Dear Editor,

The role of neurotransmitters other than serotonin in phobic-avoidance disorders has been clarified as neural science progresses.1 Among those, dopamine, both directly and by its interactions with other systems, is an excellent candidate for investigation. In agreement with the author’s hypothesis are also some important clinical correlates of the development of phobias and lack of motivational drive, and the parallel drawn between social anxiety disorder (SAnD) and studies of subordinated monkeys. There is another dimension on the role of dopamine in SAnD and anxiety disorders as a whole. Such hypothesis is founded on the idea of anxiety as a condition in which there is a conflict between appetitive/positively reinforced behaviors, closely related to dopaminergic systems,2 and escape/avoidance behaviors, related to serotonergic systems.3 Other authors have already addressed the importance of those two systems in anxiety, describing the conflict between the behavioral approach system and the fight/flight/freeze system.4 In this way, it would be logical to speculate that while serotonergic drugs would inhibit inborn fight or flight responses to proximal danger through its action in the dorsal periaqueductal gray matter,3 while dopaminergic drugs would increase motivation/appetitive behaviors through its actions in the mesolimbic system,2 augmenting thus the effect of serotonergic drugs. It is unlikely that DA could have an “anxiolitic” property by itself. Drugs enhancing dopaminergic bioavailability, such as bupropion, have little or inconsistent effects on anxiety disorders. On the other hand, augmented DA could have an impact upon treatment response.

In our group, we have found a close relation of Cloninger’s5 Self Directedness dimension and remission following behavioral treatment in SAnD. This trait may be linked to DA activity and be associated with better treatment responses.

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References

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