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Prevention of Environmental Dermatitis

Environmental dermatitis usually is the result of a contact allergen, photosensitization or atopy. Recognition that dermatitis is due to either of the first two causes, with subsequent specific therapy, often results in rapid relief of long-standing disease. Atopic dermatitis, although not environmental in origin, is often exacerbated by environmental factors and therefore can be alleviated by environmental manipulation.

When a patient asks, "Is there anything I can do to prevent this rash?" he is really asking for advice on how to prevent recurrence; initial sensitization is beyond prevention. Since this is the practicing physician's usual encounter with environmental dermatitis, the major portion of this article concerns what is known about preventing recurrence of symptoms in an already-sensitized patient.

The key to prevention lies in an accurate diagnosis of the disease process and hence a knowledge of the sequence of events leading to dermatitis. This information allows design of a therapeutic program which can break the chain of events in a manner suitable to the patient.

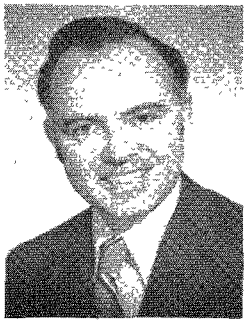
Environmental dermatitis usually falls in one of three categories: contact dermatitis, photodermatitis, or atopic dermatitis.

Contact Dermatitis

Contact dermatitis is the prototype of allergic dermatitis and is worthy of attention because removal of the offending allergen effects "clinical cure." This diagnostic coup has often resulted in a patient's being immediately relieved of years of discomfort and anguish about an unsightly skin condition which has cost him dearly in lost work time, expensive topical medications, and hours of skin care.

The allergen in contact dermatitis, a chemical of low molecular weight, combines with host protein (thought to be epidermal) to form a conjugate and hence a complete antigen. Immunization with this antigen (usually by casual exposure) produces a sensitive state wherein contact with the allergen results in allergic reaction.

This chain of events can be broken in several places. Wearing protective garments can afford considerable protection.



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Protective creams are effective only in that they stimulate patients to wash off the cream (and the allergen); hence, they should not be relied on to any great degree.

Nickel (in coins, jewelry, watchbands, etc.) is one of the most common contact allergens. Half of the battle is won when this cause of dermatitis is suspected and patch tests confirm the diagnosis. Locating less obvious environmental sources of nickel occasionally takes considerable acumen.

Nickel-plated articles can be sprayed or painted with plastic to prevent release of the ion. Frequently, nickel allergy becomes apparent only when a rise in temperature causes sweating and the nickel leaches from articles such as jewelry in contact with the skin. Patients should be warned to avoid exposure of the skin to nickel-containing articles at times when they perspire freely. For example, a patient can wear a favorite wristwatch (containing nickel) while working in an air-conditioned office but should remove it before playing tennis.

Entry of the free allergen into the epidermis can be specifically blocked by several methods. Nickel dermatitis can be prevented by spraying the exposed skin with isopropyl myristate or palmitate, which seems to block nickel's penetration of the skin.[1] Chromium, a notorious contact allergen with a valence of 6, enters the skin readily. Topical ascorbic acid solutions and other reducing agents can reduce chromium's valence to 3 and block its ability to enter the skin.[2] Chromium dermatitis occurs nearly always in males, especially construction workers who are sensitized by chrome in cement. Chrome is used in tanning leather, and often what appears to be tinea pedis clears rapidly when the patient switches to shoes made of leather tanned without chrome.

If all other methods of protection fail and the allergen enters the skin, a long-term possibility is hyposensitization or "hardening." Large-scale attempts at hyposensitization to *Rhus* (poison ivy, oak, etc.) and ragweed dermatitides have had varying degrees of success. Hardening, an increase in tolerance following continued exposure to a contact allergen, has long been considered a definite possibility and now appears to be a reality in some industrial cases.[3]

Photodermatitis

Photodermatitis is a cutaneous reaction to light, resulting from chemical structures which absorb light and interact with immune or nonimmune systems that produce tissue damage. Photoallergic reactions involve the immune system; phototoxic reactions occur in the absence of antibodies or cellular immunity. Photosensitizing chemicals can be endogenous, as in porphyria, or exogenous, as in photodermatitis associated with drug ingestion, cosmetics, or topical medications.

Photodermatitis is increasing in frequency in the United States and deserves emphasis because it is preventable. An aid to recognition is the distribution of the rash on sun-exposed areas of the body such as the face, dorsa of hands, and V of the neck. Occasionally, contact dermatitis due to airborne allergens (e.g., ragweed) mimicks photodermatitis. The latter is distinguishable in that it usually spares the shaded areas of the face (eyelids, upper lip directly below the nose, area beneath the chin).

A number of drugs are well-known photosensitizers, the most prominent ones being the sulfonamides and demethylchlortetracycline. After use of the drug is stopped, sun exposure is permissible when

the blood levels have become insignificant. Should the drug be necessary, the patient can be tested (with appropriate precautions) with ultraviolet light. If his skin does not burn when exposed to such light through window glass but does burn when the lamp is unshielded, the patient is sensitive to the typical sunburn spectrum. In this case, he can drive an automobile during daylight hours provided the windows filter out all short ultraviolet rays. If he burns when tested through glass, he should remain indoors during the hours of maximal ultraviolet radiation (9 A.M. to 3 P.M.). Sunscreens can be prescribed which selectively absorb damaging wavelengths and thus allow pursuit of necessary activities during daylight hours.

Bath soaps containing antibacterial substances have accounted for numerous cases of photodermatitis in recent years. Some of these substances, such as bithionol and tetrachlorosalicylanilide, have proved to be potent photosensitizers. The diagnosis is confirmed by the "photopatch test," which combines the technic of patch testing with irradiation of the test site with ultraviolet light filtered through window glass. Discontinuing use of such soaps brings slow improvement and increased tolerance of ultraviolet light. A few patients retain sensitivity for many months after they stop using the soap. These "persistent light reactors" require specialized treatment to increase their tolerance of ultraviolet light.[4]

Atopic Dermatitis

Atopic dermatitis is an eruption of unknown etiology. Except for occasional infants in whom certain foods have been noted to exacerbate atopic dermatitis, efforts to prove allergic causes have been largely futile. These patients often have respiratory allergies due to environmental allergens (hay fever, extrinsic asthma, etc.), but a good correlation between the respiratory symptoms and the atopic dermatitis has been lacking.

For the most part, there is no solid evidence that atopic dermatitis results from an allergic process. However, most physicians and their patients agree that anything that makes the atopic skin itch and results in scratching or rubbing can cause a flare of quiescent dermatitis. Patients often say that as long as they do not scratch themselves the dermatitis is circumscribed. If they scratch unrestrainedly, a flare often ensues.

Good skin care seems to offer the most hope for preventing recurrences of atopic dermatitis. The patient with dry flaking skin must care for it with emollients and must curtail unnecessary bathing. This becomes of paramount importance during cold, dry winter weather. The patient must be taught to recognize the premonitory signs of conditions which lead to flares and to treat them assiduously.

Recurrent dermatitis predisposes the patient to superinfection with yeast, fungi or bacteria. An awareness that inflamed skin is a fertile site for seeding of infectious agents aids in diagnosing infection. Treatment of the superinfection often clears stubborn "dermatitis."

Altogether, prevention of dermatitis rests with a team effort by patient and physician. An educated patient will avoid contact allergens, photosensitizers, and excessively dry skin. He will realize the importance of contacting his physician when the skin eruption appears atypical in distribution, extent or severity. The physician should be alert to adverse potentialities of any drug he prescribes and should look for features in the patient's skin which herald an outbreak of recurrent dermatitis.

Most of these technics require only a few minutes of the physician's time and are extremely rewarding.

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