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The genetics of morality and prosociality

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Genetically informed research of phenotypes related to morality has proliferated rapidly in the last few years, sparking paradigm shifts from theories based solely on socialization toward ones integrating biological influences. Here, we review recent genetic research in the area of morality that has received the most attention in genetic studies: prosociality — positive emotions, attitudes, and behaviors directed toward others. Individual differences in prosociality emerge early in life, increase in heritability as children develop, and are related to variation in genes regulating neurotransmitter systems central to social affect, cognition, and behavior. The majority of molecular genetic studies have been candidate-based, however genome-wide studies are emerging, with the potential to elucidate novel biological pathways associated with individual differences in morality.

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Introduction

People vary markedly in what they appraise to be right from wrong; in how they experience or report emotions that motivate moral action; in the extent to which these moral intentions override self-oriented preferences; and in the degree by which they behave consistently with their morals. Understanding the causes behind this heterogeneity in moral sentiment and behavior continues to be a topic of profound interest across the social sciences. Genetically informed research of phenotypes related to morality has proliferated rapidly in the last few years in psychology, economics, and political science, sparking paradigm shifts from theories focused on socialization toward ones integrating biological influences [1,2••]. The phenotype that has received by far the most attention in genetic research on morality is prosociality — positive emotions, attitudes, and behaviors directed toward others. Prosociality is a compelling model phenotype because it shares phylogenetic origins with other primates and, in humans, it is amenable to measurement in both children and adults [3].

Measurement of genetic factors contributing to prosociality

Like morality, measurement of prosociality is multidimensional, spanning affect, cognition, attitude, and behavior [4•]. At the affective level, prosociality includes empathy — an other-oriented affective tendency to comprehend and share the emotional states of others [5••]. At the cognitive level, aspects of Theory of Mind — the ability to appreciate others' mental states and to understand that others have beliefs, intentions, and perspectives that are different from one's own — may be necessary for some prosocial behaviors [6]. At the attitudinal level are values such as benevolence — regard toward the welfare of close contacts, and universalism — regard for the welfare of all people and for nature [7]. Finally, at the behavioral level is prosocial-behavior — voluntary behavior intended to benefit another [5••]. Fitting the multidimensionality of this construct, multiple modes of measurement (self-report and informant-report, experimental decision making tasks, brain imaging) are regularly employed to observe how genetic effects relate to individual differences in prosociality. The bulk of this research has relied on quantitative-genetic methods, however rapid advancements in the feasibility of genotyping has spurred findings based on molecular genetic techniques. The integrative picture emerging from these research programs is one of individual differences in prosociality emerging early in life, increasing in heritability as children develop, and related to variations in genes regulating neurotransmitter systems central to social affect, cognition, and behavior [3]. Research examining the synergistic processes by which genes and the environment together influence prosociality is also underway.

Most quantitative research partitioning the variance for morality phenotypes into genetic and environmental factors has traditionally relied on the twin design. In this design, the covariance between scores for a given phenotype is compared between identical (monozygotic) twins — who share virtually all of their genetic sequence — and fraternal (dizygotic) twins — who share, on average, half of their genetic variance. Assuming that MZ and DZ twins are equal in terms of how similar their environments are, then greater MZ twin concordance indicates a genetic basis for the measured phenotype (*heritability*). Similarity beyond this genetic effect is attributed to environmental influences making siblings similar (*shared environment effect*), and any differences

between twins not due to genetic differences are ascribed to *non-shared environmental effects* and measurement error [8]. Estimates of genetic and environmental effects in this variance partitioning approach are context specific; restricted to a study sample's population, age, and culture. For reviews regarding the related question of morality as an evolutionary adaptation, that is, the extent to which brain systems involved in judging what is right and wrong advantaged natural selection, were the product of gene-culture co-evolution, or an emergent property of mankind's advanced intellectual ability, the reader is referred to the following Refs. [9–11].

The genetics of prosocial development

Prosocial development is characterized by pronounced individual differences in children's compliance, conscience development, empathy-related emotions, sharing, moral sensitivity, and moral judgment [5••]. The etiology of these individual differences is generally thought to be influenced by some combination of cultural values and practices, parenting and other social environmental factors such as peers and experiences in school, and genetics. With notable exception [12], twin studies examining prosociality in children have consistently found evidence for a significant role of both heritable and environmental factors, characterized by a systematic pattern of increasing heritability. For example, in studies examining individual differences in toddlers' cognitive and affective response to another's distress, genetic effects tend to be negligible in the first year of life but increase to account for nearly half of the variance by age 3 [13•]. In contrast, the shared environment effects on children's prosociality decrease sharply as children age, plunging from 50% in toddlerhood to less than 10% by age 7 [14], a period coinciding with the transition to formal schooling. This pattern of increasing heritability and decreasing shared environment is common to other morality constructs, however the timing of change is phenotype specific. For example, for political attitudes and religious values [15,16], a shift has been observed during the transition from adolescence to adulthood, a period marked by increased independence after leaving the home.

There are at least three non-exclusive reasons for the observed rise in heritability estimates. First, genetic effects may account for a greater portion of the variance as the frequency and persistence of shared environmental experiences decrease. Second, new genetic factors related to moral behavior may arise with or be mediated by children's developing cognitive skills, capacity for abstraction, and ability to internalize social experiences [17,18]. And third, heritability may increase as a result of environmental influences that enhance or trigger the expression of predisposed prosocial tendencies. Specifically, these are active gene–environment correlations (rGE), whereby individuals select into environments in accordance with their genetic propensities, and evocative rGE, whereby individuals evoke environmental responses that correspond with their own genetic tendencies [19]. For example, children's inherited prosociality is associated with receiving more warm and supportive parenting [20]. This reciprocal process suggests genetic factors catalyze accumulating differences in the quality and frequency of environments children are exposed to, amplifying pre-existing differences in prosociality.

Genetic research allows not only for estimating the heritability of single aspects of prosociality, but also for testing theories regarding the relations of theoretical constructs and the degree to which constructs have common or distinct roots. Individuals who rate higher in affective measures of prosociality, such as empathy, are more likely to exhibit prosocial behaviors [21]. A study using mother reports of different facets of children's prosociality found that social concern, kindness, helping, and empathic concern all inter-correlate positively, consistent with the notion of a single prosociality trait. This study revealed that the common prosociality factor had high heritability (69%) [22]. Importantly, there were also genetic effects specific to each of the facets, suggesting that different genetic factors differentiate among helping, sharing and the other facets.

Hypothesis-based molecular genetic studies

While twin studies can estimate the overall contribution of genetic factors to a given phenotype (or common genetic paths between phenotypes), they do not provide an indication of biological mechanism. For this, we turn to molecular genetic studies. Until a few years ago, the dominant model has been the candidate gene study, which examines association between prosociality phenotypes and genetic variants of known function (or genetic variants that lie in or near genes of known function) relevant to the biological systems hypothesized to play a role in prosociality. The overwhelming focus of candidate gene studies of prosociality has centered on common genetic variation in four systems of neurobiological relevance. These are the dopaminergic system, characterized by its important role in executive function, learning, and reward; the serotonergic system, linked to mood and inhibition; and the oxytocinergic and vasopressinergic systems, regulators of social cognition and behavior. Genetic variants in each of these systems have been implicated across the gamut of prosociality phenotypes, including empathy [23–25], Theory of Mind [26,27], and self-reported altruism [28,29]. Experimental economic games devised to elicit preferences for prosocial behaviors such as giving, and fairness have also observed associations with these genes and behavior [30–34], but see also [29,35]. Moral judgments have recently been examined as well [36,37]. Complementing this research and offering a window into mediating mechanisms are studies examining intermediate phenotypes, for example