In response to: AMPA receptor potentially participates in the mediation of the increased brain-derived neurotrophic factor following chronic ketamine use by Yang, Jianjun; Zhou, Zhiqiang; Yang, Chun

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In this letter, the authors suggest the possibility that α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor participates in the mediation of the increased brain-derived neurotrophic factor observed in chronic ketamine users. The fact that increased intracellular glutamate release may activate AMPA receptor and this, in turn, may upregulate BDNF gene expression is certainly a plausible hypothesis and is not in contrast with our interpretation. The authors themselves acknowledge that the possibility that glutamate activates AMPA [instead of N-methyl-D-aspartate (NMDA)] receptors is caused by (or linked to) ketamine's antagonist action at NMDA receptor which we proposed as the basic mechanism (Ricci et al. 2011). The activation of AMPA receptors plasticity pathways has also been proposed to play a critical role in ketamine's rapid antidepressant effect. These rapid

effects on depressive symptoms seem to take place through a direct trafficking of AMPA receptors (fast-acting ionic glutamergic receptors) into the postsynaptic membrane (Machado-Vieira et al. 2009). Thus, we believe that the mechanism suggested by the authors is a valid compelling hypothesis to the interpretation of our data.

References

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