Short report

Intellectual ability of adults after lifelong intestinal malabsorption due to coeliac disease

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SUMMARY The intellectual impact of lifelong intestinal malabsorption was examined in a consecutive series of 19 adults (mean age, 48 ± 11 years) with untreated coeliac disease. Using a comprehensive test battery no consistent signs of cognitive impairment were found and in the light of recent observations on coeliacs, aspects of nutritional deficiency may prove more pertinent to other forms of cerebral dysfunction.

Coeliac disease is known as a lifelong malabsorption syndrome originating in the childhood of susceptible individuals. Unless gluten-containing cereals (for example, wheat flour) are withdrawn from the diet, the intestinal lesions produced will persist and account for continuous impairment in the absorption of essential nutrients in the diet. Consequently, a wide range of nutritional deficiencies is typically encountered in neglected coeliacs presenting for the diagnosis in adult life.¹

The full clinical implication of longstanding intestinal malabsorption is yet to be determined. Little is known about the impact on intellectual properties, and in the light of the number of various deficiency states implicated in the pathogenesis of organic brain syndrome,² recent case reports^{3 4} on coeliacs with disabling organic central nervous dysfunction are intriguing. A study was therefore carried out on newly diagnosed adult coeliac patients to see whether intellectual impairment is an accompanying feature of neglected disease.

Patients and Methods

Nineteen consecutive adults, 12 women and 7 men aged 28– 64 years (mean, 48 ± 11 years) were studied over a 15 month period while being investigated for later proven coeliac disease at the Department of Internal Medicine, Regional Hospital, Linköping, Sweden. The diagnostic criteria for

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Received 28 June 1982. Accepted 3 September 1982 adult coeliac disease used have been discussed elsewhere.⁵ All patients showed a flat or nearly flat jejunal mucosa at presentation, and so far 16 of 18 re-biopsied patients have shown definite improvement on the gluten-free diet. Eight patients gave a history of overt childhood coeliac disease, mostly periods of unexplained diarrhoea, and three had been treated in hospital. Steatorrhoea ($69 \pm 44 \text{ mmol/day}$, normal, less than 20 mmol/day) was present in 15 of 18 (83%), as was a low serum folate level.

The psychometric examination was done by means of a test battery⁶ comprising an integral part of that previously used in the assessment of adults with hydrocephalus and brain atrophy.⁷ The following tests were used (figure): The Synonyms Reasoning Block (SRB) test (which includes the Koh's block design test) measuring level of intelligence, Thurnstone's memory test, Reaction time with simple stimulation and 3-choice stimulation, Figure identification, Finger dexterity, Figure rotation, Design, Street, Organic Integrity Test (OIT),⁸ and Benton's visual retention test.⁹

The test procedure was carried out in one session lasting about two hours with appropriate pauses. The results are expressed by scores on a stanine scale, except for OIT and Benton. With OIT, raw scores are given, with the score increasing with form dominance and perception, maximum 100. The more negative score in Benton, the more normal perception. Comparisons were made with results achieved by general population samples,^{6 8 9} and when appropriate, within the coeliac series using the Mann-Whitney U-test for statistical evaluation.

Results

The results of the entire series are presented in the figure. The mean scores on a stanine scale ranged between $4\cdot20-6\cdot44$, which by definition is scored by about half the subjects in a general population sample. Similar results were obtained with the OIT

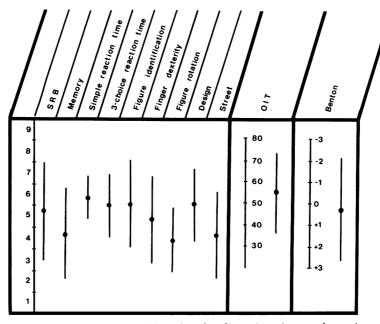


Fig Scores by 19 untreated adults with coeliac disease in various psychometric tests. In SRB – Street, the scores are expressed on a stanine scale. OIT is constructed with a mean score of 66 ± 20.0 (mean \pm SD), and Benton with 0.40 ± 1.14 .

and the Benton test, and the scoring pattern yielded no evidence for consistent signs of intellectual impairment in adult coeliac disease. When examined individually, none of the patients scored in a way indicating definite organic brain disease, whereas in four (21%) the overall pattern was in the borderline range.

Not unexpectedly those coeliacs aged 50 years and above (56 ± 5 years) tended to score generally less than a younger sub-sample of remaining nine patients

TablePsychometric test results by adult coeliac patients(A) over and (B) under the age of 50 years

	Group A (n=10)		Group B (N=9)	
	Mean	SD	Mean	SD
SRB	5.40	2.41	6.11	2.20
Memory	5.10	2.38	4.11	1.83
Simple reaction time	6.56	0.53	6.33	1.41
3-choice reaction time	5.78	1.48	6.38	1.41
Figure identification	5.80	1.99	6.22	2.44
Finger dexterity	5.00	2.07	5.56	2.40
Figure rotation	4.17	0.98	4.22	1.72
Design	6.14	2.54	5.89	1.69
Street	4.57	2.37	4.89	2.09
OIT	56.60	20.71	62.89	20.97
Benton	-0.30	2.50	1.11	1.97

The results are expressed as scores on scales depicted in the figure. No difference between the groups is statistically significant. $(38 \pm 8 \text{ years})$ (table) but the pattern was not consistent and the differences were never significant.

Discussion

Our observations of unaffected intellectual ability of adult coeliacs were made on patients living in an area of Sweden where the disorder has proved to be common, occurring at a rate of at least 1:1,000.⁵ With the patients under study closely resembling others in the area as concerns signs of malabsorption,⁵ our findings may reasonably have reference to coeliacs in general, insomuch as those eight in the series with overt illness in their childhood showed no features distinguishing them from others in the series.

Among the various vitamin deficiencies encountered in untreated coeliacs, low folates is conspicuous. In recent years, folate deficiency has been seriously thought to induce intellectual deterioration and eventually dementia.^{10 11} The present study would seem to disprove such a risk, although admittedly, the use of serum determinations is not ideal in establishing folate deficiency. It is however apparent from other series¹² that untreated coeliacs show also depressed red cell folates, generally regarded as a better index of folate stores.

About 10% of adult coeliac patients will face

neurological complications, mostly in the form of peripheral neuropathy and various myopathies.^{13 14} Seizures are not rare and a rate of 5% reported in a British series suggests a tenfold increase in the prevalence of epilepsy among coeliacs. The reason for this remains obscure, and like coeliac patients in general, twelve examined in the present series showed unremarkable EEG tracings.

Our results should not be taken to preclude the possibility of significant impact on brain functions by nutritional deficiency. Refuting co-existent schizophrenia-like symptoms in coeliac disease, two studies¹⁷¹⁸ show agreement on a consistent depressive personality pattern in adult patients that is not readily explained by the physical complaints. Mental depression was in fact the major reason for granting disability pension in a recent series,¹⁹ and recalling the role monoamines are thought to play in depression, it is interesting that untreated coeliacs show reduced levels in CSF of the major metabolites of serotonin and the catecholamines.²⁰ The underlying defect is not known in detail, but the abnormalities may be closely related to concomitant nutritional deficiency induced by the intestinal malabsorption since they revert to apparently normal levels on strict dietary gluten withdrawal parallelling the recovery of the damaged jejunal mucosa.²¹

Bearing in mind the basic distinction between coeliac disease and common food intolerence, frequently associated with cognitive symptoms,²² there is no evidence of signs of intellectual impairment preponderating in adult coeliac patients. Admitting the critical opinions raised by others²³ ²⁴ regarding infant malnutrition, we feel that ideas relating cognitive inability to poor nutrition need to be reexamined.

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