Emerging behavioral, genetic, pharmacological and neuroimaging data indicate that the neural and molecular mechanisms that guide behavioral change in reaction to positive and negative feedback can be dissociated, albeit, not fully (van Duijvenvoorde et al., 2008; Klein et al., 2007; Robinson et al., 2010; Pizzagalli et al., 2008). This dissociation informs our understanding of addictions, where sensitivity to incentive outcomes appears to be increased, while sensitivity to negative outcomes is diminished (Belin et al., 2008; Bjork et al., 2008). Computational models have provided a testable framework for relating these molecular and neural mechanisms to behavior (Frank et al., 2004).

Piray (2011) comments on our recent work that demonstrated that individual differences in behavioral sensitivity to positive, but not negative, feedback during discrimination learning and reversal were related – inversely – to dopamine D_2 receptor availability, measured with micro-positron emission tomography in adult vervet monkeys (Groman et al., 2011). Piray implements a computational model to test some of our conclusions. The model rests on a few assumptions: 1) that dopamine receptor availability, measured with PET, is equivalent to receptor number and/or function, 2) that greater negative prediction error necessarily means enhanced sensitivity to positive feedback and 3) that initial acquisition and reversal learning differ in that negative prediction errors loom large in reversal but are negligible in acquisition. Though none of these assumptions are necessarily empirically correct, the model, which posits that D_2 receptor levels/function are equivalent to the negative prediction error, appears to generate data that are consistent with our observation of an association between D_2 receptor availability and sensitivity to positive feedback (Figures 1A and 1B from Piray [2011]).

Several of our reported observations, however, are not consistent with the assumptions of the model, or its application here. First, the observed association between D_2 receptor availability and sensitivity to positive feedback was present in both initial acquisition and reversal learning, even though Piray (2011) proposes that negative prediction errors are absent during initial learning. Therefore, according to the model, there should be no relationship between D_2 function and sensitivity to positive feedback during the acquisition stage. In addition, although Piray's results propose that D_2 receptor levels/function should vary with sensitivity to negative feedback (see Figure 1C in Piray [2011]), we found no evidence of this, despite having ample range of individual differences in this measure to capture a relationship if it existed.

Our results suggest that the contributions of D_1 and D_2 receptor systems to reinforcement learning may not be as dissociable as previously proposed (Frank et al., 2004). Future studies examining the relationship between D_1 and D_2 receptor systems and reinforcement learning measures, including tests that allow estimations of learning rate parameters or that incorporate feedback-based analyses, will clarify the unique and collective influences these receptor systems exert on reinforcement learning.

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