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REVIEW ARTICLE

Diet low in fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) in the treatment of irritable bowel syndrome: Indications and design☆

Ana Zugasti Murillo[a,*], **Fermín Estremera Arévalo**[b], **Estrella Petrina Jáuregui**[a]

[a] *Sección Nutrición Clínica y Dietética, Complejo Hospitalario de Navarra, Pamplona, Spain*
[b] *Servicio de Aparato Digestivo, Complejo Hospitalario de Navarra, Pamplona, Spain*

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Abstract In recent years, there has been growing interest in diet low in FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) as an important mainstay in the treatment of irritable bowel syndrome (IBS). This model of diet was developed by a multidisciplinary team from the Monash University in Melbourne and became well-known after the publication of a study in 2008 showing that dietary FODMAPs acted as causing factors in patients with IBS. Since then there have been several randomized controlled trials which, although with small sample sizes, have again shown the benefits of this dietary pattern.

PALABRAS CLAVE
FODMAP;
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FOS

Dieta pobre en FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) en el síndrome de intestino irritable: indicación y forma de elaboración

Resumen En los últimos años está creciendo el interés en la dieta pobre en *fermentable oligosaccharides, disaccharides, monosaccharides and polyols* (FODMAP) como pilar importante en el tratamiento del síndrome de intestino irritable (SII). Este modelo de dieta lo desarrolló un equipo multidisciplinar de la Universidad de Monash, en Melbourne, y empezó a ganar notoriedad a partir de la publicación de un estudio en 2008 que demostraba que los hidratos de carbono fermentables (FODMAP) de la dieta actuaban como causantes de síntomas en los

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* Corresponding author.
E-mail address: ana.zugasti.murillo@cfnavarra.es (A. Zugasti Murillo).

pacientes con SII. Desde entonces se han llevado a cabo varios ensayos controlados aleatorizados que, aunque con muestras reducidas de pacientes, han vuelto a demostrar las ventajas de este modelo dietético.

Introduction

In Spain, intestinal bowel syndrome (IBS) affects 2.3–12% of the population, and is more common in females and patients under 55 years of age.[1] IBS is the most common cause of consultation in primary care for abdominal pain. The pathophysiology of IBS is unknown, and involves many things, from bowel motility changes to impaired peripheral perception and central processing of nociception in the gastrointestinal tract, known as ''visceral hypersensitivity''.[2] IBS is defined, using Rome III criteria, as consisting of pain or abdominal discomfort associated with two or more of the following criteria: (a) symptom improvement with defecation; (b) a change in stool frequency; or (c) a change in stool characteristics. These symptoms should have been present at least two days per week during the previous 3 months, and should have started at least 6 months before. There are four subtypes of IBS depending on the predominant symptoms: (a) constipation subtype; (b) diarrhea subtype; (c) alternating subtype; and (d) indeterminate subtype.[3] The complex and unknown pathogenesis and the differences between the patients who meet Rome III criteria make it difficult to develop effective strategies for treating IBS.

Sixty-percent of patients associate the intake of some food to symptom occurrence or exacerbation. Clinical signs and symptoms occur within 15 min–3 h of intake in 28–93% of patients respectively. The food items involved may trigger symptoms by different mechanisms (the activation of mast cells and mechanoreceptors, enzyme deficiencies, impaired bowel motility, abdominal distention, or chemosensitive activation by bioactive molecules). The restriction of some foods (gluten, fiber, fructose, lactose, caffeine, amines, fructose, sorbitol, etc.) has traditionally been used in IBS management. Food is not the cause of the disease, however, but the factor that triggers the symptoms.

FODMAP intake dose-dependently induces symptoms in patients with IBS. These symptoms are more marked when foods are taken together.[4,5] Research on the subject suggests that up to 70% of patients who follow a low FODMAP diet experience a significant improvement in symptoms, particularly those related to abdominal pain and distention.[6–13] The symptom showing the least improvement is constipation, which may be related to the low fiber provision in this dietary model.

The Low FODMAP diet was included in the guidelines for the treatment of IBS of the British Dietetic Association in 2010,[14] and in 2011 in the Australian guidelines.[15] Spanish gastroenterologists use drugs (antispasmodics, antidiarrheal, fiber, laxatives, anxiolytics) more frequently than dietary measures for the treatment of the main symptoms,[16] despite the fact that dietary changes are the measure most commonly used by patients for symptom self-management.[17] The most recent Spanish guidelines for the clinical management of this disease date back to 2005, and do not refer to this novel dietetic approach.[18]

This article will examine the concept of FODMAPs and their presence in various foods, and explain how to prepare a dietary plan.

General characteristics

FODMAPs have a number of important, unique characteristics with regard to the genesis of symptoms in IBS:

- *They are only absorbed with difficulty in the small bowel*. Fructose (a monosaccharide present in fruit), for example, is absorbed at half the speed of glucose; lactose (a disaccharide found in dairy products) must first be hydrolyzed to glucose and galactose, and many people suffer from a lactase deficiency; non-digestible oligosaccharides, such as fructans and galactans (mainly found in vegetables), usually accumulate in the distal small bowel and the proximal large bowel, thus becoming susceptible to metabolization by intestinal flora.
- *They are osmotically active*. When present in the intestinal lumen, they stimulate the mobilization of a large amount of water, which alters normal intestinal peristalsis, causing abdominal distention and pain and stools of decreased consistency.[19]
- *They are rapidly fermentable*. They are often an ideal substrate for normal and pathological intestinal flora. The product of this fermentation is gas, which causes distention and the activation of some nociceptive pathways.[13]

The FODMAP acronym was devised by an Australian group in 2001 to refer to fermentable, short-chain carbohydrates that cause gastrointestinal symptoms in subjects with visceral hypersensitivity.[20] This food group includes:

- Oligosaccharides: fructans and galactans. These cannot be digested by intestinal enzymes; they are always fermented by intestinal bacteria.
  - *Fructans*: these are fructose polymers with a glucose molecule at their end. Fructans with a degree of polymerization of 2–10 are commonly called fructooligosaccharides (FOS). FOS, especially as inulin, are frequently added to foods such as beverages, sauces, and energy bars to increase the fiber content and to