no longer than three months in the first instance. Evaluation of relapse may, but does not necessarily have to, be confirmed histologically; such confirmation may not be needed when clinical and laboratory evidence (including a rise in antigliadin antibodies titre) is clear.

These considerations and recommendations are reported as a matter for further discussion and in the hope that they will stimulate comments and criticism from all paediatricians who take part in investigation or clinical management, or both, of young patients with coeliac disease.

The data summarised here were collected at the following centres: Clinica Pediatrica dell’Università, Ancona; Istituto di Pediatría Clinica e Sociale, Università di Bari; Divisione di Pediatrica, Ospedale Maggiore ‘GA Pizzardi’ Bologna; Istituto Clinico e Biologica Età’ Evolutiva dell’Università Cagliari; Clinica Pediatrica dell’Università di Cattania; Clinica Pediatrica Ia dell’Università; Istituto G. Gaslini, Genova; Divisione Pediatrica, Ospedale Generale, Mantova; Clinica Pediatrica IIa dell’Università di Messina; Clinica Pediatrica Ia, Università di Milano; Clinica Pediatrica IIa, Università di Milano; Clinica Pediatrica IIIa, Università di Milano; Clinica Pediatrica IVa—Ospedale ‘L. Sacco’, Milano; Clinica Pediatrica Ia dell’Università di Modena; Divisione Pediatrica, Ospedale G Salesi, Ancona; Divisione Pediatrica, Ospedale degli Esposti, Bologna; Divisione Pediatrica, Ospedale Bufalini, Cesena; Divisione Pediatrica, Ospedale S Michele, Cagliari; Divisione Pediatrica, Ospedale S Carlo, Milano; Dipartimento di Pediatrica dell’Università di Napoli; Divisione Pediatrica, Ospedale SS Annunziata, Napoli; Clinica Pediatrica, Università di Padova; Divisione Pediatrica, Ospedale dei Bambini, ‘G Di Cristina’, Palermo; Clinica Pediatrica dell’Università di Parma; Clinica Pediatrica dell’Università di Pavia; Clinica Pediatrica I, Università ‘La Sapienza’, Roma; Clinica Pediatrica III, Università la Sapienza, Roma; Clinica Pediatrica dell’Università di Torino; Clinica Pediatrica dell’Università di Trieste; Servizio Pediatrico Speciale, Ospedale Borgo Trento, Verona; Divisione Pediatrica, Ospedale infantile Alessandrini, Verona; Clinica Pediatrica dell’Università, Palermo; Clinica Pediatrica IIIa Università di Bologna; Ospedale Bambino Gesù, Roma; and Clinica Pediatrica dell’Università, Verona.

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Commentary

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The ‘cause’ of coeliac disease is not yet known in molecular terms. The diagnosis of coeliac disease remains empirical and is currently based on two well established facts. The first is that gluten causes a malabsorption syndrome with small bowel enteropathy in susceptible subjects. Secondly, withdrawal of gluten from the diet leads to complete restoration to normal of the patient and his intestinal mucosa.

A third important contribution to our thinking about coeliac disease was provided by the (then) European Society for Paediatric Gastroenterology (ESPGA) in its publication of the ‘Interlaken criteria’. In these criteria, coeliac disease was affirmed to be a permanent condition of gluten intolerance, and it was implied that, in order to substantiate an initial diagnosis of coeliac disease, the permanence (or at least persistence) of gluten intolerance should be confirmed in each individual
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patient by carrying out one or more gluten ‘challenges’. Such challenges, stated ESPGA, should lead to a recurrence of the mucosal lesion within two years.

This expanded definition of coeliac disease has been influential and many European centres have used it to the letter. There have been problems, however, both theoretical and practical. Firstly, colleagues in France showed that sensitivity of the mucosa to gluten can vary with age, and Egan-Mitchell et al in Ireland described children in whom the mucosal lesion of coeliac disease did not recur until five or more years after gluten reintroduction. Secondly, and at a practical day to day level, there has been an understandable reluctance by both parents and paediatricians to reintroduce ‘forbidden fruit’ into the diet, both because of the harm it might cause and the habits it might engender. Surreptitiously, corners have been cut.

The Italian Working Group for Paediatric Gastroenterology has rendered a valuable service in the present paper by analysing what has actually been happening in 33 paediatric centres and what the outcomes have been. The authors are able to compare an unprecedentedly large series of 2400 patients diagnosed as coeliac by the full ESPGA(N) criteria, with 738 whose diagnosis was made by a simplified route. Even though allowance must be made for the fact that no attempt was made to standardise observations among centres (for example, the histological reports were accepted at face value by the Group, and different assay systems for antigliadin antibodies were used), the sheer volume of data commands serious attention.

The conclusions and recommendations are eminently sensible. Firstly, if the clinical picture suggests coeliac disease (and cows’ milk intolerance can be excluded) and the jejunal mucosa shows crypt hyperplastic subtotal villous atrophy, and secondly, if there is an obvious clinical response to a gluten free diet, the diagnosis can confidently be assumed to be coeliac disease and a lifelong gluten free diet should be instituted. My only warning to those who wish to adopt this practice is to make sure that the histological lesion is confirmed by a histopathologist who is accustomed to interpreting paediatric intestinal biopsy specimens. The commonest reason seen in our clinic in Birmingham for the erroneous diagnosis of coeliac disease made elsewhere has been overinterpretation of minor histological changes in the mucosa by a histopathologist more accustomed to viewing tissue from adults.

In theory this ‘no gluten challenge, no further biopsies’ approach could mistakenly diagnose as coeliac disease a case of temporary gluten intolerance. Such cases are, however, rare, and becoming rarer, and usually arise in young infants with a short history, and sometimes with evidence of other food sensitivities as well.

Lastly, the authors confirm the observation of others that the rise and fall of antigliadin antibodies in the blood can be useful in the initial diagnosis, in monitoring the response to gluten withdrawal, and in measuring the response to a gluten challenge. There is no evidence in the literature to suggest how, or if, antigliadin antibodies are causally related to the signs and symptoms of coeliac disease, but even if these antibodies prove to be epiphenomena, their serial measurement can give useful diagnostic help in difficult cases.

However the diagnosis of coeliac disease is reached, it is essential to undertake long term follow up by handing on one’s patients with coeliac disease to an adult gastroenterologist at an appropriate age. Dietary compliance over a lifetime is probably not achieved in many cases and the long term morbidity in those who break their diet is still uncertain.

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