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A Corrigendum on   
[Mechanisms Underlying Serotonergic Excitation of Callosal Projection Neurons in the Mouse Medial Prefrontal Cortex](https://doi.org/10.3389/fncir.2018.00002)

*by Stephens, E. K., Baker, A. L., and Gulledge, A. T. (2018). Front. Neural Circuits 12: 2. doi:* [*10. 3389/fncir. 2018. 00002*](https://doi.org/10.3389/fncir.2018.00002)

In the original article, there was an error. The original text wrongly suggested that one of our manipulations increased the driving force for potassium by “ six-fold”. Instead, while the amount of potassium was lowered six-fold (from 3 mM to 0. 5 mM), the driving force for potassium, as measured at the action potential threshold, was approximately doubled.

A correction has been made to the *Results* , subsection *Role of M-current in Serotonergic Excitation* , paragraph three:

“ The results above suggest that 5-HT acts via at least three distinct mechanisms (K V 7 suppression, the ADP conductance, and a calcium-sensitive calcium conductance) to enhance the excitability of COM neurons. To test whether M-current is the dominant potassium conductance contributing to serotonergic excitation, we enhanced the driving force for potassium by lowering the external potassium concentration ([K + ] o ) six-fold to 0. 5 mM (replaced with equimolar sodium; Figure 7). By increasing the driving force for potassium, this manipulation will enhance the impact of M-current suppression by 5-HT, but will also act to reduce the net current through potassium-permeable non-specific cation conductances. In neurons recorded with control intracellular solution, lowering [K + ] o revealed a brief inhibition occurring immediately after 5-HT application that was absent in control conditions (Figures 7A, C); these inhibitory responses are likely G q -driven hyperpolarizations (mediated by SK-type potassium channels) that occur regularly in pyramidal neurons following M1 muscarinic receptor activation ( [Gulledge et al., 2009](#B1) ), but which are only rarely observed in response to 5-HT in control conditions. Lowering [K + ] o enhanced this early potassium conductance, and reduced the magnitude of serotonergic excitation by 31 ± 9% ( *n* = 10, paired). In control conditions (e. g., 3 mM [K + ] o ), 5-HT generated peak responses of 82 ± 14% with integrals of 157 ± 44 Hz•s. After reducing extracellular potassium to 0. 5 mM, peak excitation was 61 ± 15% ( *p* = 0. 003 relative to control conditions) with integrals of 117 ± 47 Hz•s ( *p* = 0. 057, Figure 7D). Because the larger driving force for potassium is expected to increase 5-HT excitation by enhancing the contribution of M-current suppression, the observed reductions in response magnitudes and integrals suggest the participation of potassium-permeable non-specific cation conductances, such as the ADP conductance ( [Haj-Dahmane and Andrade, 1998](#B2) ).”

The authors apologize for this error and state that this correction does not change the scientific conclusions of the article in any way. The original article has been updated.

## References

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