Thus when endogenous vasopressin is supressed or congenitally absent, oxytocin exhibits a weak antidiuretic action possibly as a consequence of its structural similarity to vasopressin. However, in the presence of vasopressin this action is masked and a diuretic action revealed perhaps due to the competitive displacement of the more potently antidiuretic vasopressin.

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Inhibition of angiotensin-induced drinking by ergot alkaloids

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Angiotensin, injected into the cerebral ventricles, causes water replete rats to drink (Fitzsimons, 1972). There is evidence that dopamine is involved in this response and the dipsogenic action of angiotensin is blocked by neuroleptic drugs (Fitzsimons & Setler, 1975). In the present study we have studied the effect of ergometrine and of other ergot alkaloids on the dipsogenic response to angiotensin and carbachol.

Drugs (in 2–4 µl of 0.9% NaCl) were injected unilaterally into the lateral ventricles of male Wistar rats via permanently-implanted cannulae and water intake was measured (Sumners, Woodruff, Poat & Munday, 1979).

Following angiotensin injections, the rats commenced drinking within 2 min of injection and drinking was usually completed within 20 minutes. The maximum response was produced by angiotensin (1 nmol); at this dose the mean amount of water drunk (ml) was 20.8 ± 0.9 (± s.e. mean, $n = 26$). A dose of 200 pmol angiotensin (the dose used in all antagonist studies) produced a drinking response of 9.4 ± 0.7 ml ($n = 37$). Ergometrine maleate, injected 5 min before the angiotensin, inhibited angiotensin-induced drinking in a dose-dependent manner. The threshold dose for ergometrine was 1 nmol which caused a 24.9 ± 4.0% ($n = 9$) inhibition of the angiotensin response. At a dose of 22 nmol ergometrine, angiotensin-induced drinking was inhibited by 76.1 ± 7.3% ($n = 10$). Other ergot alkaloid derivatives were less active, producing the following inhibitions of angiotensin-induced drinking: Lysergic acid diethylamide tartrate (31 nmol), 42.8 ± 1.9% ($n = 12$); methysergide bimaleate (21 nmol), 36.5 ± 7.1% ($n = 10$). BOL-148 (50 nmol) had no significant effect on the angiotensin response.

The dipsogen carbachol (1.1 nmol) caused a drinking response of 8.7 ± 0.8 ml ($n = 24$). This response was not significantly affected by ergometrine (1, 2 or 22 nmol).

These results further demonstrate that the dipsogenic action of angiotensin is produced by a different mechanism from that of carbachol. Ergometrine is known to act as an agonist or antagonist on dopamine receptors (Woodruff, 1978). Further experiments are required to determine whether the inhibition of angiotensin-induced drinking produced by ergometrine is related to the dopamine receptor-blocking activity of the latter.

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