HISTAMINE FORMATION IN PSORIATIC SKIN

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A greater understanding of the molecular mechanisms involved in reactions mediated by histamine, particularly those regulating its biosynthesis, could lead to more effective treatment of disorders in which histamine is implicated. An increased histamine forming capacity (HFC) has been demonstrated in tissues undergoing rapid cell division (Kahlson et al 1962). Psoriasis is a skin disease characterized by proliferation of capillaries in the dermal papillae and hyperplasia of the epidermis with rapid cell turnover, often accompanied by pruritus. As data on histamine formation in human skin is extremely limited, it was decided to study this in normal and psoriatic subjects. Clinically normal skin was obtained from adult surgical patients and skin biopsies were taken from currently untreated lesions of psoriatics. Informed patient consent was given in all cases. Skin samples were incubated with ^{14}C-histidine and assayed for ^{14}C-histamine using a modification of the method of Kahlson et al (1963).

The HFC of abdominal skin was determined in 21 adults of both sexes. In 5 of these the HFC was just measurable, mean value 0.4 ng histamine/3h/g wet weight of skin, but in the remaining 16, and in mammary skin from 5 females, it was undetectable. Skin from upper arm and lower leg (6 patients) did not have a measurable HFC but samples from elbow and knee both produced 2 ng/3h/g. The highest HFC (5 ng/3h/g) came from two samples of healing abdominal skin wounds. The HFC of currently untreated psoriatic lesions (8 patients) was markedly elevated; mean values, abdominal, 22 ng/3h/g; elbow, 10 ng/3h/g. Urinary histamine was measured in some of these psoriatic patients and in volunteers with clinically normal skin, using a modification of the method of Bergström and Hansson (1951). The urinary histamine content was higher in psoriatic patients than in control subjects, mean values 75 µg/24h and 9 µg/24h respectively.

Allowing for dissimilarities of dietary intake, the increased histamine output in the urine may well reflect increased production in psoriatic skin. Psoriasis has been likened to wound healing where the repair process becomes self-perpetuating (Jablonski et al 1979) and our results, showing an increased HFC in wound tissues and psoriatic skin, reinforce this idea. Also of interest is the apparent elevation of the HFC in normal skin from the elbow and knee, areas not only subject to more friction than others, but also the most common sites of psoriasis.

Although Marks et al (1980) suggested that the antihistamines may have some role in disorders involving heightened epidermal cell production, they appear to be of little value in the treatment of psoriasis. However steroids, when used, are of considerable short-term benefit. Graham et al (1964) have shown that cortisone reduces the HFC of rat skin. Whether or not the more promising current therapies have a similar action on the HFC of human psoriatic skin has yet to be determined.

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