CellPress

Current Biology
Dispatches

Social Behavior: A Penny for Your Shocks

Hayley M. Dorfman[1] and Joshua W. Buckholtz[1,2,*]
[1]Department of Psychology & Center for Brain Science, Harvard University, 52 Oxford Street Cambridge, MA 02138, USA
[2]Department of Psychiatry, Massachusetts General Hospital, 55 Fruit Street, Boston, MA 02114, USA
*Correspondence: joshuabuckholtz@fas.harvard.edu
http://dx.doi.org/10.1016/j.cub.2015.05.036

Antisocial behavior is an enormously costly social problem, but its origins are poorly understood. A new study shows that prosocial and antisocial behaviors arise from individual differences in how we represent the value of others' pain relative to our own potential gain, rather than from variability in the capacity for effortful inhibitory control.

> "Well there are good guys and there are bad guys
> And there are crooks and criminals
> There are doctors and there are lawyers
> And there are folks like you and me"
>
> Camper Van Beethoven, 1986

Human social behavior runs the gamut: from 'good guys' to 'bad guys', with folks like you and me in between. The existence of inter-individual variation in human moral behavior is self-evident; much less so are the biological mechanisms that drive such variability. Traditionally, cooperation and other forms of prosocial behavior have been thought of as reflecting the ability to 'put the brakes' on inherently selfish or self-interested responses. By extension, moral transgressions and antisocial behavior result when this inhibitory brake is compromised [1,2]. More recently, however, an alternative account of how we make decisions to help or hurt other people has emerged [3–5]: a key component of this model is the idea that people make decisions based on the subjective value of different choice options. Prosocial behavior arises when people place higher value on options associated with benefits to others versus self, while antisocial behavior reflects the opposite [4]. New work by Crockett *et al.* [6], reported in this issue of *Current Biology*, provides strong support for the notion that prosocial and antisocial behaviors arise from individual differences in how we represent the value others' pain relative to our own potential gain, rather than from variability in the capacity for effortful inhibitory control. Further, it sheds important new light on the neurobiological mechanisms that underlie such decision-relevant social value representations.

The experimental analysis of antisocial behavior has been stymied by a very basic measurement problem: how do we recreate, in the lab, the conditions under which people are incentivized to hurt others in real life? Crockett *et al.* [6] addressed this issue with an ingenious experimental design. On each trial in their task, participants played the role of 'decider' in a series of scenarios that required balancing the value of monetary gain for themselves against the value of avoiding causing physical pain for either themselves (50% of trials) or an anonymous 'receiver' (50%). Choice options were constructed as combinations of shocks and money, and the task was structured such that subjects chose between a 'default' option and an 'alternative' option. On half the trials, the alternative option signaled more shocks/more money; on the other half, the alternative option signaled fewer shocks/less money. This clever task feature allowed the authors to separately examine choices in which action versus inaction was required to reap the benefits of causing another more pain.

Another issue with tasks that putatively measure antisocial behavior or aggression is the cognitive opacity of their output variables, such as choice proportion and/or reaction time. Crockett *et al.* [6] skillfully employed a novel computational framework to finely parse task performance. This allowed them to distinguish between multiple latent components of decision-making, including the negative utility of causing harm (here, indexed by the parameter κ) to oneself (κ_{Self}) or to another (κ_{Other}). Prior work using this task produced the important finding that, on the mean, people will sacrifice more money to avoid causing pain to others compared to themselves (a phenomenon termed 'hyperaltruism' by the authors) [7].

In their new work, Crockett *et al.* [6] employed the harm aversion task as a framework to examine the causal biology of antisocial behavior, using pharmacological manipulations to disambiguate the roles of serotonin and dopamine in this phenomenon. Both neurotransmitters have long been implicated in antisocial behavior and aggression. For example, preclinical and human data have suggested that lowered serotonergic transmission predisposes aggression [8], but the nature of this linkage remains unclear [9]. Likewise, there is evidence that elevated dopaminergic function is associated with higher levels of impulsive antisocial behavior [10,11], but the underlying mechanisms for this effect have been difficult to resolve. In the new study [6], model-based behavioral parameters were compared between subjects who were given either citalopram (a serotonin-selective reuptake inhibitor), levodopa (which is metabolized centrally into dopamine), or placebo.

Crockett *et al.* [6] found that subjects given citalopram were willing to forgo larger amounts of money in order to avoid the administration of painful electric shocks to both themselves and another person, compared to subjects who received a placebo. In other words, transiently increasing central serotonin levels increases harm aversion for both self and other. However, modulating serotonin levels had no effect on hyperaltruism: the citalopram subjects

showed no difference in their degree of harm aversion for self versus other. By contrast, levodopa administration selectively reduced hyperaltruism, such that the typical pattern of increased harm aversion for others compared to self was abolished. Considering the known role of monoamines in motor function and response inhibition, it might be tempting to attribute this effect to shifts in response vigor or inhibitory control. However, the clever inclusion of action versus inaction trials (as noted above) permitted the authors to definitively rule out this alternative explanation.

These findings show that monoaminergic transmission influences prosocial and antisocial behavior by modulating how we represent and integrate value representations of outcomes for ourselves and others. Of course, no single study can do everything, and the systems-level mechanisms underlying these results remain unclear. Future pharmaco-fMRI and PET studies in humans could further illuminate the large-scale circuits and specific signal transduction pathways through which monoamines act to influence valuation during social decision-making. More work is needed to confirm the intriguing possibility, raised here and elsewhere [12], that social behavior is motivated by the same fundamental, domain-general mechanisms for value-learning and updating that drive non-social decision-making. It will be particularly important to explore how interactions between explicitly 'social' aspects of cognition (such as theory of mind and empathic resonance) and domain general valuation processes influence the mirrored representation of harm costs outside of self. That said, this work offers a strong rebuttal to inhibition-based 'brakes' accounts of social decision-making, and sheds important new light on the manner by which serotonergic and dopaminergic signaling shape social behavior.

REFERENCES

1. Baumeister, R.F. (2014). Self-regulation, ego depletion, and inhibition. Neuropsychologia *65*, 313–319.
2. Knoch, D., and Fehr, E. (2007). Resisting the power of temptations: the right prefrontal cortex and self-control. Ann. NY Acad. Sci. *1104*, 123–134.
3. Zaki, J., and Mitchell, J.P. (2013). Intuitive prosociality. Curr. Dir. Psychol. Sci. *22*, 466–470.
4. Buckholtz, J. (2015). Social norms, self-control, and the value of antisocial behavior. Curr. Opin. Behav. Sci. *3*, 122–129.
5. Crockett, M.J. (2013). Models of morality. Trends Cogn. Sci. *17*, 363–366.
6. Crockett, M.J., Siegel, J.Z., Kurth-Nelson, Z., Ousdal, O.T., Story, G., Frieband, C., Grosse-Rueskamp, J.M., Dayan, P., and Dolan, R.J. (2015). Dissociable effects of serotonin and dopamine on the valuation of harm in moral decision making. Curr. Biol. *25*, 1852–1859.
7. Crockett, M.J., Kurth-Nelson, Z., Siegel, J.Z., Dayan, P., and Dolan, R.J. (2014). Harm to others outweighs harm to self in moral decision making. Proc. Natl. Acad. Sci. USA *111*, 17320–17325.
8. Duke, A.A., Begue, L., Bell, R., and Eisenlohr-Moul, T. (2013). Revisiting the serotonin-aggression relation in humans: a meta-analysis. Psychol. Bull. *139*, 1148–1172.
9. Buckholtz, J.W., and Meyer-Lindenberg, A. (2014). Genetic perspectives on the neurochemistry of human violence and aggression. In The Oxford Handbook of Molecular Psychology, T. Canli, ed. (Oxford University Press).
10. Buckholtz, J.W., Treadway, M.T., Cowan, R.L., Woodward, N.D., Benning, S.D., Li, R., Ansari, M.S., Baldwin, R.M., Schwartzman, A.N., Shelby, E.S., *et al*. (2010). Mesolimbic dopamine reward system hypersensitivity in individuals with psychopathic traits. Nat. Neurosci. *13*, 419–421.
11. Couppis, M.H., Kennedy, C.H., and Stanwood, G.D. (2008). Differences in aggressive behavior and in the mesocorticolimbic DA system between A/J and BALB/cJ mice. Synapse *62*, 715–724.
12. Buckholtz, J.W., and Marois, R. (2012). The roots of modern justice: cognitive and neural foundations of social norms and their enforcement. Nat. Neurosci. *15*, 655–661.

Chromosome Segregation: A Spatial Code to Correct Kinetochore–Microtubule Attachments

Julie K. Monda[1,2] and Iain M. Cheeseman[1,2,*]
[1]Whitehead Institute for Biomedical Research, Cambridge, MA, USA
[2]Department of Biology, MIT, Nine Cambridge Center, Cambridge, MA 02142, USA
*Correspondence: icheese@wi.mit.edu
http://dx.doi.org/10.1016/j.cub.2015.05.056

Erroneous kinetochore–microtubule interactions must be detected and corrected before a cell enters anaphase to prevent chromosome mis-segregation. Two new studies describe an Aurora A-mediated error correction mechanism based on the spatial position of a chromosome within the mitotic spindle.

Faithful segregation of the chromosomes to each daughter cell is an essential feature of cell division. Failure to accurately distribute the genomic material can result in aneuploidy and can have catastrophic consequences for the viability of a cell or organism. To segregate the chromosomes, microtubules emanating from the spindle poles must attach to the DNA through the kinetochore, a large, multi-protein complex assembled at the centromere of each chromosome. Proper segregation relies on the attachment of each replicated sister chromatid (or, in meiosis I, each homologous chromosome)

