Table I. *Ranked prevalence of skin disorders in 584 elderly institutionalized persons*

	Number of conditions[a]	Prevalence (%)
Pityriasis of the scalp	259	44.3
Asteatosis (including localized and widespread eczema craquelé)	169	28.9
Seborrhoeic dermatitis (face, scalp, chest)	41	7.0
Stasis dermatitis	40	6.9
Xanthelasma palpebrarum	27	4.6
Contact dermatitis	22	3.8
Psoriasis vulgaris	17	2.9
Pressure sores	13	2.2
Leg ulceration	9	1.5
Vitiligo	7	1.2
Rosacea cum acne	1	0.2

[a] 605 skin conditions were present in 452 patients, i.e. several patients had more than one skin disorder.

of senile keratoses, eczemas and seborrhoeic keratoses. The differences in frequency and rank of dermatoses in the two selected series and in the present population study is obviously due to selection of the patients who seek dermatological aid. Elderly patients are not frequent visitors to dermatological clinics. Epstein (2) found that patients over 70 years of age comprised only 8% of his private patient group.

The observed 2.9% prevalence of psoriasis in old-age is close to 2.84% in the normal population of the Faroe Islands, reported by Lomholt (6).

Vitiligo was present in 1.2% of the old-age population group, which is close to 1.4% prevalence reported among patients admitted to a Danish county hospital (3). Age-specific prevalence of vitiligo was studied on the Isle of Bornholm, Denmark, by Howitz et al. (5). They found a prevalence of 0.9% in the ages of 60 to 70 and 0.6% after the age of 70. These figures are inexplicably lower than observed in the present investigation.

Clinical evidence of contact dermatitis was found in 3.8% of the old people, which is within the range of prevalence in normal populations reported from other parts of Scandinavia (1.5 to 4.8%) (4).

In Droller's study (1) 25% of the old people living in their homes had leg ulceration. The low prevalence of leg ulcers (1.5%) and of decubitus (2.2%) reported here must be viewed in the light of the careful nursing and the good nutritional status of the group as a whole.

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Dermatitis Herpetiformis and Gluten-Sensitive Enteropathy in Monozygotic Twins

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Abstract. Of two monozygotic twins, one had dermatitis herpetiformis, while the other had gluten-sensitive enteropathy. The literature on twins is reviewed.

Key words: Dermatitis herpetiformis; Gluten-sensitive enteropathy; Twins

The high familial incidence of gluten-sensitive enteropathy (GSE) is well known (7). It has been stated that the pathogenesis of GSE is multifactorial, the genetic basis of susceptibility being

polygenic and interacting with environmental factors (4, 7, 12). However, David et al. (1) concluded in their study that the pattern of inheritance was compatible with an incompletely penetrant autosomal dominant gene. Townley (11) suggested autosomal recessive inheritance because of new evidence of lack of an intestinal enzyme, a peptidase.

Several twins with GSE have been reported. In 1956, Ebbs summarized the data on twins in the literature, including his own cases comprising 11 pairs of twins (2). Five pairs were identical and both twins had GSE. Of six pairs of non-identical twins three pairs exhibited concordance and three pairs were discordant for GSE. The non-identical twins, both having GSE, reported by Evensen, were not included (7).

Most of these case reports, however, antedated current diagnostic criteria for zygosity. Carter et al. (7) reported a pair of monozygotic twins, based on blood group typing, and a pair of dizygotic twins both discordant for GSE. More recently, MacDonald published a report of monozygotic twins, both having GSE (7). Hoffman et al. (3) reported on discordance in a monozygotic twin pair. McCrae (7) mentioned identical twins, of whom only one had GSE. Walker-Smith (12) reported on discordance in both monozygotic and dizygotic twin pairs. Shipman et al. (10) reported on dizygotic twins with one twin affected. Two pairs of monozygotic twins both concordant for GSE have recently been reported by David et al. and Penna et al. (1, 8).

Dermatitis herpetiformis (DH) is often associated with GSE. The pathology of GSE occurring with DH and that in GSE without DH is essentially the same, although the lesions in GSE without DH are usually much more severe (4). The familial occurrence of DH seems to be rather rare. Few familial cases have been reported (6, 9, 13). Marks et al. (6) reported on one pair of monozygotic twins, both of whom had DH. In their report, they mentioned other family cases of DH in which first-degree relatives were affected. The occurrence of DH and GSE in one and the same family also seems to be rare (9, 13). In the above-mentioned family studies on GSE, no family members were mentioned as having DH. However, many relatives of patients with DH have intestinal mucosal abnormalities without subjective symptoms (5). In this report, a pair of monozygotic twins, one having GSE and one with DH, are described.

CASE REPORTS

The patients are two male twins, born in 1943. Twin A was 33 years old, when in 1976 he was admitted because of 2 years of progressive weakness of arms and legs. Myelopathy with isolated anterior horn cell (paresis) and autonomous (localized anhidrosis) nerve affection was found. The patient reported a 10-year history of diarrhoea, and X-ray examination revealed flattened mucosa in the proximal jejunum. A biopsy showed maximum villous atrophy. He had steatorrhoea and macrocytic anemia with normal vitamin B_{12} in serum and extremely low erythrocytic folic acid. Calcium and albumin in serum were subnormal. The absorption of glucose and *d*-xylose was depressed. A diagnosis of GSE was made and he was given gluten-free diet. The diarrhoea disappeared and the above-mentioned parameters were normalized. A repeated jejunal biopsy showed less villous atrophy. Clinically he had no dermatitis herpetiformis and immunofluorescence of the skin was negative. The myelopathy of unknown etiology, however, was still progressing.

Twin B was 28 years old, when he was submitted to hospital in 1971 with an itching papulovesicular skin eruption symmetrically located, mainly on the face, neck, extensor surfaces, and axillae. A clinical diagnosis of dermatitis herpetiformis was made.

Histopathological examination of clinically involved skin showed a subepidermal bulla, and immunofluorescence of clinically uninvolved skin revealed granular deposits of IgA and complement C 3 in the dermal-epidermal junction, especially in the tops of the papillae. A patch test with potassium iodide was positive.

He had no intestinal symptoms. Biopsies from jejunum were normal. Laboratory tests for malabsorption showed normal values including concentrations of hemoglobin, folic acid, vitamin B_{12} in serum as well as the lactose- and *D*-xylose tests.

Table 1. *Coeliac disease in twins*

C = concordance, D = discordance

Author (year)	Monozygotic C	Monozygotic D	Dizygotic C	Dizygotic D
Evensen (1936)			1	
Ebbs (1956)	5		3	3
Carter et al. (1959)		1		1
MacDonald et al. (1965)	1			
Hoffman et al. (1966)		1		
McCrae (1969)		1		
Walker-Smith (1973)		1		1
Mylotte et al. (1974)				2
David et al. (1975)	1			
Shipman et al. (1975)				1
Penna et al. (1979)	1			
	8	4	4	8
Total	12		12	
% concordant	66.6		33.3	

He was treated with dapsone 200 mg daily and the skin cleared.

A blood sample from each twin was examined at the Institute of Forensic Medicine in Copenhagen, and the HLA type for both was HLA-$A_1$9; B8; Dw3, which confirmed that they were monozygotic.

COMMENT

The monozygotic twins described here with GSE and DH, respectively, were HLA-B8 and Dw3, in agreement with previous studies. There is an increased frequency of B8 in GSE (80–90%) and in DH (80%) as compared with only 20–30% in normal controls (4). Recently, HLA-Dw3 has been shown to occur in GSE and DH in a higher frequency than HLA-B8 (4).

Twin cases tabulated to date demonstrate 67% concordance in ostensibly monozygotic twins and 33% in dizygotic twins (Table 1). This demonstrates that the theory that GSE is due to a single gene is untenable.

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Some Unusual Findings in Porokeratosis Mibelli

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Abstract. Light microscopy in a case of unilateral porokeratosis Mibelli revealed a cyst-like dilatation in the upper corium containing two cornoid lamella complexes. Among other findings, elastic globes as part of thickening and fragmentation of elastica, which in this case may be related to microangiopathia deserve to be mentioned.

The cornoid lamella in porokeratosis Mibelli has always been found to be located in funnel-like epidermal invaginations both in connection with epidermal adnexa and in regions free of adnexa. The parakeratotic nature of cornoid-lamella cells has been suggested on the basis of histochemical and ultramicroscopical findings (2, 6). This is consistent with a deficient granular layer, perinuclear vacuolation and nuclear pyknosis in the epidermis below the cornoid lamella. The occurrence of the cornoid lamella in places where no relations to adnexa could be found, was the reason for the term of "parakeratosis centrifuga atrophicans" suggested by Miescher (7) and basically accepted by Braun-Falco & Balsa (2).

A focal disturbance afflicting a clone of epidermal cells resulting in aberrant keratinization was an alternative suggestion to the theory of adnexal origin (4, 8).

The reason for the present publication is the unique detection of two cornoid lamella complexes in a cystic dilatation of an intradermally located hair follicle.