therapy's long-term risk. We would further suggest that such follow-up must include all members of a defined cohort. I commend Dr. Leone's efforts and would encourage him and other dermatologists to follow all patients treated with PUVA carefully.

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Idiopathic leukonychia totalis and partialis

To the Editor:

Leukonychia has been classified into four separate categories: (1) leukonychia striata, (2) leukonychia punctata, (3) leukonychia partialis, and (4) leukonychia totalis. Leukonychia totalis, in which the nail is totally white, and leukonychia partialis, in which the nail is mostly white but maintains distal transverse bands of normal pink color, have traditionally been considered as independent entities. We report a patient with idiopathic leukonychia, who demonstrated a progression of his leukonychia from partialis alone to combined partialis and totalis. The spectrum and evolution of nail findings in this patient lend support to Butterworth's assertion that leukonychia partialis represents a phase of leukonychia totalis, and as such is not a separate entity.

Case report. The patient was a 23-year-old man, first seen at UCSD dermatology clinic in 1981, with a history of white fingernails dating back to 1975. The disorder began with central white spots on the fingernails that progressed over 1 year's time to almost total nail discoloration. The growth of the nails was otherwise normal. He was in excellent health, denied any past medical or dermatologic problems, and denied exposure to any systemic or topical medications or chemicals. There was no history of mechanical trauma to the nails, nor was there a family history of leukonychia or other dermatologic disorders.

On the initial physical examination in 1981, both thumbnails had white discoloration over the proximal half, while the distal half was normal pink in color with medial and lateral white areas (Fig. 1). The other fingernails were predominantly white with distal transverse bands of pink nail color. There were distal thin transverse white bands on the second toenails bilaterally; all the other toenails were normal. No ridging, pitting, or splitting of the nails was evident, and there was no subungual hyperkeratosis. The periungual area was normal.

The rest of the physical examination was unremarkable.

Laboratory studies, including complete blood count with differential, serum electrolytes, creatinine, calcium, magnesium, aspartate transaminase, lactate dehydrogenase, alkaline phosphatase, total protein, albumin, and globulins were all within normal limits. Repeated potassium hydroxide preparations and fungal cultures of the nails were negative.

Since being followed in our clinic, the patient has remained in excellent health but has shown a progression in his nail discoloration. In June 1984, 3 years after his first visit, both thumbnails and the right first fingernail were completely white. In addition, superimposed opaque yellow-white discoloration was noted on the distal aspect of both thumbnails (Fig. 2). The remaining fingernails and toenails were not significantly changed from the initial examination.

Comment. Our patient presented initially with leuk-
Correspondence

onychia partialis that evolved over 3 years into leukonychia totalis involving three fingernails, combined with leukonychia partialis of the other fingernails. To the best of our knowledge, this represents only the third reported case of leukonychia partialis and totalis observed in a single patient and is the only one without a hereditary etiology. Harrington\(^3\) described a patient with both leukonychia totalis and partialis of the fingernails, and Butterworth\(^2\) reported a patient with leukonychia totalis and partialis at different times. However, the leukonychia in both of these patients was hereditary. Histologically, leukonychia is associated with large nucleated cells and keratohyaline granules, suggesting that abnormal keratinization is responsible for the appearance of this condition.\(^1,2\)

Almost all the reported cases of leukonychia partialis or totalis are either familial or associated with underlying systemic disease such as typhoid fever, hepatic cirrhosis, ulcerative colitis, and leprosy.\(^1,4\) Some cases have been associated with exposure to certain agents such as emetine, or local exposure to salty solutions.\(^1,5\) Our patient is unusual in that we have not been able to implicate any of these factors in association with his leukonychia.

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REFERENCES

Reply
To the Editor:
The term leukonychia has various definitions to different physicians. I evaluate a patient with leukonychia as follows:

1. Is it congenital or acquired?
2. Is the color change caused by an aberration mainly of the nail plate (parakeratosis) or elsewhere?
3. Is it endogenously induced or idiopathic (more often multiple nails) or due to exogenous or nonsystemic factors (more often fewer nails)?
4. What is the pattern (partial, total, location, number of nails, etc.)?

I see acquired, exogenously induced leukonychia partialis of the nail plate most frequently. This is usually caused by trauma to the matrix. Acquired, endogenously produced leukonychia partialis (nail plate normal) is less common; Muehrcke’s lines is an example of this.

Acquired, idiopathic, leukonychia totalis of the nail plate is much less common. This obviously must begin at some point as the entire nail plate does not instantaneously change de novo. I cannot explain why the concomitant partial and total forms of this disorder have not been reported more frequently.

In this day of trying to simplify dermatologic terms and make them more logical, one does not like to suggest the use of modifiers when describing a disorder. But in the case of leukonychia, I feel that it is helpful. This letter just touches on the subject of leukonychia and further reading is recommended.\(^1,3\)

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REFERENCES

Peeling skin syndrome
To the Editor:
Peeling skin is a rare condition, although it has been reported under such names as keratolysis or skin shedding,\(^1\) skin shedding (keratolysis exfoliativa congenita),\(^2\) unusual congenital ichthyosiform erythroderma,\(^3\) deciduous skin,\(^4\) familial continual skin peeling,\(^5,6,8\) idiopathic deciduous skin,\(^7\) and peeling skin syndrome.\(^8\)

The patient described by Stone\(^1\) had episodic shedding in the summer only, and the episodes were associated with malaise, chills, and fever. That type of shedding probably is related to infection and is not the peeling skin syndrome. The patients reported by Fox,\(^2\) Kurban and Azar,\(^3\) and Abdel-Hafez et al\(^8\) appear to be similar in that they had peeling skin at an early age, the peeling continued and did not show seasonal variation, and no inflammation or pruritus was present.