COMMENTS AND OPINIONS

Ethics, the Prison System, and Dermatology

A recently published book raises several issues of relevance to dermatologists. In the book, entitled *Acres of Skin: Human Experiments at Holmesburg Prison,*[1] Allen M. Hornblum describes in detail medical research using prison volunteers in a prison in Philadelphia, Pa, starting in the 1950s. The research included not only relatively harmless procedures such as ordinary patch testing but also testing with some associated risks, such as the application of dioxin to the skin of volunteers. The principal researchers in the program were dermatologists.

See also pages 383, 457, and 477

The book emphasizes that prisoners have been exploited in the past, and that while this was happening, the medical community did not express concern for them. Since prisoners do not have the rights or opportunities afforded nonprisoners, they need to have more protection. However, to say categorically that research should not be done on prisoners, or to say that when such research is stopped the problem is resolved, misses the important points here. One point is that patients and subjects need to understand the facts, risks, and benefits behind any research project. For prisoners, in addition to this, there should be some type of ethics board.

The second important point is that the book brings us closer to an understanding of a major problem in our society today: the caste system we are creating, and the way people are treated in prison. Funding for education, health, and housing for the poor and for people of color is being cut, and at the same time more prisons are being built. This is creating a society where more and more poor people and people of color are being housed within the prison system without opportunities for success. One man who dared to confront the injustices of our system to people of color is Mumia Abu Jamal, a news broadcaster who is currently in jail facing the death penalty.[2] There is a huge amount of evidence that he has not been allowed to have a fair trial, and has undergone extremely inhumane treatment for many years in the very prison system that we read about in his books.[3,4] His case is another example of the unethical and inhumane way in which we treat our prisoners–the use of prisons for dermatologic research.

However, even if we had never done those research projects, there would still be an inhumane prison system; there would still be a system that is growing because our society is not willing to deal with the real problems it faces.

The book brings up some important points, but I am not sure what is the best response to them. If we just stop doing research in prisons, which is what seems to have happened in this case, we eliminate this one abuse of prisoners, but on the other hand, we deny prisoners an opportunity for contact with the outside and to possibly do some good, and to (in an experiment with no abuse) let them make some money in a reasonable way.

Another theme in the book is to lay blame on a few individuals. It may make a society feel good to blame these individuals; however, the book also points out that we are all culpable. Should we punish everyone? Very often, when an entire group is at fault and one person accepts all the blame, the group "feels better," but they really have not learned from the experience, which, in essence, is that one person becomes a scapegoat. The real lessons to learn from this are not simple ones. Although we are all responsible for what we do, simply blaming specific people may only have the effect of exonerating everyone else.

One question raised in the book is what the American Academy of Dermatology should do about the issue. Perhaps a statement that research on prisoners should follow the American Civil Liberties Union guidelines itemized on pages 199 and 200 of Hornblum's book would be significant.

James Krivo, MD
516 Dogwood Ave
Franklin Square, NY 10010

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Chronic Actinic Dermatitis Is Not a Viable Concept

The term "chronic actinic dermatitis" (CAD), coined by Hawk and Magnus,[1] is used synonymously with the term "photosensitivity dermatitis/actinic reticuloid syndrome" coined by Frain-Bell and colleagues.[2] Chronic actinic dermatitis encompasses 4 diagnoses: actinic reticuloid, photosensitive eczema, photosensitivity dermatitis, and more recently, persistent light reactivity,[3] originally described as distinct disorders but generally accepted as variants of CAD. Burry[4] questions the validity of the term CAD and the concept of a unifying diagnosis. In particular, he suggests that diagnos-

tic errors will be made and diagnoses of airborne contact dermatitis (particularly Compositae dermatitis), photoallergic contact dermatitis, and allergy to photosensitizing medication will be missed and managed inappropriately.[4] This is not the case.

Phototesting, generally with an irradiation monochromator, is essential for a confident diagnosis of CAD, and patch and photopatch tests are essential ancillary investigations.[3,5] This enables the distinction between CAD alone (abnormal monochromator light test and negative patch and photopatch test results) and CAD with concomitant allergic or photoallergic contact dermatitis (abnormal monochromator light test and abnormal patch and/or photopatch test results). This is the only way of making a confident diagnosis; clinical features such as the sparing of the so-called Wilkinson triangle behind the ear being, in our opinion, unreliable in distinguishing photosensitivity from airborne allergy. While sun protection measures are the keystone to the management of patients with CAD, avoidance of relevant associated contact allergens or photocontact allergens is of equal importance. Either measure on its own, sunlight avoidance or allergen avoidance, will not control the disease.

The diagnosis of CAD is distinct from that of Compositae dermatitis or other types of contact dermatitis and from photoallergic contact dermatitis. However, both contact and photoallergic contact dermatitis seem to predispose to, and are associated with, the development of sensitivity to UV light, as indeed do other forms of eczema such as endogenous eczema, such that the patient then reacts to UV light alone in the absence of allergen.[5] The whole point of the "unifying concept"[3] is that the result is the same (ie, eczema following exposure to UV light) irrespective of the disease associations (whether exposure to contact or to photocontact allergens and whether in a European garden, farm, or city, in the Australian bush, or in the United States) and irrespective of the morphologic features of the eruption (predominantly pseudolymphomatous or eczematous features) and the precise UV wavelengths implicated. Indeed, we might expect the associated contact allergens to reflect local exposure and to differ worldwide. This may well be the case with Compositae dermatitis, which is strongly associated with the disease in the population in Scotland,[5] less so in the population in the United Kingdom,[5] and not associated in the population in America.[6]

Immunohistochemical studies have confirmed that CAD is a T-cell driven, type IV immune response.[5] Early hypotheses on the pathogenesis of CAD included the theory that photoallergic chemicals may persist in the skin after the original application; this has not been substantiated and, in any event, would not explain the association with contact or endogenous eczema. Only up to 75% of patients with CAD have associated contact and/or more rarely photocontact allergy[5]; thus, contact or photocontact dermatitis may not have a direct role in the pathogenesis of CAD, although this remains possible. It is perhaps more likely that heightened immune reactivity, due to any form of eczema, may then predispose to reaction to UV light–induced antigen(s). Experimental models in guinea pigs have shown that injection of adjuvant alone (without hapten) in the presence of UV light may subsequently lead to UV light sensitivity mimicking CAD.[7] Alternatively, we may speculate that both the associated contact allergies and the type IV response to UV light reflect immune dysregulation; there is no evidence that refutes or supports this.

The role, if any, of photosensitizing medication in the development of CAD is unclear. However, we agree with Burry that it is sensible to discontinue treatment with such medication,[4] although there is a case for restarting the drug therapy if there is no improvement within 6 months. Systemic drug-induced photosensitivity is associated with normal cutaneous irradiation test findings or abnormalities in the UV-A range only, and withdrawal of the drug will resolve the abnormal clinical and positive phototest results within 6 months.[5]

Until the pathogenesis of CAD is fully elucidated, it may be sensible to remain aware of the route leading to the CAD process, whether associated with contact allergy or preceding photoallergic contact dermatitis and so forth; thus, one may here mention CAD arising, albeit rarely from persistent light reactivity. However, we are strongly of the opinion, in light of experience to date, that persistent light reactivity, actinic reticuloid, photosensitive eczema, and photosensitivity dermatitis are all part of the CAD spectrum of disease and that the development of a unifying diagnosis has clarified management of and research into this unusual disorder.

Hélène du P. Menagé, BSc, MRCP
John L. M. Hawk, MD, FRCP
Department of Photobiology,
St John's Institute of Dermatology
St Thomas' Hospital
Lambeth Palace Road
London SE1 7EH, England

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In reply

If "phototesting . . . with an irradiation monochromator" takes precedence over patch and photopatch testing in diagnosis and thereby prevention of environmental photodermatitis, something has gone wrong in the practice of clinical dermatology. Patch and photopatch testing, the continuation of the clinical processes of history taking and physical examination should not be relegated to "ancillary investigations," and a confident diagnosis of chronic actinic dermatitis (CAD) should not be made as a result of ab-

normal monochromatic light test and negative patch and photopatch test findings. Since the clinical evidence and the initial clinical assessment have indicated the need for such tests, the absence of positive test results indicates the need for further investigations, which might lead to previously unknown causative agents through further patch testing.

Do those confident diagnoses of "CAD alone" represent a "dustbin" of "too difficult" cases and missed opportunities for the discovery of unknown etiologies, or do they represent conditions entirely unrelated to those that have been explained satisfactorily as a result of past patch test research, and which du P. Menagé and Hawk place within the "CAD spectrum of disease?" No matter what the answers to these questions, the last thing able-bodied dermatologists should do is jump to the conclusion that a common factor explains all, especially if it is based on the "unifying concept"[1] of a "hypothetical endogenous photoallergen."[1]

The just-so story of the unifying concept of an endogenous photoallergen cannot be sustained without faith and is the direct opposite of evidence-based medicine, which demands proof of treatment or clinical explanation through epidemiological survey. The suggestion in the du P. Menagé and Hawk letter that they already know the result of a world epidemiological survey of photodermatitis, based on experience of CAD coming from monochromators in London, England, and Dundee, Scotland, is reminiscent of Plato's cave metaphysics, tarot cards, and astrology.

They have faith in what I consider the pseudoscience of irradiation monochromator-driven morphologic reductionism, and I am by no means certain, in spite of their confidence, that it does not constitute a barrier to sound practice. If the "CAD-alone" "diagnosis" means anything, it means complacency, which is why I think du P. Menagé and Hawk, who obviously do not approach their work complacently, should have done with it.

If my memory serves me correctly, idiopathic erythroderma and exfoliative dermatitis were once thought in the 1940s and 1950s to be due to "endogenous allergens." In 1966, Russell wrote "[i]n the management of patients with eczema it is sound practice not to accept a diagnosis of idiopathic eczema until a determined attempt has been made to discover some material and perhaps specific cause."[2] Should we more than 30 years later in the midst of our modern chemicalized culture accept that idiopathy constitutes any "diagnosis" at all, especially if an endogenous element is postulated and exogenous causes have already been proven in abundance?

I am grateful for the opportunity to enlarge on my opinion first expressed in 1984 "that too much technology has been used in the study of photosensitivity and not enough clinical medicine."[3]

John N. Burry, FRCPE
PO Box 7177, Hutt St
Adelaide, 5000
South Australia, Australia

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The Changing Spectrum of the Cutaneous Manifestation of HIV Disease

In their editorial, Costner and Cockerell[1] mention several skin conditions related to human immunodeficiency virus (HIV) that improve during highly active antiretroviral treatment (HAART). They do not mention, however, the potential adverse dermatological effects of such therapy.

The protease inhibitor indinavir sulfate, for example, may cause alopecia, dry skin, pruritic skin eruptions, and paronychia.[2,3] We observed 2 patients treated with indinavir who developed a vertical lip fissure during the summer. The fissure of the lips probably was related to the dryness of the lips caused by the indinavir. We also observed a patient who lost all the hair on his legs and axillary region during indinavir, stavudine, and lamivudine treatment. His hair started growing again when the indinavir was replaced by nelfinavir. Allergic skin eruptions can be seen with all antivirals but may be particularly severe, leading to a Stevens-Johnson syndrome, with abacavir.[4]

In future cohort studies, not only the beneficial effects of HAART but also the potential adverse effects should be studied.

Robert Colebunders, MD, PhD
Marc Vandenbruaene, MD
Institute of Tropical Medicine
Nationalestraat 155
B-2000 Antwerpen
Belgium
(e-mail: bcoleb@itg.be)

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Follow-up Letter From Tanzania

In 1994, the ARCHIVES kindly published my report on our 6-month sabbatical at the Regional Dermatology Training Centre (RDTC) in Moshi, Tanzania.[1] In July 1996 and again in July 1998, my wife and I returned to the RDTC for short visits. During each visit, I served as the external examiner for the year-end oral examinations of the students. This year, the students did well in the examinations, and once again all passed. I continue to be impressed with the success of this 2-year dermatology training program, and so I would like to provide this update on its present status.

The program was started in 1992 under the sponsorship of the International Foundation for Dermatology, chaired originally by Alfred W. Kopf, MD, and currently by Terence J. Ryan, DM, FRCP. Both have played extraordinary roles in establishing and financing this training facility.

The students are experienced medical assistants and nurses who come from many English-speaking sub-Saharan African countries for a 2-year training program in dermatology. Many do so at great personal sacrifice, leaving spouses and children at home in distant countries. Through my initial sabbatical and subsequent service as external examiner, I have met all the students who have participated in this program. To date, 49 students have graduated and returned to their homes in a variety of professional positions. Many of the countries have few or even no dermatologists, and so many of the students serve as important dermatology resources in their regions. Several graduates have been assigned to national posts, eg, 2 graduates of the first class became national directors for the sexually transmitted diseases programs in their respective countries, Malawi and Zambia. From my encounters with the most recent students, I continue to be impressed with their intellect, enthusiasm, congeniality, and dedication. In October 1998, 13 new students (including 2 from 2 new countries in the program—Cameroon and Ghana) will enter the program to join the 13 second-year students who return from their summer research projects.

The physical facilities for the training program have improved dramatically in the past 2 years. With funding from the International Foundation for Dermatology, a beautiful new RDTC building was built; it was dedicated in January 1997. This 2-story facility includes a number of examination rooms, 2 outpatient surgery rooms, a large and beautiful library, conferences rooms, and faculty offices with views of Mount Kilimanjaro, as well as a fountain in the lobby. The new building is spectacular, particularly in contrast with the old facility, which was located in the Kilimanjaro Christian Medical Centre, which is the adjacent hospital. (Under new directorship, however, improvements in the previously deteriorating hospital facility are also now noticeable.) Dermatology inpatients are still admitted to the dermatology wards of the Kilimanjaro Christian Medical Centre.

Last year, a second medical school for Tanzania, a country of 27 million people, was opened at the Kilimanjaro Christian Medical Centre. The medical students will receive their dermatology training at the RDTC, and this will provide another challenging opportunity for teaching dermatology in this part of the world.

While the new RDTC facility is impressive, the dedicated faculty are the heart and soul of the program. Enormous credit is due to Henning Grossmann, MD, who continues his critical work as founding director of the program, and Barbara Leppard, MD, who has been invaluable in coordinating the educational curriculum. In addition, Tanzanian faculty members are playing increasingly important roles. They are John Masenga, MD, who serves as vice principal of the RDTC; Anthony Kazoka, MD, an epidemiologist and senior registrar in the RDTC; and Alfred Naburi, who graduated in the first class in 1994. This past year, James Nordlund, MD, also deserves high praise for his enthusiastic and effective work. He and his wife Mary spent the whole of 1998 on sabbatical at the RDTC, and both have made important contributions to the program.

The Nordlunds had been preceded by other volunteers who worked for at least 6 months at the center. These include Sandy McBride, MD, from the United Kingdom, Peter Schmid, MD, and his wife from Switzerland, Lorne Albright, MD, from Canada, and Sue Conley, MD, and Marvin Weinreb, MD, and his wife from the United States. More volunteers are needed in the future. I can attest that the experience is enriching, rewarding, and unforgettable, and I recommend it highly. I would be more than pleased to provide further details to anyone who might be interested. Individuals who might be inspired to help with the ongoing needs for funding should contact either Dr Kopf or Dr Ryan. Spaces for bronze plates that recognize contributors to the "500 Club" are still available on a plaque hanging in the RDTC.

In addition to the teaching program in Tanzania, an outreach program was established for treating and providing preventive services to the many individuals in the region who have albinism and who suffer from skin cancers.[2] Thanks to the efforts of Dr Leppard, this project is still operational. It has now been ongoing for 5 years and continues to provide valuable service to the many albino patients in the Kilimanjaro region. It also provides an excellent educational opportunity for the dermatology trainees.

In summary, I am pleased to report that the RDTC in Tanzania is alive and well. I continue to be greatly impressed with the students and their accomplishments after completion of their training. The stunning success of the program is due in large part to the incredible efforts of a small number of dedicated people, many of whom are mentioned above. All concerned should feel proud of this remarkable program in east Africa. Its past has been highly productive, and its future appears bright.

Donald P. Lookingbill, MD
Mayo Clinic Jacksonville
Department of Dermatology
4500 San Pablo Rd
Jacksonville, FL 32224

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VIGNETTES

Textural Change Following Treatment of Facial Telangiectasias With the Tunable Pulsed-Dye Laser

Facial telangiectasias (spider veins) have been attributed to a variety of etiologies—most commonly chronic UV exposure, hormone therapy or changes, drugs or alcohol, heredity, and as a secondary phenomenon to many diseases. Therapeutic modalities have included electrodesiccation, dermabrasion, argon laser, and, more recently, the flash lamp–pumped pulsed-dye lasers.[1] Initially, it was thought that these instruments had no potential to cause scarring. However, as with any device that delivers photon energy to

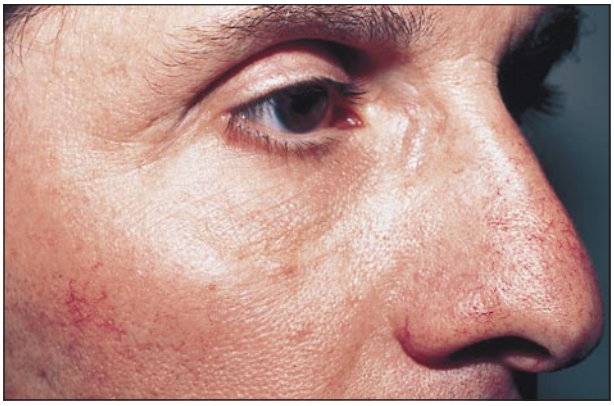

Figure 1. *Preoperative appearance revealing telangiectasias and photo damage.*

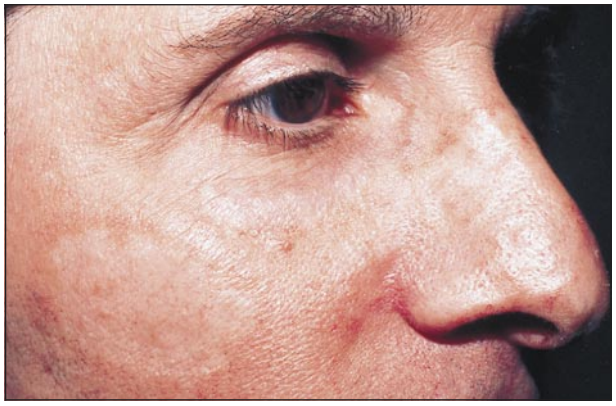

Figure 2. *Textural change on right cheek and in right nasolabial fold.*

the skin, untoward consequences, such as textural change, increased or decreased pigmentation, persistent erythema, or various forms of scarring are certainly possible.

The earlier Candela dye laser, the SPTL-1 (Candela Corporation, Weyland, Mass), was employed principally for facial telangiectasias and port-wine stains at fluences of 5 to 7 J/cm^2, 585 nm, and a 5-mm spot, with resultant purpura as an end point. A subsequent version, the Candela ScleroPlus laser, has 4 wavelengths between 585 and 600 nm, 6 spot sizes, fluences of 2 to 30 J/cm^2, and is intended for treatment of a greater variety of skin diseases, including telangiectasias of the legs.[2] Initial guidelines circulated by the manufacturer recommended fluences of 5 to 20 J/cm^2 with a 5-mm spot for treatment of facial telangiectasias, without the production of a purpuric end point.[3]

Subsequent information released by the manufacturer recommends fluences of 8 to 9 J/cm^2, 595 nm, with a 5-mm spot for facial veins. An intraoperative hydrogel dressing is recommended only for leg vein therapy or for settings above 13 J/cm^2.[4]

Report of Cases. On November 22, 1996, the Candela ScleroPlus laser was used to treat 2 patients with small red superficial facial telangiectasias. The first patient, a 25-year-old white man, was treated with settings of 12 J/cm^2, 585 nm, with a 5-mm spot, using 534 pulses. He experienced no immediate postoperative purpura and healed uneventfully with disappearance of the vessels. The second patient, a 49-year-old white man with nearly identical telangiectasias but more pronounced photodamage, was treated with identical settings of 12 J/cm^2, 585 nm, and a 5-mm spot using 534 pulses. Additionally, in separate areas, "dark circles" on each lower lid were treated with the Candela pigment laser, 3 J/cm^2, 755 nm, and a 5-mm spot using 56 pulses (**Figure 1**). Immediately after surgery, a slight grayish pink appearance was noted; Vigilon dressing (CR Bard Inc, Murray Hill, NJ) was applied, and a 4-mg, 6-day dose pack of methylprednisolone (Medrol) was prescribed.

An examination 3 days after surgery revealed the typical purple discoloration with minimal edema. However, 7 days after surgery, considerable crusting and some oozing was seen in areas treated with the vascular laser. Cefadroxil (Duricef), 500 mg, 2 times per day was prescribed for 10 days. At the 18-day postoperative visit, the areas had healed, although some textural change and lightened pigmentation were noted (**Figure 2**), which persisted 4 months after surgery.

Comment. Several explanations might be proposed for the untoward outcome noted in these illustrations.[5] Secondary infection is certainly a possibility, either with *Staphylococcus aureus* or herpes simplex virus. Trauma may be considered, although the patient denied any trauma. Preexisting atrophy, photodamage, and poikiloderma may have contributed to this occurrence, or perhaps the energy fluence of 12 J/cm^2 with a 5-mm spot was too high for facial telangiectasias. However, identical settings were employed in the patient treated just prior to the subject of discussion, and a near-perfect outcome ensued in that case. The lack of an obvious visible end point to ScleroPlus laser therapy (such as purpura) is definitely a hindrance to therapy; these "invisible pulses" lead to the possibility of overlapping or uneven treatments. Laser equipment malfunction is also, of course, a possibility, even though the textural changes were noted in all treated areas. Further study and experience are certainly needed with respect to tunable dye laser parameters, settings, and indications. Only when large-scale studies have been published can predictable outcomes and safe treatment parameters be developed for each machine and in each disease state.

James M. Swinehart, MD
Denver, Colo

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The Importance of Early Cushing Disease Diagnosis

I am writing to convey a small reminder to the many dermatologists whom this publication reaches. It is not meant as a criticism, but is submitted with the hope that a few people may be spared some of the pain and physical deterioration my mother endured because of a much-delayed diagnosis of Cushing disease.

The earliest and ultimately most exasperating symptom that my mother experienced was tissue paper–thin skin that bruised extremely easily. This was most marked on the dorsi of her forearms and hands, and the lesions took months to heal. Something as trifling as the brush of her infant granddaughter's fingernail could literally tear her skin as if it were wet tissue paper.

My mother, while in her early 60s, consulted 5 board-certified dermatologists over a 2-year period and came away with the diagnosis of purpura, presumably due to age. The worst result of the delayed diagnosis was the severe osteoporosis my mother now suffers from, though hopefully this, like her skin, will improve with time. She also went through surgery in much worse physical condition than she would have if diagnosed only 6 months earlier.

Cushing disease, the excess secretion of corticotropin from a pituitary adenoma that stimulates excess corticosteroid secretion from the adrenal glands, occurs primarily in women, aged 25 to 45 years. Easy bruisability is a commonly reported symptom. Orth and colleagues[1] further describe the indications: "The skin is atrophic, the stratum corneum is thinned, and there is loss of subcutaneous fat. The skin becomes fragile and, in extreme cases, peels off with adhesive tape like damp tissue paper (Liddle's sign)."

Though Cushing disease is uncommon, the syndrome is not. Though gradual in their development, the buffalo hump, moon facies, new-onset hypertension, proximal muscle weakness, hirsutism, weight gain, and other signs and symptoms were present in my mother. Her eventual diagnosis elicited a flashback to medical school for me, to the chairman of medicine bellowing his mantra to listen to the patient, imploring us to look at the whole patient.

My family and I want to reiterate the important role that dermatologists have in the diagnosis of systemic disease. Aging is the cause of many changes in our bodies, but the assumption of such should be made only after careful consideration and exclusion of other, pathologic, possibilities.

Susan Molchan, MD
8723 Ridge Rd
Bethesda, MD 20817

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Comparison of Calcipotriene (Dovonex) With a Coal Tar Emulsion (Exorex) in Treating Psoriasis in Adults: A Pilot Study

Coal tar, a mixture of at least 10 000 components, has long been shown to have efficacy in the treatment of psoriasis.[1] Exorex (IMX Pharmaceuticals Inc, Boca Raton, Fla) is a new coal tar–based, Food and Drug Administration–sanctioned psoriasis medication deemed safe for sensitive skin. Calcipotriene (Dovonex; Westwood-Squibb Pharmaceuticals, Buffalo, NY) is an active cholecalciferol analog, useful in psoriasis therapy, but which can cause dermatitis in sensitive areas.[2] We conducted a pilot study comparing the therapeutic efficacy and safety of Dovonex (hereinafter, "calcipotriene") and Exorex (hereinafter, "1% coal tar emulsion") in treating psoriasis.

Patients and Methods. Twenty adult patients with plaque-type psoriasis (16 men and 4 women, aged 18-75 years) were enrolled in this single-blind intrapatient comparison study. Five dropped out after baseline evaluation. Each patient underwent a blood chemistry analysis, a complete blood cell count, and urinalysis at baseline and monthly through the study's duration. Two similar 50-cm^2 psoriatic lesions were selected in each patient for twice-daily treatment with either 0.1 g of calcipotriene or 0.1 g of the 1% coal tar emulsion followed 2 minutes later by application of moisturizing cream (Exorex Stabilizing Cream; IMX Pharmaceuticals Inc). All treated lesions were photographed at baseline and at every visit. Erythema, scaling, and plaque thickness of the treated lesions were evaluated using a 4-point scale (0, no lesion; 2, mild; 4, moderate; 6, severe). After 2 months, 4-mm punch biopsy specimens were taken of the lesions treated with calcipotriene, those treated with the 1% coal tar emulsion, and normal skin.The patients were treated for a mean (± SD) 64.4 ± 7.92 days (range, 12-125 days). Statistical significance was assessed by the 2-tailed Student *t* test for paired differences.

Results. The mean ± SD pretreatment severity scores for erythema, scaling, and plaque thickness were 4.1 ± 0.3, 4.1 ± 0.4, and 3.7 ± 0.4, respectively. After treatment with the 1% coal tar emulsion, the scores for erythema, scaling, and plaque thickness decreased to 2.2 ± 0.3 (a 41% ± 8% decrease; $P<.001$), 1.9 ± 0.5 (a 56% ± 10% decrease; $P<.01$), and 2.4 ± 0.4 (a 31% ± 13% decrease; $P<.05$), respectively. After treatment with calcipotriene, scores for erythema, scaling, and plaque thickness decreased to 2.1 ± 0.2 (a 42% ± 8% decrease; $P<.001$), 1.6 ± 0.4 (a 63% ± 9% decrease; $P<.001$), and 1.7 ± 0.3 (a 47% ± 9% decrease; $P<.001$). No significant inter-treatment difference was observed in improvement of scaling, erythema, or plaque elevation. No adverse effects or laboratory abnormalities were noted after treatment with either medication.

Comment. This pilot study suggests that this preparation of 1% coal tar emulsion is about as effective as calci-

potriene in treating psoriasis. In this study, which was not vehicle controlled, the clinical improvement with either medication was comparable and substantial after 1 month of treatment. Long-term effects of 1% coal tar emulsion are not yet known, and further studies are indicated.

Irini E. Veronikis, MD
Alan O. Malabanan, MD
Michael F. Holick, PhD, MD
Department of Medicine
Boston University School of Medicine
715 Albany St, M-1013
Boston, Mass 02118

Supported in part by grant M01RR00533 from the National Institutes of Health, Bethesda, Md, and funding from IMX Pharmaceuticals Inc, Boca Raton, Fla.

Presented in part at the 58th Annual Meeting of the Society for Investigative Dermatology, Washington, DC, April 26, 1997.

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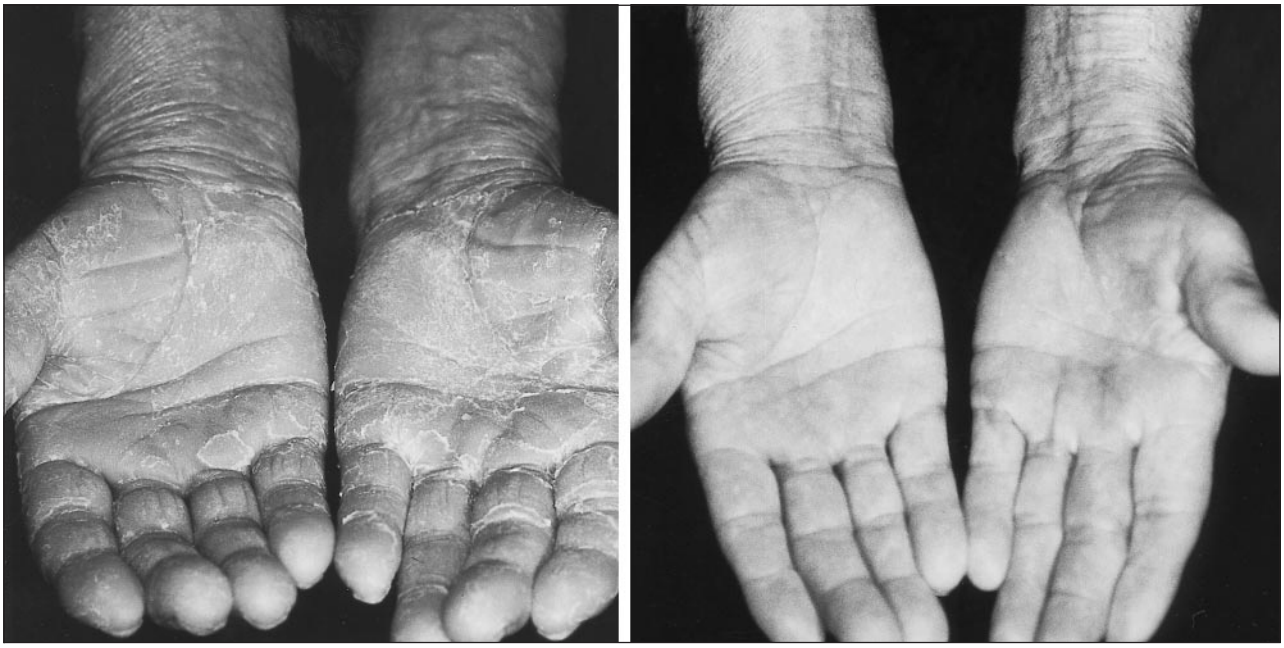
Extracorporeal Photochemotherapy for the Treatment of Erythrodermic Pityriasis Rubra Pilaris

Pityriasis rubra pilaris (PRP) is a rare papulosquamous disorder of unknown origin. Although PRP usually resolves within 1 to 3 years,[1] treatment of the disease is generally difficult. Encouraged by good results with extracorporeal photochemotherapy (ECP) in patients with erythroderma of other origin,[2-4] we treated 2 patients with erythrodermic PRP type I using ECP in combination with systemic retinoids and cyclosporine, respectively.

Report of Cases. *Case 1.* A 65-year-old white man presented in April 1995 with a 3-week history of generalized erythroderma with several spared, roundish areas of normal skin on the trunk, hyperkeratotic and yellowish palms (**Figure**, left) and soles, and thickened fingernails and toenails. A biopsy specimen from the left forearm revealed psoriasiform epidermal hyperplasia with dilated infundibula plucked by cornified cells, alternating orthokeratosis, and parakeratosis of the stratum corneum, and a sparse perivascular lymphocytic infiltrate and dilated vessels in the stratum papillare of the dermis. The histological findings were consistent with a diagnosis of PRP. Treatment with psoralen and UV-A in combination with acitretin for 6 weeks was without any therapeutic success. In July 1995, we started the patient on ECP with 150 mg of methoxsalen that was placed directly into the treatment bag of the ECP apparatus (UVAR II System; Therakos, Exton, Pa). Extracorporeal Photochemotherapy was applied twice a week every 2 weeks for 1 month and once a month thereafter. Therapy with systemic retinoids was continued. A first improvement of the skin rash was noted at 6 weeks after the first ECP treatment. At the end of August 1995, we first detected an enlarged prostate, and biopsy gave the diagnosis of prostatic carcinoma. After suprapubic prostatectomy, the patient noted continuous improvement of the skin rash. The therapy with systemic retinoids was discontinued, but ECP was continued at 4 week-intervals. In January 1996 (6 months after the first ECP and 4 months after prostate surgery) the erythroderma had cleared and palms (Figure, right) and soles as well as fingernails and toenails appeared



A 65-year-old man with pityriasis rubra pilaris. Before extracorporeal photochemotherapy, the patient exhibited hyperkeratotic and yellowish palms (left). Six months after starting treatment with extracorporeal photochemotherapy, the patient showed complete remission of hyperkeratosis on palms (right).

normal. Therefore, treatment with ECP was discontinued. By July 1998, follow-up still revealed complete remission.

Case 2. A 71-year-old white man presented in March 1996 with a 4-month history of generalized erythroderma with some spared, roundish areas of normal skin, hyperkeratotic yellowish palms and soles, and thickened fingernails and toenails. The histology of a biopsy specimen taken from the abdomen was consistent with a diagnosis of PRP. Retinoid psoralen and UV-A given from January to March 1996 was without clinical success. Since the therapeutic response to cyclosporine 200 mg daily from April to May was also negligible, ECP was added in June 1996 and given twice a week every 2 weeks for 1 month, and once a month thereafter. During ECP and cyclosporine therapy, the patient's skin rash gradually improved within several weeks. By March 1997, the patient's erythroderma and hyperkeratotic changes of the palms and soles as well as nails had nearly completely resolved. In June 1997, the dosage of cyclosporine could be gradually reduced to 25 mg daily, and the frequency of ECP was decreased to 1 set of 2 treatments given 8 weeks apart. Since then the patient's skin changes have remained in remission under continued combination therapy with cyclosporine and ECP.

Comment. The skin rashes in both of our patients fulfilled the criteria of classic adult-onset PRP type I. In our first patient, a paraneoplastic origin of the PRP was possible since the PRP was associated with cancer of the prostate. Indeed, PRP is previously reported in association with squamous cell carcinoma of different origin and adenocarcinoma of the prostate.[5]

Our 2 patients responded to ECP in combination with other therapeutic agents. Since both patients earlier had no therapeutic response to systemic retinoids, psoralen and UV-A, or cyclosporine alone, ECP seemed to be the most significant factor for therapeutic response in both cases. However, in our first patient, the erythrodermic PRP may have been paraneoplastic in origin, and, thus, removal of his prostate cancer may have at least contributed to the resolution of the skin disease. Nevertheless, our observations indicate that ECP may represent an alternative and effective treatment modality for erythrodermic PRP.

Angelika Hofer, MD
Robert Müllegger, MD
Helmut Kerl, MD
Peter Wolf, MD
Department of Dermatology
University of Graz
A-8036 Graz, Austria

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