small voltage gradient, 1 V/cm across the stem of the 'negative' plant could transport adequate water for maintenance of the plant from the soil. The soil at this stage appeared dry and hard. In the case of the 'positive' plant, (soil 'negative'), the moisture was transferred from the plant to the soil, hence the plants 'complete' dehydration on the 4th day of the 'voltage gradient' experiment. The 'voltage gradient' experiment on the broad beans was performed 3 times with identical results during 2 years, at times when the atmospheric conditions were considered right for producing stress conditions in plants.

No measurements were made of the amount of moisture transported in the broad bean plants. The reason being that the high transportation rate of the plants, subjected to the very arid conditions in the glass house, 23-40 °C and relative humidity which fluctuated between 40 and 20%, made such measurements meaningless.

Measurements were made on the influence of a weak potential gradient on the transportation of fluid in vine plants during the dormant period, June 1981 (winter in the southern hemisphere). Three plants of the cultivar Pinotage were used in the experiment. The plants were approximately similar in size and were grown in plastic pots in sandy loam soil and kept in the glass house, the temperature fluctuated between 10 and 20 °C. The stems were cut off 18 cm from the soil and 2 pairs of 9 V batteries connected in series to supply 18 V, connected to the soil and the stems of the plants. In 1 instance the plant was 'positive'; the Pt wire was inserted in the stem at the position where it was cut; the soil was 'negative'. In a 2nd instance the plant was made 'negative' and the soil 'positive'. The 3rd plant was the control. The plants were watered daily for 3 weeks. After 24 h exposure to the gradient, the 'negative' plant (soil 'positive') started to emit fluid from the 'wound'. A chute was made of 'Bostic' gum to direct the exuded fluid into a plastic centrifuge tube attached to the stem. 30 ml were collected in 48 h. The experiment was terminated after 21 days. At that stage the 'positive' plant was dry and never became viable again. The 'negative' plant started to bud while the control plant was still dormant. On the assumption that stationary ionizable acidic, viz. carboxyl and, or basic, viz. imino and amino groups are present in the plant tissues, the transport of moisture by a weak potential gradient may be due to electro-endosmosis; the direction of migration of the fluid being dependant upon the net charge density.

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Relaxation of isolated rabbit veins mediated by latent histamine H_2-receptors

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Summary. Isolated rabbit veins preconstricted by either norepinephrine, methoxamine or potassium were relaxed by histamine in the presence of mepyramine, a histamine H_1-antagonist. The relaxation was not antagonized by atropine, propranolol and indomethacin but by an H_2-antagonist cimetidine. It is likely that histamine relaxes the rabbit veins through H_2-receptors.

It is now well established that the effect of histamine on the cardiovascular system, including the heart and arterial side, is mediated by 2 distinct specific receptors H_1 and H_2[2-4]. However, there is little information regarding the mechanism of histamine action, especially about H_2-receptors, on the venous side. The current study reports the existence of H_2-receptors in veins and the relaxation mediated by the receptors.

Male Japanese white rabbits weighing 2-3 kg were killed by a blow on the head and rapid exsanguination. The saphenous, cephalic, ear, facial, external jugular, azygos, pulmonary, portal, splenic and renal veins and posterior vena cava were removed and ring segments (4 mm in length) were prepared under a dissecting microscope. A ring segment preparation[5] was suspended in an organ bath containing 50 ml of modified Krebs-bicarbonate solution which was aerated with 95% O_2 + 5% CO_2 and maintained at 37 °C. The composition of the modified Krebs-bicarbonate solution was: NaCl 119, KCl 4.7, $CaCl_2$ 2.5, KH_2PO_4 1.18, $MgSO_4$ 1.17, $NaHCO_3$ 24.9 and glucose 11.7 (all mM). A passive load of 0.5 g was applied for large veins with an OD of 1.5 mm or more, and 0.3 g for smaller veins. Isometric contraction and relaxation were recorded on an ink-writing oscillograph (Nihon Kohden Kogyo, Tokyo, Japan) by means of force displacement transducers (Nihon Kohden Kogyo). During the equilibration period of 2 h, submaximal contractions were elicited twice by histamine 10 μM, and bathing media were renewed approximately every 20 min before the experiment on drug effects.

Drugs used were histamine dihydrochloride, l-norepinephrine bitartrate, methoxamine hydrochloride, mepyramine maleate, cimetidine[6], atropine sulfate, dl-propranolol hydrochloride and indomethacin.

Histamine induced contractions in all veins studied. As shown in figure 1, the histamine concentration-response curve was shifted êö , tlel to the right, dose-dependently, by mepyramine, a selective H_1-receptor antagonist. pA_2-Values[7] were around 9 in most veins, the values comparing well with those obtained with the guinea-pig ileum[7].

On the other hand, an H_2-receptor antagonist cimetidine 10 μM hardly affected the contractile responses of veins to histamine (data not shown). The result was in accordance

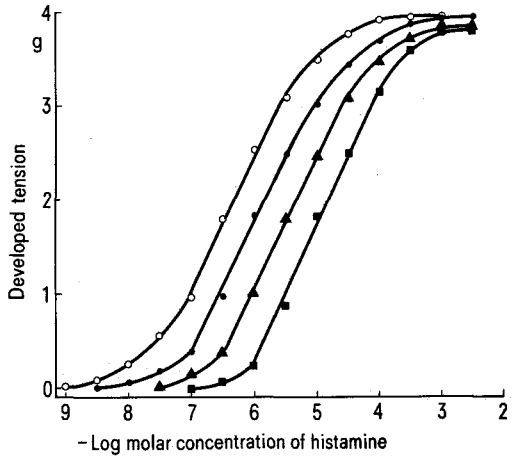

Figure 1. A typical concentration-response curve for histamine and antagonism between histamine and mepyramine obtained from the rabbit saphenous vein. Histamine was cumulatively added to the organ bath. Mepyramine 1 nM (●), 3 nM (▲) and 10 nM (■) shifted the curve in parallel to the right. The figure shows that the contractile response to histamine was specifically and competitively antagonized by mepyramine.

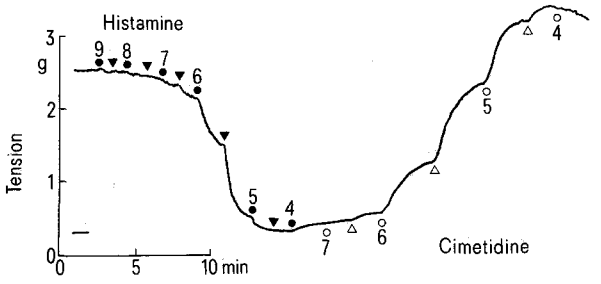

Figure 2. A typical record of histamine-induced relaxation and antagonism between histamine and cimetidine obtained from the rabbit saphenous vein. The vein was preconstricted by methoxamine 2 μM in the presence of mepyramine 1 μM. After the maximum relaxation was obtained by cumulative administration of histamine, cimetidine was added cumulatively into the bath. Cimetidine reversed the histamine-induced relaxation stepwise. Numerals are negative logarithms of molar concentrations of histamine (●) and cimetidine (○). ▲ and △ show 3-fold the previous concentrations. Horizontal line just left of tracing represents the resting level before the addition of methoxamine.

with the findings of Powell and Brody[8] and Konishi et al.[4]. Therefore, the contractile response to histamine seemed to be mediated exclusively by H_1-receptors. Histamine also contracted the facial vein, which is known to have the peculiarity that it produces spontaneous tone and that adrenergic nerve stimulation and exogenous norepinephrine produce relaxation[9,10].

When the veins were preconstricted by either norepinephrine 0.1–1 μM, methoxamine 1–10 μM, or potassium 30–60 mM, histamine relaxed veins dose-dependently in the presence of mepyramine 1–10 μM (fig. 2). The relaxation induced by histamine was antagonized by pretreatment with cimetidine 3–10 μM and reversed stepwise by cumulative administration of cimetidine as shown in figure 2. Cimetidine seemed to antagonize the relaxant action of histamine at lower concentrations in the veins than in the arteries, since cimetidine 10 μM has shown to shift the histamine concentration-relaxant response curve in parallel by about one log unit in the canine coronary and renal arteries[4]. The mean pA_2-value of cimetidine was calculated as 6.42 (±0.17, SEM, n = 6) with the saphenous vein, the value being in approximate accordance with those obtained with the guinea-pig atria and the rat uterus[11].

Neither atropine 1 μM, propranolol 1 μM nor indomethacin 10 μM affected the relaxation at all. The above-mentioned phenomena were observed in all veins studied. Although Powell and Brody[8] concluded that H_2-receptors were absent from veins, the results presented here demonstrate clearly that there are H_2-receptors in rabbit veins, and that the receptors mediate relaxation.

Regarding the paradox that H_2-antihistamines alone have no apparent antihistamine activity in the cardiovascular system, Powell and Brody[8] presented a hypothetical mechanism for the interaction between histamine and histamine antagonists with vascular histamine receptors. The mechanism would involve activation of H_1- and H_2-receptors by histamine. However, the activation of the H_1-receptor would be predominant. The present results also seem to favor their hypothesis.

Furchgott and Zawadzki[12] and Altura and Chand[13] revealed that the relaxation of arterial smooth muscle by acetylcholine and bradykinin was dependent on intact endothelial cells. Thus, the possibilities of involvement of endothelial cells and of other mediators in the histamine-induced relaxation of rabbit veins remain to be elucidated.

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