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acid. Plasma was separated by centrifugation and was applied on to a Centricon-10®. The filtrate was obtained by centrifugation at 5000 ×g. A 100 μl portion of the filtrate was used for RIA. The filtrate subjected to alkaline hydrolysis served as blank. Under these bleeding conditions, AChE activity in the blood was confirmed to be completely inhibited. The recovery ratio of ACh added to plasma was 103.6±5.0%. Plasma concentrations of ACh were 639±60 in rabbits (n=6), 526±68 in men (n=6) and 542±115 pg/ml in women (n=8). These data demonstrate that small amount of ACh is present in the blood of rabbits and humans. The origin of ACh in the blood is under investigation.

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Enhancement of Acetylcholine Release and Contraction Response by Pirenzepine and Atropine in the Longitudinal Muscle Strips of Guinea-pig Ileum

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We have recently shown that acetylcholine (ACh) released from the longitudinal muscle strips of guinea-pig ileum can be direstly measured by a radioimmunoassay (RIA). The strips were suspended in an organ bath and perfused with normal or drug containing Kreds solution (0.4 ml/min). The strips were pretreated with methanesulfonyl fluoride, an irreversible cholinesterase (ChE) inhibitor. Electrical stimulation produced a contraction response and an increase in ACh release. Perfusion with pirenzepine (0.1—10 μM) and atropine (1—100 nM) increased ACh release from the strips upon electrical stimulation. The contraction response was enhanced by perfusion with lower concentrations of both pirenzepine (0.1 and 1 μM) and atropine (1 and 10 nM), while the enhancement of the contraction response was abolished at higher concentrations of these drugs. The data indicate that pirenzepine and atropine increase ACh release through the action on M_1-muscarinic autoreceptors under the present experimental conditions. M_1-muscarinic receptor in the myenteric neurons appears to be involved in the regulation of ACh release.

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