keratin. Contours of a rolled hair were visible within the keratin mass. At points of exposure, the rolled hair showed a normal cuticle (Fig. 3).

DISCUSSION
Although rolled hair was already mentioned as early as 1894 by Unna (5), it has not received much attention in the literature. Ferguson et al. (2) reported 4 patients, of whom 2 patients had received systemic corticosteroid. They concluded that corticosteroids might be involved in the formation of rolled hairs. Adatto (1) reported that rolled hairs are frequently found in men aged 50 and over, without follicular abnormality. This would indicate that rolled hairs ought to be a regular feature in everyday dermatologic practice. Our study, however, shows that in Finnish patients this is a rare phenomenon, since it was not found in any patient in an examination of 100 out-clinic patients.

REFERENCES

Factors influencing the formation of rosacea have been discussed for a long time. They have been the objective of various investigations, but conceivable explanations are based on clinical observations only. A possible genetic disposition is discussed, as well as disorders of the gastrointestinal tract (1).

Another possibility which is discussed and which might be relevant is the role of the sebaceous gland in the aetiology of rosacea. Investigations have shown that there were no significant differences in the quantity or quality of skin surface lipids of rosacea patients compared with those of a control group (2, 8). On the other hand, the clinically often impressive appearance of seborrhoea, and the success of treatment with tetracycline (11) point to an altered function of the sebaceous glands as being one of the various conditions for the onset of rosacea. This is suggested by the results of treatment of rosacea with 13-cis retinoic acid (6, 7, 10), which is known to suppress sebaceous gland functions very effectively.

The dubious role of the sebaceous gland for the pathomechanism of rosacea, as well as the appearance of the disease in middle age when hormonal changes take place in the organism, led to the conclusion that hormonal influences might contribute to the origin of the disease—a conclusion which was based mainly on clinical observations.

The purpose of the present investigation was to study estrogen and androgen receptors in normal skin and in rosacea lesions, since a previous report (9) has shown an influence of sex steroids at the cellular level. In addition, 17β-estradiol and testosterone serum levels were estimated in order to correlate them with receptor contents.

MATERIAL AND METHODS
Skin was obtained from 11 male and 4 female rosacea patients between 40 and 82 years of age. Two 6-mm
biopsies were taken under local anesthesia from skin lesions on the face and from a normal skin region—mainly the hip. Skin slices were frozen in liquid nitrogen immediately after biopsy and stored until receptor assays were performed. Venous blood samples were taken for the assay of hormone serum levels. Determination of receptor contents was performed by saturation analysis (9). Cytosol protein was determined ad modum Lowry (4), and receptor contents are reported as fmol per mg cytosol protein. Determination of hormone serum levels was carried out as described previously (3).

RESULTS AND DISCUSSION

Data displayed in Table I show that no differences in incidence were found between rosacea lesions and normal skin of each patient. The receptor levels were nearly identical for male and female patients. Moreover, estradiol and testosterone serum levels did not differ from normal range values. Furthermore, data shown in Table I do not suggest any endocrine control of rosacea lesions. However, possible hormone influences cannot be ruled out completely on grounds of the present data. It may well be that hormones other than estrogen and testosterone are involved. This notion is suggested by the observation of rosacea in menopausal females.

The data in Table I do not suggest endocrine control of rosacea lesions. Flush symptoms which precede stadium erythematosum and subsequently lead to the initial teleangiectasies might not only be interpreted as a consequence of oral thermoflexes (12) but probably indicate a possible hormonal involvement in the disease on a probably much more differentiated and complex level, which is not measurable by our present methods. A possible level of hormone action might be in the small vessels. Electron microscopic investigations of teleangiectasies in Morbus Weber-Rendu-Osler showed remarkable alterations of the intima of the vessels during estrogen and progestagen therapy (5). Similar mechanisms could become active during the development of rosacea. Further investigations are necessary to clarify this point.

REFERENCES

Epithelial Cell Proliferation of Oral Lichen Planus in Patients Treated with an Aromatic Retinoid

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Abstract. In 4 patients with lichen planus mucosae (l.p.m.) punch biopsies were taken from the involved and uninvolved buccal sides before treatment and from the affected side after a 3-week systemic therapy with aromatic retinoid (Ro 10-9359/50 mg/die). After biopsy, the specimens were incubated in Minimum Essential Medium (MEM, Flow Lab.) containing tritiated thymidine (spec. act. 5 μCi/μl) for 60 minutes. Subsequently serial section histo-autoradiography (5 μm cutting width, stripping film Kodak AR 10) was performed. Evaluation of the histo-autoradiographs was performed by copying each serial section using a microprojector. Further details have been published elsewhere (6, 8).

The following data were determined:
1. Number of labelled cells/100 basal cells (L_{basal})
2. Number of labelled suprabasal cells/100 basal cells (L_{suprabasal})

With the development of oral retinoids and their successful application to many dermatoses showing aberrant proliferation (2, 3, 4, 13), a new era of dermatologic therapy began which also proved beneficial in erosive lichen planus mucosae (l.p.m.) (1, 7, 11). Due to the improved clinical course, a normalization of the disturbed proliferation and keratinization has been attributed to this therapy in l.p.m. To our knowledge, however, studies concerning epithelial cell proliferation in patients receiving this medication are lacking so far. Hence this study was aimed to determine, before and during therapy with an aromatic retinoid, some cell kinetic parameters in patients with l.p.m.

MATERIAL AND METHODS

Punch biopsies from the buccal mucosa of 4 patients (1 male, 3 female, age 37-64 years) with histologically confirmed l.p.m. were investigated. From each patient, following informed consent, three specimens (4 mm Ø, local anaesthesia: 1% solution of Scandicain without vasoconstrictor) were taken. Before treatment, one biopsy from the involved and the uninvolved (contralateral) buccal mucosa were gathered simultaneously at morning time. After a 3-week therapy with the aromatic retinoid (Ro 10-9359/50 mg/die) another tissue sample was taken from the originally affected buccal mucosa.

Immediately after biopsy, the specimens were incubated in Minimum Essential Medium (MEM, Flow Lab.) containing tritiated thymidine (spec. act. 5 μCi/μl) at 37°C and 215.8 kPa (=2.2 bar). O2-partial pressure for 60 minutes. Subsequently serial section histo-autoradiography (5 μm cutting width, stripping film Kodak AR 10, exposure time 14 days at 4°C) was performed. Evaluation of the histo-autoradiographs was performed by copying each serial section using a microprojector. Further details have been published elsewhere (6, 8).

The following data were determined:
1. Number of labelled cells/100 basal cells (H-Index, L_{basal})
2. Number of labelled basal cells/100 basal cells (L_{basal})
3. Number of labelled suprabasal cells/100 basal cells (L_{suprabasal})

Abbreviations used
l.p.m.: lichen planus mucosae
L_{basal}: number of labelled cells/100 basal cells
L_{suprabasal}: number of labelled suprabasal cells/100 basal cells
L_{basal}/L_{suprabasal}: mean quotient of individual values of L_{basal} and L_{suprabasal}

Key words: Histo-autoradiography; Lichen planus mucosae; Aromatic retinoid

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