

Correspondence

Polymerase chain reaction

To the Editor: I read with interest the recent report by Zhu et al. (J AM ACAD DERMATOL 1992;26:710-4). Powerful techniques such as the polymerase chain reaction (PCR) have the ability both to illuminate and to confuse. With the advent of extraordinarily sensitive probes for DNA has come the casual assumption that by identifying the DNA, we have understood the infection itself. Unfortunately, in vitro experimentation often fails to correspond to in vivo reality.

Zhu et al. applied therapeutic agents (20% podophyllin, 40% trichloroacetic acid [TCA], and liquid nitrogen) directly to freshly excised fragments of condylomas. The tissue specimens were then examined for the presence of human papillomavirus (HPV) DNA. HPV DNA was not found in warts after exposure to podophyllin or TCA, whereas liquid nitrogen-frozen tissue continued to contain intact HPV DNA. The authors argue that these data suggest that podophyllin and TCA "act in part by damaging DNA." They propose that liquid nitrogen acts by causing physical removal of infected tissue, without destruction of HPV DNA.

These arguments are flawed. Exposure in vitro to 20% podophyllin or 40% TCA in no way realistically models their actions in vivo. Viable epidermis is a remarkably effective barrier to chemical penetration, in contrast to the "raw" surfaces exposed on the excised tissue specimens.¹ One could predict that only a tiny fraction of podophyllin or TCA applied topically in vivo would actually penetrate the epidermis. Once there, it would be further diluted by the dermal circulation, giving levels far below the 20% and 40% concentrations used. The experiments of Zhu et al. are analogous to suggesting that because rubbing alcohol kills bacteria when applied to a tissue culture plate, topical application of alcohol would be an effective treatment of cellulitis. The model also fails to account for the effects of local tissue destruction and the accompanying inflammatory and possibly immunologic reactions to HPV.

Available evidence suggests that podophyllin is in fact a relatively poor treatment of condylomas, in part because of the substantially higher recurrence rates after treatment when compared with treatments such as cryotherapy.² The suggestion that podophyllin is a superior treatment on the basis of its ability to destroy HPV DNA is not supported by the evidence.

It is likely that after any successful treatment of condylomas, HPV DNA will be found to persist.³ Such persistent DNA does not necessarily correlate with the presence of clinically identifiable lesions; whether such persistent sites of DNA are infectious is not known. Until the significance of persistent HPV DNA is better understood, and until more relevant models for the effect of

treatment on the presence of such DNA exist, data from in vitro PCR analysis will remain of questionable relevance.

Mark R. Ling, MD, PhD
Department of Dermatology
Emory University School of Medicine
5001 Woodruff Memorial Building
Atlanta, GA 30322

REFERENCES

1. Arndt KA, Menzies PV. The pharmacology of topical therapy. In: Fitzpatrick TB, Eisen AZ, Wolff K, et al, eds. *Dermatology in general medicine*. New York: McGraw-Hill, 1987:2532-40.
2. Ling MR. Therapy of genital human papillomavirus infections. Part II. Methods of treatment. *Int J Dermatol* (In press.)
3. Ling MR. Therapy of genital human papillomavirus infections. Part I. Indications for and justification of therapy. *Int J Dermatol* (In press.)

Dermatopathia pigmentosa reticularis

To the Editor: It seems likely that dermatopathia pigmentosa reticularis described by Heimer et al. (J AM ACAD DERMATOL 1992;26:298-301) is the same condition as autosomal dominant hereditary diffuse hyperpigmentation that I described in a family in 1980.¹ A 33-year-old woman had scalp hair thinning, congenital mottling of the skin, smooth fingertips, brittle fingernails (some split at the free end), and palmar keratoses. She also gave a history of heel blisters in early life. Sweating and the condition of her teeth were normal.

Julian L. Verbov, MD
Dept. of Dermatology
Royal Liverpool University Hospital
Liverpool, L7 8XP, U.K.

REFERENCE

1. Verbov J. Hereditary diffuse hyperpigmentation. *Clin Exp Dermatol* 1980;5:227-34.

Reply

To the Editor: We are grateful to Dr. Verbov for calling our attention to his report of a woman with a reticulate hyperpigmentation that he termed *hereditary diffuse hyperpigmentation* (HDH).¹ We believe Dr. Verbov's case represents dermatopathia pigmentosa reticularis (DPR) as first reported by Hauss and Oberste-Lehn² and later by others.³⁻¹⁰ Because the cause of this unusual ectodermal dysplasia is still unknown, it is clinically helpful to adhere to a guideline of criteria to assist in making the diagnosis

of DPR. Several comparative tables have been published.⁸⁻¹⁰ Naegeli-Francescetti-Jadassohn syndrome most closely resembles DPR, but one sees dental anomalies and a fading of the hyperpigmentation with age, neither of which have been reported with DPR.¹⁰

One difference between previous cases of DPR (including our case) and the case presented by Dr. Verbov is the "absence of sweating problems." No quantitation of sweating was reported in Dr. Verbov's case. It is possible that an asymptomatic mild hypohidrosis existed. This point emphasizes the need for quantifying sweating by an accepted method when these patients are examined, regardless of history.

CPT William L. Heimer II, MC, USA
COL William D. James, MC, USA
Dermatology Service
Walter Reed Army Medical Center
Washington, DC 20307-5001

REFERENCES

1. Verbov J. Hereditary diffuse hyperpigmentation. *Clin Exp Dermatol* 1980;5:227-34.
2. Hauss H, Oberste-Lehn H. Dermatopathia pigmentosa reticularis. *Dermatol Wochenschr* 1958;138:1337.
3. Flegel H. Dermatopathia pigmentosa reticularis. *Hautarzt* 1960;11:262-5.
4. Gahlen W. Dermatopathia pigmentosa reticularis hypohidrotica et atrophica. *Dermatol Wochenschr* 1964;150:193-8.
5. van der Lugt L. Dermatopathia pigmentosa reticularis hyperkeratotica et mutilans. *Dermatologica* 1970;140:294-302.
6. Lunder M, Fettich J. Beitrag zum Begriff der Dermatopathia Pigmentosa Reticularis. *Z Hautr* 1973;48:857-63.
7. Rycroft RJG, Calnan CD, Allenby CF. Dermatopathia pigmentosa reticularis. *Clin Exp Dermatol* 1977;2:39-44.
8. Maso M, Schwartz R, Lambert W. Dermatopathia pigmentosa reticularis. *Arch Dermatol* 1990;126:935-9.
9. Sparrow GP, Sammam PD, Wells RS. Hyperpigmentation and hypohidrosis (the Naegeli-Francescetti-Jadassohn syndrome): report of a family and review of the literature. *Clin Exp Dermatol* 1976;1:127-40.
10. Heimer WL II, Brauner G, James WD. Dermatopathia pigmentosa reticularis: a report of a family demonstrating autosomal dominant inheritance. *J AM ACAD DERMATOL* 1992;26:298-301.

Pool palms

To the Editor: For many years I have been devoted to all aspects of the effects of water on the skin.¹⁻³ I have found the observations of Blauvelt et al. on "pool palms" interesting (*J AM ACAD DERMATOL* 1992;27:92), but perhaps the presentation was too brief and not particularly detailed.

I do have some questions. (1) Are calluses or a callus-like reaction being described? (2) Why are the lesions described as red? (Calluses are not red.) (3) Have they

been observed in more than one race or only in white persons? (I understand that the number of patients reported is limited, and larger series may be necessary to better determine a racial predisposition.) (4) How is the "linear" configuration explained? (5) Do they occur in "young swimmers" only? (Perhaps they do, but the observations may be biased considering the age of the patient population that most of the authors see in their pediatric dermatology practices.) I wonder which material was used to build the floors and walls of the pools. If cement was used, perhaps the erythema could result from contact with this rough surface. If tile or other smooth materials were used, then I find it difficult to explain the erythema. We can exclude both chlorine and sodium hypochlorite because they rarely produce irritant, allergic, or urticarial reactions.⁴

Most probably we are dealing with a "wet dermatitis."⁵ We coined this term to describe persons with special susceptibility who, when exposed to a combination of prolonged water contact, friction, chemicals, and microbes, develop a chronic dermatitis. This phenomenon may be responsible for the erythema noted in "pool palms."

I hope that my comments are helpful. Obviously we have much to learn about this new aquatic dermatosis.

Ricardo M. Mandojana, MD
1525 Kensington Dr.
Knoxville, TN 37922

REFERENCES

1. Mandojana RM. Dermatoses of the aquatic environment (aquatic dermatology). In: Moschella SL, Hurley HJ, eds. *Dermatology* 3rd ed. Philadelphia: WB Saunders, 1992: 2003-22.
2. Mandojana RM. Aquatic dermatoses of the tropics. In: Canizares O, Harman RM, eds. *Clinical tropical dermatology*. 2nd ed. Boston: Blackwell Scientific Publications, 1992:707-21.
3. Mandojana RM, ed. *Aquatic dermatology*. *Clin Dermatol* 1987;5:3.
4. Fisher AA. Contact dermatitis to diving equipment, swimming pool chemicals and other aquatic denizens. *Clin Dermatol* 1987;5:838.
5. Mandojana RM, Simms JK. Miscellaneous dermatoses associated with the aquatic environment. *Clin Dermatol* 1987;5:139.

Reply

To the Editor: We appreciate the interest in our article on "pool palms," and we will attempt to address the comments made by Dr. Mandojana. The initial lesion is more appropriately described as an abrasion (explaining the erythema we observed) and not as a callus; however, the repeated scraping of the palms on gritty pool surfaces leads to callus formation with time. We have now seen patients in both the early and late stages. The lesions appear linear because the abrasion spares the slightly