Genetic and Environmental Influences on Prosocial Behavior

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Abstract

The obvious importance to human social functioning of prosocial behavior (behavior intended to benefit others) has led to extensive research on the sources of individual differences in prosociality, empathy, helping, cooperation, and altruism. At the species level, this markedly human trait has been explained in genetic terms. At the individual level, however, only about a dozen studies have directly addressed the origins of individual differences in prosocial behavior using genetically informed research designs. In contrast, there are more than 50 studies of the genetic influences on individual differences in aggression and antisocial behavior. Responding to this imbalance, researchers have recently begun to explore the genetic architecture of prosocial behavior. In this chapter we present evidence from our own research (e.g., Knafo & Plomin, 2006) and others’ work for the role of genes in prosocial behavior. Moreover, we present evidence for a developmental pattern in which the heritability of prosocial behavior increases with age, and we discuss potential explanations for this increase, such as changes in the role of theory-of-mind abilities needed for effective prosocial responding. Finally, recent evidence from molecular genetic studies (Knafo, Israel, et al., 2008) is presented to illustrate the role of genes in human altruism, in correspondence with parallel findings in other mammals. The correlations and interactions between genetic and environmental influences on prosocial behavior are discussed, and they are illustrated by research showing higher heritability in families with additional siblings.
Genetic and Environmental Influences on Prosocial Behavior

Prosocial behavior is central to human social functioning. Extensive research has been conducted on individual differences in various forms of prosociality, including empathy, helping, cooperation, and altruism (e.g., Fehr & Fischbacher, 2003; Graziano et al., 2007; Mikulincer & Shaver, 2005; Penner, Dovidio, Piliavin, & Schroeder, 2005; Singer et al., 2004; Staub, 1979). At the species level, this distinctively human trait has been explained in genetic terms since Darwin (1871) and Hamilton (1964). However, at the individual level only about 15 studies have directly explored genetic influences on individual differences in prosocial behavior, whereas more than 50 studies have been published on the genetics of aggression and antisocial behavior (Hur & Rushton, 2007). Researchers have recently begun to redress this imbalance (e.g., Cesarini et al., in press), and in this chapter we present evidence that supports the role of genes in prosocial behavior. We discuss developmental patterns in the influence of genes and environment, and present new evidence illustrating the interaction of genetic and environmental influences on prosocial behavior.

Prosocial behaviors can be defined as voluntary behaviors made with the intent of benefiting others (Eisenberg & Fabes, 1998). Typical prosocial behaviors include volunteering, sharing personal resources, instrumental help, costly help (sometimes at the risk of one’s life), and emotionally supporting others in times of distress. There has been considerable debate, which is beyond the scope of this paper to review, concerning the extent to which prosocial behaviors reflect altruism (behaviors that benefit an unrelated individual for intrinsic reasons or at a cost to oneself; e.g., Batson, Chapter 1, this volume; Batson et al., 1988). Similarly, the conceptual and operational boundaries
between prosocial behavior and empathy (a response that involves sharing the affective state of another person) or sympathy (feeling concern for someone who is experiencing distress) are not easily demarcated (see Eisenberg, Fabes, & Spinrad, 2006, for definitions of each of these concepts). Most researchers, however, view empathy and sympathy as providing a cognitive and affective basis for prosocial behavior (e.g., Hoffman, 2000; Knafo, Zahn-Waxler, et al, 2008; de Waal, 2007). For current purposes, we treat empathy, altruism, and prosocial behavior as related aspects of a single prosocial tendency.

*Environmental Influences on Prosocial Behavior*

Although the focus of this paper is genetic influences on prosocial behavior, it is important to acknowledge the importance of the environment. Most of the genetic studies we describe here refer to environmental influences without specifying their nature. That is, the amalgamation of psychobiological and social influences that affect prosocial behavior is described in terms of proportions of variance not accounted for by genetic factors. In this section we briefly discuss these factors and refer readers to the vast literature on the topic.

A multitude of environmental forces may influence prosocial behavior, for example: siblings and peers (Hastings, Utendale, & Sullivan, 2007); close friends, especially if the affective quality of the friendship is high (Barry & Wentzel, 2006); schools and teachers (Fraser et al., 2004). A five-year longitudinal study showed that training teachers to promote children’s prosociality, self control, and personal commitment to rules and values increased children’s prosocial behavior (Solomon, Watson, Delucchi, Schaps, & Battistich, 1988). There is also some evidence that
television programs designed to increase children’s prosocial behavior and attitudes can be effective (Calvert & Kotler, 2003; Cole et al., 2003).

Most of the environmental research on individual differences in prosocial behavior has focused on parental influences. Several excellent reviews of the literature exist (e.g., Eisenberg, Chapter 7, this volume; Eisenberg & Fabes, 1998; Eisenberg et al., 2006; Grusec, Davidov, & Lundell, 2002; Hastings et al., 2007; Staub, 1979); here we will summarize only the most consistent findings. Children’s prosocial behavior is longitudinally predicted by parenting style. Prosocial behavior increases when parents are warm, supportive, responsive, and sensitive to their children’s needs. In contrast, less prosocial behavior is found among children whose parents are authoritarian, strict, or punitive (Eisenberg & Fabes, 1998; Hastings et al., 2007).

Environmental variables can also have biological effects. In one study that distinguished between genetic and other prenatal biological factors, Hur (2007) investigated monozygotic (MZ) twin similarity. She grouped genetically identical twin pairs according to whether they shared their chorion or not. Chorion sharing can be seen as a proxy variable for a variety of hormonal and other influences that tend to be shared more extensively by monochorionic twins. Hur (2007) found no influence of chorion sharing on South Korean twins’ parent-rated prosocial behavior. However, evidence for hormonal influences on social behavior, whether the hormones are measured prenatally (Hines et al., 2002) or administered pharmacologically in adulthood (Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005; Thompson, George, Walton, Orr, & Benson, 2006), suggests that the role of biological environmental factors should be studied further.

Genetic Influences at the Individual and Species Levels
At first glance, it might seem paradoxical to seek an evolutionary justification for prosocial behavior. In a world where selfish players are rewarded with increased biological fitness and altruistic players are punished by sacrificing theirs, one would presume that Darwinian forces act strongly against the preservation of altruistic traits (Sesardic, 1995). As mentioned by Quine (1969, p. 126), “Inveterately altruistic creatures have a pathetic tendency to die before reproducing their kind.” Yet altruism does exist; and by examining mechanisms that incorporate prosocial behavior within a broader array of successful survival strategies, we can make an evolutionary argument for altruistic behavior.

Prosocial behavior is often viewed in terms of weighing of costs and benefits for altruistic acts against some criterion, such as genetic relatedness (kin selection), likelihood of future interaction (direct reciprocity), or maintaining one’s reputation (indirect reciprocity) (see Nowak, 2006, for a review). Although there is still some disagreement concerning genetic versus cultural contributions to the large-scale emergence of cooperative social adaptations in human societies (Richerson & Boyd, 2005), many of the underlying cognitive and affective processes that contribute to these adaptations, such as serial processing, abstract representational abilities, and neural “fear circuits,” were likely to have been under selective pressure (Simpson & Beckes, 2008). These evolutionary considerations suggest that altruism could have evolved as a human capacity.

Evolutionary pressures might also have increased diversity in the genetic tendencies to behave more or less prosocially. Computer simulations using multi-agent systems show that when individuals’ cooperation levels are evident to some degree (“signaled”) to
other individuals in the population, cooperative norms can emerge (Nowak & Sigmund, 1998). At the same time, these models highlight one of the central challenges in developing evolutionarily stable strategies: Although cooperators achieve higher fitness when interacting with one another, they are also susceptible to predation by free-riding noncooperators (who receive the benefits of cooperation without paying its costs). Thus, the genetic composition of a population with polymorphic equilibria of cooperation and selfishness would seem to be more robust than compositions leading consistently to cooperation or selfish defection (Cesarini et al., in press).

Nettle (2006) has expanded this argument by providing a framework for understanding the evolutionary forces that maintain variability in heritable behavior. Each of the Big Five dimensions of human personality (Costa & McCrae, 1992) – extraversion, neuroticism, openness to experience, conscientiousness, and agreeableness – can be viewed as the result of a tradeoff between fitness benefits and costs. The case of agreeableness, the trait most likely to relate to prosocial behavior (Eisenberg et al., 2006; Graziano et al., 2007; Penner et al., 2005), is especially relevant. Agreeableness balances the social benefits of empathy and trust with the costs of increased susceptibility to cheating and failure to maximize selfish advantages. High levels of agreeableness would have been more advantageous in certain evolutionary contexts and less so in others (Nettle, 2005).

As a case in point consider the gene locus for the human dopamine receptor D4 (DRD4). Bachner-Melman, Gritsenko, et al. (2005) reported that the most common 4-repeat variant in the third exon (the part of the gene translated to RNA for coding protein) is associated with prosociality. But an alternative genotype may also have advantages.
Ding and colleagues (2002) estimated that the 7-repeat variant in the third exon (associated with behavioral phenotypes such as extraversion, novelty seeking, and ADHD) is the result of a relatively recent and rare mutation event (40,000 to 50,000 years ago) that increased in prevalence due to positive selection. The study’s authors proposed that individuals with a DRD4 7-repeat allele may have had personality and cognitive traits that endowed them with evolutionary advantages (multiple sexual partners, higher probability for mate selection, etc.), which in certain cultural milieus that made up for the imposed costs of the variant (increased physical risk and family instability) would have expanded the frequency of the 7-repeat. Bachner-Melman, Gritsenko, et al. (2005) also suggested that the need for diverse behavioral phenotypes in human populations resulted in a balanced maintenance of both alleles.

**Individual Differences in the Tendency to Behave Prosocially Are Partly Heritable**

There are two major methods for assessing genetic influences on individual differences in human behavior and personality. One approach, to be discussed later in this chapter, establishes relationships between a trait and variability in observed DNA sequences. The other approach uses one of several possible genetically informative research designs to disentangle environmental and genetic effects on prosocial behavior. These designs (such as the separated twins design, or the adoption design) are intended to account for variance in the measured or observed (phenotypic) trait by looking at differences in the similarity between family members as predicted by differences in their genetic and/or environmental relatedness (Plomin, DeFries, McClearn, & McGuffin, 2001). The results are usually reported in terms of proportion of the variance accounted for by genetic and environmental factors.
Most studies of genetic and environmental contributions to individual differences in prosocial behavior have used the twin design, which compares monozygotic (MZ) twins, who share 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, that MZ and DZ twins growing up in their biological families are equal in terms of how similar their environments are, in which case greater similarity of MZ twins indicates genetic influence (heritability). Similarity beyond this genetic effect is attributed to the environment the twins share, and any further differences between the twins are ascribed to non-shared environment and measurement error (Plomin et al., 2001). One advantage of the twin design is that it simultaneously points to the importance of environmental and genetic influences accounting for individual differences. The heritability, shared environment and non-shared environment estimates sum up to 100% of the phenotypic variance.

From the few studies that have directly addressed the genetic and environmental contributions to individual differences in prosocial behavior, a meaningful developmental pattern emerges. Across roughly a dozen studies, there appears to be an increase in the heritability of prosocial behavior as children grow up, accompanied by a decrease in the importance of shared environment contributions.¹

¹ In our literature review, we often reviewed studies with a limited (e.g., 292, Volbrecht et al., 2007) number of twin pairs. In such studies model-fitting procedures often find that dropping shared environment effects does not result in worsening model fit, and an alternative model is chosen. But the model-fitting decision often depends on the statistical power of a particular study. Since for the purposes of this literature review we were interested in comparing studies, we describe results in terms of the "raw", unfitted models. In addition, we chose not to review evidence concerning genetic and environmental contributions to prosocial behavior that occurs between twins or siblings (e.g., Lemery & Goldsmith, 2001), because it is impossible to disentangle genetic and environmental contributions. Twin similarity in such behaviors can be due to mutuality in their behavior (see Knafo & Plomin, 2006a).
Overall, a majority of studies conducted with children find both genetic and shared environment effects. Deater-Deckard et al. (2001) reported modest heritability (.15) and modest shared environment effects (.18) based on maternal reports using the prosocial behavior scale of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). They used a step-family sibling design. A British twin study (Knafo & Plomin, 2006a) revealed genetic and shared environment effects on mother-reported prosocial behavior of children aged 2 to 7 years, and similar findings using teacher reports at age 7. Evidence for genetic effects and some shared environment effects on prosocial behavior as measured with the SDQ scale was reported for 5- to 16-year-old British twins (Scourfield, John, Martin, & McGuffin, 2004), but for 2- to 9-year-old South Korean twins (Hur & Rushton, 2007) the genetic effects were not accompanied by shared environment effects. As we explain below, such effects may be age dependent.

Two studies have addressed genetic and environmental influences on children’s reactions to adults in distress. Of particular interest are children’s empathy displays, which include both a cognitive aspect labeled hypothesis-testing, whereby the child actively tries to understand the other person’s problem, and an affective aspect labeled empathic concern, which requires one to experience a vicarious emotional response to another person’s expressed emotions (Zahn-Waxler, Radke-Yarrow, Wagner, & Chapman, 1992). Volbrecht et al. (2007) estimated the heritability of empathic concern for mother shown by children aged 19-25 months to be .30; for hypothesis testing it was .40. Shared environment effects accounted for 19-24% of the variance. A study of twins followed from 14 to 36 months of age also found moderate genetic effects on empathic
concern and hypothesis testing (Zahn-Waxler et al., 2001), as well as evidence for both shared and non-shared environment influences.

In these two studies children’s prosocial acts (trying to help or comfort a distressed victim) were observed. Volbrecht et al. (2007) reported a heritability of .22 and a shared environment effect of .43 on toddlers’ helping behavior toward their mothers. Zahn-Waxler et al. (2001) found genetic effects that were not consistent across different ages. A later report with a fuller sample found genetic effects for both hypothesis testing and empathic concern, but no genetic effect for prosocial behavior (Knafo, Zahn-Waxler, et al., 2008). In this study, shared and non-shared environment effects almost equally accounted for the positive relationship between empathy and prosocial behavior.

Studies of adults generally find genetic influences on prosocial behavior. Three self-report twin studies in Western countries all found genetic effects and no shared environment effects (the participants were adult men, aged 42 to 57; Matthews, Batson, Horn, & Rosenman, 1981; twins of both sexes aged over 50; Gillespie, Cloninger, Heath, & Martin, 2003; male and female high school seniors; Davis, Luce, & Kraus, 1994; see Knafo & Plomin, 2006a, for a review). In a British study of 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) (Rushton, Fulker, Neale, Nias, & Eysenck, 1986). The only shared environment effect found was for men’s (but not women’s) empathy (.23).

A Japanese study of 617 pairs of adolescent and young adult twins (Ando et al., 2004) included the cooperativeness subscales of the Temperament and Character Inventory (TCI) by Cloninger et al. (1993). For the empathy subscale, heritability was
estimated at .00, and a meaningful (.27) shared environment effect was found. In contrast, genetic effects of .41 and .34 were found, respectively, for helpfulness and compassion, and the rest of the variance was accounted for by non-shared environment and error.

Thus, most twin studies using questionnaire measures converge on the finding that, at least in adolescence and adulthood, genes influence prosocial traits, as does the environment. The environmental influences, however, tend to be of the non-shared type, meaning that they cause twins growing up in the same family to be dissimilar rather than similar. One American study reached a different conclusion: Krueger, Hicks, and McGue (2001) found a moderate effect of shared environment on altruism (.35) and little genetic effect.

Questionnaire studies are valuable when one needs to collect data from large samples to meet statistical power criteria. But evidence from observational and experimental studies is also important. In a study by Cesarini and colleagues (in press), Swedish and American twins participated in an economic game called *The Trust Game* (Berg, Dickhaut, & McCabe, 1995), with real monetary rewards. In this game, the first player (the investor) receives an initial allocation of monetary units and has the option of transferring all or some of them to the second player (the trustee). Any amount transferred is tripled by the experimenter, and the trustee can then either keep the tripled sum or choose to transfer all or part of it to the other player. The “rational” prediction for the game is for investors to transfer nothing because the trustees are not obligated to reciprocate. Twins were paired with unknown other players. Investors’ transfer of funds can reflect either trust (about getting their money back), a desire to increase their own funds (as the sum is tripled), or a desire to benefit the trustee by transferring funds to him
or her. In contrast, trustees’ return of funds (“trustworthiness”) is considered altruistic or cooperative in the sense that the other player (in the Swedish sample) could not reciprocate their return of funds (although trustworthiness is also a function of the investor’s decision). In other words, trustees could elect to keep any amount of their gains without repercussions. In the Swedish sample, the heritability of trustworthiness was estimated at .18 and the influence of the shared environment at .17. Similar results were obtained in the United States with a modified version of the game (Cesarini et al., in press).

*Genetic Effects on Prosocial Behavior Increase with Age*

Most of the literature reviewed above suggests that shared environment influences on prosocial behavior steadily decrease from childhood to adolescence and into adulthood. A different pattern emerges for genetic effects. In their literature review, Knafo and Plomin (2006a) noted that the heritability of prosocial behavior tended to be higher in adolescence and adulthood than in childhood. This is in line with the notion that the influence of shared environment usually decreases, and that heritability increases from infancy towards adulthood (McGue, Bouchard, Iacono, & Lykken, 1993; Plomin et al., 2001).

Generalizing from these trends, Knafo and Plomin (2006a) expected a decline throughout childhood in the importance of the shared environment in predicting prosocial behavior. Using British teacher reports on the SDQ, Scourfield et al. (2004) found shared environment effects in their ages 5-10 subsample, but none in their adolescent (11-16 years) subsample (.30 vs. .00). Similar (although nonsignificant) differences were found using parental reports of twins’ prosocial behavior. Scourfield et al. (2004) found lower
Comparing genetic and environmental influences on children’s parent-reported prosocial behavior with a longitudinal design, Knafo and Plomin (2006a) found that shared environment effects decreased from .47 on average at 2 years of age to .03 at age 7, and genetic effects increased from .32 on average to .61. In another longitudinal study (Knafo, Zahn-Waxler, et al., 2008), genetic influences on children’s empathy (a common factor derived from children’s affective and cognitive empathy toward their mother and an examiner) were negligible at 14 and 20 months and increased by 24 to 36 months. In contrast, shared environment effects were large at the earlier ages but small by 36 months.

To make cross-cultural comparisons, we plotted the results from four studies in which parents rated twin children’s prosocial behavior (with either the SDQ or a similar scale from Hogg, Rutter, & Richman, 1997). Since we were interested in age effects, we used age-specific correlations whenever they were available. British correlations were reported by Knafo and Plomin (2006a) and Scourfield et al. (2004), and Korean correlations by Hur and Rushton (2007). Israeli correlations were computed from data from the first 491 pairs participating in the Longitudinal Israeli Study of Twins (LIST; Knafo, 2006). We estimated the degrees of heritability by doubling the difference between the MZ correlation and the DZ correlation (to reflect the difference in genetic relatedness between MZ and DZ twins). The difference between the DZ correlation and
the heritability (divided by two) indicated a shared environment effect, and the remaining variance was accounted for by the non-shared environment (and error). For example, in Israel the MZ correlation was .54, and the DZ correlation was .07. Heritability is therefore estimated as around 54% (possibly representing a genetic dominance effect, because the DZ correlation is so low), and the remaining 46% is attributed to the non-shared environment and error.

Figure 1 shows both cross-cultural similarities and differences in the contributions of genetics and the shared environment to individual differences in children’s parent-rated prosocial behavior. In both British samples the younger ages appear more to the right, indicating larger shared environment effects in the younger age periods (although the shared environment is estimated to be more influential by Knafo & Plomin, 2006a, than by Scourfield et al., 2004). The same is true for the Korean samples, based on comparing children ages 2 and 3 with older children. Both British samples show higher heritability in older children, but the Korean sample does not. Finally, interesting cross-cultural variability is demonstrated when results from age 3 are compared. In all three countries, heritability at this age is estimated to fall between .43 and .58. However, the differential positions of the three subsamples on the X axis represent the finding of no shared environment effects in Israel, modest effects in Korea, and substantial effects (.26) in the UK. This calls for further cross-cultural consideration of the interaction of genetics and the environment with the cultural and ethnic context.

*Genetics and the Environment Cause Both Continuity and Change in Prosocial Behavior*

Having established that prosocial behavior is partly heritable, and that the genetic effects tend to increase with age (while shared environment effects decrease with age),
we now address the dynamics of these changes. With a longitudinal adaptation of the Cholesky method (e.g., Gillespie & Martin, 2005) it is possible to use within-twin and between-twin multivariate variance-covariance matrices to decompose the variance within and between ages into a set of genetic, shared environment, and non-shared environment factors. The variance is decomposed so that at each age, genetic, shared environment, and non-shared environment components are estimated, and their reappearance at later ages can be examined. To the extent that scores at later and younger ages load on the same factors, this indicates continuity. To the extent that scores at later ages do not load on the same factors as those at younger ages, this indicates change.

We are aware of only two studies in which the investigators attempted to estimate the environmental and genetic contributions to change and stability in children's prosocial or empathic behavior. Both found that genetics contributed to both change and stability, shared environment contributed to stability but became less important with age, while the non-shared environment (which includes measurement error) contributed mainly to change (Knafo & Plomin, 2006a; Knafo, Zahn-Waxler, et al., 2008; the latter study following up on earlier analyses by Zahn-Waxler et al., 2001).

Regarding empathy, the earlier publications from Zahn-Waxler and colleagues reported that genetic effects were partially responsible for continuity (Plomin et al., 1993; Zahn-Waxler et al., 2001). However, new genetic effects emerged at the different ages, accounting for change as well. With a larger sample, it was possible to pinpoint the developmental period in which changes in the genetic influences occurred (Knafo, Zahn-Waxler, et al., 2008). At 14 months, no genetic effect was found on the overall empathy measure. The first genetic effects appeared at 20 months, accounting for 9% of the
variance. These genetic effects were carried on, accounting for 16% of the variance at age 24 months. At that age a new genetic effect, distinct from the earlier genetic effect, emerged. This effect accounted for an additional 8% of the variance in empathy at 24 months (resulting in a heritability of .24 at this age). The genetic effect derived at 24 months accounted fully for the 25% heritability estimated at 36 months. This means that the main change in the genetic factors influencing empathy occurred in the period between 20 and 24 months, which makes sense from a developmental standpoint, because this period includes major transitions in self-other differentiation, children’s affective regulation, and cooperative play (e.g., Brownell & Carriger, 1990; Eckerman, Davis, & Didow, 1989; Hay, 1979; Nielsen & Dissanayake, 2004; Zahn-Waxler et al., 2001), all psychological variables relevant to empathy (Knafo, Zahn-Waxler, et al., 2008).

A study of parent-rated prosocial behavior also found that genetics accounted for both change and continuity (Knafo & Plomin, 2006a). While Knafo, Zahn-Waxler, et al. (2008) found no new genetic effects on empathy from 24 to 36 months, with regard to prosocial behavior new genetic effects continued to emerge at 3, 4, and 7 years. This is not surprising, because prosocial behavior has other predictors besides empathy (e.g., norm following). An interesting sex difference emerged in the transition from 4 to 7 years. The heritability of parent-rated prosocial behavior was similar for boys (.57) and girls (.55) at this age. However, most of the genetic variance in girls’ prosocial behavior was stable and represented earlier genetic effects (.41), with the new genetic effects accounting for only 14% of the variance. In contrast, the new genetic effects for boys at this age accounted for 32% of the variance, more than half of the genetic influence at age 7 (Knafo & Plomin, 2006a). The dynamics of change in genetic effects are therefore quite
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different for girls and boys. Research on this phenomenon is still needed, focusing on potential moderators such as the experience of the transition to school that occurs in this period.

Environmental influences can also contribute to change as well as continuity. In the empathy study, a single strong (.43) shared environment effect was estimated at 14 months and was carried over, but it became increasingly weak with age, until it contributed only 9% to the variance at 36 months (Knafo, Zahn-Waxler, et al., 2008). In the prosocial behavior study, a substantial effect of shared environment at age 2 for boys (.44) and girls (.56) was also carried over but waned in importance in later years. There was also evidence for new effects at 3 and at 4 years, but they were progressively smaller in size, and at age 7 no new significant effects were found (Knafo & Plomin, 2006a).

Thus, both studies found early substantial and stable shared environment factors that became weaker with age. In both studies, the main contributions of the non-shared environment were unique at each age. This is not surprising because non-shared environment estimates include a component of error. Knafo and Plomin’s (2006a) longitudinal analysis included 2,901 pairs, so they were able to detect minimal (1-2%) contributions of the non-shared environment to continuity that are unlikely to reflect longitudinal measurement error.

Where are the Genetic Effects to be Found?

Despite overwhelming evidence supporting the role of genetic factors in predicting prosocial behavior, we know very little about the specific genes involved. As with other traits (Plomin, DeFries, Craig, & McGuffin, 2003), this can be explained by in terms of the idea of quantitative trait loci (Plomin, Owen, & McGuffin, 1994). Many genes with
small effects are responsible for genetic influence on complex traits (Cardon & Bell, 2001). They may influence prosocial behavior through small effects on different behavioral, cognitive, and affective components, in an additive or interactive manner. It would be unlikely for a single gene to have a strong influence on the normal variation in a trait (see Plomin et al., 2001).

Nevertheless, there has been some progress in molecular genetic research into prosocial behavior and related traits. Hastings et al. (2006) proposed to study genes associated with serotonergic systems, because they are relevant to various affective processes (Hariri & Weinberger, 2003), which are likely to be involved in empathy. In one study, 17 of 59 examined genes were associated with individual differences in cooperativeness in a sample of 204 males, each accounting for less than 5% of the variance (Comings et al., 2000).

Research suggests that the genes involved in the dopaminergic system associated with temperament dimensions such as novelty seeking, extraversion, and reward would be strong candidates for explaining variability in prosocial behavior (Bachner-Melman, Gritsenko, et al., 2005). Many dopaminergic genes have been associated with conduct disorder and antisocial behavior (Tahir et al., 2000), and the researchers reasoned that genetic variants protective against these traits might also predict increases in prosocial behavior. In a study of 354 families, significant associations were observed between self-reported altruism (based on the Selflessness scale; Bachar et al., 2002) and genetic polymorphisms in the dopamine receptor D4 (DRD4) and D5 (DRD5) genes, as well as in the insulin-like growth factor 2 gene (IGF2), which is involved in development and growth and is associated with body-mass index and eating disorders (Bachner-Melman,
Gritsenko, et al., 2005). More research is needed to replicate these groundbreaking findings.

Recent findings regarding the oxytocin (OT) and arginine vasopressin (AVP) neuropeptide systems illustrate a new way to view genetic influences on prosocial behavior. These two nonapeptides are synthesized in the hypothalamus, are released into the blood as hormones, and act as neurotransmitters at synaptic targets in the brain. OT and AVP also serve important social functions, such as social bonding, parental care, stress regulation, social communication, and emotional reactivity (Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005). Several studies have found that pharmacological administration of OT and AVP profoundly affects social behavior. For example, OT administration attenuates fear responses to emotional faces (Domes, Heinrichs, Glascher, et al., 2007; Kirsch et al., 2005), increases people’s willingness to trust anonymous partners in an economic game (Kosfeld et al., 2005), and improves the ability to infer other people’s mental states (Domes, Heinrichs, Michel, Berger, & Herpertz, 2007). AVP administration has been found to modulate responses to facial expressions, with patterns differing between men and women (Thompson et al., 2006).

Genes involved in the regulation of OT and AVP, notably the oxytocin receptor gene (OXTR) and vasopressin receptor gene (AVPR1a) are therefore likely candidates for genetic association with social behavior. Variability in these genes has been related to behavioral and social differences within and between other mammalian species (Hammock & Young, 2005). For example, in the vole, a small rodent resembling a mouse, differences in AVPR1a microsatellites (a base pair repeat of DNA with a high degree of variability) in the promoter region have profound effects on social behavior,
mate preference, bonding, and affiliative behavior (Lim et al., 2004; Young, Nilsen, Waymire, MacGregor, & Insel, 1999). Prairie voles (with longer variants of the AVPR1a polymorphism) form lifelong attachments, rear pups together, and show high levels of social interest. In contrast, the closely related montane vole (with a shorter variant) does not pair-bond; the males do not contribute to parental care and they appear socially indifferent. Notably, in a comparison of the promoter region of this gene in humans, chimpanzees, and their more social and gregarious cousin, the bonobo, there was a longer microsatellite shared by humans and bonobos but absent in chimpanzees (Hammock & Young, 2005).

Ebstein and colleagues conducted research suggesting that the AVPR1a gene is especially relevant to human social behavior (Bachner-Melman, Zohar, et al., 2005). Analyses of postmortem brain samples indicate that the length of RS3, a microsatellite in the vicinity of this gene, is associated with the amount of mRNA produced, strengthening the hypothesis that the polymorphism is associated with gene function. (Knafo, Israel, et al., in press). In one study, very short versions of the microsatellite were more likely to be found in autistic individuals (Yirmiya et al., 2006). Other studies (reviewed by Israel et al., in press) found associations with a variety of social phenomena, from sibling relationships to creative dance and musical memory.

Many of these studies fall along a dimension of social ability, from autism to socially sensitive self-expression, strengthening the hypothesis that genetic variation in OT and AVP receptors have associations with a broad domain of social behavior (Israel, et al., in press). Altruism can be viewed as situated at the higher end of this social dimension. Together with Richard Ebstein, Gary Bornstein, and other colleagues, we
investigated the relationship between altruism and the length of the AVPR1a RS3 region. We assigned individuals the role of either Player A or Player B in the Dictator Game. Player A received the equivalent of $12 in Shekels. He (or she) was then asked if he wanted to share with Player B, a person he would never meet and who would never learn his identity. More than 60% of the participants who had two copies of the “long” version or the RS3 region were “generous,” in that they donated at least half of their endowment to the other player. In comparison, less than 30% of individuals with two copies of the “short” version of RS3 donated generously (Knafo, Israel, et al., in press).

Other genes relevant to altruism, empathy, and other aspects of prosocial tendencies are likely to be discovered in the next few years. One way to proceed would be to look at genetic influences on traits that are correlated with prosocial behavior. In twin studies, as described above, it is possible to estimate the genetic contribution to the correlation between traits. For example, Knafo (2007) reported a negative relationship between children’s mother-reported shyness and their experimentally induced and mother-reported prosocial behavior. Data from a twin sample indicated that the association between shyness and mother-reported prosocial behavior was wholly due to the genetic influences common to both traits (bivariate heritability). Most (60%) of the association between prosocial behavior and children’s sociability was due to bivariate heritability, and the rest was due to bivariate nonshared environment effects. To the extent that there is a strong genetically influenced correlation between prosocial behavior and another trait, genes that are reliably related to the other trait are likely to be relevant to prosocial behavior as well. It is important not only to find associations between genes and behavior, but also to
identify the biological processes underlying them. After all, genes do not code for behaviors; they code for proteins involved in biological processes involved in behavior.

*Genetic and Environmental Effects Are Often Intertwined*

Great effort has been devoted to dividing variability in human behavior into its constituent genetic and environmental components, but the two should not be seen as opposing explanations for behavior (Plomin et al., 2001). Focusing on only one of them is comparable to watching a multicolor movie in black and white. The richness of development is derived in part from the multitude of possible correlations and interactions between genes and varied environments.

In relation to gene-environment correlations and interactions, consider, for example, the steady age-related increase in the heritability of prosocial behavior. As Knafo and Plomin (2006a) noted, this increase in may reflect in part environmental processes. Parents’ behaviors are often influenced by their children’s temperamental tendencies (Kochanska, 1995). Plomin et al. (1977) referred to these processes, in which children’s genes operate through their behavior on the environment, as evocative gene-environment correlations. Such influences are more likely as children grow up (Scarr & McCartney, 1983), and they end up contributing to the heritability estimate (Knafo & Plomin, 2006a).

The issue of gene-environment correlations was addressed in the same sample (Knafo & Plomin, 2006b). At ages 3, 4, and 7, children’s low prosociality was related to parents’ use of negative discipline and withdrawal of affection. These associations were due mainly to children’s genetic tendencies, implying that the genetically influenced low prosociality evokes a negative reaction from parents. In contrast, parents’ positive discipline and affection showed a moderate positive correlation with children’s prosocial
behavior. This correlation was due mainly to shared environment processes, and to a lesser extent to genetic processes (Knafo & Plomin, 2006b).

Genetic and environmental effects are likely to interact in determining behavior, although only recently has strong evidence begun to accumulate (Bakermans-Kranenburg & van IJzendoorn, 2006; Caspi et al., 2002; Lau & Eley, 2008). One approach to assessing gene-environment interactions (Caspi et al., 2002) compares the association between an environmental variable (e.g., child abuse) and a measured behavioral or psychological phenotype (e.g., aggression), across different genetic profiles. For example, a Dutch study looked at the interaction between insensitive parenting and children’s DRD4 third exon. Bakermans-Kranenburg and van IJzendoorn (2006) reported a sixfold increase in externalizing behaviors in children who did not have the 7-repeat allele and were exposed to insensitive care, in comparison with children without this combination of risk factors. This is an important finding, especially as this polymorphism has been associated with self-reported altruism (Bachner-Melman, Gritsenko, et al., 2005).

Another approach to gene-environment interactions involves investigating the relative genetic contributions to individual differences across different levels of a measured environmental variable, such as parental discipline (Lau & Eley, 2008) or socioeconomic status (Turkheimer, Haley, Waldron, D’Onofrio, & Gottesman, 2003). Changes in heritability across levels of the environmental variable suggest that the magnitude of genetic effects is influenced by the environment.

With regard to prosocial behavior, the presence of other siblings is an especially interesting environmental variable. First, older or younger siblings are among the first
people children have to share with, from a very early age (Hay, 1994). Second, some older siblings, especially those with better social cognitive abilities, are often expected to take care of their younger siblings, thereby providing the latter with a model of prosocial behavior (Eisenberg et al., 2006). Third, there is no simple relationship between number of siblings or birth order and prosocial behavior (i.e., no main effect for number of siblings on prosocial behavior). For example, sharing may be more common, and spontaneous help less common, in large families (see Eisenberg et al., 2006, for a review).

As an illustrative case of gene-environment interactions, we focused on the presence or absence of additional siblings in the first 491 families of the Longitudinal Israeli Study of Twins. In this study the families of all Hebrew-speaking twins born in Israel in 2004 and 2005 were contacted, and sent questionnaires around the time of the twins’ third birthday (see Knafo, 2006, for details concerning study design and zygosity assessment). In our questionnaire, mothers of 3-year-old twins reported the age and sex of all their children. For current purposes the number of siblings was reduced to three levels (no additional siblings, one additional sibling, and two or more additional siblings). Mothers rated their twins’ prosocial behavior using the SDQ scale. No association was found between the number and sex of additional siblings and children’s prosocial behavior.

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2 It may seem counterintuitive to consider the presence of additional siblings in families of twins, where no child is a single child. However, the fact that the additional siblings are older or younger than the twins is important: Older siblings are often expected to behave prosocially toward their younger siblings, and they can be viewed as role models (Garner, Jones, & Palmer, 1994). In addition, it has been suggested that taking the perspective of the younger sibling contributes to the development of prosocial behavior (Eisenberg et al., 2006). Research shows that the presence of an additional sibling of the same age (i.e., a twin) does not improve children’s theory of mind, but having an older or younger sibling does (Cassidy, Fineberg, Brown, & Perkins, 2005).
Table 1 presents the twin correlations with mother-reported prosocial behavior, separately for MZ and DZ twins. As is clear from the table, MZ correlations tended to be substantially higher than DZ correlations, indicating a genetic influence on prosocial behavior. Next we ran a series of model-fitting procedures, using MZ and DZ data to estimate genetic and environmental effects, with twins’ sex and age as covariates. The best-fitting model appears in Figure 1.

Although there is evidence for genetic influence on prosocial behavior in this sample, an additional gene-by-environment interaction emerges from the findings. Clear differences were found between families in which twins were the only children and families in which there were additional siblings. In the former, an additive genetic effect of .23 and a nonshared environment (plus error) effect of .77 accounted for individual differences. In families with additional children, a larger genetic effect of .64 to .72 was found (this effect was estimated as a dominant, non-additive genetic effect, due to the strong MZ correlation and the negligible DZ correlation). Further analyses showed that splitting these families into two groups, those with younger or older siblings and those with different numbers of additional siblings, had little effect, indicating that the mere presence of additional siblings was associated with increased heritability.

This finding illustrates how the environment can moderate the effects of genes on children's behavior. One possibility is that in families with additional children, parents’ attention is divided among more children, and the parents are constrained in their ability to influence children consistently based on their own preferences and values. This may result in increased differences between children due to their genetic propensities. Another possibility is that when families include more children, the number of opportunities
children have to demonstrate prosocial behavior increases due to more frequent social interactions with different people (e.g., siblings and their friends), in which case their decisions to help or share represent their genetically influenced tendencies more purely. Alternative explanations may be proffered, but we should not lose sight of the central point that GXE interactions may be important and should be investigated in greater detail.

Conclusions

We have reviewed six studies of children and seven of adults, and all but one of them provides evidence for the heritability of prosocial behavior. Initial evidence from molecular genetic studies supports this conclusion, although more research elucidating the psychobiological processes responsible for the genetic effects is still needed. The heritability of prosocial behavior tends to increase with age, although the reasons for this increase remain largely unknown. The environmental influences are also important, and our cross-cultural comparison suggests a need for further twin research in additional countries. Such studies will increase our understanding of the variable effects of shared environment across cultures. Finally, genetic and environmental influences should not be seen as competing explanations. They are complementary explanations of individual differences in prosocial behavior.
References


Table 1

*Table Correlations in Mother-rated Prosocial Behavior according to Presence of Additional Siblings*

<table>
<thead>
<tr>
<th>Siblings</th>
<th>Twin correlation</th>
<th>Genetic influence (95% CI)</th>
<th>Non-shared environment influences and error (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MZ twins</td>
<td>DZ twins</td>
<td></td>
</tr>
<tr>
<td>No additional siblings</td>
<td>.25</td>
<td>.10</td>
<td>.23 (.01-.44)</td>
</tr>
<tr>
<td>One additional sibling</td>
<td>.78**</td>
<td>.02</td>
<td>.64 (.31-.81)</td>
</tr>
<tr>
<td>Two or more additional siblings</td>
<td>.71**</td>
<td>.21</td>
<td>.72 (.56-.82)</td>
</tr>
<tr>
<td>One, two, or more additional siblings</td>
<td>.73**</td>
<td>.09</td>
<td>.70 (.56-.79)</td>
</tr>
<tr>
<td>Total sample</td>
<td>.54**</td>
<td>.10</td>
<td>.54 (.40-.65)</td>
</tr>
</tbody>
</table>

Note: Numbers in parentheses represent 95% confidence intervals. Best-fitting models are presented (details of each analysis and model fitting procedure are available from the authors.) No significant shared environment influences were found. All genetic effects (except for the group with no additional siblings) are non-additive (dominant genetic influences).

*p < .05; ** p< .01.*
Cross-cultural comparison of genetic and shared environment effects on children’s parent-rated prosocial behavior. Numbers in the figure represent the age (in years) of each subsample. The United Kingdom (UK) estimates are based on Knafo and Plomin (2006a) (ages 2-7) and Scourfield et al. (2004) (ages 5-16); South Korean estimates are based on Hur and Rushton (2007); Israeli estimates are unpublished data from the sample described by Knafo (2006).