The Effects of Modafinil on Motivation and Salience of Pleasure in Healthy Individuals: Quantitative Evidence From the Cognitive Neurosciences

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The article by Kjærsgaard (2015) discusses enhancing motivation by use of prescription stimulants: the ethics of motivation enhancement. The article is very interesting in that it focuses on how enhancing the psychological process of motivation with psychostimulants may raise ethical concerns.

The article makes the assumption that stimulants actually enhance motivation and goes on to evaluate the ethical issues that may arise from this assumption. However, the article uses very limited qualitative evidence and it cites opinion articles to support the view that psychostimulants enhance motivation in healthy individuals. The evidence used to support this assumption is limited and needs further support from the cognitive neuroscience literature.

In what follows, I describe quantitative evidence from modafinil studies showing that psychostimulants do not actually improve motivation in healthy individuals but they increase the perceived judgment and confidence in performing better psychological tasks, and they increase salience of pleasure, which makes boring tasks look more interesting, even though task performance is not actually improved.

Motivation can be defined as a behavior that an organism displays even in the face of barriers or contingencies. Nonetheless, motivation consists of several cognitive and affective components, and stimulant medications influence these processes in a complicated manner. For instance, Volkow and Swanson (2008) argue that stimulants amplify the activity of the neurotransmitter dopamine (DA) and modulate salience pleasure, incentive motivation, and reward, making cognitive tasks and everyday activities seem more interesting and rewarding.

Drugs of abuse such as amphetamines increase perceived confidence of ability without improving cognitive performance. For example, cocaine leads to the release of large amounts of DA into the reward systems of the brain,
which elicit powerful reinforcements and reward effects that eventually lead to escalation of use, abuse, and addiction (Koob and Volkow 2009).

Modafinil is a more recent drug licensed for the treatment of narcolepsy and sleep apnea, and has been reported to be used by healthy individuals as a cognitive enhancer. Despite the high level of reported off-label use of modafinil as a putative cognitive enhancer (Mohamed and Sahakian 2012), the mechanisms by which modafinil modulates cognitive and affective processes in healthy non-sleep-deprived neurotypical individuals are currently unknown (Mohamed 2014a) and need to be thoroughly investigated (Mohamed 2012a; Mohamed 2012b; Mohamed 2014b; Mohamed 2014c).

Furthermore, although positron emission tomography studies with both rhesus monkeys and healthy male human participants have shown that modafinil acts on brain areas that are associated with reward and reinforcement (i.e. the caudate, nucleus accumbens, and putamen; Volkow et al. 2009), according to the reviews of modafinil very limited cognitive and behavioral evidence exists on its effects on motivation in healthy individuals (Lynch, Palmer, and Gall 2011; Repantis et al. 2010). A recent behavioral study by Stoops and colleagues (2005) showed that modafinil had a stimulant-like reinforcement effect in healthy individuals. Stoops and colleagues (2005) gave participants who took modafinil or placebo the chance to rest or perform a mathematical problem solving task. Participants who took modafinil preferred to solve the mathematical problems and worked significantly more in order to earn more modafinil tablets, to enable them to work even more on the mathematical problems. The study suggests that modafinil had reinforcement effects, which is different from increasing the motivation to perform a task in a straightforward manner because participants behaved as though taking more tablets would help them perform the task better. These reinforcement effects are known to be elicited by drugs of abuse (Koob and Volkow 2009), and are consistent with the finding that modafinil is acting in the reward centers of the brain (Volkow et al. 2009). This indicates that the use of modafinil is more likely to lead to recreational use, rather than increasing motivation for higher academic achievement (Mohamed 2012a).

This is also consistent with early work by Baranski and colleagues (2004), who argued that the cognitive enhancing effects of modafinil are due to increased overconfidence. Baranski and colleagues (2004) administered 300 mg modafinil to healthy non-sleep-deprived adults and found that participants who took modafinil reported a significant increase in overconfidence in their judgment to perform the cognitive tasks administered during the study. It is important to note that the study found no cognitive-enhancing effects of modafinil on some of the tasks, including addition and line discrimination.

Interestingly, those taking these stimulants are reported to be underachieving students who might be taking them to enhance their academic performance (for review, see Mohamed 2014a and references therein). It is likely that these individuals are ameliorating some underlying undiagnosed psychological problems or academic under achievement rather than seeking increased motivation per se.

It is also unclear what kind personal or psychological problems students and academic who take these medications are facing in their lives. Therefore, it is very simplistic to argue, based on narrow qualitative evidence, that these stimulants improve motivation in healthy, non-sleep-deprived neurotypical individuals. The picture is very likely to be more complex than presented by the current article.

Given the lack of previous research investigating the behavioral effects of modafinil on motivation, we aimed to study the effects of modafinil on salience of pleasure and motivational reinforcement of reward learning in healthy non-sleep-deprived, neurotypical participants. Sixty-four healthy participants participated in a study that employed a randomized, double-blind, placebo-controlled, parallel-group design methodology. We found that modafinil significantly increased the ratings of pleasure, as measured by computerized visual analogue scales, during the performance of the entire study period without improving cognitive tasks that measured executive functions and working memory performance. On a task of motivational reinforcement learning, known as the Cued Reinforcement Reaction Time, modafinil increased latency of response and impaired the ability for participants to learn motivational contingencies associated with positive reward. The results from our study indicate that modafinil increases the subjective pleasure gained through behaviorally demanding situations without improving tasks that tap into higher order cognitive processes. Furthermore, they show that modafinil does not improve the learning of motivational tasks and argue against the notion that modafinil increases motivation in healthy, non-sleep-deprived individuals.

The lack of enhancing effects of modafinil on the motivational task is further consistent with our recent study (Mohamed and Lewis 2014), which demonstrated that administration of single 200-mg doses of modafinil to healthy, neurotypical non-sleep-deprived individuals increased the latency of responses in the performance of the Hayling Sentence Completion Test, a task that is highly sensitive to prefrontal executive function, without enhancing accuracy of performance of this task. In fact, a trend toward significance was observed for the members of the modafinil group, who made more errors on the task.

We also recently reported a double-blind placebo controlled randomized trial that investigated the effects of modafinil on divergent and convergent thinking tasks of creativity in healthy non-sleep-deprived neurotypical participants (Mohamed 2014c). The results for this trial showed that modafinil impaired the performance of the convergent thinking tasks of creativity in participants who scored high in the creativity personality trait while also
significantly reducing the performance of the divergent thinking tasks of creativity in all participants who took 200 mg modafinil, indicating that modafinil might reduce creativity thinking in healthy individuals (Mohamed 2014b).

Taken together, these results provide important clues to defining the limitations of modafinil as a putative cognitive enhancer and suggest that rather than being a more general cognitive enhancer that might increase motivation, modafinil might have negative effects on higher order cognitive processes in healthy, neurotypical non-sleep-deprived individuals.

Hence, the evidence to support the notion that stimulants such as modafinil, as suggested by Kjersgaard (2015), improve motivation in healthy individuals is limited in the quantitative neuroscience literature. What the limited qualitative evidence cited by Kjersgaard might describe is at best a placebo effect or salience of reward effect where healthy individuals perceive that they are being enhanced without their performance on cognitive tasks actually being enhanced. This commentary describes quantitative evidence from the cognitive neurosciences supporting this argument. Thus, it is clear that discussing the ethics of enhancing motivation with stimulants is currently premature because the quantitative evidence from cognitive neuroscience studies does not seem to support this argument (Mohamed 2014c).

Rather than rely on very few qualitative evidence and opinion articles to support the notion that stimulants increase motivation in healthy individuals, and argue that this warrants ethical discussion, the randomized controlled trials reported here point to the view, based on the real evidence, that these medications might impair cognition in healthy individuals. Hence, both neuroscientists and ethicists need to be discussing the ethical implications that may arise when stimulants impair cognition in healthy individuals. Thus, we might need to rethink and reevaluate the debate on cognitive enhancement and reframe ethical discussion based on the hard and unbiased evidence from the cognitive neurosciences (Mohamed 2014a).

REFERENCES


