not only reduced the inhibition, but occasionally produced histamine release by itself.

Another effect that this compound possessed was its ability to inhibit the uptake of labelled histamine by isolated human leucocytes, in a dose-dependent fashion. This is a property of histamine antagonists, recently reported by one of us (Assem, 1976).

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Anti-allergic properties of a new coumarin compound (BM 15,100)

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BM 15,100, a compound chemically defined as 1-(4-Chlorobenzyl)-4 3-(3,4-dimethylcoumarin-7-yl-oxy)-propyl -piperazine dihydrochloride combines two types of pharmacological activity useful for medical therapy of anaphylactic diseases such as atopic asthma and hay fever: (1) inhibition of mediator release from tissue mast cells and (2) antagonism to the effect of mediators.

The passive cutaneous anaphylactic (PCA) reaction in rats mediated by reaginic antibodies (Goose & Blair, 1969) was significantly inhibited by BM 15,100 (0.75 mg/kg i.v.). The same dose of this compound significantly suppressed degranulation of mesenteric mast cells (MD) (Fügner, 1973) and the cutaneous reaction elicited by intradermal injection of histamine. The PCA- and MD-inhibitory activity of BM 15,100 was similar to that of disodium cromoglycate (DSCG). Mepyramine antagonized PCA at a dose of 3 mg/kg i.v. whereas 0.38 mg/kg i.v. were sufficient to inhibit the histamine reaction. These results suggest that the PCA inhibition by BM 15,100 observed in our experiments was caused mainly by suppression of MD and only partly by the antihistamine activity. In contrast to the lack of activity of DSCG on oral administration, oral doses of BM 15,100 inhibit PCA in the rat significantly at doses nearly identical with the corresponding intravenous doses.

The oral antianaphylactic activity was also demonstrated in experiments with guinea pigs. Conjunctival oedema and hyperaemia in anaphylactic conjunctivitis (Dwyer, Turk & Darougar, 1974) were inhibited significantly by BM 15,100 at a dose of 0.75 mg/kg, anaphylactic bronchospasm (Davies & Johnston, 1971) was suppressed significantly with 0.4 mg/kg orally.

The naturally occurring allergy of monkeys to ascaris-antigen can be considered as a reliable *in vivo* model of human reagin mediated anaphylaxis. Active cutaneous anaphylactic (ACA) reactions were evoked in stump tail monkeys by intradermal injections of different dilutions of ascaris antigen (Perper, Sanda & Lichtenstein, 1972), and one cutaneous reaction in every experiment by intradermal injection of histamine. We measured the skin reaction by multiplying the diameter of the wheal by an arbitrary score for colour intensity. BM 15,100 in a dose as low as 0.1 mg/kg, given orally, significantly and strongly inhibited ACA whereas the histamine reaction was suppressed to a lesser degree. With doses up to 0.8 mg/kg there was no further increase in ACA inhibition but a clear dose-response relationship for the histamine antagonism could be seen. This finding is in agreement with the above-mentioned result from the intravenous injection into rats in so far as the histamine antagonism does not seem to be decisive for the inhibition of the anaphylactic reaction by BM 15,100.

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