EXPANDED ABSTRACT

Ca^{2+} mobilization by nicotine through synaptic activation in rat parotid acini

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Abstract : Nicotine has been reported to increase the intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$) in sublingual acini due to neurotransmitter release from nerve terminals associated with the cell preparation (1). However, it is unclear whether or not the same reaction exists in parotid cells. Therefore, we investigated effect of nicotine on Ca^{2+} mobilization in digested parotid acini from rats. After removing the parotid gland from Wistar rats, the tissues were minced and digested with collagenase. Then, the intracellular Ca^{2+} indicator fura-2 was added to the preparation, and the change in $[Ca^{2+}]_i$ was monitored using fluorescent microscope. In many but not all parotid acini, K^+ stimulation induced transient increases in $[Ca^{2+}]_i$. The K^+-induced Ca^{2+} response in parotid acini was completely blocked by Cd^{2+}-containing solution. These results suggest that the parotid cell preparation has nerve terminals. In all high-K^+-sensitive parotid acini, over 3 μM of nicotine increased $[Ca^{2+}]_i$, and the response was blocked by a Cd^{2+}-containing solution and nicotinic receptor antagonists. All high-K^+-insensitive acinar cells were resistant to the effect of nicotine on Ca^{2+} mobilization. These results suggest that nicotine induces increases in $[Ca^{2+}]_i$ in parotid acini due to neurotransmitter release from associated nerve terminals. J. Med. Invest. 56 Suppl. : 376, December, 2009

Keywords **:** *nicotine, nerve terminal, parotid cells*

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