Moral bioenhancement: a neuroscientific perspective

Molly J Crockett

Can advances in neuroscience be harnessed to enhance human moral capacities? And if so, should they? De Grazia explores these questions in ‘Moral Enhancement, Freedom, and What We (Should) Value in Moral Behaviour’.1 Here, I offer a neuroscientist’s perspective on the state of the art of moral bioenhancement, and highlight some of the practical challenges facing the development of moral bioenhancement technologies.

The science of moral bioenhancement is in its infancy. Laboratory studies of human morality usually employ highly simplified models aimed at measuring just one facet of a cognitive process that is relevant for morality. These studies have certainly deepened our understanding of the nature of moral behaviour, but it is important to avoid overstating the conclusions of any single study. De Grazia cites several purported examples of ‘non-traditional means of moral enhancement’, including one of my own studies. According to De Grazia, we showed that ‘selective serotonin reuptake inhibitors (can be used) as a means to being less inclined to assault people’. In fact, our findings are a bit more subtle and nuanced than implied in the target article, as is often the case in neuroscientific studies of complex human behaviour. In our study, we tested the effects of the selective serotonin reuptake inhibitor (SSRI) citalopram on moral judgments about hypothetical scenarios, and on behaviour in an economic game. In the hypothetical scenarios, we found that citalopram made people less likely to judge it morally acceptable to harm one person in order to save many others. In the economic game, citalopram made people less likely to reduce the payoffs of other people who behaved unfairly toward them. We interpreted these results as evidence that serotonin enhances the aversiveness of harming others—either imagined harms (in the case of the hypothetical scenarios) or economic harms (in the case of the economic game).2 While our findings are consistent with the idea that SSRIs could reduce people’s inclination to assault others, to my knowledge this has not yet been demonstrated in the laboratory in healthy volunteers (and indeed would be quite difficult to implement, practically and ethically speaking). Clinical research has shown that SSRIs can be useful for treating aggressive behaviour, but only in certain types of patients; serotonin appears to be involved more in reactive, impulsive aggression (eg, as seen in personality disorders) than in premeditated aggression (eg, as seen in psychopathy).3 Far more research is needed before we fully understand the role of serotonin in aggression, and how serotonin interventions might be used to reduce individuals’ propensities towards harming others. The same caution should be applied to many of the other examples cited. For instance, a recent comprehensive genome-wide association study of 10 000 individuals casts substantial doubt on whether single genes can significantly predispose people towards, for example, fairness or altruism,4 despite the enthusiasm generated by initial studies in much smaller samples. We must be careful not to draw premature conclusions about potential avenues for moral bioenhancement.

However, for the sake of argument, suppose we were to amass a body of evidence that a single neurotransmitter (eg, serotonin) reliably and substantially reduced people’s propensity to physically harm others. Before we pull out the

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prescription pads, it will be important to consider the potential unintended consequences of altering the function of that neurotransmitter, beyond the desired effects on moral behaviour. Most neurotransmitters serve multiple functions and are found in many different brain regions. For example, in addition to its involvement in social behaviour, serotonin plays a role in a variety of other processes, including (but not limited to) learning, emotion, vision, sexual behaviour, appetite, sleep, pain and memory, and there are at least 17 different types of serotonin receptors that produce distinct effects on neurotransmission. Thus, interventions that affect moral behaviour by globally altering neurotransmitter function may have undesirable side effects, and these should be considered when weighing the costs and benefits of the intervention. Of course, it is plausible that advances in technology could minimise the possibility of side effects. One could imagine sophisticated drug delivery systems that target only specific receptor types in specific brain regions. While such technologies could counteract the issue of unintended side effects, De Grazia argues that highly selective and targeted forms of moral bioenhancement may pose a greater threat to freedom. Thus, in developing moral enhancement technologies, we may face a tradeoff between minimising undesirable side effects on the one hand, and minimising threats to freedom on the other.

Finally, De Grazia distinguishes between the enhancement of moral motivation, moral cognition and moral behaviour. From a neuroscientific perspective, the evidence so far suggests that targeting moral motivation may be the most promising avenue for promoting moral behaviour. For instance, my colleagues and I compared the effects of the SSRI citalopram with the effects of the noradrenaline reuptake inhibitor atomoxetine on moral judgment and behaviour. Atomoxetine could be described as a ‘cognitive enhancer’; in our study, it improved performance on tasks requiring sustained attention, and other studies have shown that it enhances the ability to control one’s actions. Our results suggested that citalopram affected moral judgment and behaviour through a motivational channel, by increasing harm aversion. In contrast, atomoxetine did not significantly alter moral judgment or behaviour, despite its beneficial effects on cognitive function. Further support for the primacy of motivational processes in moral behaviour comes from studies of psychopaths. Although psychopaths engage in morally inappropriate behaviour, their ability to distinguish right from wrong appears to be intact, suggesting that moral transgressions in psychopaths are caused by deficits in moral motivation. Similar, albeit less severe, motivational deficits may contribute to everyday acts of immorality. Future research is needed to identify the specific types of motivational processes that contribute to moral behaviour, and to uncover their neurobiological mechanisms.

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