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Dopey dopamine: high tonic results in ironic performance

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Financial incentives are commonly used as motivational tools to enhance performance. Decades of research have established that the neurotransmitter dopamine (DA) is the fuel that propels reward-motivated behavior, yet a new PET study questions whether dopamine is beneficial to performance, showing that tonic DA synthesis predicts performance decrements when incentives are high.

Money is a powerful motivator: it is what gets most of us out of bed to do monotonous jobs and motivates us to work harder and longer. The promise of money, and the drive to pursue it, is fuelled by a primitive set of neurotransmitters, most notably DA. A large body of research from the fields of behavioral neuroscience, primate neurophysiology, and human neuropharmacology converged on the finding that DA is involved in reward seeking and reward learning. DA is a simple chemical, yet its effects are complex. For example, questions still remain concerning how DA relates to impulsivity in attention deficit hyperactivity disorder (ADHD), how DA neurons generate prediction errors, or how DA neurons interact with nonDA neurons. A recent study by Aarts *et al.* [1] adds to the debate by showing that DA may not always be beneficial to reward seeking and may in fact impair performance.

Aarts and colleagues used PET imaging to estimate baseline DA synthesis in the striatum. Subjects performed a modified Stroop task requiring focused attention and significant cognitive demand and monitoring to inhibit prepotent responses and to respond quickly and accurately to cues. Just before a trial, a potential reward amount and information regarding the congruency of the stimulus was presented to participants. Then, subjects were presented a word, 'LEFT' or 'RIGHT', embedded in an image of a right-pointing arrow and instructed to press a button with either their right index finger (indicating left) or right middle finger (indicating right), corresponding to the word. A successful trial resulted in receiving the indicated reward and required the subject to provide both the correct response and within a specified amount of time. Participants with greater DA synthesis capacity as measured by fluoro-L-*m*-tyrosine (FMT) in the striatum (left caudate) had slower latencies on higher reward incongruent trials in which they were not provided information about congruency and, hence, could not prepare before stimulus presentation.

The results of Aarts and colleagues support a growing body of literature showing the paradoxical effect of high incentive=induced impairments on performance [2–4]. For example, a functional MRI study by Mobbs and colleagues [4] showed midbrain activity-dependent performance decrements in a high incentive context, implying a potential role for dopaminergic signaling in modulating performance; the authors theorized that overmotivation may result in conscious monitoring of otherwise efficient automatic processes. A later study by Chib *et al.* [3] suggested that incentives paired with performance on a task are initially encoded as a potential gain reflected by increased ventral striatal activity, but shift to loss processing represented by decreases in activity when subjects perform a task. Aarts and coworkers extend on these studies by being the first to implicate DA directly in performance impairments (Figure 1).

The elegant adaptation of the Stroop paradigm used by Aarts *et al.* [1] falls squarely into the domain of tasks that require conflict monitoring. Under the conflict hypothesis, the anterior cingulate cortex is involved in monitoring conflict, and activity in this region predicts an ensuing increase in right dorsolateral prefrontal cortex (dlPFC) activity, presumably to engage cognitive control mechanisms that modulate attention [5]. The cognitive conflict system may compete for capacity-constrained attentional resources with the dopaminergic reward and arousal systems, where individuals with higher DA synthesis capacity exhibit a greater degree of interference in cognitive control. However, is that all there is to it? The vast distributed networks involved in cognitive control, attention, arousal, reward processing, and action selection involve several neurotransmitters in addition to DA. For example, attention networks in the PFC are innervated by catecholaminergic inputs, including norepinephrine (NE) innervation from the locus coeruleus and DA inputs from the striatum. Furthermore, the NE system is strongly implicated in attentional shifting through interaction with the medial PFC [6]. Together, these studies point to a larger set of interacting and competing neural circuits, and propose a more complex explanation for why high incentives negatively impact performance.

A potentially remarkable result of the Aarts *et al.* [1] study is that task incentives, which are not particularly high in their study, result in performance impairments. The largest possible single trial gain was US$0.15 and the

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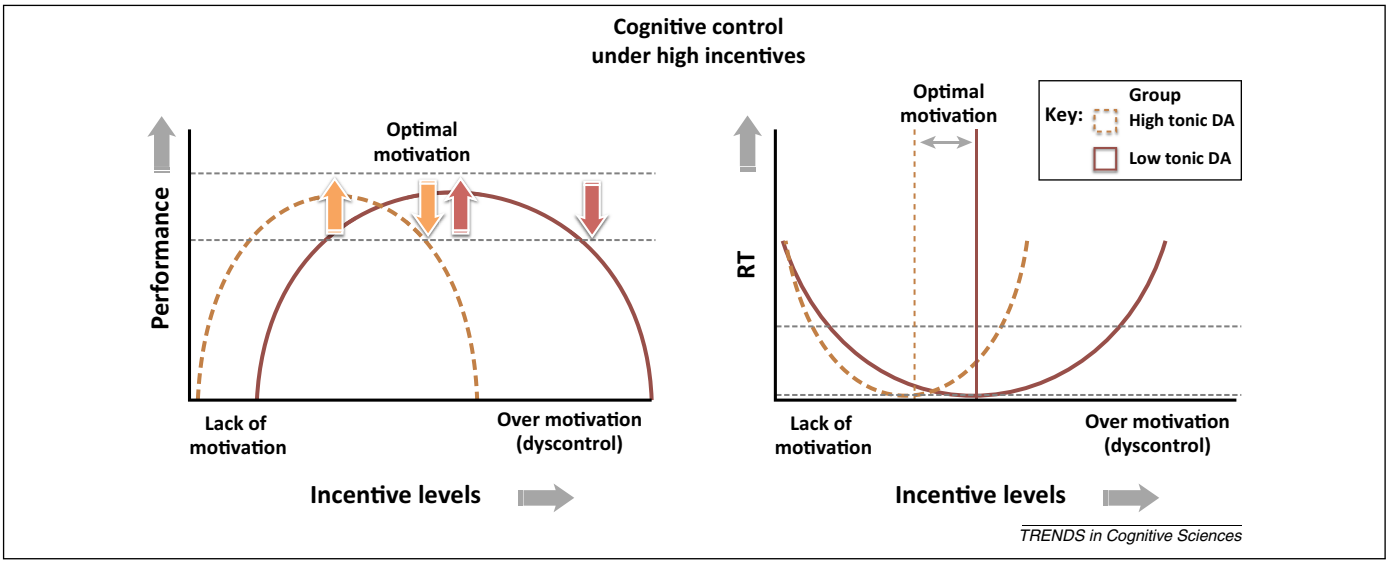

Figure 1. The DA-dyscontrol shift. The left panel shows the hypothetical ''Inverted-U-shaped'' function of low tonic DA (red line) showing that high tonic DA (broken curve) may result in a faster shift to dyscontrol. The right panel illustrating the U-shaped curve for the reaction time (RT) showing that high tonic DA should result in faster switches to dyscontrol, whereas low tonic DA results in slower shift to motivated behavior and dyscontrol. Adapted from [10].

smallest a penny, representing a spread of outcomes in which the maximum reward was 15 times greater than the smallest. These rewards would hardly motivate any working person in a developed country to get out of bed in the morning. Although other research paradigms have used similar spreads, including Mobbs and colleagues [4], who used a 10:1 payout ratio or the 100:10:1 ratio used by Ariely *et al.* [2], these studies have evoked similar performance decrements with much larger rewards. For example, Mobbs and colleagues [4] used a minimum of approximately US$1 and maximum payout of US$10, whereas Ariely *et al.*'s study [2] conducted in India, used a dramatic 4, 40, or 400 Indian Rupees, with 495 Rupees being the average monthly wages for that region. The findings of Aarts *et al.* [1] imply that it is not the magnitude of money that evokes performance impairments, but the relative difference in possible rewards, the processes involved in maximizing them, as well as the type of task being performed.

The impressive results of Aarts *et al.* transcend the industrial or sporting repercussions by identifying some of the neural mechanisms that are impaired in individuals with aberrant DA functioning. DA abnormalities have been associated with several conditions, including Parkinson's disease (PD), schizophrenia, Tourette's syndrome, and ADHD [7]. Response selection is commonly modeled in terms of direct and indirect 'Go' and 'NoGo' pathways associated with D1 and D2 receptors, respectively. A bias towards Go learning is observed in some patients with PD taking L-Dopa, manifesting as impulsivity [8], including increased gambling behaviors. High tonic DA levels are believed to magnify the 'Go' pathway and facilitate expedited responses via stimulus loading into working memory, making it more difficult to inhibit prepotent responses. Other deficits include impaired response inhibition is observed in schizophrenia, impulsivity in ADHD, and excess excitability in the striatal Go pathway has been hypothesized in Tourette's syndrome [9]. Given that DA levels alone are not sufficient to produce impulsive behavior or account for the range of observed deficits, an approach that takes into account the broader circuitry and temporal dynamics will likely be required to better explain these deficits and performance decrements. However, Aarts *et al.* [1] have taken the first important step in answering these questions.

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