Family studies of coeliac disease in Cuba

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SUMMARY Peroral jejunal biopsies were performed on 69 of 85 1st-degree relatives of 20 index coeliac patients in 20 families. Three had mucosal changes typical of coeliac disease, giving an incidence of 4.3%. This figure is similar to that for family studies in England, and it confirms the familial nature of coeliac disease in a population with different racial characteristics from countries where coeliac disease is common.

The familial nature of coeliac disease is well known.¹ MacDonald *et al.*² were the first to make a family study of coeliac disease using intestinal biopsy as a criterion for diagnosis.³ Family studies of coeliac disease have been done in Ireland,⁴ England,⁵ and elsewhere, but not previously in Latin America.

Material and methods

Twenty families of coeliac patients, chosen at random, were invited to co-operate in the study. The diagnosis of coeliac disease in each patient was made following these criteria: (1) a suggestive clinical history, (2) evidence of intestinal malabsorption, (3) total or severe villous atrophy with hypertrophic crypts and increased cellularity of the lamina propria on light microscopical examination of an intestinal biopsy, (4) clinical and morphological response to a gluten-free diet. Coeliac disease was confirmed in 7 patients by gluten challenge.

If the family members wished to co-operate a jejunal biopsy was carried out under fluoroscopic control, taking the specimen at the ligament of Treitz. The biopsies were processed for routine histology and examined by light microscopy. No other test for malabsorption was done.

A total of 74 relatives from the 20 families were studied: 20 mothers, 14 fathers, and 40 siblings. Six fathers and 5 siblings were not studied, either because they did not wish to take part or because they were away.

Results

Forty of the 74 relatives were male (Table 1); there were 50 white, 15 black, and 9 mestizos (Table 2).

In 5 of the 74 relatives the biopsies were technically unsatisfactory. Of the remaining 69, 24 had

Table 1 *Sex distribution of the relatives studied*

	Male		Female	
	No	%	No	%
Parents	14	42	20	50
Siblings	26	65	14	35
Total	40	54	35	46

Table 2 *Racial distribution of the relatives studied*

Race	Relatives	
	No	%
White	50	68
Black	15	20
Mestizo	9	12
Total	74	100

minor abnormalities, 7 of them with evidence of *Giardia lamblia* in faeces or duodenal juice.

Only 3 relatives (all siblings and 2 from one family) had mucosal changes typical of coeliac disease (flat mucosa). No coeliac was found among the 34 parents. This gives an overall incidence of 4.3% of 69 1st-degree relatives. Two of the 20 families thus had another coeliac member, giving an overall incidence of 10% of 20 families. In one of these two families, the proband had diabetes mellitus too, a disease recorded as coexisting with coeliac disease.⁶ This patient had a brother with coeliac disease; another brother was schizophrenic and did not wish to co-operate; it has been suggested that schizophrenia has a relationship with dietary wheat gluten.⁷ The other family had, besides the proband, 2 sisters who also suffered from coeliac disease. The father and the other 3 brothers did not wish to co-operate.

The members of one family were black immigrants from the Caribbean island of Grenada.

Discussion

The reported incidence of coeliac disease among 1st-degree relatives of coeliac patients varies widely between 0.4 and 16.2%. Our figure of 4.3% is close to that given by Rolles *et al.*³ in England. In both studies, all the patients were children.

There was one family with 3 coeliacs with a further 3 members who were not biopsied.

Our results confirm the familial nature of coeliac disease in a population with racial characteristics different from such countries as the UK, Australasia, and USA where coeliac disease is fairly common. Although coeliac disease may be more common in people of Caucasian origin, it must be accepted that it does occur in Indians and negroes when gluten is present in their diet.

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