In the present study experiments were made on Norfolk White rabbits using skin removed from New Zealand Whites. Six homografts were made on one leg or both legs of each recipient as described previously (Jasani & Lewis, 1971). Each graft was weighed prior to grafting and subsquently on removal postoperatively. Then it was subjected to dry weight analysis using a modification of the method of Schneider (1945) and DNA estimation (Burton, 1956). Moisture content was estimated from the difference between the fresh weight and the total dry weight of each graft. The final values for each parameter were compared with those obtained from a similar analysis of a portion of non-grafted donor skin.

In the first series of experiments all the six grafts were removed at the same time interval after grafting. Although there was a statistically significant increase in all three parameters in each of the six grafts from the fourth day onwards, the extent of the increases in dry weight and moisture content of grafts on the medial aspect were significantly lower than those in the lateral grafts. These findings indicated that these two parameters and to a lesser extent DNA content, were significantly influenced by the anatomical position of the graft.

This was shown more clearly in a second series of experiments in which pairs of grafts from the same anatomical site on both legs were removed at daily intervals after grafting. When these were compared there was no statistically significant difference between the dry weight, moisture content or DNA content of homologous pairs.

Since changes in the three parameters did not parallel one another, even in the grafts taken from the same site, it is concluded that each index may represent a separate tissue event. Changes in dry weight may parallel the increases in vascularity of the graft, an event which comes to a halt with the onset of rejection; increases in the moisture content may reflect changes in vascular permeability and those in the DNA content may indicate increases in the cellularity of the graft.

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Effects of frusemide on cyclic AMP binding in the toad bladder

D. R. FERGUSON and B. R. TWITE*

Department of Pharmacology, Medical School, University of Bristol

Frusemide, a rapidly acting diuretic, is known to antagonize the stimulation of active sodium transport produced by vasopressin in toad bladders (Ferguson, 1966).

Using a modification of Gilman's (1970) isotope displacement assay for cyclic AMP, we have investigated the effect of frusemide on cyclic AMP binding to rabbit muscle cyclic binding protein. There was a significant reduction in cyclic AMP binding in the presence of $2{\cdot}5 \times 10^{-3}$M frusemide ($P<0{\cdot}01$).

When the experiments were repeated using toad bladder epithelium cyclic binding protein, we found a significant displacement of cyclic AMP at a lower frusemide concentration.

Although the mode of action of cyclic AMP in mediating the natriferic effects of vasopressin is unknown, it is of interest that the effects of frusemide on cyclic AMP binding are seen at concentrations which inhibit natriferic effects of vasopressin in the intact tissue.

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Release by kinin of a substance contracting rabbit aorta (RCS) from guinea-pig lung

JOAN PICKENS, G. B. WEST and C. J. WHELAN

Department of Applied Biology, North East London Polytechnic, Dagenham, Essex

Rabbit-aorta contracting substance (RCS) is released from chopped guinea-pig lung by stirring with a blunt nylon rod (Palmer, Piper & Vane, 1970). Its release is inhibited by low concentrations of anti-inflammatory drugs such as aspirin. These drugs have no effect when superfused over the rabbit aortic strip and thus modify the release of RCS from the lung (Piper & Vane, 1969).

Piper & Vane (1969) showed that bradykinin released RCS from perfused unsensitized guinea-pig lung and this led Collier (1969) to suggest that such an action occurred *in vivo* especially as anti-inflammatory agents had been shown to antagonize some of the *in vitro* actions of bradykinin. The present study was designed to test and extend this proposal.

Krebs solution containing hyoscine, phenoxybenzamine and mepyramine (each at 0·5 μg/ml) with propranolol and cyproheptadine (each at 2 μg/ml) was passed at a rate of 10 ml/min at 34°C through a perspex vessel containing chopped guinea-pig lung. The effluent was made to superfuse a rat stomach strip (to detect prostaglandin-like activity), a rabbit aortic strip (to detect RCS), and a rat duodenum (to detect kinin).

When the lung tissue was stirred for 30 s with a blunt nylon rod, RCS was released and the rabbit aortic strip contracted. It was possible to obtain up to 8 such responses from a single preparation. When aspirin (2–20 μg) was administered to the lung tissue 5 min before stirring, the release of RCS was inhibited (5 experiments). However, a complete inhibition of the release of RACF was obtained (5 experiments) with aprotinin (500–1,000 K.I.U.) and with soya bean trypsin inhibitor (50–100 μg), and this lasted for more than 20 min. When bradykinin (10–100 μg) was given to the lung tissue, stirring resulted in an increased output of RCS which was dose-dependent. The release of prostaglandin, as measured on the rat stomach strip, was not significantly altered during these procedures, and stirring failed to release detectable amounts of bradykinin.

Thus, the potentiation of the RCS release from lung tissue by bradykinin is consistent with the hypothesis that kinins are involved in this process. Furthermore, inhibition of the release of RCS by aprotinin and soya bean trypsin in indicative of the involvement of proteases.

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