

tion available concerning CO inhibition and AA pathways in different cell types. Platelet AA metabolism has been found normal.⁵ However, CO from nasal polyp cells and AA metabolism in stimulated alveolar macrophages have been found defective.¹

All these findings, taken together, suggest that the beneficial effect of NSAIDs in our patient is not dependent on abnormal AA metabolism of platelets. We suggest that this exceptional bronchial response is probably due to an idiosyncratic reaction occurring in the respiratory tract. Szczeklik¹ has also postulated an intrinsic defect of AA metabolism restricted to the lung in aspirin-induced asthma.

Future experiments tending to assess the contribution of different metabolites derived from AA and the role of various cell types are necessary to clarify

the pathophysiology of idiosyncratic reactions to NSAIDs.

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Reproducibility of skin prick test reactions to common allergens in patients with atopic eczema

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Prick testing is the simplest, cheapest, and most reliable method to diagnose IgE-dependent allergic reactions. Patients with atopic eczema often react in this test to a wide battery of allergens. It is, however, uncertain whether such tests are reproducible and whether they might be influenced by unspecific inflammatory events of the eczematous skin, as is the case with the well-known angry back syndrome during patch testing for delayed-type contact eczema.¹

To clarify this issue, we have performed prick tests at weekly intervals and in duplicate on opposite arms in 19 patients with atopic eczema and in nine patients with allergic rhinitis. The test agents included always

the same battery of allergen extracts to common inhalants and food allergens (Bencard, Neuss, Germany), as well as histamine and NaCl as controls.

The severity of disease was graded as mild (involvement of body folds only) in four patients, moderate (some additional body sites involved) in eight patients, and severe (generalized eczema or erythroderma) in seven patients with atopic eczema. Six of these patients suffered also from asthma and 12 from allergic rhinitis. Before prick testing, none of the patients had received antihistamines for 3 or more days previously, depending on the pharmacokinetics of the specific agent used, and none had received oral steroids for 3 or more weeks previously. All prick tests were performed by one of the authors (S. D. W.) on the extensor surfaces of both upper arms. Repeated tests could not be performed in three patients because of worsening of the eczema after the prick testing and the need for antihistamines. Wheals were measured 15 minutes after pricking with allergens and 10 minutes after pricking with histamine at their largest diameter and at perpendicular (90 degrees) angles, dis-

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TABLE I. Means (square millimeter) $\pm$ 1 SD of all skin prick test reactions to allergens or histamine

	Allergens	Histamine
Atopic eczema		
Test 1 (n = 19)	47.55 $\pm$ 20.31	35.93 $\pm$ 24.56
Test 2 (N = 16)	51.35 $\pm$ 35.78	28.94 $\pm$ 22.82
Allergic rhinitis		
Test 1 (N = 9)	59.48 $\pm$ 52.59	20.78 $\pm$ 7.10
Test 2 (N = 9)	55.80 $\pm$ 34.98	26.55 $\pm$ 20.52

Tests were repeated at weekly intervals in only 16 of the 19 patients with atopic eczema because of exacerbations of the skin disease after testing in three patients. The values of repeated tests did not differ significantly.

regarding pseudopods. Values were expressed as the product of these two values. A positive test was defined as an area of ≥ 9 mm². Statistical significance was tested with the Mann-Whitney U test.

The pattern of skin test reactivity was similar in both patient groups, with most of the patients reacting to grass and rye pollens, followed by reactivity to house dust mite and other inhalants. There were no reactions to commercial food extracts. No significant differences were observed between the two test sites and the reactions at weekly intervals (Table I) in the same patient, with regards to reactions to allergens as well as to histamine. Patients with milder forms of eczema were found to have a better reproducibility of the skin tests to allergens ($\alpha = 0.02$), but not to histamine. Of a total of 224 positive reactions to allergens in all patients, 30 were not reproducible on repeated testing. The coefficient of variation for weak reactions (51.7) was significantly different ($\alpha = 0.01$) from that for strong reactions (29.0).

On the basis of these findings, it can be concluded that the overall reproducibility of prick tests to allergens is good in patients with atopic eczema and allergic rhinitis regarding both contralateral reactivity and reactions at different time intervals. Tests with a very small wheal (≤ 30 mm²) were less reproducible, but the relevance of such reactions is questionable. However, caution should be practiced in the interpretation of reactions in patients with severe eczema because there was a lower reproducibility compared to that in mildly affected patients. This finding might be due to the effect of mediators, such as histamine, that

are known to be released into the circulation and that can cause false positive tests when they are injected in close proximity to sites of allergen skin tests.² An influence of increased use of topical steroid in these patients is unlikely, since it should have reduced, rather than increased, the reactivity. The state of atopy itself has previously been demonstrated not to influence the size of histamine whealing,³ although these data need confirmation.

There is general consensus in the literature that skin prick tests have a high diagnostic value in the diagnosis of allergic skin diseases.⁴ The present data, demonstrating a good reproducibility of skin testing, would allow counseling of patients regarding the avoidance of specific allergens, taking always into account the clinical history. It is, however, advisable to postpone testing in severely affected patients until sufficient healing of the skin has taken place, since the results are then more reliable and relevant and the testing is better tolerated.

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