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DIFFERENCE OF Ca DEPENDENCY BETWEEN NICOTINIC AND MUSCARINIC  
RECEPTOR MEDIATED CATECHOLAMINE SECRETIONS IN  
PERFUSED ADRENAL GLAND OF GUINEA PIG

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1. The relationship between the Ca dependency of ACh-induced catecholamine release and the ACh-receptor types involved was studied using isolated and perfused adrenal glands of the guinea pig.
2. In the presence of extracellular Ca, catecholamine release induced by ACh started to appear at  $10^{-6}$ M and increased with increasing concentration until it attained a maximum at  $10^{-3}$ M.  $ED_{50}$  of ACh was  $6.8 \times 10^{-5}$ M.
3. ACh ( $10^{-5}$ M), but not nicotine ( $5 \times 10^{-5}$ M), was partially effective in releasing catecholamine during perfusion with Ca-free Locke solution containing EGTA ( $10^{-5}$ – $10^{-3}$ M).
4. When chromaffin cells were completely depolarized by perfusion with isotonic KCl (KCl : 159.6mM) solution, ACh ( $10^{-5}$ M), but not nicotine ( $3 \times 10^{-5}$ M), caused almost the same amount of catecholamine release as that obtained during perfusion with the standard Locke solution.
5. During perfusion with the isotonic KCl solution, ACh ( $10^{-5}$ – $10^{-4}$ M) failed to increase catecholamine release in a concentration dependent manner.
6. Secretory response to ACh ( $10^{-5}$ – $10^{-4}$ M) in the isotonic KCl solution was abolished by atropine ( $10^{-5}$ M), but not by hexamethonium ( $10^{-3}$ M).
7. ACh ( $10^{-4}$ M) caused simultaneous release of catecholamine and DBH during perfusion with the Ca-free solution containing EGTA. The molar ratio of DBH to the amount of catecholamine released was almost the same as that contained in isolated chromaffin granules.
8. It is suggested that ACh causes catecholamine release from adrenal chromaffin cells of guinea pig by increasing Ca influx through a nicotinic receptor-linked, voltage dependent Ca channel, and a muscarinic receptor-linked, voltage independent Ca channel, or by mobilizing Ca from some muscarinic receptor-linked intracellular Ca pool.