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Coeliac disease

This article forms part of a series looking at the relationship between diet and good health, and the role of the dietitian in the primary health care team. This article discusses the assessment and dietary management of coeliac disease, a T-cell mediated reaction to gluten.

■ **Coeliac disease (CD), gluten enteropathy, is a T-cell mediated reaction that affects up to 1% of the population. Gluten from wheat, rye or barley, as well as a genetic predisposition, is essential for expression of CD. Life long avoidance of gluten is necessary for recovery.**

Coeliac disease is characterised by destruction of resorptive (villous) epithelium in the small intestine, which reduces the absorptive area and results in malabsorption of virtually all nutrients. Classic childhood symptoms include: weight loss, failure to thrive, abdominal cramping, bloating, flatus, nausea, vomiting, muscle wasting, diarrhoea, and steatorrhea.¹

Coeliac disease, in both adults and children, is a diagnostic challenge as it is now known that it can cause a broad range of signs and symptoms including: constipation, fatigue, headaches, mild gastrointestinal complaints, iron deficiency, miscarriage, bone fractures and dermatitis herpetiformis, but may also be asymptomatic (*Table 1*).

Mechanism of action

The presence of gluten in the small intestine is essential for expression of epithelial damage in genetically susceptible individuals. Gluten peptides (such as gliadin) penetrate between the cells of the epithelium where they are deamidated by free tissue transglutaminase (tTG). This deamidation of gliadin allows binding to the gene loci, human lymphocyte antigen (HLA) DQ2 or DQ8 molecules, which activates cytotoxic T-cells, stimulating both damage to the epithelium and production of antibodies to gliadin and tTG.²

It has been estimated that 30–40% of caucasians carry DQ2 or DQ8, but less than 3% of these people will develop CD.³ As the HLA-DQ2 and HLA-DQ8 genes can be present without expression of CD, the current view is that the pathogenesis of CD is a result of interactions between genetic, immunological and environmental factors.¹

Table 1. Features of coeliac disease

Presentation	<ul style="list-style-type: none"> • Fatigue • Gastrointestinal symptoms <ul style="list-style-type: none"> – bloating – cramps – diarrhoea – constipation • Anaemia • Osteoporosis NB: May have no symptoms
Age of onset	<ul style="list-style-type: none"> • Any age
Family history	<ul style="list-style-type: none"> • HLA gene association <ul style="list-style-type: none"> – coeliac disease – type 1 diabetes – thyroid disease
Reaction timing	<ul style="list-style-type: none"> • Hours → days
Reaction reproducibility	<ul style="list-style-type: none"> • Reproducible
Mechanism	<ul style="list-style-type: none"> • Immune (inflammatory T-cells)
Food triggers	<ul style="list-style-type: none"> • Gluten (wheat, triticale, rye, barley)
Tests (must be eating gluten at time of testing)	<ul style="list-style-type: none"> • Antibodies to tissue transglutaminase • Small bowel biopsy
Dietary management	<ul style="list-style-type: none"> • Gluten free diet (strict)
Outcome	<ul style="list-style-type: none"> • Life long immune reactivity • Bowel pathology and antibodies usually return to normal as a result of a gluten free diet

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Prevalence

Coeliac disease varies geographically and has a slight female preponderance due only to health care seeking behaviour. The accuracy of the tTG serological screening test, the high prevalence of silent CD (ie. biopsy proven, active disease without recognised symptoms) among the general population, and the recognition of nonclassic symptoms, has raised the prevalence of CD to approximately 1% in developed countries worldwide.^{4–6}

Table 2. Common ingredients and their approximate gluten content

Gluten free (<3 mg gluten/kg final food product)		Low or residual gluten (<200 mg gluten/kg of final food product)		Gluten containing
No possible gluten	No detectable gluten*	Trace sources of gluten*	Minor sources of gluten	Major sources of gluten
<ul style="list-style-type: none"> • Arrowroot • Buckwheat • Corn/maize • Lupin • Maize cornflour • Millet • Modified maize starch • Polenta • Psyllium • Rice • Sago • Seeds • Sorghum • Soy • Tapioca • Wine vinegar 	<ul style="list-style-type: none"> • Caramel colour • Dextrose • Fructose • Glucose • Glucose powder • Glucose syrup • Sorbitol 	<ul style="list-style-type: none"> • Beverage whitener • Thickeners 1400–1450 • Dextrin • Maltodextrin • Pre-gel starch 	<ul style="list-style-type: none"> • Cornflour* • Malt • Malt extract • Malt vinegar • Modified starch* • Oatmeal • Oats uncontaminated • Starch* • Wheaten cornflour • Wheat starch 	<ul style="list-style-type: none"> • Barley • Bran* • Biscuits* • Couscous* • Flour* • Noodles* • Pasta* • Rye • Semolina • Spelt • Triticale • Wheat • Wheatgerm • Wheat flour • Wheatbran • Wheatmeal
* If derived from wheat				
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Diagnosis

For diagnosis to be valid, adequate gluten (2–4 slices of wheat or rye bread for adults and two slices for children daily) is necessary for 6 weeks before investigations are carried out. The serological screening to investigate CD includes measuring total IgA to evaluate for selective IgA deficiency, IgA class anti-tTG antibodies and anti-gliadin antibodies IgA and IgG.⁴ Although the tTG test is highly specific, this test alone is insufficient for CD diagnosis and a positive blood test must be followed by small bowel biopsy. The diagnosis is confirmed if there is evidence of villous atrophy while eating gluten and a follow up biopsy shows improvement or normalisation of villous architecture after changing to a gluten free diet.⁷

Dietary management

Treatment involves the life long elimination of all gluten containing grains (ie. wheat, rye, triticale, barley) from the diet. Oats (25 g/day in children up to 50 g/day in adults) that have not been contaminated with gluten grains may be tolerated by many people, but not all.⁸

Since 2002 it has been compulsory for food manufacturers to disclose to consumers all foods and ingredients derived from gluten containing grains. While this provides consumers with confidence to avoid products that contain gluten containing flours and their derivatives, some, such as wheat derived glucose syrup, do not need to be avoided as the most sensitive test for detecting gluten (currently <3 – 5 mg gluten/kg total food) can find no detectable gluten in wheat derived glucose syrup. *Table 2* lists examples of ingredients and

their approximate gluten content according to the Australian Food Standards. An Accredited Practising Dietitian (APD) experienced in the area of CD has knowledge of products and food standards to assist patients in achieving a nutritionally adequate gluten free diet with maximum variety of foods and minimum restrictions.

Resources

- To find an APD with an interest in CD, visit the 'Find an APD' section of the Dietitians Association of Australia website at www.daa.asn.au or telephone 1800 812 942
- The Coeliac Society of Australia has support societies in all states and territories. Information about your nearest society can be found at www.coeliacsociety.com.au or by emailing info@coeliacsociety.com.au.

Conflict of interest: none declared.

References

1. Ciclitira PJ. Coeliac disease. In: Textbook of gastroenterology. Yamade T, Alpers DH, Owyang C, Powell DW, Silerstein F, editors. Philadelphia: Lippincott-Raven, 1999; Ch 74.
2. Schuppan D, Hahn EG. Gluten and the gut – lessons for immune regulation. *Science* 2002;297:2218–20.
3. Green PH, Jabri B. Celiac disease. *Ann Rev Med* 2006;57:207–21.
4. Fasano A, Catassi C. Current approaches to diagnosis and treatment of celiac disease: An evolving spectrum. *Gastroenterology* 2001;120:636–51.
5. Maki M, Mustalahti K, Kokkonen J, et al. Prevalence of celiac disease among children in Finland. *N Engl J Med* 2003;348:2517–24.
6. Tommasini A, Not T, Kiren V, et al. Mass screening for coeliac disease using anti-human transglutaminase antibody assay. *Arch Dis Child* 2004;89:512–5.
7. Maki M, Collin P. Coeliac disease. *Lancet* 1997;349:1755–9.
8. Kagnoff MF. Overview and pathogenesis of celiac disease. *Gastroenterology* 2005;128:S10–8.

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