BIOCHEMISTRY
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Addition and Correction

Correction to Interaction of Selective Serotonin Reuptake Inhibitors with Neuronal Nicotinic Acetylcholine Receptors [(2010) *Biochemistry 49,* 5734. DOI: 10.1021/bi100536t]. Hugo R. Arias,* Dominik Feuerbach, Katarzyna M. Targowska-Duda, Megan Russell, and Krzysztof Jozwiak

Page 5734. The corrected abstract, with a change in point (1), follows.

We compared the interaction of fluoxetine and paroxetine, two selective serotonin reuptake inhibitors (SSRIs), with the human (h) α4β2, α3β4, and α7 nicotinic acetylcholine receptors (AChRs) in different conformational states, using Ca^{2+} influx, radioligand binding, and molecular docking approaches. The results established that (1) fluoxetine was more potent than paroxetine in inhibiting agonist-activated Ca^{2+} influx on hα4β2 and hα7 AChRs, whereas the potency of both SSRIs was practically the same in the hα3β4 AChR. (2) SSRIs bind to the [^{3}H]imipramine locus with a higher affinity when the AChRs are in the desensitized states compared to the resting states. (3) The different receptor specificity for fluoxetine determined by their inhibitory potencies or binding affinities suggests different modes of interaction when the AChR is in the closed or activated state. (4) Neutral and protonated fluoxetine interacts with a binding domain located in the middle of the AChR ion channel. In conclusion, SSRIs inhibit the most important neuronal AChRs with potencies and affinities that are clinically relevant by binding to a luminal site that is shared with tricyclic antidepressants.

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