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Ca^{2+}/calmodulin-dependent cyclic nucleotide phosphodiesterase in cGMP metabolism in rabbit parotid acinar cells

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ABSTRACT

Muscarinic cholinergic receptor activation provokes cGMP formation in parotid acinar cells. We investigated the involvement of Ca^{2+}/calmodulin-dependent cyclic nucleotide phosphodiesterase (PDE1) in cGMP breakdown in rabbit parotid acinar cells. The muscarinic agonist carbachol stimulated cGMP formation in the cells. The carbachol-induced cGMP formation was enhanced in the presence of 8-methoxymethyl-3-isobutyl-1-methylxanthine (MM-IBMX), a PDE1 inhibitor. cGMPPDE activity in rabbit parotid acinar cells was reduced by about 25% in the absence of Ca^{2+}/ calmodulin or in the presence of MM-IBMX. Ca^{2+}/calmodulin-dependent cGMP-PDE in rabbit parotid acinar cells was purified using Calmodulin-Sepharose 4B and Mono Q ion-exchange column chromatography. Two dominant fractions with cGMP-PDE activity, referred to as the P-1 and P-2 fractions, were eluted from the Mono Q ion-exchange column. The *K*m values for cGMP of PDE in the P-1 and P-2 fractions were 0.82 μM and 0.40 μM, respectively, which were much lower than that for cAMP. The EC_{50} for Ca^{2+} and calmodulin of PDEs in the P-1 and P-2 fractions were 458 nM and 426 nM, respectively, and 32 nM and 137 nM, respectively. Protein bands that crossreacted with anti-PDE1A antibody were detected. These results suggest that Ca^{2+}/calmodulin-dependent PDE, PDE1A, is involved in cGMP breakdown in rabbit parotid acinar cells.

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