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Commentary

Unselfish genes? The quest to uncover genomic influences on prosocial behavior

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In the current commentary, we discuss Stoltenberg and colleagues' finding (reported in this issue) that variation in the serotonin transporter gene (5-HTTLPR) is associated with prosocial behavior via effects on anxiety in social situations. We note how their results are consistent with evidence from the psychopharmacological literature and illustrate how a mediational framework can inform understanding of genetic and psychological associations, and we suggest that future studies that manipulate social context could further elucidate the relationship between genes involved in serotonin regulation and prosocial behavior.

Keywords: 5-HTTLPR; Serotonin; Prosocial behavior.

In 1976, Richard Dawkins made the provocative argument that genes are selfish. He argued that natural selection occurs at the level of the gene and that much prosocial behavior is a consequence of efforts to perpetuate genetic continuity across generations. Dawkins' theorizing is consistent with research showing that altruism, in many different species, tends to be directed toward kin (conspecifics who closely share one's genes) and other organisms who are likely to reciprocate helpful acts (Hamilton, 1964; Trivers, 1971). Modern humans, however, often live far from their families and routinely demonstrate acts of kindness toward strangers with no expectation of reciprocity. Thus, one of the challenges of contemporary research and theorizing about prosocial behavior has been to explain how genes contribute to seemingly unselfish behavior when there is arguably no benefit to oneself or genetic continuance.

In the current issue, Stoltenberg, Christ, and Carlo (2013) provide important insight into genetic influences on prosociality. They examined commonly occurring variation in the serotonin transporter gene, specifically the 5-HTTLPR along with an adjacent polymorphism that can moderate the effects of the L allele (Hu et al., 2006; Wendland, Martin, Kruse, Lesch, & Murphy, 2006) They found that this genetic variation (the 5-HTTLPR triallelic genotype) was associated with prosocial tendencies through effects on anxiety in social situations. Specifically, they found that carriers of the S′ allele were less likely than individuals homozygous for the L′ allele to help others and this effect was partially mediated by S′ allele

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carriers construing social situations as more threatening to their well-being. This finding suggests that genetic variation influences the extent to which people weigh self (and genetic) preservation concerns when making decisions about whether or not to help others.

The association between the 5-HTTLPR triallelic genotype and social anxiety found by Stoltenberg and colleagues (2013) is entirely consistent with evidence that drugs that inhibit the serotonin transporter, such as selective serotonin reuptake inhibitors (SSRIs), reduce symptoms of social anxiety disorder (Stein & Stein, 2008). Therefore, a logical extension of Stoltenberg and colleagues' model is that SSRI administration would increase prosocial behavior by reducing self-focused anxiety, which in turn, allows one to experience concern for others. In line with this prediction, work has shown that acute administration of the SSRI citalopram increases prosocial behavior by making people more sensitive to harming others (Crockett, Clark, Hauser, & Robbins, 2010). That both pharmacological manipulation of serotonin transporter functioning and variation in the serotonin transporter gene are related to prosocial behavior, albeit in slightly different ways, provides greater confidence in the findings by Stoltenberg and colleagues. This is especially relevant for the 5-HTTLPR literature because the precise cellular mechanisms by which the 5-HTTLPR influences serotonin neurotransmission has not been conclusively identified (Mann et al., 2000).

An impressive feature of the work by Stoltenberg and colleagues is that they go beyond simply reporting a genetic association with prosocial behavior and use a meditational model to delineate how serotonin transporter variation might exert its effect via social anxiety. Such a process-based framework has the potential to lay the foundation for future research that examines how social anxiety interfaces with other psychological motivators of prosocial acts, including empathy, compassionate goals, and specific moral values (Batson & Coke, 1981; Crocker & Canevello, 2011; Eisenberg, 1986; Schwartz & Howard, 1984). This work will be critical because each of these other drivers of prosocial behavior likely involve their own genetic pathways.

Another factor contributing to the complexity of understanding genetic contributions to prosocial behavior is the social context. Building on Stoltenberg and colleagues' finding that the 5-HTTLPR triallelic genotype is associated with social anxiety, it is theoretically conceivable that, depending on the context, the S′ allele could be negatively, positively, or not at all related to prosocial behavior. The majority of the items in the prosocial behavior questionnaire used by Stoltenberg and colleagues involve social interactions with strangers (e.g., "I have given a stranger a lift in my car" or "I have helped carry a stranger's belongings, books, parcels, etc."). Thus, it is to be expected that that prosociality was negatively related to the S′ allele because prosociality was largely assessed via scenarios that people who experience anxiety during interactions with strangers would actively avoid. In a different social context, such as one involving an in-group member, friend, or romantic partner whose presence elicits less social anxiety, 5-HTTLPR variation might not be related to helping behavior. Similarly, 5-HTTLPR genotype may not relate to a prosocial act like writing a check for a charity, which could create anxiety over forgone income, but does not involve the anxiety of a social interaction. It is even possible that in some situations, higher levels of 5-HTTLPR-associated social anxiety could lead to greater rather than less helping. For instance, when socially anxious people are unable to avoid an interaction, they might exhibit more prosocial behavior because social situations that elicit distress can activate a social monitoring system that heightens attunement to others' social cues (Pickett & Gardner, 2005). These scenarios underscore how the relationship between genetic variation and a particular behavior could change considerably based on the social context.

More broadly, the differential influences of contextual factors could contribute to the variable results seen in many genetic association studies. For this reason, knowledge derived from social psychology's rich tradition of examining situational influences on behavior could greatly benefit the design and conduct of genetic association studies. Thus, contextual factors should be added to the long list of variables (e.g., epigenetics, early life experience) that can influence the relationship between genetic variation and a phenotype (Ratner & Kubota, 2012; Way & Gurbaxani, 2008; Way & Taylor, 2010).

In sum, research into the relationship between genetics and prosocial behavior has made considerable advances since Dawkins and others began to posit a function of genes in promoting altruism. The current ability to examine variation in specific genes combined with a theoretical shift in focus from outcomes to process and from universality to context-specificity has the potential to lead to new breakthroughs. Such insight will be informative as people continue to grapple with how best to increase compassion and social support for other members of society.

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