

This article was downloaded by: [New York University]

On: 08 September 2013, At: 14:12

Publisher: Routledge

Informa Ltd Registered in England and Wales Registered Number: 1072954 Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Social Neuroscience

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/psns20>

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

Kyle G. Ratner^a & Baldwin M. Way^{ab}

^a Department of Psychology, The Ohio State University, Columbus, OH, USA

^b Institute for Behavioral Medicine Research, The Ohio State University School of Medicine, Columbus, OH, USA

Published online: 02 Sep 2013.

To cite this article: Social Neuroscience (2013): Unselfish genes? The quest to uncover genomic influences on prosocial behavior, Social Neuroscience, DOI: 10.1080/17470919.2013.832372

To link to this article: <http://dx.doi.org/10.1080/17470919.2013.832372>

PLEASE SCROLL DOWN FOR ARTICLE

Taylor & Francis makes every effort to ensure the accuracy of all the information (the "Content") contained in the publications on our platform. However, Taylor & Francis, our agents, and our licensors make no representations or warranties whatsoever as to the accuracy, completeness, or suitability for any purpose of the Content. Any opinions and views expressed in this publication are the opinions and views of the authors, and are not the views of or endorsed by Taylor & Francis. The accuracy of the Content should not be relied upon and should be independently verified with primary sources of information. Taylor and Francis shall not be liable for any losses, actions, claims, proceedings, demands, costs, expenses, damages, and other liabilities whatsoever or howsoever caused arising directly or indirectly in connection with, in relation to or arising out of the use of the Content.

This article may be used for research, teaching, and private study purposes. Any substantial or systematic reproduction, redistribution, reselling, loan, sub-licensing, systematic supply, or distribution in any form to anyone is expressly forbidden. Terms & Conditions of access and use can be found at <http://www.tandfonline.com/page/terms-and-conditions>

Commentary

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

Kyle G. Ratner^{1*} and Baldwin M. Way^{1,2*}

¹Department of Psychology, The Ohio State University, Columbus, OH, USA

²Institute for Behavioral Medicine Research, The Ohio State University School of Medicine, Columbus, OH, USA

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

Correspondence should be addressed to: Baldwin M. Way, 100G Lazenby Hall, 1827 Neil Avenue, Columbus, OH 43210, USA. E-mail: way.37@osu.edu

This project was supported in part by Award Number Grant [8KL2TR000112-05] from the National Center for Advancing Translational Sciences. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Center for Advancing Translational Sciences or the National Institutes of Health.

*These authors contributed equally to this work.

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 mediational 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.

REFERENCES

- Batson, C. D., & Coke, J. S. (1981). Empathy: A source of altruistic motivation for helping? In J. P. Rushton & R. M. Sorrentino (Eds.), *Altruism and helping behavior* (pp. 167–187). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Crocker, J., & Canevello, A. (2011). Egosystem and ecosystem: Motivational perspectives on caregiving. In S. E. Brown, M. Brown, & L. A. Penner (Eds.), *Moving beyond self-interest: Perspectives from evolutionary biology, neuroscience, and the social sciences* (pp. 211–223). New York, NY: Oxford University Press.
- Crockett, M. J., Clark, L., Hauser, M. D., & Robbins, T. W. (2010). Serotonin selectively influences moral judgment and behavior through effects on harm aversion. *Proceedings of the National Academy of Sciences*, *107*(40), 17433–17438.
- Dawkins, R. (1976). *The selfish gene*. New York, NY: Oxford University Press.
- Eisenberg, N. (1986). *Altruistic emotion, cognition, and behavior*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Hamilton, W. D. (1964). The genetical evolution of social behaviour. *Journal of Theoretical Biology*, *7*(1), 1–52.
- Hu, X., Lipsky, R. H., Zhu, G., Akhtar, L. A., Taubman, J., Greenberg, B. D., . . . Kennedy, J. L. (2006). Serotonin transporter promoter gain-of-function genotypes are linked to obsessive-compulsive disorder. *American Journal of Human Genetics*, *78*(5), 815.
- Mann, J. J., Huang, Y., Underwood, M. D., Kassir, S. A., Oppenheim, S., Kelly, T. M., . . . Arango, V. (2000). A serotonin transporter gene promoter polymorphism (5-HTTLPR) and prefrontal cortical binding in major depression and suicide. *Archives of General Psychiatry*, *57*(8), 729.
- Pickett, C. L., & Gardner, W. L. (2005). The social monitoring system: Enhanced sensitivity to social cues as an adaptive response to social exclusion. In K. D. Williams, J. P. Forgas, & W. von Hippel (Eds.), *The social outcast: Ostracism, social exclusion, rejection, and bullying* (pp. 213–226). New York, NY: Psychology Press.
- Ratner, K. G., & Kubota, J. T. (2012). Genetic contributions to intergroup responses: A cautionary perspective. *Frontiers in Human Neuroscience*, *6*.
- Schwartz, S. H., & Howard, J. A. (1984). Internalized values as motivators of altruism. In E. Staub, D. Bar-Tal, J. Karylowski, & J. Reykowski (Eds.), *Development and maintenance of prosocial behavior: International perspectives on positive morality* (pp. 229–253). New York, NY: Plenum Press.
- Stein, M. B., & Stein, D. J. (2008). Social anxiety disorder. *Lancet*, *371*(9618), 1115–1125.
- Stoltenberg, S. F., Christ, C. C., & Carlo, G. (2013). Afraid to help: Social anxiety partially mediates the association between 5-HTTLPR triallelic genotype and prosocial behavior. *Social Neuroscience*.
- Trivers, R. L. (1971). The evolution of reciprocal altruism. *Quarterly Review of Biology*, *46*(1), 35–57.
- Way, B. M., & Gurbaxani, B. M. (2008). A genetics primer for social health research. *Social and Personality Psychology Compass*, *2*(2), 785–816.
- Way, B. M., & Taylor, S. E. (2010). Social influences on health: Is serotonin a critical mediator? *Psychosomatic Medicine*, *72*(2), 107–112.
- Wendland, J., Martin, B., Kruse, M., Lesch, K., & Murphy, D. (2006). Simultaneous genotyping of four functional loci of human SLC6A4, with a reappraisal of 5-HTTLPR and rs25531. *Molecular Psychiatry*, *11*(3), 224–226.