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## Celiac Disease: What Dietitians Can Do to Effectively Treat a Growing Problem

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**CELIAC DISEASE: WHAT DIETITIANS CAN DO TO  
EFFECTIVELY TREAT A GROWING PROBLEM**

by

**Meagan Roxanne Wade**

**Thesis submitted in partial fulfillment  
of the requirements for the degree**

of

**DEPARTMENTAL HONORS**

in

**Dietetics  
in the Department of Nutrition and Food Sciences**

**Approved:**

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**UTAH STATE UNIVERSITY**  
**Logan, UT**

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## Author's Biography

Meagan Wade, born in Lubbock, Texas and raised in Silverthorne, Colorado graduated in 2002 from Summit High School in Breckenridge, Colorado. She pursued her interest in photography at the University of North Texas in Denton, Texas as a freshman. Deciding her interests had changed to nutrition, she transferred to Utah State University in 2003 to pursue a degree in dietetics. With a thirst for the challenging, Meagan applied to the coordinated program in dietetics and had the opportunity to receive clinical instruction from dietitians at McKay-Dee and Logan Regional Hospitals, Primary Children's Medical Center, Wasatch Dialysis Center, and the Diabetes Care Center. Passionate about building long-term relationships with clients and patients, Meagan looks to work in a dialysis center and help patients with kidney disease better understand and comply with their therapeutic diets.

Meagan has had the opportunity to work as an intern and staff assistant for the Food Stamp Nutrition Education Program for almost two years. Through her work, she has given lectures at statewide conferences and as satellite broadcasts. She has also had the opportunity to act as a student clinical preceptor for her younger peers in their internship experiences during the spring semester of 2007. She is a co-author of *Eat Right! Healthy Eating in College and Beyond* by Janet B. Anderson, Nedra K. Christensen, Emily Hoffman, Heidi LeBlanc, Kim McMahan, Tamara S. Vitale, Meagan Wade, and Heidi Wengreen (Benjamin Cummings/Pearson Education, San Francisco, CA:2007). She received a nomination for the American Dietetic Association student of the year for 2006-2007.

After she graduates in May 2007, Meagan will likely pursue a Master of Nutritional Science at Utah State University.

1 **Celiac Disease:**  
2 **What Dietitians Can do to Effectively Treat a Growing Problem**  
3

4 **Introduction**

5 The symptomology of celiac disease, also known as gluten-sensitive enteropathy or celiac  
6 sprue, was first described in AD 50 by Aretaeus, a noted ancient Greek physician who  
7 specialized in isolating and explaining disease states (1-3). Clinical manifestations of the disease  
8 were distinctly defined by Samuel Gee in 1888 who theorized that diet therapy was likely the  
9 treatment for the disease: “the allowance of farinaceous must be small, but if the patient can be  
10 cured at all, it must be by means of diet (1,2).” Farinaceous foods included those rich in starches  
11 or mealy in texture. Treatment at the time consisted of a dietary regimen of rice, bananas, and  
12 cream (2). Approximately 60 years later, a Dutch pediatrician named Willem Carol Dicke,  
13 isolated gluten as the “toxic fraction” and cause of the enteropathy associated with the disease  
14 (1,2). This discovery was made with the coinciding rise and fall of hospitalizations caused by  
15 celiac disease and the fluctuation in bread and wheat rationing during World War II (2). In the  
16 1980s, the causative factor of celiac disease was further determined to be an allergy to the gliadin  
17 fraction of gluten (1). Until recently, the disease was classified as a malabsorption disorder in  
18 children; it is now recognized to occur at any age and onset of symptoms may occur in  
19 individuals that are 50 to 60 years of age (4,5).

20 **Etiology and Characteristics of Celiac Disease**

21 Celiac disease is an inflammatory disorder of the small intestine that causes  
22 malabsorption secondary to flattening of the intestinal mucosa and microvilli as a result of the  
23 ingestion of gluten (3,6). Gluten is a complex protein and is classified generally as a prolamin  
24 protein as the storage form of protein in wheat, rye, and barley (1). The associated inflammatory  
25 response is a result of the intestine’s T-cell-mediated immune reaction specifically to the gliadin

1 fraction of gluten; gluten is comprised of the smaller proteins gliadin and glutenin (2, 5, 7-10).  
2 Gluten can be isolated as the proteins line up in a wheat flour and water combination; when  
3 water is passed through the product, the starch molecules are released from the protein matrix  
4 leaving a ball of pure gluten (1). Although ball formations of barley or rye and water cannot be  
5 manipulated in the same way as wheat, they elicit the same inflammatory response in the  
6 gastrointestinal system of those diagnosed with celiac disease and are classified also as gluten-  
7 containing (1).

8         The glutenin fraction is particularly apt to forming covalent bonds and allowing for the  
9 establishment of a matrix to trap water and create a dough-like texture, providing for the  
10 elasticity of the product in which it is used (1,10). The main amino acid complexes in gluten are  
11 glutamine and proline residues. Glutamine's role in the growth of the seedling is found in the  
12 high availability of nitrogen; proline is thought to have a protective role (10). Gluten abounds in  
13 the typical American diet and ingestion of rye, barley, wheat or related components insult the  
14 gastrointestinal tract and initiate inflammation (7). The disease tends to spread progressively  
15 through the bowel with the proximal small intestine as the first affected area (11).

16         Celiac disease is not classified as a food allergy but rather an inflammatory disease  
17 because the reaction is not immunoglobulin-E antibody mediated (1). Celiac disease has a multi-  
18 faceted effect on the body's immune systems. Innate immunity, or the immunity with which a  
19 child is born that allows for natural protection against harmful microbes, is comprised of both  
20 chemical (enzymes) and physical barriers (skin) (12). Adaptive or acquired immunity is initiated  
21 when an individual's innate immunity does not have the resources to overcome an insult;  
22 adaptive immunity results in the activation of B and T cells and antibodies to resolve the attack

1 (12). Although it is well known that celiac disease causes adaptive or acquired immunity, it is  
2 also thought to have an effect on innate immunity (10).

3 The National Institute of Health (NIH) has divided celiac disease into four different  
4 classes: 1) Classical celiac disease, which includes individuals who exhibit typical symptoms  
5 like diarrhea, bloating, abdominal pain, and weight loss; 2) Celiac disease with atypical  
6 symptoms, which includes those who experience few intestinal symptoms but exhibit symptoms  
7 like anemia, osteoporosis, neurological damage; 3) Silent celiac disease, which includes those  
8 who are asymptomatic but test positive through serologic testing and biopsy; and 4) Latent celiac  
9 disease which includes those who have positive serologies but negative biopsies who are likely  
10 to eventually manifest with the aforementioned symptoms and intestinal changes (2).

### 11 ***Gastrointestinal Signs and Symptoms***

12 Celiac disease is often not suspected without the presence of overt symptoms including  
13 diarrhea, abdominal discomfort and abdominal distention (7). Other hallmark symptoms include  
14 episodic diarrhea, steatorrhea, short stature, anemia, unintentional weight loss, abdominal  
15 distention edema, and dermatologic disorders; patients may present with an array of mild to  
16 severe symptoms (1,2,7,8). Only about one-half of all individuals diagnosed with celiac disease  
17 exhibit symptoms that involve the gastrointestinal tract (8). The classic manifestation of an  
18 infant with celiac disease is one in which the infant was considered thriving until the introduction  
19 of cereals into the diet after which the infant developed a distended abdomen, wasted extremities,  
20 loose stools, and generalized failure to thrive (5).

### 21 ***Nutritional Signs and Symptoms***

22 One of the most common ways that celiac disease is diagnosed is as a result of the  
23 unexplainable presence of anemia manifest secondary to gastrointestinal bleeding and iron

1 malabsorption (8). Because the small bowel is intimately involved in the absorption of vitamins  
2 and minerals, deficiencies of the fat-soluble vitamins A, D, E, and K and the water-soluble  
3 vitamins folate, B12 and the mineral calcium (8,11,13). Because the proximal small intestine is  
4 generally affected first, the initial nutrient deficiencies observed are often iron, folate, and  
5 calcium; if the disease progresses to the ileum, B12 becomes a concern, particularly for the  
6 elderly (11,13). A secondary lactose intolerance is not uncommon as the unhealthy intestine has  
7 an impaired ability to produce lactase. As the disease spreads throughout the small bowel, other  
8 nutrient deficiencies often manifest including carbohydrate, fat, and fat-soluble vitamins (11).

### 9 *Other Signs and Symptoms*

10 The gastrointestinal system is not the only system affected by celiac disease. The  
11 neurologic, hematologic, endocrine, orthopedic, and dermatologic systems are also included  
12 (11). With increasing research conducted in regards to celiac disease, neurological symptoms  
13 are found to be increasingly common with the condition. Neurologic manifestations include  
14 learning disorders, headache, and hypertonia (5). Dermatitis herpetiformis (DH) is a common  
15 dermatologic symptom of celiac disease; while not all those with celiac disease have DH, all  
16 those with DH have celiac disease (13). DH manifests as “severe, pruritic blistering skin” that is  
17 found on the buttocks, elbows, or knees and is more often found in adults than in children (13).  
18 The rash will likely clear up in six months if a gluten-free diet is followed (13). Left untreated,  
19 celiac disease increases the risk for developing autoimmune diseases, osteoporosis secondary to  
20 the malabsorption of calcium and vitamin D, reproductive disorders, and lymphoma (1,4,11).

21 Autoimmune diseases occur anywhere from 3 to 10 times more often in patients with  
22 celiac disease and include type 1 diabetes, primary biliary cirrhosis, Sjogren’s syndrome and  
23 peripheral neuropathy (1). Type 1 diabetes is often found to coexist with celiac disease as their

1 autoimmune etiologies are similar. Because of the complexity and difficulty in adhering to a  
2 gluten-free and a diabetic diet, these patients benefit greatly from education from registered  
3 dietitians (11). Malignancies are also increasingly observed in the celiac population (1).

#### 4 **The Challenge of Diagnosis**

5       Diagnosis of celiac disease in children is particularly notable as they begin to manifest  
6 failure to thrive and pass pale, malodorous stools (1,7,11). In adults diagnosed with celiac  
7 disease, symptoms vary greatly; some adults may exhibit several symptoms while others only  
8 experience a few (11). Because the symptoms are so generalized and varied, celiac disease is  
9 often misdiagnosed as other conditions, including irritable bowel syndrome, fibromyalgia,  
10 chronic fatigue syndrome, or anxiety disorders. Because celiac disease is often misdiagnosed as  
11 other diseases, many patients have visited with a number of different specialty physicians in  
12 order to determine the cause of their symptoms by the time they are finally diagnosed with celiac  
13 disease (11). Diagnosis of the disease can occur at any age with the mean age of diagnosis  
14 between 40 and 50 years of age. Women are diagnosed two to three times more than men with  
15 diarrhea as the most predominant symptom (1). Diagnosis has become increasingly common in  
16 the past 50 years; however, celiac disease is still considered to be underdiagnosed (4).

17       Villous atrophy is the affirmative diagnostic marker of the disease (2). There are three  
18 factors that are considered to positively diagnose celiac disease: 1) a positive serologic test; 2) a  
19 biopsy sample of the small intestine; and 3) a positive response to a gluten-free diet (4,8). The  
20 serologic tests look specifically for levels of immunoglobulin-A endomysial antibody (IgA-  
21 EMA) and IgA tissue transglutaminase antibody (IgA-tTGA) (8). Antigliadin antibodies are  
22 generally not sensitive enough to indicate celiac disease (8). A biopsy indicative of celiac  
23 disease will show villous atrophy and lymphocytic infiltration of the intestinal mucosa; it is also



1 considered the “gold standard” for diagnosis because of the notable histologic features of the  
2 disease (1,2,8,11). The villous atrophy exhibited in this condition is progressive and the extent  
3 of the atrophy may influence the presence or absence of the serum antibodies (11). Conflicts  
4 arise if both serologic tests and the biopsy do not indicate celiac disease (8). If the serologic test  
5 is positive and the biopsy is negative, the biopsy should be reviewed and a second biopsy should  
6 be considered (8). If the biopsy yields positive results and the serologic test is negative, it is  
7 possible that there is an IgA deficiency or that there is another cause of the enteritis (8). A single  
8 negative biopsy does not necessarily indicate that the individual does not have celiac disease as  
9 atrophy progresses over time (5). It is critical that a gluten-free diet not be followed until after  
10 the serological testing is done to ensure a proper diagnosis (11).

11         The extent of intestinal damage can be based on a continuum as established by Marsh (1).  
12 The initial (Marsh I) lesion manifests in “intraepithelial lymphocytosis in normal appearing villi”  
13 (1). Further progression to Marsh II lesions manifest in “crypt hyperplasia in addition to the  
14 intraepithelial lymphocytosis” (1). However, the majority of those diagnosed with celiac disease  
15 have progressed to the Marsh III stage of the continuum at the time of diagnosis with “villous  
16 atrophy, crypt hyperplasia, and intraepithelial lymphocytosis” (1). The Marsh IV stage includes  
17 all degrees of villous atrophy from partial to total atrophy encompassing all hallmarks of the  
18 previous stages.

19         Four different antibodies can be tested and are considered nonexistent in the serum of  
20 those individuals who have followed a gluten-free diet for six months or longer (8). Early  
21 diagnosis is beneficial for the patient as he/she does not experience his/her symptoms for a  
22 lengthy period of time which will result in a more favorable outcome (11). Surveys recently

1 taken by individuals with celiac diseases indicate that the mean length of time suffered by  
2 patients until diagnosis is about 11 years (2,11).

3         There is an overall difficulty for primary care physicians in diagnosing celiac disease  
4 with the majority of diagnoses resulting from a consultation with a gastroenterologist (4). Zipser  
5 et al. discovered that of 2440 patients diagnosed with celiac disease, only 11% were diagnosed  
6 by primary care physicians (4). Those diagnosed by primary care physicians had only suffered  
7 symptoms for about 1 year prior to diagnosis. Those who were eventually diagnosed by a  
8 gastroenterologist had suffered the symptoms for an average of at least seven years; the delay in  
9 diagnosis could not be directly attributed to a delay on the part of the referring physician or the  
10 gastroenterologist. However, a study conducted in Ireland found that about two-thirds of  
11 patients with celiac disease are diagnosed by primary care physicians using endomysial antibody  
12 testing which has been found to yield 75-98% sensitivity and provide an accurate diagnosis (4).  
13 This study demonstrated that only 44% of US physicians were aware that endomysial testing was  
14 an effective method to diagnose celiac disease (4). Additionally, most physicians were unaware  
15 of the range of symptoms from abdominal pain to osteoporosis (4).

#### 16 **The Profile of Those with Celiac Disease**

17         Individuals are thought to be genetically predisposed with 1 in 120-300 individuals in  
18 North America and Europe being affected (6). The disease occurs largely in those of Caucasian  
19 ancestry and is rarely found in individuals of African-Americans, Japanese, and Chinese descent  
20 (1,5). The prevalence in first-degree relatives of an individual with celiac disease is 1 in 22;  
21 prevalence among second-degree relatives is 1 in 39 (11). Other groups of individuals are  
22 considered more susceptible to celiac disease than the general population include those with  
23 Down's syndrome, Turner syndrome, and rheumatoid arthritis (5). Celiac disease has long been

1 thought to occur mainly in childhood, especially after cereals containing gluten were introduced  
2 into the diet (11). In more recent studies and observations, however, it has been found that in  
3 those who are genetically predisposed, celiac disease can manifest at any age (5,11).

#### 4 **The Intestinal Impact of Ingesting Gluten**

5 More than one gene is implicated in the occurrence of celiac disease (10). In individuals  
6 without celiac disease, gluten is broken down in the small intestine into peptide fractions; in the  
7 individual with celiac disease, the 33-amino acid sequence fails to be broken down by the  
8 digestive process (2,10). The inflammatory cascade is initiated when the undigested protein  
9 sequence enters the lamina propria that causes the release of T cells. The release of T cells spurs  
10 the release of cytokines, which in turn activate the inflammatory response. As the villi are  
11 inflamed, they are unable to appropriately absorb nutrients (2). The gliadin in the undigested  
12 proteins promotes the action and accumulation of lymphocytes to the site of inflammation (7).  
13 The reaction is T-cell mediated and occurs in the lamina propria (1,10).

#### 14 **Medical Treatment**

15 The NIH has identified 6 main interventions in the general medical treatment of  
16 individuals with celiac disease that follow the acronym CELIAC: 1) Consultation with a  
17 registered dietitian; 2) Education; 3) Lifelong adherence to a gluten-free diet; 4) Identification  
18 and treatment of nutritional deficiencies; 5) Availability of a support group; 6) Continuous long-  
19 term follow-up by a multidisciplinary team, including a registered dietitian (2,5).

#### 20 **Nutritional Treatment**

21 The only known treatment for celiac disease is the adherence to a gluten-free diet for life  
22 and in a "wheat-laden world" (2), it presents a hefty challenge (2,11). Strict adherence to a  
23 gluten-free diet is mandatory for recovery and maintenance of proper health and nutrition: as

1 little as 1/8<sup>th</sup> of a teaspoon of flour will cause notable damage to the mucosa (2). After a gluten-  
2 free diet is followed for a period of time, the intestinal villi will return to a normal and absorptive  
3 state (3).

4 A comprehensive approach must be taken when counseling patients with celiac disease  
5 with particular note made of both the emotional and social impacts of the disease (2). On all  
6 quality of life questions answered in a survey done by Lee and Newman, participants with celiac  
7 disease indicated that they felt compromised by a gluten-free diet (3,14). In a study done by  
8 Sverker et al., patients admitted to experiencing a variety of emotions including fear, isolation,  
9 and shame (14). One of the participants stated, "I am often reminded at meals with friends and  
10 family. They eat something else, and it looks so good.... but I can't eat it. It can happen several  
11 times a week and I think it is so sad" (14). Another participant considered eating at restaurants  
12 with friends a family a "tragedy" as it often took a visit to three or four restaurants to find one  
13 that would serve a gluten-free dish (14). Additionally, contention can arise in a restaurant setting  
14 in which workers are unaware of the needs of a gluten-free diet or are unwilling to cater to the  
15 needs of the gluten-free diner. Many individuals also worry about being a burden to those with  
16 whom they associate (14).

17 A variety of foods contain gluten, including unusual and less-obvious foods that typically  
18 would not be considered (11). Wheat is one of the primary grains used in preparing foods in the  
19 United States and is a major component of typical daily food items like breads and cereals.  
20 Other food items that contain gluten or wheat derivatives include sauces, soups, and  
21 thickening/stabilizing agents. Wheat starch may be found in products like prepared meats,  
22 including luncheon meats and hot dogs, soy sauce, candy, and some medications (11). Wheat

1 flour and malt from barley are common components of gravies, soups, processed cheeses, and  
2 icing (2).

3 Food labels must be read thoroughly. Food manufacturers in the United States are only  
4 required to include the eight major allergens on food labels; this only includes wheat and does  
5 not include a mandatory statement as to whether or not it is gluten-free. It is important to  
6 understand that “gluten-free” and “wheat-free” are not synonymous (11). If ingredient lists are  
7 ambiguous, the manufacturer should be contacted to confirm the content of the product (2).  
8 “Hydrolyzed proteins” and “modified food starch” are generic terms that imply that wheat may  
9 have been used but is not necessarily a component listed on the ingredient list (11). Because of  
10 this ambiguity, the products that contain these ingredients must be avoided.

11 A gluten-free diet is often considered unpleasant to follow as many of the product  
12 replacements, like gluten-free bread, have unpleasant texture and taste and storage problems  
13 (10). Additionally difficult is the mixing of gluten-free and gluten-containing products. For  
14 example, a cereal may contain only gluten-free grains but the grains are contaminated in a plant  
15 that also processes gluten-containing cereal (10).

16 Although it may appear that many foods must be excluded from the gluten-free diet, there  
17 are also many food items that are naturally gluten-free including meats, fruits, vegetables, fish,  
18 poultry, eggs, nuts and seeds, milk, yogurt and cheese (11). Ancient grains such as quinoa,  
19 buckwheat, and millet are considered acceptable as they are not related to wheat and provide a  
20 good source of B-vitamins that may be difficult to acquire in a diet without wheat, rye, and  
21 barley (2). A mixture of gluten-free flours are often used to effectively create products that are  
22 similar to those made with gluten-containing flour (11). For example, a gluten-free yeast bread

1 can be effectively made with a combination of xanthan gum and rice, tapioca, and potato starch  
2 flours (15).

3 Following a strict gluten-free diet is advisable to maintain adequate health; in addition to  
4 an individual food component, cross-contamination with gluten-containing products must be  
5 avoided (11). Separate pans, utensils, and even toasters are suggested for the individual with  
6 celiac disease who lives in a family or group setting. An individual with celiac disease should  
7 have his or her own condiments so there is minimal risk of contamination with gluten-containing  
8 products (11).

9 Because following a gluten-free diet has a significant impact on dietary patterns, it in turn  
10 has extreme impact on all aspects of life. In individuals with celiac disease who adhere to a  
11 gluten-free diet, research has documented a general decrease in social interactions travel and  
12 dining at restaurants (11). Teenagers are often noncompliant to a following a gluten-free diet  
13 which is of particular concern considering that the teenage years are those in which nutrient  
14 demand for bone deposition and overall growth is greatest. Follow-up and personalized attention  
15 by both physicians and dietitians is critical in the success of patients with celiac disease (11).

#### 16 **Are oats appropriate?**

17 Only recently have oats in moderate amounts been deemed acceptable for individuals on  
18 a gluten-free diet (11). Because oats contain avenin which is classified as a prolamin protein  
19 (like gluten), it has been thought that oats may trigger a similar reaction (2,9). However, the  
20 current and more accurate issue with oat consumption is that oats are usually contaminated with  
21 barley, rye, or wheat (2,11). Companies are now attempting to keep oats from being  
22 contaminated in transport, milling, and growing (11). While oats have typically been avoided by  
23 those on a gluten-free diet, recent feeding studies have indicated that oats are considered safe for

1 those with celiac disease to consume because they do not share the same amino acid sequence as  
2 rye, barley, and wheat (2,9). In vitro studies done with intestinal biopsies have shown no  
3 reaction to the avenin fraction of oats (9). However, studies have shown that some individuals  
4 are chronically sensitive to oats while others are not; it cannot be generally concluded that all  
5 patients with celiac disease can consume oats (9).

6 In a study done by Arentz-Hansen et al., nine patients with celiac disease were shown to  
7 have quite different reactions to oats. Three of the participants experiencing Marsh III  
8 classifications, three patients with absolutely no intestinal reaction, 2 patients meeting Marsh II  
9 and one patient meeting Marsh I criteria (9). Though small, the study indicates that some  
10 patients with celiac disease have a sensitivity to oats. It is currently unknown what percentage of  
11 those with celiac disease also have a sensitivity to oats. It is important that clinicians are aware  
12 that some individuals with celiac disease may experience villous atrophy even if they are  
13 adhering to a gluten free diet because of sensitivity to oats (9).

#### 14 **A Dietitian's Role**

15 Nutrition assessment for those with celiac disease is based mainly on weight  
16 measurements and laboratory values; registered dietitians should also be aware of common  
17 nutritional deficiencies and the potential need for supplementation, the differences in  
18 presentation of the disease and the emotional and social burden of the disease (2). Iron, mean  
19 cell volume, calcium, electrolytes and albumin should be watched particularly closely. The  
20 registered dietitian should be sensitive to patient information overload and a patient's willingness  
21 to learn. It is generally recommended that education be given in two general sessions. The first  
22 session should cover survival skills, grocery shopping, and social situations and would be a  
23 perfect opportunity for the dietitian to offer hands-on experience by going to the grocery store

1 with the client. The second session should include information about exercise, alternative grains,  
2 and overall health and nutrition. Adequate and appropriate education for the patient with celiac  
3 disease is critical. Unless a patient feels empowered and that a gluten-free diet is possible for  
4 him/her to follow, adherence is not likely. In fact, some patients admit to intentionally  
5 “cheating” on their diet with only 68% of individuals following a gluten-free diet all of the time  
6 and 30% following it most of the time (2). When away from home, 26% of individuals violate  
7 their diet and 21% tend to violate their diet at social functions (3). Individuals on other  
8 restrictive diets have some flexibility in making food choices without long-term consequences  
9 for noncompliance; however, those with celiac disease will suffer consequences if they do not  
10 adhere strictly to a gluten-free diet (2).

11 Lee et al. conducted a survey that found that 49% of individuals with celiac disease  
12 considered their health to be “good” before following a gluten-free diet; 71% of individuals  
13 surveyed considered their health to be “excellent” after following a gluten-free diet (3). Only  
14 13% of individuals surveyed had reported receiving diet instruction from a registered dietitian of  
15 which only 21% found the instruction helpful (3). Perhaps the greatest issue involving registered  
16 dietitians is that some registered dietitians may have little or no knowledge about celiac disease  
17 which causes them to give ineffective education (3).

18 Many support groups locally and on the internet offer an avenue for reinforcement, ideas,  
19 recipes, and encouragement. Many websites offer email updates of new findings or  
20 manufacturing changes that benefit or change the habits of those following a gluten-free diet.  
21 The Celiac Disease Foundation in particular strives to maintain and disperse information on the  
22 most recent political and scientific movements that impact those with celiac disease (15). Other  
23 support groups for those with celiac disease can be accessed by typing “support group for celiac



1 disease” into a search engine. Both national and local results provide individuals with an  
2 opportunity to network with those who are experiencing a similar situation. It is critical for a  
3 registered dietitian to be aware of local support groups in which the client might be interested  
4 based on his/her age at diagnosis and/or corresponding complications.

5         Because celiac disease can only be effectively treated through diet, registered dietitians  
6 are an essential part of the treatment of celiac disease and the outcome of the patients who have  
7 the disease. Without an effective registered dietitian who focuses on evidence-based, individual  
8 treatment, adherence to a gluten-free diet will be minimal. It is critical that the dietitian realize  
9 the social and emotional impact that will occur in the newly-diagnosed individual. It is essential  
10 to help them transition effectively through active learning and not allow them to feel alienated by  
11 only providing them with reading materials to take home and decipher for themselves.

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