Nicotinic α7 acetylcholine receptor-mediated currents are not modulated by the tryptophan metabolite kynurenic acid in adult hippocampal interneurons

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The tryptophan metabolite, kynurenic acid (KYNA), is classically known to be an antagonist of ionotropic glutamate receptors. Within the last decade several reports have been published suggesting that KYNA also blocks nicotinic acetylcholine receptors (nAChRs) containing the \alpha7 subunit (\alpha7*). Most of these reports involve either indirect measurements of KYNA effects on \alpha7 nAChR function, or are reports of KYNA effects in complicated in vivo systems. However, a recent report investigating KYNA interactions with \alpha7 nAChRs failed to detect an interaction using direct measurements of \alpha7 nAChRs function. Further, it showed that a KYNA blockade of \alpha7 nAChR stimulated GABA release (an indirect measure of \alpha7 nAChR function) was not due to KYNA blockade of the \alpha7 nAChRs. The current study measured the direct effects of KYNA on \alpha7-containing nAChRs expressed on interneurons in the hilar and CA1 stratum radiatum regions of the mouse hippocampus and on interneurons in the CA1 region of the rat hippocampus. Here we show that KYNA does not block \alpha7* nAChRs using direct patch-clamprecording of \alpha7 currents in adult brain slices.

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