# Fingerprint Changes in Coeliac Disease

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**Summary: Study of the fingerprints of 73 patients with coeliac disease, taken carefully, showed changes varying between moderate epidermal ridge atrophy and actual loss of fingerprint patterns. Of the patients 63 had these abnormalities, compared with 3 out of 485 controls. A high degree of correlation existed between ridge atrophy and changes in the clinical state of patients with coeliac disease.**

## Introduction

It is well appreciated that certain diseases affect the condition and clarity of fingerprints. These include eczema, leprosy, hypohidrotic ectodermal dysplasia, and adult acanthosis nigricans (Verbov, 1970), scleroderma (Chatterjee, 1967), and Darier's disease and skin lesions due to radiation (Cherrill, 1950). Extremes of age also affect the clarity of fingerprints. During a survey of hospital inpatients unexpected abnormalities were detected in patients with coeliac disease.

## Methods and Subjects

Rolled and plain impressions of fingerprints were taken as directed by the Home Office (1960), after full explanation had been made to the patients. Ether was used in every case to remove perspiration, which can otherwise interfere with obtaining good clear prints. Fingerprints of infants were obtained by lifting latent impressions from glazed white tiles as described by the Federal Bureau of Investigation (1963), using "Bristol black" powder and standard lifting materials.§ Fingerprints were retaken on each visit to the outpatient department. One to eight sets of fingerprints were obtained from each patient over a period of two years. All prints were taken by the same person (T.J.D.).

*Patients with Coeliac Disease.*—Every patient had been fully investigated in hospital and diagnosed as having coeliac disease. All of them had had a jejunal biopsy which showed subtotal villous atrophy. The average age of the 73 patients (25 males and 48 females) studied was 35 years; 12 were under 15 years of age.

*Controls.*—These consisted of 275 healthy women (hospital medical staff), 45 healthy men (hospital medical staff), 35 male inpatients, 85 female inpatients, 5 hospital domestic staff (not using gloves for washing-up), and 40 patients who had lost weight (those who had lost 1 to 8 st. (6·4 to 51 kg.) due to illness).

## Results

Of the 73 subjects, 63 had abnormal fingerprints—21 men (10 on gluten-free diet) and 42 women (31 on gluten-free diet). Ten patients had normal prints—four males (three on gluten-free diet) and six females (five on gluten-free diet)—seven were children. The abnormalities found were as follows: (1) ridge atrophy, with the appearance of white lines (see below), and (2) further ridge atrophy, with loss of visible ridges and disappearance of white lines.

In five patients their fingerprints improved when they were treated with a gluten-free diet and in two there was no improvement when treated with a gluten-free diet, one of these having had no clinical improvement either. In two other patients who relaxed their gluten-free diet their fingerprints deteriorated (in the reverse of the sequence above); this corresponded with a clinical deterioration. The fingerprints of eight patients deteriorated when they relapsed while still on a gluten-free diet; in one case these changes occurred 10 days before the patient suddenly deteriorated clinically and required emergency admission to hospital. Five patients already on a gluten-free diet showed improvement of their fingerprints when their clinical condition improved, and in two of these only after corticosteroids had been given.

Only three controls had the same changes as those found in the patients with coeliac disease. Of these, two had primary biliary cirrhosis and one chronic active hepatitis. Other patients with these two diseases had normal fingerprints. All the domestic staff had abnormal prints, but the changes were different from those in patients with coeliac disease, and consisted of irregular breaks and cracks in the ridges with white lines but no ridge atrophy.

## Discussion

The changes in coeliac disease (Figs. 1 to 3) are distinguishable from:

(*a*) *Eczema* (Fig. 4): Some fingers may be normal and others affected. In one fingerprint some parts of the pattern may be normal and other parts completely obscured. The patchiness of the changes in eczema makes it easily differentiated from ridge atrophy, though in a very bad case the fingerprint patterns may be completely obscured, in which case the disease should be clinically obvious.

(*b*) *Trauma:* Usually localized to one or two digits, and is commonest on the left forefinger (Cherrill, 1954, p. 98). Burns and cuts leave quite characteristic scars, as do certain occupations such as carpentry and tailoring (Galton, 1965).

(*c*) *Skin grafts:* Individual digits affected only.

(*d*) *Severe mental subnormality:* As a cause of occasional pronounced ridge atrophy should be self-evident.

(*e*) *Dotted ridges* (Fig. 5): Found in Darier's disease; extremely rare in normal people.

(*f*) *Warts:* Easily visible to the naked eye.

(*g*) *Ridge dissociation* (Fig. 6): Also wrongly called dysplasia. Excessively rare and quite distinctive. Thumb most affected of all fingers in ridge dissociation, whereas little finger worst affected in patients with coeliac disease.

(*h*) *Early infancy, old age:* White lines are normally present. In old age the skin becomes dry and the ridges appear less distinct.

(*i*) *Denervated hand:* Pronounced ridge atrophy may occur (Cummins, 1967); indistinguishable from changes in coeliac disease.

(*j*) *Sweating:* This obscures inked prints and may smudge latent prints.

(*k*) *Ridge aplasia:* Complete absence of fingerprints. Very rare, and found mainly in Japan and the United States of America; inherited as a Mendelian dominant (Cummins, 1970).

In this preliminary survey 86% of 73 patients with coeliac disease had ridge atrophy of their fingerprints. If the adults are taken alone then 95% have ridge atrophy. The number of children studied is insufficient to ascertain whether untreated coeliac disease affects their fingerprints, since 10 of the 12

§ Obtainable from the Institute of Applied Science, 1920. Sunnyside Avenue, Chicago, Illinois 60640, United States of America.

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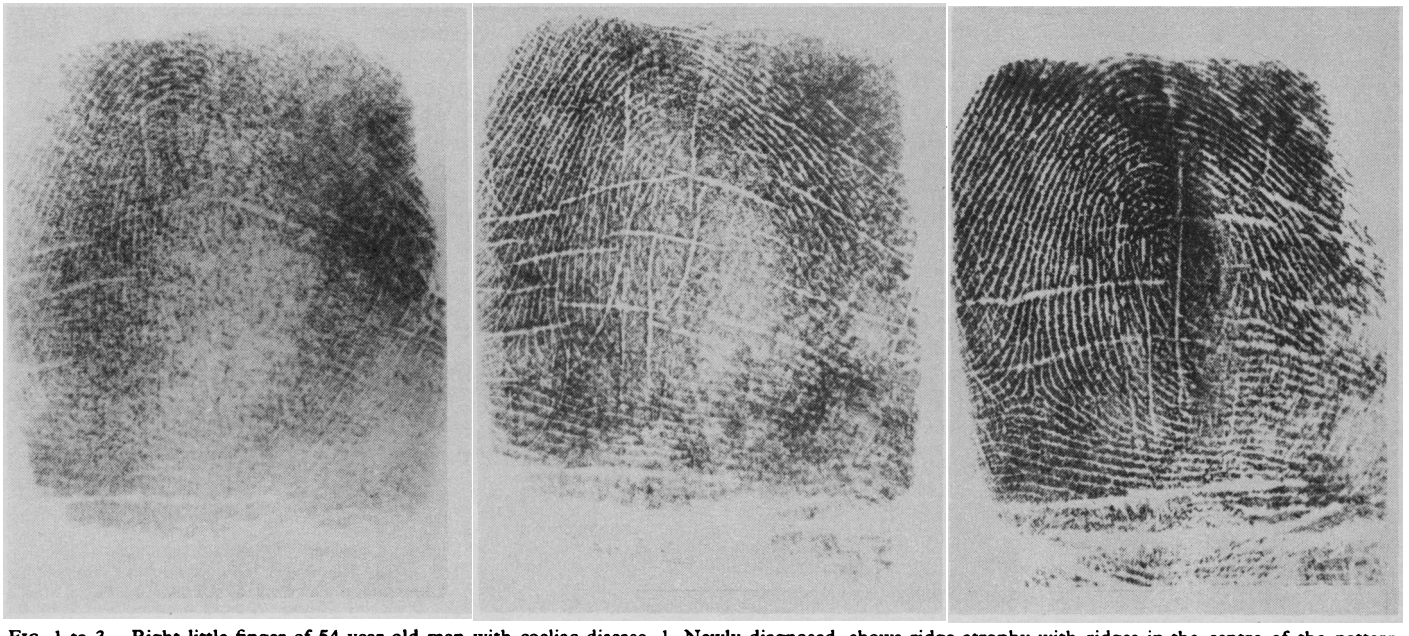

FIG. 1 to 3.—Right little finger of 54-year-old man with coeliac disease. 1, Newly diagnosed, shows ridge atrophy with ridges in the centre of the pattern not discernible. 2, After one month's treatment with a gluten-free diet. Shows partial ridge atrophy with appearance of white lines. 3, After 11 months on a gluten-free diet. Ridges almost completely regrown, with almost complete disappearance of white lines. Pattern can now be classified as a whorl with an outer ridge tracing.

FIG. 4.—Right ring-finger of 6-year-old girl with eczema. Lower part of fingerprint worse affected than upper part, where the ridges are clearer.
FIG. 5.—Right thumb of 20-year-old girl. Ridges are regularly broken up into dots.
FIG. 6.—Right thumb of 43-year-old man. Pronounced ridge dissociation.

children studied were already on a gluten-free diet and clinically well. The two untreated children were infants of one year old, in whom the disease had been recently diagnosed, and both had abnormal prints. There are not enough controls in this age group to assess the significance of this. One girl aged 11 informed us that when playing "fingerprints" at school two years ago she was the only member of her class who could not make nice clear prints. Only 3 out of 485 controls had the same changes as the patients with coeliac disease.

To rule out weight loss as a cause of ridge atrophy, we took fingerprints from 40 patients with wasting diseases (carcinomatosis, leukaemia, steatorrhoea not due to coeliac disease, Crohn's disease, ulcerative colitis, thyrotoxicosis, cirrhosis, and protein-losing enteropathy). Even a patient who had lost 8 st. (51 kg.) in weight had normal prints recorded at necropsy. Patients with iron-deficiency anaemia, folate-deficiency anaemia, and pernicious anaemia all had normal fingerprints.

The improvement in fingerprints after treatment with a gluten-free diet has been started appears rapidly and is easily detected within a month. Ridge atrophy seemed to precede clinical deterioration by some weeks in the few patients whose fingerprints were taken frequently enough to show such changes. We cannot explain, however, the finding of normal fingerprints in three adults with coeliac disease.

Galton (1965) suggested that when the skin becomes thin the ridges simultaneously subside in height. Though this might account for ridge atrophy in coeliac disease it does not explain why patients with wasting diseases and thin skin have

normal fingerprints. Cummins and Midlo (1961) suggested that white lines are due to buckling of the skin. An examination of the fingers with white lines in coeliac patients does not confirm this, though we have observed two patients with Marfan's syndrome where buckling of the skin undoubtedly did lead to white line formation. Verbov (1970) has stated that "ridge distortion" may also be seen in dry or atrophic skin "due to many causes." Though some patients with coeliac disease do have dry skin this is by no means always the case. From our findings we think that the white lines in patients with coeliac disease are merely secondary to ridge atrophy, and this is supported by the finding that white lines disappear when ridge atrophy is almost complete and reappear when the ridges begin to grow again. Possibly ridge atrophy is a manifestation of abnormal cell turnover in the skin, paralleled by similar changes of the cell turnover in the small bowel (Croft *et al.*, 1968).

Fingerprinting of patients with coeliac disease may have two possible uses, subject to further long-term studies being made at present: (1) As a diagnostic feature in new cases of adult coeliac disease, where it is apparently the only wasting disease where ridge atrophy is seen; (2) as a measure of a full response to a gluten-free diet. If fingerprint changes correlate closely with villous atrophy in the gut, then fingerprinting might also spare patients a repeat small intestinal biopsy. The changes in coeliac disease need to be carefully distinguished from other ridge abnormalities, but this has in practice been found to be fairly simple, provided that the prints are taken carefully by an experienced operator with the correct materials. It would be quite impossible to detect subtle ridge changes with inkless methods (Cherrill, 1954, p. 151), which are barely adequate for pattern classification let alone for a study of fingerprint minutiae. The type of paper used is also important, as if the ink is excessively absorbed then the ridges will not be clear enough for close study.

Further long-term studies are being made with quantitative methods of assessing ridge changes in coeliac disease and correlating these with changes in the small-bowel villi and other clinical criteria.

We are grateful to the many doctors who permitted us to study patients under their care, and to the patients themselves, who were most helpful.

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# Fibrosing Alveolitis Associated with Renal Tubular Acidosis

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**Summary: The discovery of a case of renal tubular acidosis and fibrosing alveolitis led to the investigation of 19 further patients. Abnormal pulmonary function tests were found in a further four patients with overt renal tubular acidosis and in four out of eight patients with "incomplete" renal tubular acidosis. The response to an ammonium chloride test in seven patients with cryptogenic fibrosing alveolitis was normal. Those patients with a defect of both renal acidification and pulmonary gas transfer had concurrent autoimmune diseases such as Sjögren's syndrome and primary biliary cirrhosis. It is suggested that the renal and pulmonary abnormalities may be part of a systemic disorder capable of affecting many organs. Moreover, hyperglobulinaemia and autoantibodies in these patients further suggests that immunological mechanisms are concerned in the pathogenesis of these abnormalities.**

## Introduction

Fibrosing alveolitis may be associated with hyperglobulinaemia (Hobbs and Turner-Warwick, 1967) and the presence of autoantibodies (Turner-Warwick and Doniach, 1965). Possibly autoimmune processes are concerned in the pathogenesis of this disorder (Mackay and Ritchie, 1965), and the concurrence of fibrosing alveolitis with other autoimmune diseases such as rheumatoid arthritis, Sjögren's syndrome, Hashimoto's thyroiditis, and active chronic hepatitis has been reported (Turner-Warwick, 1968; Scadding, 1969).

Renal tubular acidosis similarly may be associated with hyperglobulinaemia (Morris and Fudenberg, 1967) and autoantibodies (Talal *et al.*, 1968). Diseases recorded as occurring with renal tubular acidosis include Sjögren's syndrome (Shearn and Tu, 1965), Hashimoto's thyroiditis (Clinico-Pathological Conference, 1968), active chronic hepatitis (Read *et al.*, 1963), and hyperglobulinaemic purpura (Greenspan, 1949). The coexistence of all of these conditions with renal tubular acidosis has recently been reviewed (Mason and Golding, 1970).

In this paper the clinical and immunological investigation of one patient with renal tubular acidosis and fibrosing alveolitis is reported in detail. Because of this previously undescribed association, a further four patients with overt renal tubular acidosis and eight with "incomplete" renal tubular acidosis had their lung function and chest radiographs studied. The response to an acid load was measured in seven patients with cryptogenic fibrosing alveolitis. Immunological studies and investigations for the presence of other autoimmune diseases were performed in all patients.

## Patients and Methods

The patients selected for investigation were divided into three groups. Group A consisted of five patients with overt renal tubular acidosis—one of these (Case 1) is described in

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