regarding the adrenal, pituitary, or ovarian source of increased androgen production. We await more detailed information on Kasick's promising therapeutic studies with spirinolactone and other antiandrogens.

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#### References

- Hamilton JB. Male hormone substance. A prime factor in acne. J Clin Endocrinol Metabol 1941; 1:570-592.
- Lobo RA, Paul WL, Goebelsmann U. Dehydroepiandrosterone-sulfate as an indicator of adrenal androgen function. Obstet Gynecol 1981; 57:69–73.
- Paulson JD, Keller DW, Wiest WG, Warren JC. Free testosterone concentration in serum: Elevation is the hallmark of hirsutism. Am J Obstet Gynecol 1977; 128:851-857.
- Kirschner MA, Zucker IR, Jepperson D. Idiopathic hirsutism—an ovarian abnormality. N Engl J Med 1976; 294:637–640.
- Pochi PE, Strauss JS. Endocrinologic control of the development and activity of the human sebaceous gland. Invest Dermatol 1974; 62:191–201.

# The Value of Persistence and Thoroughness in Searching for the Allergen in Contact Dermatitis

The description of 3 cases of dermatitis in this issue by Taylor et al (page 123), "Contact Dermatitis to Knee Patch Adhesive in Certain Types of Boys' Jeans," emphasizes the value of thoroughness, insight, and persistence in pursuing the identity of an allergen. On a superficial level the authors identified epoxy resin as the cause of chronic dermatitis on both knees of three boys. On closer examination, there are important lessons to be learned regarding moral responsibilities and vigilance.

A rare or common allergen? Three patients were identified as having epoxy clothing dermatitis in a limited geographic area. It is highly improbable that these were the only children who developed such an allergy from these jeans. The investigative work of the authors identified the etiology in spite of the difficulty in obtaining information. Most physicians confronted with this dermatitis presumably muddled on, allowing children to continue with chronic dermatitis until the jeans were discarded or until repeated washing decreased the allergen level. Often, identification of an allergen in a few cases proves the problem instead to have been widespread. The ethylene-diamine in nystatin-neomycin sulfate-gramicidin-

triamcinolone acetonide (Mycolog) cream and its subsequent demonstration as a common allergen is possibly analogous to this epoxy episode.<sup>1</sup>

Exogenous versus endogenous dermatitis. When examining patients with dermatitis, we define the cause by appropriate history, morphology, distribution, and, when indicated, by patch testing. Many of us undoubtedly saw similar patients, and assumed that the process was endogenous in origin. The diagnosis of atopic dermatitis is all too often applied to children with eczema of unknown origin. However, these authors looked further, identifying the allergen by appropriate patch testing, thus immediately stopping a chronic dermatitis. The value to the public surely is greater. When the allergen was identified, the manufacturers altered the product.

An adequate corporate scientific staff or an inefficient system? It would be easy in retrospect to fault the clothing manufacturer for this dermatitis. Unfortunately, this is not an unusual event. Manufacturers and scientists often learn after the fact to be more careful and thorough in their dermatotoxicologic approach to product safety. In this case the manufacturer first claimed ignorance on the basis of inadequate information from his supplier. Proprietary data are often used as an excuse. Until more adequate legislation mandates away such secrecy, manufacturers must be demanding and persistent in obtaining the chemical composition from suppliers.

Because few manufacturers can justify the services of a full-time dermatotoxicologist, even if adequate numbers were trained, they must learn to seek part-time consultation in making risk assessments. An ever-expanding library of reference works simplifies the task significantly.<sup>2,3</sup>

Credibility of the manufacturer. When the authors first contacted the manufacturer, the presence of epoxy was denied. This all too often is the case. A classic example is that of Wilkinson,<sup>4</sup> who, with the aid of a black light, identified a fluorescent compound, a biocide in soap, as the cause of an epidemic of photoallergy in England.

We in no way wish to attack the credibility of manufacturers. We believe that it is the responsibility of manufacturers to know what they are selling. It is only recently that some corporations have insisted on knowing the compositions of fragrances they utilize. There is much room for improvement in defining this aspect of the chemical environment.

Allergen alternatives. Often manufacturers claim that they must use an allergen because no alternatives exist. Fortunately, with ingenuity and

determination, this can often be overcome, as was done here. A recent summary of structure-function relationships of chemicals and allergic contact dermatitis by Dupuis and Benezra<sup>5</sup> should make identification of allergen alternatives far more efficient.

Cutaneous versus systemic toxicity. This miniepidemic related to a localized problem, knee dermatitis. Yet, the mechanism is clearly systemic, delayed hypersensitivity or cell-mediated immunity. A possible future direction of dermatotoxicologic research is the identification of possible internal organ involvement. Unfortunately, clothing and adhesive suppliers have not considered this alternative seriously or accumulated data to deny or define its possible existence.

Relevance of dermatotoxicologic assays. Would this allergen have been identified if the manufacturer, even not knowing the chemical composition, had performed appropriate routine allergic contact dermatitis assays? We will not know until this is done retrospectively. Yet, it is my opinion, albeit biased, that much allergic dermatitis in clothing (especially shoes) could be prevented by proper application of such assays.

## Summary

The article of Taylor et al offers lessons in responsibility for public health and in toxicologic surveillance as well as in the obvious significance and value of the diligent and careful investigation of these workers in establishing the diagnosis of contact dermatitis caused by a knee-patch epoxy resin.

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### References

- Epstein E, Maibach HI. Ethylenediamine; allergic contact dermatitis. Arch Dermatol 1968; 98:476–477.
- Foussereau J, Benezra C, Maibach H. Occupational Contact Dermatitis. Philadelphia: Saunders, 1983.
- Cronin E. Contact Dermatitis. Edinburgh, Churchill Livingstone, 1980.
- Wilkinson DS. Photodermatitis due to tetrachorsalicylanilide. Br J Dermatol 1961; 73:213–219.
- Dupuis G, Benezra C. Allergic Contact Dermatitis to Simple Chemicals. New York: Marcel Dekker, 1982.
- Marzulli F, Maibach H (eds). Dermatotoxicology. 2nd ed. New York: Hemisphere Pub, 1982.