Chapter 9

Behavior genetics of prosocial behavior

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Quantitative genetics of prosocial behavior

The field of quantitative genetics aims to understand how environmental and genetic factors independently and interactively shape behavioral traits in humans. This scientific approach has been applied to a wide variety of observed characteristics, or *phenotypes*, making this work relevant for all areas of psychology. To fully understand this body of work, we first need to introduce the quantitative genetic methodology and review the types of prosocial phenotypes that have been examined. Then, we can explain what the accumulating evidence reveals about the origins of prosocial behavior.

Overview of quantitative genetics

Quantitative genetics relies on samples of related individuals, called *genetically informative samples*. These samples include groups of adoptees, siblings, twins, and these probands' families. Twins have supplied the large majority of quantitative genetic data on compassion and related prosocial phenotypes, and so we focus heavily on them here. Twins are a sort of natural experiment. Identical, or *monozygotic* (MZ), twins share (for all intents and purposes) 100% of their genetic complement, whereas fraternal, or *dizygotic* (DZ), twins share 50% of their genetic complement. By comparing the resemblance of MZ versus DZ twins, behavior geneticists are able to estimate the degree to which genetic factors influence a given phenotype.

The primary objective of twin studies is to decompose variation in a trait into that which is caused by genetic versus environmental factors. In twin analysis, genetic effects are assumed to cause the heightened correspondence among MZ twins compared to DZ twins. In fact, the percentage of phenotypic variance that is due to genetics can be approximated by doubling the difference between the MZ and DZ twin correlations for a given trait (e.g. Falconer & MacKay, 1996). Environmental effects, in turn, are divided into two varieties in twin designs. The *shared environment* includes experiences that children reared in the same family have in common (e.g. parenting style, socioeconomic status) and that promote

sibling similarity on a given trait. In contrast, the *unique environment* encompasses environmental exposures (e.g. peer groups, marital relationships) that cause siblings growing up in the same family to differ from one another. Accordingly, quantitative genetic studies that rely on twin samples typically report the propor-tion of variation in a particular trait that is due to genetics, the shared environment, and the unique environment.

Quantitative genetic studies of self-reported prosocial attitudes

Some of the first quantitative genetic studies on compassion and related prosocial behavior focused on self-reported attitudes that are related to these constructs. Early research on this topic asked over 500 pairs of MZ and DZ twins to answer questionnaires designed to assess altruism, empathy, and nurturance (Rushton, Fulker, Neale, Nias, & Eysenck, 1986). This research revealed an even split, whereby 50% of the variability in these traits was attributable to inherited genetics and 50% was attributable to environmental factors. Foreshadowing a consistent finding from subsequent studies in adults, this study reported that the shared environment—again, those elements of the child-rearing environment that cause siblings to resemble one another—had virtually no effect on prosocial outcomes. There are exceptions to this pattern, however, as a more recent study that used the University of London Institute of Psychiatry Adult Twin Register reported that 42% of the variability in prosocial behavior (e.g. civic responsibility, conscientiousness) was due to genetics, 35% to unique environment, and a full 23% to the common environment (Rushton, 2004).

Cooperativeness has also been shown to be partly under genetic influence. For example, 2,571 Australian twins over 50 years old completed the Cooperativeness scale on the Tridimensional Personality Questionnaire (Cloninger, Przybeck, & Svrakic, 1991), and analyses revealed that the percentage of variation due to genetic differences, called the *heritability estimate* (or h^2), was 27% (Gillespie, Cloninger, Heath, & Martin, 2003). The remaining variance was attributable to differences in the unique environment. A subsequent study of 617 Japanese adolescents and young adults who completed the Temperament and Character Inventory (TCI) found that the heritability for cooperativeness was estimated at 47%, and, again, no effect was detected for the shared environment (Ando et al., 2004). In this study, cooperativeness was among the most highly heritable personality traits (heritability range: .00–.49).

Several studies followed up on Rushton and colleagues' (1986) early genetic analysis of altruism. Krueger, Hicks, and McGue (2001), for example, reported surprising results from 673 adults in the Minnesota Twin Registry who completed the Self-Report Altruism Scale (Rushton, Chrisjohn, & Fekken, 1981), which inquires about altruistic actions toward strangers and organizations. Because the MZ twin correlation did not substantially exceed the DZ twin correlation for altruism, no genetic contribution to altruism was detected for those twin pairs. In contrast, a heritability estimate of

52% was reported for antisocial behavior in this sample. A subsequent study using an independent sample from the Minnesota Twin Registry also uncovered a small genetic influence ($h^2 = 10\%$) on self-reported altruism (Koenig, McGue, Krueger, & Bouchard, 2007). Although this is a relatively small genetic effect, joint biometric analyses with reports of religiousness demonstrated a large correspondence (73%) between the genetic effects on altruism and religiousness.

Several other studies on adult twins are relevant to the genetic and environmental causes of prosociality. Consistent with results reported by Rushton et al. (1986), Matthews, Batson, Horn, & Roseman (1981) reported that 72% of the variation on a self-report measure of empathy was due to genetic variation in middle-aged male twins. Also, Kendler (1996) asked over 1,000 adult twin pairs and their parents to describe the parenting they were exposed to as children, and he found that parental warmth was heritable ($h^2 = 38\%$), whereas parental authoritarianism and protectiveness were not (see also Losoya, Callor, Rowe, & Goldsmith, 1997).

A small number of studies have examined the heritability of prosocial behavior in young people, in part to discern the relative importance of the rearing environment versus the inherited genetic complement to prosocial outcomes. In a sample of 183 seven-year-old twin pairs, Knafo-Noam and colleagues asked mothers to report on social behavior of their twin offspring in five areas: sharing, social concern, kindness, helping, and empathic concern (Knafo-Noam, Uzefovsky, Israel, Davidov, & Zahn-Waxler, 2015). These sub-domains of prosocial behavior were moderately inter-correlated (rs > .39). Moreover, evidence was found for a common prosociality factor that accounted for the correspondence among these sub-domains. Capitalizing on the twin design, the authors reported that much of the sample variation related to this overarching factor was due to genetic causes ($h^2 = 69\%$). In addition, the unique environment accounted for the remaining variance, leaving only a very small (perhaps nonexistent) role for the shared environment.

Taking a very similar approach, Scourfield, John, Martin, and McGuffin (2004) derived a common prosociality factor from parent and teacher ratings of child and adolescent prosocial behavior. This factor was moderately heritable (h^2 range: 46%-53%) in childhood (i.e. ≤ age 10) and even more so in adolescence. Furthermore, the small effect of the shared environment that was detected in childhood eroded completely as the youth transitioned into adolescence. The authors commented that this pattern of declining influence for the shared environment, and corresponding growing magnitude of genetic effects, resembles a pattern found in other substan-tive literatures, such as youth depression (e.g. Rice, Harold, & Thapar, 2002) and general intelligence (e.g. Briley & Tucker-Drob, 2017).

Quantitative genetic studies of self-reported prosocial behavior

Transitioning from studies designed to trace the genetic and environmental origins of prosocial attitudes, we now concentrate on the smaller number of quantitative

genetic studies of self-reported prosocial behavior. In this context, Son and Wilson (2010) accessed twin data from the Midlife in the United States Study to evaluate genetic contributions to the number of hours per month that people spent doing volunteer work for organizations (e.g. youth education, healthcare work). They found that volunteering was mildly heritable for women ($h^2 = 30\%$), but not at all for men. For both sexes, there was no discernible shared environmental effect, meaning that the majority of the variance was accounted for by the unique environment. A second cleverly designed study used the Danish Twin Register to investigate the heritability of donating blood, which is thought to be motivated strongly by altruism (Pedersen et al., 2015). By linking the twin register to a national database of blood donors, the authors concluded that donating blood was jointly determined by genetic (53%) and shared environmental (28%) factors.

A pair of genetically informative studies has also been published on marital fidelity, which is a phenotype akin to pair bonding in animal research. One recent study of over 7,000 Finnish twins reported that much of the variation in self-reported fidelity (i.e. absence of extra-pair mating or, more colloquially, extra-marital affairs) was due to genetics, although the percentage differed for males (62%) versus females (40%) (Zietsch, Westberg, Santtila, & Jern, 2015). Largely corroborating this result, an earlier study of female twin pairs in the United Kingdom estimated the heritability of marital fidelity at 41% (Cherkas, Oelsner, Mak, Valdes, & Spector, 2004). In a surprising twist, this same study found that *attitudes* toward infidelity, as compared to the act itself, were not at all heritable.

Experimental quantitative genetic studies of prosocial behavior

Finally, several studies have used the twin methodology in the context of experimental settings. An early study examined empathy among approximately 200 pairs of two-year-old twins (Zahn-Waxler, Robinson, & Emde, 1992). To induce empathic responses in the laboratory, these researchers asked experimenters to fake painful accidents, such as closing a finger in a suitcase or banging a knee on a table. The experimenters then simulated pain vocalizations and agonizing facial expressions. Children's facial expressions, vocalizations, and comforting actions were coded by independent raters. Ultimately, this study detected only modest evidence for the heritability of empathy to the insofar as the genetic effects were small and confined to only a few dimensions of empathy (e.g. affective facial expressions).

Using an almost identical methodology, a more recent study evaluated empathetic behavior in 400 twin pairs assessed four times between 14 and 36 months old (Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008b). Over these four assessments, the heritability estimates for empathy were 0%, 0%, 34%, and 47%. For prosocial behavior (i.e. comforting actions, such as offering the experimenter a toy or getting a bandage), the corresponding estimates were 17%, 0%, 9%, and 24%. The influence of the shared environment declined

over these intervals in concert with the rise of genetic factors such that, by the final time point, its influence on empathy was gone. Also, there was a moderate phenotypic association between empathic expressions and prosocial actions, but this relation was entirely due to overlapping environmental influences on these two outcomes. As such, the genetic factors involved in empathy and prosocial behavior were found to be entirely independent.

Summary of quantitative genetic studies

In summary, quantitative genetic techniques tell us about the proportion of sample variability that is attributable to genetic, shared environmental, and unique environmental factors. The methodology can thus give us unprecedented insight into the etiology of psychological phenotypes that is not available through traditional observational research designs. Twin studies are the workhorse of the quantitative genetic field, and most of the existing data that are relevant for understanding compassion come from twin samples.

To describe this literature, we reviewed evidence from studies in which respondents were (a) asked to report on prosocial attitudes, (b) asked to report on prosocial behavior, or (c) observed for evidence of prosociality in a laboratory setting. Although there are notable exceptions (e.g. Krueger et al., 2001), these studies generally confirm a partly genetic origin to compassion-related phenotypes. However, the magnitude of heritability estimates varies widely across studies, even among those that rely on the same sorts of samples and assessment tools. It is a truism that most personality and clinical traits have heritability estimates of approximately 50% in adulthood (Turkheimer, 2000). The studies reviewed here suggest that prosocial traits are similarly, or perhaps slightly less, heritable.

Consistent with patterns observed in other literatures, the heritability of prosociality appears to increase with age (Knafo et al., 2008b; Scourfield et al., 2004). Moreover, as genetic effects expand, shared environmental influences diminish. This increase in genetic influence has been attributed to the emergence of *gene-environment correlation*, or the process whereby genetically based preferences begin shaping the environments to which people are exposed (see Scarr & McCartney, 1983). For instance, compassionate young people may select into friendship groups of compassionate peers who further foster compassionate attitudes among their friends. Although it is tempting to label favorable social experiences as "environmental events," from a causal perspective, they are (distally) due to genetic influences. Therefore, as young people are able to exert more control over their environments, such as seeking out one's own social niche, the heritability estimates in behavior genetic studies rise.

Regardless of the reason for this increasing influence of genetic factors over time, the phenomenon raises interesting questions regarding the role the rearing environment plays in shaping long-term trajectories of prosocial behavior among offspring. Behavior genetic studies have indicated that the influence of the shared environment declines precipitously to point that, in adult samples, the influence

of the shared environment is nil. These data suggest that the rearing environment, including parenting practices, may have little-to-no long-term effect on offspring prosocial behavior. Tackling this controversial issue is beyond the scope of this chapter, but interested readers can see Harris (1998) for an introduction.

Molecular genetics of prosocial behavior

Using the quantitative genetics paradigm to partition the origins of a phenotype into genetic, shared environmental, and unique environmental sources has many advantages. Unlike much social science research, for example, this methodology enables researchers to make strong inferences about what *causes* a given trait, as opposed to merely documenting statistical associations that may or may not be causal in nature. Moreover, quantitative genetics has been largely immune to the ongoing replication crisis in psychology (Plomin, DeFries, Knopik, & Neiderhiser, 2016). This is because within a given substantive domain, such as prosocial behavior, heritability estimates tend to be relatively consistent across studies.

At the same time, quantitative genetic studies have an important limitation: they do not identify the specific genes that give rise to phenotypes. Rather, these studies yield an omnibus genetic effect, meaning that researchers can discern the effect of individual differences in the entire genome on a trait; however, the genetic variants that drive these effects are not available for analysis. As a result, quantitative genetic methods alone cannot delineate the specific genes that are involved in shaping particular phenotypes, such as compassion or prosocial behavior.

This is where molecular genetics becomes useful. Molecular genetics is the set of methodologies that illuminates how individual genes are related to a phenotype. Since the completion of the Human Genome Project in the early 2000s, researchers have had access to the approximately 30,000 genes in the human genome, and variation at some genetic loci has been linked to psychological outcomes, including prosocial attitudes and behavior. In this section, then, we review the rapidly proliferating literature on how prosocial outcomes are related to measured genes, as opposed to the unmeasured genes that contribute to omnibus genetic effects that are revealed in behavior genetic designs.

Overview of molecular genetics

First, we need to introduce some fundamental terms and concepts in molecular genetics. For starters, genes are segments of DNA that contain instructions for making proteins. Proteins, in turn, are essential for the operation of countless psychological and biological processes that help sustain life. As an example, the serotonin transporter enzyme, which regulates the efficiency of serotonin neurotransmission and is the target of many antidepressant medications, is a protein. Although our genetic code is critical for life, not everyone has the same DNA

code at a given location in the genome. Rather, there is substantial inter-individual variability in the make-up of our genes. Genes that show differences in structure across people are called genetic *polymorphisms* or *variants*.

Furthermore, we have two copies of any gene that is not transmitted on a sex chromosome; one copy is inherited from our mother and one from our father. These copies are also called *alleles*. Therefore, at any given location on the genome, three *genotypes* are possible (i.e. two copies of allele *X*, two copies of allele *Y*, or one copy of each). Individuals who have two copies of the same allele for a given gene are said to be *homozygous* for that genotype, whereas persons with one copy of each allele are called *heterozygous*. These individual differences in genotypes often correspond to differences in protein production, which can in turn produce phenotypic differences, such as varying rates of serotonin neurotransmission in the case of the serotonin transporter protein previously described.

With this background information in mind, we now turn to recent research implicating individual genetic variants in prosociality. To make the interpretation of the findings easier, we have organized this part of the review into three *candidate gene* pathways. Candidate genes are those genetic variants that are theorized to be involved in a given phenotype given their role in biological systems that are thought to support the phenotype. Since we are interested in the genetic bases of prosociality, we focus here on genes relating to (a) the oxytocin and vasopressin neuropeptides, (b) dopamine neurotransmission, and (c) serotonin neurotransmission. Each of these systems has been associated with prosociality and related traits in animal model and human research, so we now examine the extent to which the genes regulating activity in each of these systems predict prosocial physiological, cognitive, and behavioral outcomes.

Oxytocin and arginine vasopressin

Oxytocin (OT) and arginine vasopressin (AVP) are neuropeptides that are often studied in tandem. Together, they are thought to regulate the propensity to engage in affiliative behaviors involving compassion, perspective taking, empathy, and trust (see Ebstein, Knafo, Mankuta, Chew, & San Lai, 2012). Intensive human research on OT and AVP was inspired by early animal studies showing that endogenous (i.e. naturally occurring) levels of these hormones are correlated with the strength of pair bonding in prairie voles (e.g. Insel & Shapiro, 1992).

Human research examining these neuropeptides has found that intranasal administration of OT and AVP influences social interactions in the laboratory (e.g. Tabak et al., 2015). For instance, temporarily boosting OT produces increases in empathy, trust, identification of others' affective states, and limbic reactivity to emotional cues in laboratory settings (for a review, see Striepens, Kendrick, Maier, & Hurlemann, 2011). In light of the wide-ranging effects of naturally occurring OT and AVP on social cognition and behavior, several studies have

investigated how prosociality is influenced by genetic polymorphisms that are responsible for the structure and activity of these hormones.

A main focus of this work has been on examining links between prosociality and polymorphisms in the gene that encodes the oxytocin receptor, which is responsible for binding OT to the postsynaptic neuron and thus terminating the neurochemical communication process among neurons. There are many locations in the oxytocin receptor gene (abbreviated OXTR) where the DNA code varies across people. Analogously, there are two polymorphisms in the gene carrying the information for an AVP receptor protein (called AVPR1a) that are usual suspects in molecular genetic research on prosocial outcomes.

Correlational molecular genetic studies of oxytocin and arginine vasopressin

Turning first to self-reported prosocial behaviors, two studies have investigated these genes in relation to empathy. The first study found that individuals homozygous for the so-called G allele at the OXTR polymorphism (designated rs53576) endorsed more empathic attitudes than those who were heterozygous at that polymorphism or who had two copies of the A allele (Rodrigues, Saslow, Garcia, John, & Keltner, 2009). Additionally, these same G homozygotes demonstrated better inferences of others' affective states in a laboratory task. More recently, Uzefovsky and colleagues found this same rs53576 G allele to be linked with greater emotional empathy (i.e. empathic concern and personal distress over another's pain), but not cognitive empathy (i.e. perspective taking), in an undergraduate sample (Uzefovsky et al., 2015). Conversely, a polymorphism in the promoter region of the AVPR1a gene was associated with emotional, but not cognitive, empathy in this study. Specifically, those without a variant called the 327-repeat allele (to denote the increased length of the promoter region) at a polymorphism labeled RS3 reported less of an emotional response to others' distress. An independent study of mothers with two-year-old offspring corroborated this OXTR finding by showing that mothers carrying at least one rs53576 G allele were more responsive to their toddlers' cries than mothers without the G allele (Bakermans-Kranenburg & van Ijzendoorn, 2008).

Several large-scale studies have examined real-world prosocial behavior (i.e. as opposed to attitudes or personality traits). In the same female twin sample described earlier, Cherkas and colleagues (2004) found no evidence of an association between variation in the AVPR1a gene and marital infidelity. Similarly, after correction for multiple testing, a study of Finnish twins found no associations between polymorphisms in the AVPR1a gene (including the RS3 location mentioned above) and rates of marital fidelity (Zietsch et al., 2015). Finally, a study employing a nationally representative sample of unrelated individuals in the United States found no main effects of OXTR rs53576, AVPR1a RS1, or AVPR1a RS3 polymorphisms on participants' engagement in volunteer work or charitable activity (Poulin, Holman, & Buffone, 2012).

Experimental molecular genetic studies of oxytocin and arginine vasopressin

Complementing these correlational findings, an interesting line of experimental behavioral research has begun to explore compassion through individuals' economic behavior. For example, several studies have genotyped players in the Dictator Game, in which the first player (the "Dictator") decides what proportion of a fixed sum of money to keep and what proportion to donate to an anonymous "Recipient," without any consideration of reciprocity or retaliation. The donations can thus be considered completely altruistic insofar as they cannot benefit the Dictator in any way. In one study that employed this task, three out of the 15 polymorphisms examined predicted the size of the Dictators' donations (Israel et al., 2009). In a second study, Apicella et al. (2010) attempted to replicate this result but found no associations between any of the nine OXTR polymorphism tested and participants' donation rates. In a third study, Knafo et al. (2008a) examined variation in the AVPR1a gene and found that the RS3 (but not RS1) polymorphism predicted donation size, with the longer versions of RS3 (indicating larger promoter regions of AVPR1a) being associated with more generous Dictator Game allocations and higher self-reported altruism. Finally, Krueger and colleagues (2012) genotyped undergraduates at OXTR rs53576 and invited them to play a different economic game. In this "trust game," an investor and trustee both start with ten monetary units. Then, the investor donates some proportion of that amount to the trustee, at which point the donation amount is tripled and the trustee chooses how much of his or her money to return to the investor. Using this task, Krueger and colleagues found that G homozygote investors not only made larger initial transfers to trustees, but also had higher self-reported dispositional empathy.

Another useful paradigm for assessing prosocial behavior in the lab involves dyadic interactions, either between strangers or close relations. Using this approach, Feldman et al. (2012) coded minute-by-minute interactions between parents and infant offspring for instances of parental touch and gaze synchrony (i.e. parent-offspring eye contact). This study not only found that the OXTR rs1042778 polymorphism—one of those linked with Dictator donations by Israel et al. (2010)—predicted parental touch rates, but also implicated variation in the CD38 gene, which is partially responsible for brain OT secretion, in parenting behavior. No genetic effects were detected for gaze synchrony. Around the same time, an independent research group studied the gaze duration of unrelated undergraduates in a naturalistic conversation, and contradicting prior research on the OXTR rs53576 locus, this study found that undergraduates carrying an A allele maintained better eye contact with a conversation partner compared to G homozygotes (Verhagen, Engels, & Van Roekel, 2014). Other research with adults has found no direct effect of rs53576 variation on cognitive empathy tests or judgments of confederates' experience of pain (Laursen et al., 2014; Weisman et al., 2015). In a recent study of Chinese children, however, Wu and Su (2015) found that rs54576 G allele homozygotes exhibited more helping and comforting behaviors to experimenters than A allele carriers.

Along these same lines, several studies have focused on OXTR variation and romantic partner interactions. In one study, investigators observed a supportive interpersonal interaction and coded it for indices of empathic communication (Walter et al., 2012). They found that a risk score computed from variation at five OXTR polymorphisms correlated with poorer empathic support. A separate research team also found evidence of oxytocinergic involvement in prosocial romantic behavior (Algoe & Way, 2014). Specifically, this study examined CD38 variability—polymorphisms called rs6449182 and rs3796863 that underlie the expression of the CD38 protein and plasma OT levels—and found that these variants related to participants' positive responses to expressions of gratitude from their romantic partners.

Serotonin

The neurotransmitter serotonin has been associated with a vast array of behavioral phenotypes. These include such diverse processes as sleep, appetite, sexual behavior, and mood. Initial attention to the potential impact that serotonergic genetic variation might have on prosociality and antisociality was highlighted by early personality theories linking serotonin system function with aggression (Carver, Johnson, & Joormann, 2008), and a seminal demonstration of gene-environment interaction for violence and antisocial behavior involving a serotonergic gene (i.e. monoamine oxidase A; Caspi et al., 2002).

The body of research examining serotonin-related genes and prosocial behavior is far smaller than that involving OT and AVP. Moreover, nearly all of the studies on this topic involve variation in one gene that controls the transcription efficiency of the serotonin transporter gene. Specifically, there is a polymorphism (abbreviated 5-HTTLPR) in the promoter region of the serotonin transporter gene that corresponds to differences in the number of serotonin transporter enzymes on presynaptic neurons (Lesch et al., 1996). The short (S) allele of this polymorphism is less transcriptionally efficient than the long (L) allele and leads to fewer working serotonin transporters. This difference in the availability of transporter enzymes in turn translates into individual differences in rates of serotonin neurotransmission.

The S allele at 5-HTTLPR is best known for conspiring with environmental stressors to confer vulnerability to emotional disorders (e.g. Conway, Slavich, & Hammen, 2014; for a review, see Caspi et al., 2010). However, several studies have also examined its relation to prosociality. An early study (Hamer, Greenberg, Sabol, & Murphy, 1999) provided evidence supporting an association between the 5-HTTLPR S allele and lower self-reported cooperativeness (Cohen's $d = 0.35$), as assessed by the TCI. The direction of this effect is consistent with the S allele's implication in various forms of psychopathology that involve social deficits (Caspi et al., 2010).

Experimental molecular genetic studies of serotonin

Data linking the serotonin transporter gene and prosociality also come from laboratory studies of social cognition and interaction. In one study on utilitarian moral

judgments (Marsh et al., 2011), healthy volunteers were asked about whether they would cause harm to one person to save the lives of others by changing the path of a hypothetical runaway train away from five people and toward one innocent victim. S allele carriers were less likely to endorse this type of harm and were also more averse to causing unintentional harm to innocent people. A more recent study examined associations between 5-HTTLPR and females' judgments of pain experience of another participant (Laursen et al., 2014). The authors assessed how accurately raters could sense the intensity of pain that their fellow participants were experiencing after an electric shock. However, genotype at 5-HTTLPR was unrelated to the accuracy of ratings and neural activity in relevant brain regions during the rating process. In sum, then, different research methodologies have yielded inconsistent findings linking 5-HTTLPR with prosociality.

Dopamine

Finally, like serotonin, the dopamine system is involved in a variety of different behavioral outcomes. Although dopamine neurotransmission is perhaps most commonly linked with personality traits like disinhibition, reward dependence, and extraversion, some studies have explored its association with prosociality. For example, Comings et al. (2000) invited undergraduates to complete the TCI and then analyzed their responses in relation to 59 candidate genes. Variation at the dopamine transporter gene (DAT), which performs an analogous function to 5-HTTLPR, was associated with responses on the TCI Cooperativeness scale, although only 3% of the variation in self-reported cooperativeness was accounted for by DAT. In a second study, Baeken, Claes, and De Raedt (2014) examined variation in the catechol-*O*-methyltransferase (COMT) gene as a predictor of novelty seeking, as measured by the TCI. Although the hypothesized association was not found, COMT genotype was strongly related to the TCI Cooperativeness scale. The authors argued that this finding is consistent with the hypothesis that people with lower resting dopamine levels are more empathic and compassionate and, in doing so, they cited an independent study on DAT genotype showing that higher dopamine availability corresponds to low TCI Cooperativeness scores (Pelka-Wysiecka et al., 2012). Corroborating this hypothesis, another study documented gender-specific effects of a dopamine receptor gene (DRD4) on self-reported empathy (Uzefovsky et al., 2014).

Experimental molecular genetic studies of dopamine

Several laboratory studies that are similar in design to those supporting the involvement of OT and AVP in prosociality have been conducted to examine dopaminergic genetic effects on objective prosocial behavior. Two such studies investigated genetic links to children's willingness to donate money to charity. In the first study, children earned money in two computer tasks and were then invited to donate as much of their endowment to a South American charity as they liked

(Reuter, Frenzel, Walter, Markett, & Montag, 2011). Children carrying at least one COMT Val allele (named after the amino acid valine encoded by the corresponding segment of DNA) donated approximately twice as much as children who were homozygous for the COMT Met (methionine) allele. This effect was in the same direction as the association between COMT and TCI Cooperativeness reported by Baeken et al. (2014). In a second study, Bakermans-Kranenburg and van Ijzendoorn (2011) examined associations between DRD4 genotype and altruism in seven-year-olds. They found no main effect of genotype on charitable donations, although there was a complex pattern of interaction between child attachment style and DRD4 genotype in predicting helping behavior. Specifically, securely attached children gave more money, but only if they carried a so-called 7-repeat allele at DRD4.

Finally, a few laboratory studies have examined dopaminergic effects on empathy in child samples. One study found that maternal negativity interacted with DRD4 genotype to predict children's reactions to an experimenter who was simulating physical pain (Knafo, Israel, & Ebstein, 2011). A second study examined associations between dopamine-related genes and *theory of mind*, which is the ability to understand others' mental states that is thought to underlie compassionate behavior and related phenotypes (Lackner, Sabbagh, Hallinan, Liu, & Holden, 2012). Here, children carrying the 7-repeat version of DRD4 demonstrated superior theory of mind capabilities, which is consistent with the results reported by Bakermans-Kranenburg and van Ijzendoorn (2011). However, variation at DAT and COMT in this study was unrelated to theory of mind performance.

Summary of molecular genetic studies

In summary, research on the molecular genetics of prosociality is in its infancy compared to that of other personality and clinical phenotypes. Although the data are still limited, several trends are apparent. First, the studies conducted to date have focused largely on the OT and AVP systems, in large part because of the long history of animal and human research linking naturally occurring levels of these hormones to affiliative behavior. The literature on genotypes that are related to monoamine neurotransmitter systems (i.e. serotonin, dopamine) is growing but is still comparatively small, and the literature linking genotypes with other biological systems is even smaller. Second, variation at any single location in the genome explains a surprisingly small proportion of prosocial phenotypic variance. Indeed, the largest effects rarely surpass more than a few percentage points of variability explained. The incongruity between the large overall genetic (i.e. entire genome) effects reported in quantitative genetic studies and the diminutive—sometimes vanishingly small—effect sizes emerging in molecular genetic studies is termed the *missing heritability* problem. This problem also highlights that quantitative and molecular genetic study designs have complementary strengths and weaknesses. Specifically, whereas quantitative genetics is capable of detecting robust genetic effects but cannot reveal the precise locations in the genome that are

Table 9.1 Key behavior genetic findings in research on prosocial behavior

Quantitative genetics	*Molecular genetics*
• Like other personality traits, prosociality is moderately heritable • Heritability estimates range from about 0% to about 50% • Like other cognitive and personality phenotypes, the heritability of prosocial behavior increases with age • The shared environment, which includes parenting, appears to have a small influence on prosocial behavior, and this effect declines with age	• Genes regulating oxytocin, vasopressin, serotonin, and dopamine are implicated in prosocial outcomes • The influence that any single gene has on prosociality is small and sometimes infinitesimal • As in other molecular genetic research areas, reproducibility of results for prosocial behavior is poor • Through genome-wide association studies, researchers can now simultaneously examine associations between thousands of genetic polymorphisms and prosociality

responsible for behavior, molecular genetics can pinpoint specific genes, but these genes seem to have only small—and perhaps not practically significant—effects on behavioral outcomes.

Third, readers may have noticed inconsistency across molecular genetic studies of a given phenotype. For instance, the G allele at OXTR rs53576 was found to be associated with both increased and diminished prosociality, and also to have no effects on prosociality. Failures to replicate are endemic to molecular genetic research in psychology (Duncan & Keller, 2011), and the track record of irreproducibility has inspired greater attention to good research practices (e.g. adequate statistical power, replication in independent samples). Perhaps the most prominent example of inconsistency in molecular genetic research is represented by the explosion of rep-lication attempts for 5-HTTLPR gene-environment interaction in depression that has yielded many positive and negative results, along with contradictory meta-analytic results (Karg, Burmeister, Shedden, & Sen, 2011; Risch et al., 2009). These mixed results in genetic studies of prosociality, and the broader context of replication difficulties in molecular genetics, point to the importance of replica-tion in studies on prosocial phenotypes and to exploring new avenues for genetic research, some of which are reviewed in the next section.

Future directions

What future developments can we expect in molecular genetic research on prosociality? One trend that is likely to advance research on prosocial phenotypes involves the simultaneous analysis of multiple polymorphisms in the same gene or across different genes. This practice is common in more mature genetic literatures and was inspired in part by the small effect sizes observed for any one polymorphism

in isolation and, in addition, by the fact that genotypes at contiguous locations in the genome tend to be correlated (due to Mendel's law of segregation). As a result, some researchers are creating *haplotypes*, or combinations of nearby polymorphisms, to try and explain more variation in prosocial phenotypes. Other researchers are simply adding up the number of "risk" alleles in a given gene—or across a number of genes in the same biological pathway, such as the OT system—to create a *polygenic risk score* (e.g. Schneiderman, Kanat-Maymon, Ebstein, & Feldman, 2014; Tabak et al., 2014).

The ultimate example of analyzing multiple polymorphisms at the same time is the *genome-wide association study* (GWAS). Compared with hypothesis-based candidate gene research, which is driven by existing theory about the biological systems that support a given phenotype, GWAS is atheoretical. Because the cost of conducting genetic assays is falling quickly, researchers can now examine associations between thousands of polymorphisms and a particular phenotype. At the same time, though, the GWAS approach requires very large samples and oftentimes data sharing within a large research consortium. As a result, this method is starting to yield replicable genetic risk markers for cognitive ability and some psychiatric disorders (e.g. Plomin & Deary, 2015), but we are not presently aware of any studies that have used this methodology for studying the genetic bases of prosocial attitudes or behaviors.

Like GWAS, meta-analysis offers a way to enhance power to detect small genetic effects. Meta-analysis combines data from various original empirical studies to establish an effect size estimate based on all available data. This technique has been important for making sense of molecular genetic literatures for other personality and clinical phenotypes (e.g. Karg et al., 2011). However, we are aware of only one meta-analysis of molecular genetic effects on prosocial outcomes. Specifically, Bakermans-Kranenburg and van Ijzendoorn (2014) summarized evidence relating OXTR rs53576 and rs2254298 to prosocial outcomes in biological, subjective, and behavioral domains. This analysis included more than 17,000 participants for rs53576 and 13,000 participants for rs2254298, and it revealed that the overall effect size estimates are not significantly different from zero for any domain. This result calls into question the importance of OXTR variation for real-world prosocial outcomes. It also suggests that additional meta-analyses involving OXTR and other relevant genes are warranted.

Finally, to fully understand how genes cause prosocial phenotypes, it will be necessary to elucidate the entire biological pathway underlying these phenotypes. Specifically, researchers will need to identify the full set of psychological and biological mechanisms in the causal chain from distal genes to proximal prosocial outcomes. Fully integrated, multi-level models have been proposed for other outcomes, such as stress-related disorders and depression (e.g. Slavich & Cole, 2013; Slavich & Irwin, 2014), but not yet for prosocial phenotypes.

Critical for advancing this scientific strategy is the identification of intermediary processes, sometimes called *endophenotypes*, which occur downstream of gene action (Kendler & Neale, 2010). Prior molecular genetic work has identified

some possible endophenotypes for prosocial behavior. For example, Tost et al. (2010) correlated the OXTR rs53576 A allele, which in other research predicted social deficits associated with diminished empathy, with amygdala responses to images of emotional faces. Because the amygdala is involved in socioemotional information processing, altered neural dynamics in this brain region that are mediated by OXTR could potentially account for genetic effects on amygdala-dependent social behaviors and attitudes. Similarly, in the study reviewed here that asked participants to judge the pain experiences of live confederates (Laursen et al., 2014), variation at OXTR was associated with activation in the superior temporal sulcus during the appraisal of others' pain. The implication, therefore, is that OXTR may influence empathic cognitions and behaviors through its regulation of activity in the superior temporal sulcus.

Sympathetic nervous system activity is another possible mechanism linking OXTR with downstream cognitive and behavioral prosocial phenotypes. Evidence supporting this link comes from a study that examined participants' electrodermal activity (i.e. galvanic skin response) while they watched a violent mixed martial arts fight (Smith, Porges, Norman, Connelly, & Decety, 2014). Specifically, participants with the rs53576 G allele exhibited greater electrodermal activity during the fight compared to A allele homozygotes, indicating greater relative sympathetic arousal. A second study of OXTR and indices of the sympathetic nervous system presented females with the sounds of infants crying (Riem, Pieper, Out, Bakermans-Kranenburg, & van Ijzendoorn, 2011). Here, women with the rs53576 G allele exhibited greater heart rate responses to the cry sounds than A homozygotes.

In sum, the molecular genetic literature provides important new information about the exact genetic variants underlying large omnibus genetic effects documented in twin studies. Nonetheless, an imperfect track record of replication and consistently small effect sizes highlight the importance of exploring complementary and alternate methodologies. Haplotype analysis, GWAS, and meta-analysis all promise to enhance gene-hunting efforts, and the greatest advancements will likely be realized by integrating information from each of these strategies. Finally, establishing endophenotypes for prosocial behavior can simultaneously maximize genetic effect sizes and help delineate the mechanisms linking candidate genes and prosocial behavior. In this context, though, new models of prosociality are needed that identify the full set of psychological and biological mechanisms that link specific genes with prosocial behavior.

Summary and conclusions

In summary, decades of quantitative genetic research have revealed that compassion and closely related prosocial phenotypes are moderately heritable. Thus, some (but not most) individual differences in prosocial behavior are attributable to genetic differences. Perhaps unexpectedly, one of the main lessons learned from this body of quantitative genetic research involves the importance of environmental experiences in shaping prosocial attitudes and behavior. The most important

sources of environmental effects, however, may be surprising. Specifically, twin studies have shown that the influence of the home environment (including parenting styles) on prosocial behavior is small, whereas environments not shared across siblings (e.g. distinct friendship groups, romantic partnerships) typically explain the lion's share of the non-genetic variation in prosocial outcomes.

Molecular genetic research, in turn, has implicated several neurotransmitter systems in prosociality. Polymorphisms in genes regulating oxytocin and arginine vasopressin have been associated with prosocial outcomes, although the effect size estimates are variable and usually small. Genes supporting serotonin and dopamine neurotransmission have also been linked to prosocial behavior. To resolve the missing heritability problem, new molecular genetic techniques and larger sample sizes will very likely be prioritized in future research on the genetics of compassion and other prosocial behaviors.

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