Chapter 6

Diet in the Etiology and Management of Functional Dyspepsia

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1. Introduction

Functional dyspepsia (FD) is a highly prevalent disorder, characterized by persistent or recurrent pain or discomfort centered in the upper abdomen without evidence of organic disease that might explain the symptoms [1]. Epidemiologic surveys suggest that 15 – 20 % of the general population in Western countries experience dyspepsia over the course of one year.

The options for managing functional dyspepsia are limited and far from ideal [2]. Limited information concerning the underlying pathophysiologic mechanisms has hampered the development of effective management strategies and specific therapeutic agents.

Several factors have been proposed to play a role in functional dyspepsia: delayed gastric emptying, *Helicobacter pylori* infection, hypersensitivity to gastric distention, impaired gastric accommodation to a meal, altered duodenal sensitivity to lipids or acids, abnormal duodenojejunal motility, and central nervous dysfunction. None of these abnormalities are able to completely account for the dyspepsia symptom complex [3].

Abnormal gastric motility and visceral hypersensitivity are generally thought to be directly linked to FD symptoms. Other factors that directly affect physiologic function include lifestyle, diet and genetics. [4,5]

The purpose of this chapter is to provide more specific dietary patterns and avoidances of certain food items in managing functional dyspepsia

2. Physiology of human feeding

The gastrointestinal tract processes ingested food via a complex series of actions in specific organs.

The esophagus propels food into the stomach though a relaxed lower esophageal sphincter that subsequently contracts to prevent gastroesophageal reflux. The functions of the proximal and distal stomach differ remarkably. Initially, the proximal stomach relaxes, providing a reservoir function. The distal stomach regulates the gastric emptying of solids by grinding and sieving the contents until the particles are small enough to pass the pylorus. The small intestine also regulates the gastric emptying rate through a feedback mechanism mediated by vagal nerves, and by physiological changes, such as gastric relaxation and the release of gastrointestinal hormones [3].

The overall process of digestion is coordinated by interactions between the gut and brain. Hunger is the sensation that leads us to seek and consume food, whereas satiety notifies us when to stop feeding.

Food intake is influenced by several types of gastrointestinal signals. These signals, when elicited by receptors in the stomach, provide information to the brain via the vagus nerve [6]. The stomach functions as a food reservoir; its capacity limits food intake. The gastric distention associated with ingestion of food activates tension mechanoreceptors and this generates a feeling of satiety. Pyloric chemoreceptors have an important role in regulating gastric motility, a fixed energy load being emptied into the duodenum at a constant rate regardless of meal composition. Conversely, gastrointestinal peptides, secreted by the stomach and small intestine with meals, primarily exert short–term effects on food intake. The gut peptides that reduce meal size are cholecystokinin (CCK), glucagon, glucagon – like peptide 1, amylin, somatostatin, peptide YY and bombesin. In contrast, ghrelin appears to have the opposite effect, stimulating enhanced food intake [3].

3. Pathophysiology of functional dyspepsia

Abnormal gastric motility and visceral hypersensitivity are thought to be the phenomena that are most closely related to the manifestation of FD symptoms.

Postprandial gastric motility may involve two possible sites: 1. the proximal stomach (fundus) exhibiting a disordered accommodation reflex after food ingestion, and/or 2. the antrum having abnormal gastric motor contractility. Proximal gastric distention, in fact, correlates very well with dyspeptic symptoms [2, 7]. The accommodation reflex is regarded as an appropriate response by which the stomach provides a reservoir facility for ingested food. In FD, this reflex can be impaired, leading to early satiety [8]. Such impairment occurs in 40 to 50% of FD patients [9]. In addition to impaired accommodation, delayed gastric emptying is also thought to contribute to the pathogenesis of FD. Food that is delayed in leaving the stomach provides the

sensation that the stomach feels heavy. Some reports have suggested that delayed gastric emptying may be seen in up to 40% of FD patients [10].

Visceral hypersensitivity is also an important factor contributing to the feeling of dyspepsia. When a balloon is distended in the stomach of an FD patient, the threshold at which pain is perceived is significantly lower in FD patients compared to normal controls [11]. Such gastric hypersensitivity relates to symptoms of postprandial pain, belching and weight loss.

4. Nutrients and gastrointestinal function

Different nutrients and food items may modulate gastrointestinal motor and sensory functions, and so provoke gastrointestinal symptoms. The three basic nutritional components (carbohydrates, proteins and lipids) can contribute to disturbed gastrointestinal function. The individual nutritional components impact gastric emptying and the sensation of fullness differently. 1. Lipids (fat) and proteins exert a negative ("braking") effect on gastric motility. Fat releases enteric hormones (such as CCK) that increase pyloric sphincter tone and delay gastric emptying. 2.

Proteins alter gastric motility, leading to a feeling of fullness and this provides satiety. 3.

Carbohydrates and some food chemicals (like salicylates and amines) give rise to an osmotic effect with increased luminal volume. This can result in a sensation of fullness particularly in patients with visceral hypersensitivity.

In addition to the individual nutrients, the caloric content, the physical form, and the ingested volume of food affect the sensation of satiety and fullness. High meal viscosity has a greater effect on the sense of satiety, whereas high caloric foods delay gastric emptying. [12,13].

Dietary nutrients influence gastrointestinal function and seem to be related to symptom generation. Thus, it seems logical that disturbances of gastrointestinal motor and sensory functions can lead to generation of gastrointestinal symptoms after food ingestion. However, usually mixtures of food items are eaten, creating difficulties when attempting to pinpoint the individual responsible factor and limiting advice in terms of dietary restrictions in patients with dyspepsia. Dietary measures are classically prescribed in the management of patients with motility disorders, although they have not been systematically studied.

5. Role of meals in the generation of symptoms in functional dyspepsia

Epidemiologic studies, both in the USA and Europe, have shown that 50 to 80% of subjects with functional dyspepsia indicate that their symptoms are meal-related [5]. Meal ingestion is associated with diverse changes in the environment of the gastrointestinal lumen, the gastrointestinal function and potential physiopathological mechanisms. The two most cited causes of pathology are delayed gastric emptying and visceral hypersensitivity. After ingestion of a

meal, patients with functional dyspepsia experience a marked rise in the intensity of their symptoms (epigastric burning, epigastric pain, fullness, bloating, nausea and belching) that persists for 4 hours [14]. Postprandial fullness is the most severe symptom that a meal aggravates.

Disturbances in upper gastrointestinal motor functions in FD have received considerable attention. Not surprisingly, current treatments such as prokinetics are primarily directed to these abnormalities. Therapies for visceral hypersensitivity remain difficult to establish. Factors such as eating patterns (meal size and frequency, nutrient composition, overall energy intake) and intolerances to specific foods or food groups have received little attention so far.

6. Dietary factors in functional dyspepsia

6.1. Eating patterns

Patients with functional dyspepsia frequently report that they are able to tolerate only small quantities of food [15], suggesting that their eating patterns differ from healthy subjects [8]. As a result of eating smaller quantities of food with lower energy intake, over half of FD patients experience weight loss even with a tendency to snack [16]. Despite eating fewer meals and consuming less total energy and fat, patients with FD experience fullness that is directly related to the amount of fat ingested and overall energy intake, while inversely related to the amount of carbohydrates ingested. Management of FD patients therefore might be improved by consuming smaller meals with reduced fat content [17,18].

6.2. Food intolerances

Patients with functional dyspepsia appear to exhibit more food intolerances than healthy persons, although studies are limited.

The belief that food is causing or at least triggering gut symptoms has led to the application of investigations purporting to guide dietary design. Various tests for food “intolerances” are widely available, such as skin prick (allergy) tests and assays for food specific immunoglobulins, but their value is unknown. Furthermore, various diets including wheat-free, anti-candida, carbohydrate-free, and other complex exclusions diets are touted in books and on the internet but evidence for any benefit is lacking.[19] The exception is the gluten-free diet, which will be discussed later in the chapter.

Alternative health practitioners and some nutritionists have advocated many such diets and intolerance-testing.

Therefore, gastroenterologists often are left with a defensive role when patients request dietary interventions. Although gastroenterologists may appreciate that food is an undoubted trigger, it is difficult to recognize the specific food item. Tests designed to this have a poor predictive value, while the resulting diets are often overly restrictive with the potential to render the patient nutritionally compromised [20].

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How Food Triggers Gastrointestinal Symptoms?

The enteric nervous system is a major controller of multiple gut functions, such as secretion, motility, blood flow and mucosal growth. In a normal situation, low intensity stimuli from the lumen have few discernible effects on motility (as occurs in association with minimal inflation of the balloon during barostat studies).

In most patients with functional gastrointestinal disorders, there is a change in relationship between stimulus intensity and perception (the hallmark of visceral hypersensitivity) and efferent motility response. These people experience pain in response to low intensity stimuli; abnormal motility responses may ensue.

Luminal events may be initiated via two main stimuli: mechanical (associated with distention of the gut wall) and chemical stimuli. Chemical stimuli trigger specific enteroendocrine cells of the gut, releasing serotonin, which stimulates primary afferents of the enteric nervous system [21]. There is also evidence that some enteric neurons might directly respond to mechanical stimuli: the transient receptor potential (TRP) cation channels seem to be involved in most levels of control of gastrointestinal function, including visceral hypersensitivity [22]. The TRPV1 (vanilloid) channels appear to be central to the initiation and persistence of visceral hypersensitivity in an animal model. Increased expression of TRPV1 channels in neurons of the gut has been observed in patients with IBS; such expression correlates with visceral hypersensitivity, and with abdominal pain [23,24].

Food stimulates the gut through the release of enteric hormones and particularly via the enteric nervous system. A primary trigger is luminal distention, which results from the physical act of ingesting food and from secondary events such as gas production (especially bacterial fermentation). Food also contains potent chemicals. If the food constituents that stimulate the enteric nervous system were to be identified, then these would become obvious targets for dietary manipulation.

On the basis of these concepts (luminal distention, visceral hypersensitivity and chemical stimuli of the enteric nervous system), three specific areas of proven or suspected food-induced gut symptoms in patients with functional GI symptoms are important: a. FODMAPs (fermentable oligo-, di- and monosaccharides and polyols) that include luminal distention; b. food chemicals (salicylates, amines) that potentially stimulates the enteric nervous system (ENS), and c. gluten that may trigger symptoms by as yet unknown mechanisms.

a. *Targeting luminal distention: The FODMAP approach*

Carbohydrates occur across a range of foods regularly consumed including grains such as wheat and rye, vegetables, fruit and legumes. Short-chain carbohydrates with chain lengths up to 10 sugars vary in their digestibility and subsequent absorption. Those that are poorly absorbed exert osmotic effects in the intestinal lumen increasing its water volume. They are also rapidly fermented by bacteria yielding consequent gas production. These two effects may underlie many of the gastrointestinal symptoms that follows their ingestion. Only monosaccharides (glucose, galactose) can be actively absorbed across the small intestinal epithelium.

Di- and oligosaccharides must be hydrolyzed to their constituent hexoses for absorption to occur. All these molecules are plentiful in the diet and have been termed FODMAPs[1]

FODMAPs are therefore poorly absorbed, highly osmotic and rapidly fermented by gastrointestinal bacteria, leading to increased water and gas. The result is intestinal distention that also effects changes in motility, leading to symptoms of bloating and discomfort [26]. FOPMAPs induce functional symptoms in patients with IBS who have fructose malabsorption; reduction of dietary FODMAPs produces a durable symptomatic response [27, 28].

Some common food sources of FODMAPs are summarized in table 1:

	Oligosaccharides, fructans	**Lactose**	**Fructose**	**Polyols**
Fruit	Peach, persimmon, watermelon		Apple, cherry, mango, pear, Watermelon	Apple, apricot, pear, avocado, cherry, blackberries, plum, prune, nectarine
Vegetables	Artichokes, beetroot, Brussels sprouts, chicory, garlic, onion, peas		Asparagus, artichokes, sugar snap peas	Cauliflower, mushroom, snow peas
Grains , cereals	Wheat , rye, barley			
Nuts	pistachios			
Milk		Milk, yoghurt, ice-cream, custard, soft cheeses		
Legumes	Lentils, chickpeas			
Other	Chicory drinks		Honey, high fructose corn syrup	
Food additives	Inulin			Sorbitol, mannitol, maltitol, xylitol, isomalt

Table 1.

b. *Targeting food chemicals*

Plants produce a wide variety of chemicals, some of which have survival function (the bad taste for protection, odors for reproduction), along with antibacterial or preservative properties.

Potentially bioactive chemicals include salicylates (that have a protective role), amines and glutamates (that are products of protein breakdown), and common food additives such as benzoates, sulfites and, nitrates (as preservatives).

1 FODMAP is an acronym for different carbohydrates: F: fermentable; O: oligosaccharides (fructans , galacto-oligosaccharides); D: disaccharides (lactose); M: monosaccharides (fructose); A: and P: polyols (sorbitol , mannitol , xylitol , maltitol) [25].

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In general, the stronger the flavor of the food, the higher the chemical content will be. In clinical practice, food chemicals have received some attention in the pathogenesis and management of urticaria, headaches, asthma and anaphylactic reactions.

Food chemicals are major afferent stimuli to the enteric nervous system. In the presence of visceral hypersensitivity, normal physiological stimulation by such chemicals might result in exaggerated effector responses (luminal distention). [32] Plant chemicals are able to activate TRP channels. Chronic exposure to certain chemicals will lead to increased expression of TRP channels and this contributes to a higher sensitivity of the enteric nervous system, and thus to the development of functional gut symptoms. Withdrawing the offending chemicals from the diet may reverse the TRP channel overexpression with subsequent resolution of the gut symptoms.

The only food chemicals that have been systematically studied with respect to gut symptoms are salicylates and related molecules such as non-steroid anti-inflammatory drugs. 2 to 4 % of patients with irritable bowel syndrome (IBS) or food allergies are salicylate-drug intolerant [29]. Examples of food sources containing high amounts of potentially bioactive chemicals are summarized in Table 2.

	Salicylates	**Amines**	**Glutamates**
Fruits	Avocado, berries , cherry, citrus , date, grape , kiwifruit, pineapple, plum , strawberry	Redcurrant	Dried prunes, raisins, grapes, plum, sultanas
Vegetables	Mushrooms, sauerkraut, spinach, tomato, chicory, eggplant, onion, chili, ginger, herbs	Eggplant, olives	
Grains , cereals	Breakfast cereals , mueslis, dried fruit, honey, coconut, potato chips		
Nuts	Almond, hazelnut, marzipan , peanut butter , nut pasta		
Seeds	Mustard seeds, sesame seed pasta		
Milk , milk products	Milk with chocolate, strawberry or banana flavor , yoghurt	Brie , camembert, parmesan , tasty cheeses	Brie , camembert , parmesan
Legumes	Bean mixes , broad beans , canned baked beans in sauce	Surimi, soy sauce , miso, tempeh	Canned baked beans in sauce , textured vegetable protein

	Salicylates	Amines	Glutamates
Meat, fish , chicken	Beef : smoked , corned , dried Chicken : nuggets , smoked Meat pastes , fish pastes , salami	Ham , bacon , anchovies, prawns tuna , fish : pickled , salted, smoked	Beef : billong, jerky Chicken : pressed , seasoned , gravy
Fats and oils	Almond oil , extra virgin olive oil , sesame , avocado oil	Almond oil , extra virgin olive oil , sesame oil	Almond oil , extra virgin olive oil , sesame oil
Beverages	Flavored mineral waters, spirits (except gin, tonic, whisky, vodka) , wine, fruit juices, ginger beer, beer , champagne , cider , herbal tea , tea	Beer , champagne , cider , tea , herbal tea , wine Chocolate drinks , cocoa powder	Beer , champagne , cider , tea , herbal tea , wine
Other	Jam , marmalade , fruit flavored syrup , yeast extract , vinegar (cider , red and white wine) Honey , peppermints, tomato sauce , soy sauce	Jam , marmalade , yeast extract , vinegar , chocolate , sauces ,	Jam , fruit flavored sweets , yeast extract , fermented products , chicken salt , sauces (tomato , soy , fish and oyster)

Table 2. Examples of food sources with very high amounts of salicylates, amines and glutamates (reference: http://www.allergy.net.eu)

c. *Targeting gluten: A suspected molecule without a known mechanism*

A. *Celiac disease* in recent years has undergone a profound revision. Celiac disease (CD) is now considered to be a systemic immune-mediated disorder elicited by gluten. The common denominator for all patients with CD is the presence of a combination of gluten-dependent clinical manifestations, specific autoantibodies (anti–tissue transglutaminase, anti-endomysial antibodies plus serum IgA) and different degrees of enteropathy, ranging from lymphocytic infiltration of the epithelium to complete villous atrophy. [33, 34] Nevertheless, CD remains underdiagnosed in all age groups. The advent of serological testing has improved the detection of celiac disease but typical endoscopic findings for villous atrophy such as scalloping of folds, a mosaic pattern, or decreased folds are often not evident in less severe cases. Magnification tools like confocal endomicroscopy or “water immersion” techniques help characterize the abnormal duodenal mucosa and target biopsying. In many patients, particular adults, the disease features atypical symptoms or is completely silent, the so-called “celiac iceberg”. Upper abdominal symptoms, such as abdominal pain and dyspepsia, are a common primary complaint in CD [36]. 30 to 40 % of celiac patients have dyspeptic symptoms. From a different perspective, diagnostic testing for celiac disease in individuals with dyspepsia has some advocates, because of a trend to a greater prevalence [35]. Nevertheless, the prevalence of

biopsy-proven celiac disease in individuals with dyspepsia may be as low as 1%, a value similar to that amongst individuals in the general population, or markedly higher at 6% to 9% [37]. Routine screening for celiac disease therefore seems useful through serological testing and with distal duodenal biopsy during upper gastrointestinal endoscopy done to investigate dyspepsia.

B. *Gluten (wheat) sensitivity*. Gluten may also induce other pathological conditions, such as a wheat allergy. Wheat allergy is an immunoglobulin IgE–mediated disease and thus completely unrelated to celiac disease. [33] Recent attention however has been given to another entity: gluten or wheat sensitivity (also termed non-celiac gluten sensitivity). This disorder misses one or more of the key criteria: enteropathy and the presence of specific autoantibodies that define celiac disease (CD). The current working definition of non-celiac gluten sensitivity is the occurrence of irritable bowel syndrome (IBS)-like symptoms after ingesting gluten, and improvement after gluten withdrawal from the diet. Celiac disease must be excluded by negative celiac serology or a normal intestinal architecture, while wheat allergy should be negated by a negative IgE-mediated allergy test to wheat. Non-celiac gluten sensitivity (NCGS) thus encompasses a collection of medical conditions in which gluten leads to an adverse food reaction, clinically similar to some features of celiac disease, but celiac testing is negative or inconclusive [38, 39]. Such non-celiac IBS patients, in whom celiac disease is excluded, will improve on a gluten-free diet [30].

The key question is the mechanism by which gluten induces symptoms. Gluten may mediate cholinergic activation, leading to increased smooth muscle contractility and indirectly have effects on luminal water content. Another explanation might be the release of neutrally active peptides from the gluten digestion that might potentially gain access to enteric nerve endings. Gluten ingestion can precipitate duodenal tissue eosinophilia in those with wheat sensitivity [39]. Although there is no well-established mechanism for NCGS, the gluten-free diet has gained substantial popularity with the general public.

7. Dietary management strategies in functional dyspepsia

Because of the many patients with functional dyspepsia and its serious impairment to their quality of life, this entity represents an important clinical challenge. Pharmacologic therapies are limited, leaving patients and physicians to often use dietary strategies in managing FD.

Unfortunately most of the available information concerning the role of diet and food intake in FD patients is inconclusive. Several studies fortunately have shown clear differences between FD patients and healthy persons in the ability to tolerate certain types of foods including fermentable carbohydrates (FODMAPs).

FD patients often maintain regular consumption of several foods despite these being implicated with the dyspepsia. Why these patients do not avoid the majority of food components, which they link to dyspepsia, remains unclear. Possible reasons might be ignorance of this association, a lack of alternatives to replace food items, or cultural habits such as the use of

coffee in some populations. Nevertheless dietary recommendations are intrinsic for managing FD. General advice should include consuming small, frequent meals that have a low-fat content.

Although such recommendations are helpful, specific strategies more commonly become necessary.

A well-trained nutritionist should direct the patient to record a 7-day food and symptom diary. It is also important to record other variables such as stress levels and activity as these factors can also impact symptomatology. The role of the dietitian is to explain the physiological basis of the diet, provide a list of suitable alternative foods and so restrict specific FODMAPs, while promoting a nutritionally adequate diet.

A low FODMAP diet is currently the first approach for many dietitians. This relatively complex diet involves the reduction, but not the complete avoidance of FODMAPs. Foods have been classified into high and low FODMAP content, and therefore knowledge of the FODMAP status of foods is an important skill for patient education (see table below). Low FODMAP foods that are suitable alternatives to foods high in FODMAP are encouraged. For example, rather than completely restricting fruit, reduce the intake of high FODMAP fruit and encourage the intake of FODMAP fruit [32]. After 6 to 8 weeks, the dietitian should undertake a review. If there is a satisfactory improvement, then a re-challenge could be done. It is important to determine the tolerance level, and also to increase variety in the diet. If the improvement is partial or absent, than additional dietary triggers should be emphasized: avoidance of some food chemicals such as salicylates, amines and glutamates, and last but not least a gluten-free diet might be initiated.

Any diet that aims to reduce one group of components will affect other dietary components with the potential to influence the same end point. This is certainly the case with a low FODMAP diet. As gluten-containing cereals also contain a high FODMAP content, any reduction of gluten intake would be accompanied by a decrease in other potentially symptom-inducing, cereal-related proteins. Likewise, if lactose is avoided in a proportion of patients, then the intake of dairy-associated proteins concomitantly may be reduced.

Type of food	HIGH in FODMAP	LOW in FODMAP
Milk	Milk : cow, sheep, goat, soy Creamy soups with milk Evaporated milk Sweetened condensed milk	Milk : almond, coconut, hazelnut, rice Lactose free cow's milk Lactose free ice cream
Yoghurt	Cow's milk yoghurt Soy yoghurt	Coconut milk yoghurt
Cheese	Cottage cheese Ricotta cheese Mascarpone cheese	Hard cheeses : cheddar, Swiss, brie, blue cheese, mozzarella, parmesan, feta No more than 2 tablespoons ricotta or cottage cheese

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Type of food	HIGH in FODMAP	LOW in FODMAP
		Lactose free cottage cheese
Dairy-based condiments	Sour cream Whipping cream	Butter Cream cheese
Dairy-based desserts	Ice cream Frozen yoghurt Sherbet	Sorbet from FODMAPs friendly fruit
Fruit	Apples, pears Cherries , raspberries, blackberries Watermelon Nectarines, white peaches, apricots, plums Peaches Prunes Mango, papaya Persimmon Orange fruit Canned fruit Large portions of any fruit	Banana Blueberries, strawberries Cantaloupe, honeydew Grapefruit, lemon, lime Grapes Kiwi Pineapple Rhubarb Tangelos <1/4 avocado <1 tablespoon dried fruit Consume ripe fruit ; less-ripe fruit contains more fructose
Vegetables	Artichokes Asparagus Sugar snap peas Cabbage Onions Shallot Leek Onion and garlic salt powders Garlic Cauliflower Mushrooms Pumpkin Green peppers	Bok choy , bean sprouts Red bell pepper Lettuce, spinach Carrots Chives, spring onion Cucumber Eggplant Green beans Tomato Potatoes Garlic infused oil Water chestnuts <1 stick celery <1/2 cup sweet potato, broccoli, Brussels sprouts
Grains	Wheat Rye Barley-large quantities Spelt	Brown rice Oats , oat bran Quinoa Corn Gluten-free bread, cereals , pastas and crackers without honey Apple/pear juice , agave

Type of food	HIGH in FODMAP	LOW in FODMAP
Legumes	Chickpeas , hummus Kidney beans, baked beans Soy milk Lentils	Tofu Peanuts <1/3 cup green peas
Nuts and seeds	Pistachios	1-2 tablespoons almonds, pecans, pine nuts, walnuts, sunflower seeds, sesame seeds
Sweeteners	Honey Agave High fructose corn syrup Sorbitol, mannitol, xylitol, maltitol	Sugars Glucose , sucrose Pure maple syrup Aspartame
Additives	Inulin Fructose-oligosaccharides Sugar alcohols Chicory root	
Alcohol	Rum	Wine , beer Vodka , gin
Protein-rich food		Fish, chicken, turkey, eggs, meat

Table 3. FODMAP status of food

8. Summary

Functional dyspepsia is a clinical problem of considerable magnitude for the health care system due to its high prevalence and the chronic or recurrent nature of symptoms. The manifestation of FD symptoms is directly caused by physiological abnormalities: abnormal gastroduodenal motility and/or visceral hypersensitivity. The therapeutic options for a clinician are limited and far from optimal: pharmacological therapies often fail. As food ingestion commonly triggers gastrointestinal symptoms, a dietary approach would seem most effective. There is reasonable evidence to suggest that a low FODMAP diet is beneficial, while gluten sensitivity may benefit others particularly in patients with IBS features. Gastroenterologists should no longer ignore specific dietary intervention for patients with functional dyspepsia.

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