, R. (1994). A modified version of the Rutter Parent Questionnaire including extra items on children's strengths: A research note. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35,* 1483–1494.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology and Psychiatry, 38,* 581–586.

Great Britain Department for Education and Employment. (1998). Pupils under 5 years of age in schools in England: January 1998. *Statistical Bulletin, 10/98.*

Greenberg, G. (2005). The limitations with behavior-genetic analyses, *Developmental Psychology, 41,* 989–992.

Grusec, J. E., Davidov, M., & Lundell, L. (2002). Prosocial and helping behavior. In P. K. Smith & C. H. Craig (Eds.), *Blackwell handbook of childhood social development* (pp. 457–474). Malden, MA: Blackwell Publishers.

Grusec, J. E., Goodnow, J. J., & Cohen, L. (1996). Household work and the development of concern for others. *Developmental Psychology, 32,* 999–1007.

Hogg, C., Rutter, M., & Richman, N. (1997). Emotional and behavioural problems in children. In I. Insclare (Ed.), *Child psychology portfolio.* Windsor, UK: NFER-Nelson.

Knafo, A., & Plomin, R. (2006). Parental discipline and affection and children's prosocial behavior: Genetic and environmental links. *Journal of Personality and Social Psychology, 90,* 147–164.

Knafo, A., & Sagiv, L. (2004). Values and work environment: Mapping 32 occupations. *European Journal of Psychology of Education, 19,* 255–273.

Knafo, A., & Schwartz, S. H. (2004). Identity status and parent–child value congruence in adolescence. *British Journal of Developmental Psychology, 22,* 439–458.

Kochanska, G. (1995). Children's temperament, mothers' discipline, and security of attachment: Multiple pathways to emerging internalization. *Child Development, 66,* 597–615.

Krevans, J., & Gibbs, J. C. (1996). Parents' use of inductive discipline: Relations to children's empathy and prosocial behavior. *Child Development, 67,* 3263–3277.

Krueger, R. F., Hicks, B. M., & McGue, M. (2001). Altruism and antisocial behavior: Independent tendencies, unique personality correlates, distinct etiologies. *Psychological Science, 12,* 397–402.

Kuczynski, L., Marshall, S., & Schell, K. (1997). Value socialization in a bidirectional context. In J. E. Grusec & L. Kuczynski (Eds.), *Parenting and the internalization of values: A handbook of contemporary theory* (pp. 23–50). New York: Wiley.

Lemery, K. S., & Goldsmith, H. H. (2002). Genetic and environmental influences on preschool sibling cooperation and conflict: Associations with difficult temperament and parenting style. *Marriage and Family Review, 33,* 77–99.

Maccoby. E. (1998). *The two sexes: Growing up apart, coming together.* Cambridge, MA: Belknap Press.

Mathai, J., Anderson, P., & Bourne, A. (2004). Comparing psychiatric diagnoses generated by the Strengths and Difficulties Questionnaire with diagnoses made by clinicians. *Australian and New Zealand Journal of Psychiatry, 38,* 639–664.

Matthews, K. A., Batson, C. D., Horn, J., & Rosenman, R. H. (1981). "Principles in his nature which interest him in the fortune of others . . .":

The heritability of empathic concern for others. *Journal of Personality, 49,* 237–247.

McArdle, J. J., & Hamagami, F. (2003). Structural equation models for evaluating dynamic concepts within longitudinal twin analyses. *Behavior Genetics, 33,* 137–159.

McGue, M., Bouchard, T. J., Jr., Iacono, W. G., & Lykken, D. T. (1993). Behavioral genetics of cognitive ability: A life-span perspective. In R. Plomin & G. E. McClearn (Eds.), *Nature, nurture, and psychology* (pp. 59–76). Washington, DC: American Psychological Association.

Miller, P. A., Eisenberg, N., Fabes, R. A., & Shell, R. (1996). Relations of moral reasoning and vicarious emotion to young children's prosocial behavior toward peers and adults. *Developmental Psychology, 32,* 210–219.

Neale, M. C., Boker, S. M., Xie, G., & Maes, H. (1999). *Mx: Statistical Modeling.* (5th ed.) Richmond, VA: Virginia Commonwealth University, Department of Psychiatry.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families.* Dordrecht, the Netherlands: Kluwer/Academic.

Partridge, T. (2005). Are genetically informed designs genetically informative: A commentary on quantitative behavioral genetics. *Developmental Psychology, 41,* 985–988.

Pearl, R. (1985). Children's understanding of others' need for help: Effects of problem explicitness and type. *Child Development, 56,* 735–745.

Plomin, R. (1994). *Genetics and experience: The interplay between nature and nurture.* Thousand Oaks, California: Sage.

Plomin, R., DeFries, J. C., Craig, I. W., & McGuffin, P. (2003). *Behavioral genetics in the postgenomic era.* Washington, DC: American Psychological Association.

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin, 85,* 309–322.

Plomin, R., DeFries, J. C., McClearn, G. E., &Z McGuffin, P. (2001). *Behavioral Genetics* (4th ed.). New York: Worth Publishers.

Plomin, R., Emde, R. N., Braungart, J. M., Campos, J., Corley, R., Fulker, D. W., et al. (1993). Genetic change and continuity from fourteen to twenty months: The MacArthur Longitudinal Twin Study. *Child Development, 64,* 1354–1376.

Plomin, R., Owen, M. J., & McGuffin, P. (1994, June 17). The genetic basis of complex human behaviors. *Science, 264,* 1733–1739.

Price, T. S., Freeman, B., Craig, I. W., Petrill, S. A., Ebersole, L., & Plomin, R. (2000). Infant zygosity can be assigned by parental report questionnaire data. *Twin Research, 3,* 129–133.

Roberts, W., & Strayer, J. (1996). Empathy, emotional expressiveness, and prosocial behavior. *Child Development, 67,* 449–470.

Ronald, A., Eley, T. C., & Plomin, R. (2003). *A twin study of general and specific behavior problems in preschool children.* Unpublished manuscript.

Rushton, J. P. (1984). The altruistic personality: Evidence from laboratory, naturalistic, and self-report perspectives. In E. Staub, D. Bar-Tal, J. Karylowski, & J. Reykowski (Eds.), *The development and maintenance of prosocial behavior: International perspectives on positive development* (pp. 271–290). New York: Plenum Press.

Rushton, J. P., Chrisjohn, R. D., & Fekken, G. C. (1981). The altruistic personality and the Self-Report Altruism Scale. *Personality & Individual Differences, 2,* 293–302.

Rushton, J. P., Fulker, D. W., Neale, M. C., Nias, D. K. B., & Eysenck, H. J. (1986). Altruism and aggression: The heritability of individual differences. *Journal of Personality and Social Psychology, 50,* 1192–1198.

Saudino, K. J., Ronald, A., & Plomin, R. (2003). The etiology of behavior problems in 7-year-old twins: Substantial genetic influence and negligible shared environmental influence for parent ratings and ratings by same and different teachers. *Journal of Abnormal Child Psychology, 33,* 113–130.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype–environmental effects. *Child Development, 54,* 424–435.

Schwartz, S. H. (1996). Value priorities and behavior: Applying a theory of integrated value systems. In C. Seligman, J. M. Olson, & M. P. Zanna (Eds.), *The psychology of values: The Ontario Symposium* (Vol. 8, pp. 1–24). Mahwah, NJ: Erlbaum.

Scourfield, J., John, B., Martin, N., & McGuffin, P. (2004). The development of prosocial behavior in children: A twin study. *Journal of Child Psychology and Psychiatry, 45,* 927–935.

Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist, 55,* 5–14.

Spinath, F. M., Ronald, A., Harlaar, N., Price, T., & Plomin, R. (2003). Phenotypic 'g' early in life: On the etiology of general cognitive ability in a large population sample of twin children aged 2 to 4 years. *Intelligence, 31,* 195–210.

Staub, E. (1979). *Positive social behavior and morality: Socialization and development* (Vol. 2). New York: Academic Press.

Trouton, A., Spinath, F. M., & Plomin, R. (2002). Twins Early Development Study (TEDS): A multivariate, longitudinal genetic investigation of language, cognition and behavior problems in childhood. *Twin Research, 5,* 444–448.

Warden, D., & Mackinnon, S. (2003). Prosocial children, bullies, and victims: An investigation of their sociometric status, empathy, and social problem-solving strategies. *British Journal of Developmental Psychology, 21,* 367–385.

Waterman, A. S. (1999). Identity, the identity statuses, and identity status development: A contemporary statement. *Developmental Review, 19,* 591–621.

Woerner, W., Fleitlich-Bilyk, B., Martinussen, R., Fletcher, J., Cucchiaro, G., P., Lui, M., & Tannock, R. (2004). The Strengths and Difficulties Questionnaire overseas: Evaluations and applications of the SDQ beyond Europe. *European Child and Adolescent Psychiatry, 13*(Suppl.2), II47–I54.

Zahn-Waxler, C., Robinson, J., & Emde, R. N. (1992). The development of empathy in twins. *Developmental Psychology, 28,* 1038–1047.

Zahn-Waxler, C., Schiro, K., Robinson, J. L., Emde, R. N., & Schmitz, S. (2001). Empathy and prosocial patterns in young MZ and DZ twins: Development and genetic and environmental influences. In R. N. Emde & J. K. Hewitt (Eds.), *Infancy to early childhood: Genetic and environmental influences on developmental change* (pp. 141–162). New York: Oxford University Press.

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