Dihomo-γ-linolenic acid effects on platelet utilization of arachidonic acid

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Characterization of [^{14}C]-(±)-propranolol uptake by guinea-pig lung

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Vitamin C and the cholesterol-lowering effect of clofibrate

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Radioimmunoassay of bradykinin in human skin exudates

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Bradykinin has previously been estimated by bioassay, and lack of more refined assay methods have hampered investigation of its physiological and pharmacological roles. We have now developed a highly sensitive and specific radioimmunoassay for bradykinin.

Bradykinin triacetate was conjugated to ovalbumin using toluene 2,4 diisocyanate (Schick & Singer, 1961). The lyophilized conjugate was dissolved in physiological saline and the solution emulsified in Freund's complete adjuvant. The emulsion was then injected into multiple intradermal sites on the dorsal surface of 6 rabbits as described by Vaitukaitis, Robbins, Nieschlag & Ross (1971). Five rabbits produced antisera suitable for radioimmunoassay. The antiserum employed routinely is used at a dilution of 1:5572 and is specific for bradykinin (Table 1).

Table 1 Specificity of bradykinin antiserum

Analogue	Cross-reactivity %
Bradykinin	100
N.N.-Dithiopropionyl bradykinin	6.6
5-D-Phe Bradykinin	0.002
5,8 di-D-Phe Bradykinin	<0.001
5-D-Phe 8 Tyr Bradykinin	0.001
6 Cys Bradykinin	<0.001
8-D-Phe Bradykinin	0.001
Bradykinylserine	0.001
Physalaemin	<0.001

Bradykinin triacetate [2 prolyl 3,4-[^{3}H]-(N)] serves as the tracer and separation of antibody bound and free antigen is achieved using a double antibody technique. Using an 18 h incubation period a sensitivity of 50 pg is obtained. This compares favourably with previously reported radioimmunoassays (Spragg, Austen & Haber, 1966) and with bioassay.

Immunoreactive bradykinin has been measured in exudate from normal and inflamed human skin. The exudate was collected using a suction bullae technique (Black, Greaves, Hensby & Plummer, 1976) and placed into a solution containing Trasylol and EDTA.

Our preliminary results support a role for bradykinin as a mediator of inflamed human skin.

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Automatic analysis of blood pressure and electrocardiograph records

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The following communication and demonstration were given at the Middlesex meeting of the British Pharmacological Society, 4–6 January 1978.

Food-intake and locomotor activity: effects of mazindol and spiperone

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Mazindol reduces food-intake, and central dopaminergic (DA) mechanisms may play a mediating role in this action (Kruk & Zarrindast, 1976). Our results show that in male Lister rats, adapted over a 3-week period to consuming their daily food intake in a 4 h period (11.00–15.00), mazindol (2.5 mg/kg) markedly reduced food intake in the first h but not in the subsequent 3 h period (Figure 1a). The dopamine receptor blocking agent spiperone (Anden, Butcher, Corrodi, Fuxe & Ungerstedt, 1970) at a dose of 0.1 mg/kg significantly attenuated this mazindol induced anorexia within the first h of the test, but was without effect on food intake in the remainder of the test (Figure 1a). This finding is consistent with previous reports of attenuation of mazindol anorexia by other DA antagonists (Zambotti, Carruba, Barzaghi, Vicentini, Groppetti & Mantegazza, 1976; Kruk & Zarrindast, 1976). However, spiperone alone, at the dose administered, also produced a significant decrease in food intake in the first h of the test. We sought further evidence to determine if the reduction of feeding under spiperone alone was secondary to a more general depression or interference with motor responses.

Male Lister rats were run in an open-field apparatus (60 cm^2; equipped with 10 infra-red beams and photocells), and activity measured as the total number of beam interruptions at 1 min intervals for 5 minutes. Spiperone (0.1 mg/kg) produced a marked deficit in locomotor activity, while mazindol (2.5 mg/kg) increased activity (Figure 1b). A group of animals injected with both spiperone and mazindol showed a level of activity not different from that following spiperone alone (Figure 1b).

It seemed possible that the antagonism of mazindol anorexia by spiperone, leading to an increase in food intake (Figure 1a), involved a mechanism of action distinct from the non-specific depression of behav-