Background: Pinta, 1 of the 3 nonvenereal treponematoses, is supposed to be extinct in most areas in South and Central America, where it was once endemic. Only scattered foci may still remain in remote areas in the Brazilian rain forest, and the last case from Cuba was reported in 1975.

Observation: A native Austrian woman, who had lived for 7 years in Cuba and was married to a Cuban native, developed a singular psoriasiform plaque on her trunk and several brownish papulosquamous lesions on her palms and soles during a visit to her home in Austria. Positive serological findings for active syphilis and the detection of spirochetes in the trunk lesion indicated early secondary syphilis, but an extensive case history and the clinical appearance fulfilled all criteria for pinta.

Conclusion: The acquisition of a distinct clinical entity, pinta, in a country where it was formerly endemic but now is believed to be extinct raises the question of whether the disease is in fact extinct or whether syphilis and pinta are so similar that no definite distinction is possible in certain cases.

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lived for the past 7 years in an urban area of Cuba, developed a solitary erythematosquamous plaque on the left side of her trunk. This lesion had lasted for almost 1 month when she was examined at our outpatient clinic. She did not remember any fever, tick bites, changes in oral or genital mucous membranes, joint pain, or lymphadenopathy. On questioning, she told us that her husband had observed a similar lesion in his right subaxillary region about 4 months before but that it had disappeared spontaneously; a routine test of her husband’s serum before minor surgery in Cuba had disclosed a positive serological test result for syphilis, and the husband had then been treated with penicillin. Both our patient and her spouse denied having had any extramarital relations in the recent past.

Clinical inspection of the patient disclosed a solitary erythematos, hyperkeratotic annular plaque of 2.5 cm in diameter in the left thoracolumbar area, resembling tinea corporis, a psoriatic plaque, granuloma annulare, or hyperkeratotic lichen planus (Figure 1 and Figure 2). Furthermore, several discrete erythematosquamous lesions on the palms and soles (Figure 3), each about 1 cm in diameter, were found; the patient had not been aware of them. The remaining skin as well as oral and genital mucous membranes showed no abnormalities; the lymph nodes were not enlarged, and no alopecia or any other signs of secondary syphilis could be detected.

<table>
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<tr>
<th>Characteristics</th>
<th>Pinta</th>
<th>Venereal Syphilis</th>
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<tbody>
<tr>
<td>Transmission</td>
<td>&gt;95% Nonsexually (extragenital pattern)</td>
<td>&gt;95% Sexually; hematogenous; diaplastic (3-9 mo)</td>
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<tr>
<td>Clinical manifestations</td>
<td>Stage 1: Exogenous; erythematous infiltrated plaque</td>
<td>Genital/extragenital; indolent ulcer; regional lymphadenopathy</td>
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<td></td>
<td>Stage II: PIIDS (small, scaly papules that enlarge to psoriasiform plaques with peripheral accentuation); no systemic symptoms</td>
<td>Fever, joint pain, lymphadenopathy, exanthema, condylomata lata, corona veners, “plaques muqueuses,” angina specifica, alopecia areolari specifica, and luetic leukoderma</td>
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<td></td>
<td>Stage III: Vitiligo-like depigmentation; no involvement of other organs</td>
<td>Gumma; involvement of skin, bones, central nervous system, and visceral organs (liver, kidney, etc)</td>
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<tr>
<td>Histopathology</td>
<td>Histological criteria for the differentiation of treponematoses are unreliable: acanthosis, intraepidermal neutrophilic microabscesses, upper dermal inflammatory infiltration around blood vessels</td>
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<td>Molecular techniques</td>
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<td>Epidemiology</td>
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<td>Therapy</td>
<td>≤2-y duration: penicillin G benzathine, 2.4 million U intramuscularly, 1 application; &gt;2-y duration: penicillin G benzathine, 2.4 million U intramuscularly, 3 times in 1 wk (in case of allergy to penicillin: doxycycline, erythromycin)</td>
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*From Koff and Rose.† TPHA indicates Treponema pallidum hemagglutination; FTA-ABS, fluorescent treponemal antibody absorption test; and ELISA, enzyme-linked immunosorbent assay.

Figure 1. A single nummular plaque in the left thoracolumbar region.

Figure 2. Close-up view showing a hyperkeratotic annular plaque.
Wound serum was harvested from the hyperkeratotic plaque on the trunk after mechanical erosion of the plaque. On dark-field microscopy, multiple typical treponemes could be observed performing their characteristic bending and rotational movements.

Histopathological examination was done on a 4-mm punch biopsy specimen from the plaque after formalin fixation and paraffin embedding. Hematoxylin-eosin staining showed a psoriasislike picture with parakeratosis, acanthosis, spongiosis, and an infiltration of lymphocytes, eosinophils, and abundant neutrophils in the epidermis forming intraepidermal abscesses. There was edema of the papillary dermis and an obscured epidermal-dermal interface. In the dermis there was a patchy perivascular and interstitial infiltrate of lymphocytes, plasma cells, neutrophils, and some eosinophils. Blood vessels showed dilation but no substantial endothelial swelling. Several melanophages were present in the dermis. Furthermore, abundant treponemes were detectable in the epidermis by silver staining and by an immunoperoxidase staining on cryostat sections with the use of a polyclonal anti–T pallidum antibody in a 1:200 dilution (Biodesign International, Kennebunk, Me) (Figure 4).

Figure 3. Several brownish red papulosquamous lesions, each about 0.5 to 1 cm in diameter, on the palms.

Figure 4. Numerous spirochetes in the epidermis of the hyperkeratotic trunk lesion (immunoperoxidase method with polyclonal anti–Treponema pallidum antibody, oil immersion, original magnification ×1000).

The following specific and nonspecific serological test results for Treponema were positive in our patient:

- T pallidum hemagglutination test, 1:5120
- Fluorescent treponemal antibody absorption test for IgM and IgG
- Captia-Syphilis-M IgM enzyme-linked immunosorbent assay (Biotech; Gull Laboratory, Bad Homburg, Germany)
- VDRL test, 1:32
- Western blot analysis with a T pallidum IgM and IgG test kit (Marblot; VIRAMED, Munich, Germany) showed bands of 15.6, 17, 47, 61, and 83 kd in the IgG blot and 17, 37, 41, and 47 kd in the IgM blot, a pattern typical of Treponema (Figure 5). As controls, serum samples from 8 patients with syphilis and 1 healthy person were investigated. Serum from our patient (lane 1) demonstrated bands nearly identical to those of serum from patients with syphilis I and II.

The patient was treated with 2.4 million U of penicillin G benzathine according to the World Health Organization regimen for the treatment of early syphilis. The lesions disappeared within a few days, and markers of active syphilis (VDRL) became negative.

Nonvenereal treponematoses differ significantly from venereal syphilis in their mode of transmission, epidemiological characteristics, and clinical appearance, but not in their serological test results and treatment recommendations. The typical clinical manifestations of one of the nonvenereal treponematoses, pinta, however, closely resemble those of syphilis (Table). Treponema carateum, the cause of pinta, is morphologically and antigenically indistinguishable from T pallidum. In fact, despite advanced technical developments in the last decade, no differences between the subtypes of treponema can be detected by Western blot or Southern blot hybridization techniques. Treponema carateum cannot be cultured in vitro but can be transmitted to chimpanzees. Noordhoek et al reported that the T pallidum subspe-

Figure 5. Western blots showing nearly identical bands in serum from our patient with pinta (lane 1) and serum from patients with syphilis II (lane 9), syphilis I (lanes 2-6 and 8), and latent syphilis (lane 10). Lane 7 contains serum from a healthy person without syphilis.
cies *pertenue* and *pallidum* differed by a single nucleotide; these findings, however, could not be replicated by others. It has been suggested that the variations in clinical expression are entirely caused by divergent environmental conditions, such as temperature.

Since 1975, pinta seems to have been eradicated worldwide except in a few scattered areas in the Brazilian rain forest. Yet, any textbook on dermatovenereology still describes this disease, and the medical student has to learn the differential diagnosis.

Our patient fulfills all the criteria of pinta stage I to II, with the typical primary skin lesion on the thoracolumbar area in the form of an annular hyperkeratotic plaque, and secondary papulosquamous lesions on the palms and soles, spirochetes within the lesion, and serological findings indicative of second-stage syphilis. In contrast, in syphilis II, symmetrical lesions are present, and on histological examination, swelling and proliferation of endothelial cells and no melanophages but epithelioid and giant cells are seen. Neutrophilic microabscesses are more often described in yaws. The patient, like her husband, obviously acquired her disease in an area in which pinta was once endemic. Yet, neither the patient nor her husband suffered from the typical signs of stage I syphilis. In such a case the closest diagnosis is pinta. We therefore conclude that if a disease such as pinta still does exist in Cuba, our patient suffered from that disease and did not import it to Europe. If, however, the disease is extinct—except perhaps for a few scattered foci in the South American rainforest—why do we still burden our medical knowledge with such an outdated historical term?

Nonvenereal treponematoses constituted a major health problem for many underdeveloped countries before mass treatment programs were initiated in the 1950s. No further cases of pinta have been reported to the World Health Organization from Mexico or Colombia since 1979. The Centers for Disease Control and Prevention in Atlanta, GA, and the Pan American Health Organization in Washington, DC, have received no reports of pinta from Cuba in the past 20 years (G. P. Schmid, MD, MSc, Centers for Disease Control and Prevention, Atlanta, GA; and S. Talhari, MD, Department of Dermatology, Institute of Tropical Medicine, Manaus, Brazil).

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