

POSITION STATEMENT

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Brazilian Society for Food and Nutrition position statement: gluten-free diet

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Abstract

Position statement: The Brazilian Society for Food and Nutrition (SBAN) bases the following position statement on a critical analysis of the literature on the indications of a gluten-free (GF) diet. (1) There is insufficient evidence to assume that healthy individuals would experience any benefits from the consumption of a GF diet. (2) Recent studies suggest that gluten sensitivity may be confounded by sensitivity to low-fermentable, poorly absorbed, short-chain carbohydrates known as fermentable oligo-, di-, and mono-saccharides and polyols (FODMAPs). (3) Epidemiological data supports that even overweight celiac disease (CD) individuals fail to achieve weight loss under a GF diet. (4) Recent experimental data showed possible deleterious effects of GF feeding on the intestinal microbiota of healthy individuals. (5) GF diets can be healthy for the general population, as long as GF-processed foods are avoided, and the ingestion of other whole grains, and low-energy-density vegetables is assured. This position statement has been externally reviewed and approved by the board of the Brazilian Society for Food and Nutrition, and has not gone through the journal's standard peer review process.

Keywords: Diet, Gluten-free, FODMAPs, Celiac disease, Gluten sensitivity

Background

Among Western societies, nutrition has emerged as one of the most prominent and attractive fields in biomedical science. The increasingly high incidences of both obesity and chronic diseases, along with the growing interest in esthetic standards, are probably the main reasons why even non-specialized individuals (who were not previously concerned with the subject) are becoming “specialists” in general health/esthetic strategies.

We understand that the search for knowledge in this area is extremely helpful, as both health promotion and nutrition education depend on public awareness in order to be effective. However, because of the widespread dissemination by the general media, marketers, and food manufacturers, we now observe increased adherence to new “fashion diets,” which are based on restrictive interventions targeting weight loss and whole health improvement but which are usually grounded in either empirical observations or incomplete/inconclusive studies.

Following this trend of restrictive and monotonous diets, we have observed the popularization of a dietary pattern

previously accepted as treatment for celiac disease (CD) and wheat allergy (WA), characterized by exclusion of gluten from the patients' diets (a.k.a. gluten-free (GF) diet) [1, 2].

Celiac disease

Celiac patients carry a genetic mutation that leads to gut inflammation upon contact with peptides originating from the digestion of gluten from wheat, rye, or barley. Gliadin (one of the most common peptides found in gluten) is transported to the *lamina propria* of the gut wall, inducing a Th1 and Th2 immune response. As a result of Th1 response, T lymphocytes secrete a pattern of cytokines that play an important role in villous atrophy and intestinal crypt cell hyperplasia. In addition, Th2 response stimulates B cells to mature antibody-secreting plasma cells, which produce IgA antibodies against gliadin, transglutaminase, and gliadin-transglutaminase complex [3]. The resulting morphological change in the brush border leads to the defective absorption typically observed in celiac patients [4–6].

Clinical presentation

There are three main clinical forms of celiac disease: classic celiac disease with typical symptoms, atypical celiac disease, and asymptomatic or silent celiac disease [7].

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Classic celiac disease is characterized by chronic diarrhea, usually with abdominal distension and weight loss. These symptoms are often associated with loss of subcutaneous fat, gluteal muscles atrophy, anorexia, emotional lability (irritability or apathy), vomiting, and anemia. In the atypical form, gastrointestinal symptoms are completely absent in approximately 40 % of affected individuals. However, patients present relevant extraintestinal manifestations, such as low height, iron deficiency anemia refractory to oral iron treatment, vitamin B12 or folate deficiency anemia, osteoporosis, dental enamel hypoplasia, arthritis, intestinal constipation refractory to treatment, delayed puberty, sterility, recurrent miscarriage, psychiatric disorders (depression, autism, and schizophrenia), recurrent aphthous ulceration, elevated liver enzymes, weight loss, and weakness.

The asymptomatic form is characterized by positive antibody finding and pathological small intestine biopsy in the absence of disease symptoms.

Diagnosis

As a general rule, serology and histological investigations confirm the diagnosis in the majority of patients today. The most important antibodies are anti-gliadin antibodies, anti-endomysial antibodies, anti-tissue transglutaminase type-2, and anti-deamidated gliadin [7]. In addition, it is important to perform the biopsy of the small intestine to obtain at least four fragments of the distal duodenum [7–9].

Non-celiac gluten sensitivity

Besides CD, several recent evidences summarized by Mansueto et al. [10] and Czaja-Bulsa et al. [11] support the existence of a “non-celiac gluten sensitivity” (NCGS). In contrast to what happens in CD, NCGS patients exhibit preserved intestinal barrier permeability with no severe epithelial histological changes, but they still show numerous other disturbances including small lymphocyte infiltration, and gut inflammation, when exposed to a gluten-rich diet. However, NCGS is still a matter of debate. Researchers from Monash University, Australia, reported in 2011 that the positive effect of gluten-free diet in patients with NCGS could not be fully explained by a placebo effect [12]. Biesiekierski et al. showed that irritable bowel syndrome (IBS) like symptoms of NCGS was more frequent in the gluten-treated group (68 %) than in subjects receiving placebo (40 %) [12]. Despite that, in a second study with a more rigorous methodology, Biesiekierski et al. showed that there was no effect of gluten in subjects with NCGS, suggesting that gluten sensitivity might be confounded by sensitivity to low-fermentable, poorly absorbed, short-chain carbohydrates known as fermentable oligo-, di-, and mono-saccharides and polyols (FODMAPs) [13]. FODMAPs could contribute to NCGS symptoms, at least those related to IBS [13].

Although FODMAPs are found in gluten-containing grains such as wheat, rye, and barley, they can also be found in gluten-free foodstuffs including broccoli, garlic, onion, apple, and avocado. Peter Gibson, a gastroenterology professor at Monash University of Australia, and chief of this research group, in defense to criticism, he said their only interest is the truth, and the truth they only scratched the surface [14].

Treatment

Independent of the clinical condition (CD, WA or NCGS), the basic therapy for gluten sensitive patients includes the exclusion of wheat, rye, barley, and malt containing preparations. In addition, oatmeal should also be excluded because it is usually contaminated with wheat. Even if confirmed to be pure, if oats are introduced into the diet, there should be careful follow-up to monitor for signs of clinical and serological relapse [15, 16]. There is some evidence that a small number of patients with CD may be intolerant to pure oats and develop an immunological response to oat avenins. This could be related to a variation in toxicity of oat cultivars [15, 16].

Food manufacturers

Curiously, since 2004, gluten-free product sales have been growing around 30 % a year, despite no such corresponding increase in the incidence of gluten-sensitivity, and no more rigorous or widespread care of patients. This exponential increase appears to be due to a larger demand created by the adhesion of non-gluten-sensitive individuals to the GF diet [2, 17].

According to these individuals, the main reason to use GF products is the premise that they are healthier than their standard counterparts, helping with weight loss, improving pathological conditions and gastrointestinal discomfort. This premise has been supported and exploited by food manufacturers and may have led to an increased intake of processed GF products, which have been commonly reported as nutritionally poor [18].

Recommendations

Therefore, despite sensitive improvement in CD patients, there is insufficient evidence to assume that healthy individuals would experience any benefits from the consumption of a GF diet. Recent studies have also reported that gluten sensitivity might be confounded by sensitivity to FODMAPs. Regarding weight balance, for example, epidemiological data supports that even overweight CD individuals fail to exhibit weight loss under a GF diet [19]. According to a study with school children [20], there was an increase in body mass index (BMI) and in obesity prevalence among celiac children after exposure to the GF diet. It is important to note that GF diets are frequently poor in whole grains and fibers, the

intake of which is inversely proportional to BMI [21]. This could partially explain the source of the reported weight gain.

Regarding gastrointestinal health, recent experimental data showed possible deleterious effects of GF feeding on the intestinal microbiota of healthy individuals [22]. Due to the exclusion of foods containing wheat, there was a significant decrease in the ratio of good to bad intestinal bacteria in the stools of ten young adults, an outcome probably related to the lower intake of oligofructose and inulin, two types of soluble fiber essential to the maintenance of a healthy microbiota [22, 23]. Because of its prebiotic effect, one can even affirm that whole-wheat flour intake by non-gluten-sensitive individuals is associated with a reduced risk of intestinal cancer, inflammatory conditions, dyslipidemias, and cardiovascular disease.

Nevertheless, it is also important to note that GF diets can be healthy for the general population, as long as GF-processed foods are avoided, and the ingestion of other whole grains, and low-energy-density vegetables is assured. This does not, however, imply that gluten withdrawal is responsible for any of the possible benefits observed.

Finally, we reaffirm that the lack of solid scientific evidence along with some epidemiological data suggests that exclusion of gluten per se probably fails to improve overall the condition in healthy subjects and that wrong planning can even negatively affect the health of the population.

Conclusions

- There is insufficient evidence to assume that healthy individuals would experience any benefits from the consumption of a GF diet.
- Recent studies suggest that gluten sensitivity might be confounded by sensitivity to low-fermentable, poorly absorbed, short-chain carbohydrates known as fermentable oligo-, di-, and mono-saccharides and polyols (FODMAPs).
- Epidemiological data supports that even overweight celiac disease (CD) individuals fail to exhibit weight loss under a GF diet.
- Recent experimental data showed possible deleterious effects of GF feeding on the intestinal microbiota of healthy individuals.
- GF diets can be healthy for the general population, as long as GF-processed foods are avoided, and the ingestion of other whole grains, and low-energy-density vegetables is assured.

Abbreviations

BMI: body mass index; CD: celiac disease; FODMAPs: fermentable oligo-, di-, and mono-saccharides and polyols; GF: gluten-free; IBS: irritable bowel syndrome; NCGS: non-celiac gluten sensitivity; SBAN: Brazilian Society for Food and Nutrition; WA: wheat allergy.

Competing interests

All authors of this article have roles in the Brazilian Society for Food and Nutrition: Lucas Carminatti Pantaleão is Deputy Member of the Communication Committee, Marcelo Macedo Rogero is the Second Treasurer, and Olga Maria Silverio Amancio is the President.

Authors' contributions

LCP and MMR contributed to the manuscript drafting and final revision of the study. OMSA carried out the manuscript conception, manuscript drafting, and final revision of the study. All authors read and approved the final manuscript.

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References

1. Gaesser GA, Angadi SS. Gluten-free diet: imprudent dietary advice for the general population? *J Acad Nutr Diet*. 2012;112:1330–1.
2. NPD group. Percentage of U.S. adults trying to cut down or avoid gluten in their diets reaches new high in 2013, reports NPD. 2015. <https://www.npd.com/wps/portal/npd/us/news/press-releases/percentage-of-us-adults-trying-to-cut-down-or-avoid-gluten-in-their-diets-reaches-new-high-in-2013-reports-npd/> (accessed November 2015).
3. Sollid LM, Lie BA. Celiac-disease genetics: current concepts and practical applications. *Clin Gastroenterol Hepatol*. 2005;3:843–51.
4. Castillo NE, Theethira TG, Leffler DA. The present and the future in the diagnosis and management of celiac disease. *Gastroenterol Rep*. 2015;3:3–11.
5. Di Sabatino A, Corazza GR. Coeliac disease. *Lancet*. 2009;373:1480–93.
6. Ludvigsson JF, Leffler DA, Bai JC, Biagi F, Fasano A, Green PH, et al. The Oslo definitions for coeliac disease and related terms. *Gut*. 2013;62:43–52.
7. Sdepanian VL, Galvão LC. Doença celíaca. In: Lopez FA, Campos Jr D, editors. *Tratado de pediatria*. 2nd ed. Barueri: Manole; 2009. p. 819–27.
8. Fasano A, Catassi C. Current approaches to diagnosis and treatment of celiac disease: an evolving spectrum. *Gastroenterology*. 2001;120:636–51.
9. Hill ID, Dirks MH, Liptak GS, Colletti RB, Fasano A, Guandalini S, et al. Guideline for the diagnosis and treatment of celiac disease in children: recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition. *J Pediatr Gastroenterol Nutr*. 2005;40:1–19.
10. Mansueto P, Seidita A, D'Alcamo A, Carroccio A. Non-celiac gluten sensitivity: literature review. *J Am Coll Nutr*. 2014;33:39–54.
11. Czaja-Bulsa G. Non coeliac gluten sensitivity: a new disease with gluten intolerance. *Clin Nutr*. 2014;34:189–94.
12. Biesiekierski JR, Newnham ED, Irving PM, Barrett JS, Haines M, Doecke JD, et al. Gluten causes gastrointestinal symptoms in subjects without celiac disease: a double-blind randomized placebo-controlled trial. *Am J Gastroenterol*. 2011;106:508–14.
13. Biesiekierski JR, Peters SL, Newnham ED, Rosella O, Muir JG, Gibson PR. No effects of gluten in patients with self-reported non-celiac gluten sensitivity after dietary reduction of fermentable, poorly absorbed, short-chain carbohydrates. *Gastroenterology*. 2013;145:320–8.
14. Levinovitz A. *A mentira do gluten*. Porto Alegre: CDG. 2015. (ISBN: 978-85-68014-16-5).
15. Silano M, Dessi M, De Vincenzi M, Cornell H. In vitro tests indicate that certain varieties of oats may be harmful to patients with coeliac disease. *J Gastroenterol Hepatol*. 2007;22:528–31.
16. Comino I, Real A, de Lorenzo L, Cornell H, López-Casado MA, Barro F, et al. Diversity in oat potential immunogenicity: basis for the selection of oat varieties with no toxicity in coeliac disease. *Gut*. 2011;60:915–22.
17. Sapone A, Bai JC, Ciacci C, Dolinsek J, Hadjivassiliou M, Kaukinen K, et al. Spectrum of gluten-related disorders: consensus on new nomenclature and classification. *BMC Med*. 2012;10:13.

18. Satudacher HM, Gibson PR. How health is a gluten-free diet? *Br J Nutr.* 2015;114:1539–41.
19. Dickey W, Kearney N. Overweight in celiac disease: prevalence, clinical characteristics, and effect of a gluten-free diet. *Am J Gastroenterol.* 2006;101:2356–9.
20. Valletta E, Fornaro M, Cipolli M, Conte S, Bissolo F, Danchielli C. Celiac disease and obesity: need for nutritional follow-up after diagnosis. *Eur J Clin Nutr.* 2010;64:1371–2.
21. Marcason W. Is there evidence to support the claim that a gluten-free diet should be used for weight loss? *J Am Diet Assoc.* 2011;111:1786.
22. De Palma G, Nadal I, Collado MC, Sanz Y. Effects of a gluten-free diet on gut microbiota and immune function in healthy adult human subjects. *Br J Nutr.* 2009;102:1154–60.
23. Moshfegh AJ, Friday JE, Goldman JP, Ahuja JKC. Presence of inulin and oligofructose in the diets of Americans. *J Nutr.* 1999;129:1407S–11S.

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