Food Engineering Series
Series Editor: Gustavo V. Barbosa-Cánovas

Navneet Singh Deora
Aastha Deswal
Madhuresh Dwivedi *Editors*

Challenges and Potential Solutions in Gluten Free Product Development

Springer

Food Engineering Series

Springer's *Food Engineering Series* is essential to the Food Engineering profession, providing exceptional texts in areas that are necessary for the understanding and development of this constantly evolving discipline. The titles are primarily reference-oriented, targeted to a wide audience including food, mechanical, chemical, and electrical engineers, as well as food scientists and technologists working in the food industry, academia, regulatory industry, or in the design of food manufacturing plants or specialized equipment.

More information about this series at http://www.springer.com/series/5996

Navneet Singh Deora
Aastha Deswal • Madhuresh Dwivedi
Editors

Challenges and Potential Solutions in Gluten Free Product Development

Springer

Editors
Navneet Singh Deora
Chief Technology Officer
Bluetribe Foods
Mumbai, India

Aastha Deswal
Bright LifeCare Private Limited
Gurugram, Haryana, India

Madhuresh Dwivedi
Department of Food Process Engineering
National Institute of Technology
Rourkela, Odisha, India

ISSN 1571-0297
Food Engineering Series
ISBN 978-3-030-88696-7 ISBN 978-3-030-88697-4 (eBook)
https://doi.org/10.1007/978-3-030-88697-4

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

Recent epidemiological studies have shown that 1 in 100 people worldwide suffer from gluten intolerance. The worldwide average of celiac sufferers has been predicted to increase in the next number of years. This will result in a rapid-growing market for high-quality gluten-free cereal products. However, due to the unique properties of gluten, it is a major challenge for food scientists and industries to manufacturer quality gluten-free products at present.

Additionally, gluten absence results in major complications for product developers. Currently, many gluten-free products available in the market are of low quality and limited shelf life, and exhibit inferior mouthfeel and overall acceptability as compared to the products containing gluten. This presents a major challenge to the cereal technologist and baker alike, and has led to the search for alternatives to gluten in the manufacture of gluten-free bakery products. This book aims to provide the possible solutions for the gluten-free product development.

The only treatment for celiac disease is the total lifelong avoidance of gluten ingestion. Patients have to follow a very strict diet and avoid any products that contain wheat, rye, or barley (some authors also include oats). Avoidance of these cereals leads to a recovery from the disease and significant improvement of the intestinal mucosa and its absorptive functions. Patients with celiac disease cannot eat some common foods such as bread, pizzas, and biscuits or drink beer. This book also aims to cover the nutrition aspects of gluten-free products. Since protein is of paramount importance, this book will also serve to include the alternative protein in gluten-free product development in form of case study.

The overall aim of this book is to provide the reader a chance to take a journey through all aspects related to product development for patients suffering from celiac disease. As such, this book is unique in its form and hopes to represent a technical guide to the readers working in the related areas. It aims to summarize and critically review the works and knowledge gained so far in the area of gluten-free product development.

Mumbai, India — Navneet Singh Deora
Gurugram, Haryana, India — Aastha Deswal
Rourkela, Odisha, India — Madhuresh Dwivedi

Acknowledgment

This book is a result of the combined efforts of highly qualified scientists. Each contributor was responsible for researching and reviewing subjects of immense depth, breadth, and complexity. Care and attention were paramount to ensure technical accuracy for each chapter discussed in this book. This book is unique as stated earlier, and it is our sincere hope and belief that it will serve as an essential reference for gluten-free products.

We wish to thank all the contributors for sharing their expertise throughout our journey. We also thank the reviewers for giving their valuable comments, leading to improvements in the content of each chapter.

The production of this current book could not have been accomplished without the hard work and excellent suggestions of professionals in the production team assigned by Springer to manage the project.

Disclaimer

All information in this book is based on practical knowledge gained by the author while working in factories as well as theoretical knowledge gained during his studies and should not be used as the basis for any legal claims. Hence, all information stated is not intended to credit or discredit any manufacturer of equipment or additives and is based purely on the opinion of the authors.

Contents

Contributors

R. Anand Kumar Department of Food Process Engineering, NIT Rourkela, Rourkela, Odisha, India

Saptashish Deb Department of Food Engineering and Technology, Sant Longowal Institute of Engineering & Technology, Sangrur, Punjab, India

Center for Rural Development and Technology, Indian Institute of Technology Delhi, New Delhi, India

Madhuresh Dwivedi Department of Food Process Engineering, NIT Rourkela, Rourkela, Odisha, India

Payel Ghosh Department of Food Technology, Vignan's Foundation for Science Technology and Research, Vadlamudi, Andhra Pradesh, India

Rewa Kulshrestha Department of Food Processing and Technology, Atal Bihari Vajpayee Vishwavidyalaya, Bilaspur, Chhattisgarh, India

Chitrangada Das Mukhopadhyay Centre for Healthcare Science and Technology, Indian Institute of Engineering Science and Technology, Shibpur, Howrah, West Bengal, India

Mohona Munshi Department of Food Engineering and Technology, Sant Longowal Institute of Engineering & Technology, Sangrur, Punjab, India

Department of Chemical Engineering, Vignan Foundation for Science, Technology, and Research, Vadlamudi, Guntur, Andhra Pradesh, India

Surabhi Pandey Bioresource Engineering Department, McGill University, Ste-Anne-de-Bellevue, Canada

Sumit Sudhir Pathaka Department of Food Process Engineering, National Institute of Technology Rourkela, Rourkela, Odisha, India

Rama Chandra Pradhana Department of Food Process Engineering, National Institute of Technology Rourkela, Rourkela, Odisha, India

Sandeep Singh Rana Department of Food Technology, Vignan's Foundation for Science, Technology and Research, Vadlamudi, Andhra Pradesh, India

E. J. Rifna Department of Food Process Engineering, NIT Rourkela, Rourkela, Odisha, India

Winny Routray Department of Food Process Engineering, NIT Rourkela, Rourkela, Odisha, India

Murakonda Sahithi Department of Food Process Engineering, NIT Rourkela, Rourkela, Odisha, India

Mahipal Singh Tomara Department of Food Process Engineering, National Institute of Technology Rourkela, Rourkela, Odisha, India

About the Editors

Navneet Singh Deora is a food engineer with a PhD from the Agricultural & Food Engineering Department, Indian Institute of Technology (IIT) Kharagpur, India; has a master's degree in food technology from CSIR-CFTRI, Mysore, India; and is a Bachelor of Engineering in Food Processing Technology from AD Patel Institute of Technology (ADIT), Gujarat. In the past, he was involved with RPSG Goenka Group (*Extrusion Expert*, 2019), Jubilant Food Works (*Open Innovation*, 2018), and Nestle R&D (*Cereal Specialist*, 2017). His research interests focus on open innovation in the food industry, sustainability, plant-based proteins, equipment design, and advancements in cereal science. He is an author or co-author of multiple research papers, books, and patents. He is currently chief technology officer of Bluetribe Foods.

Aastha Deswal is a PhD from the Agricultural & Food Engineering Department, Indian Institute of Technology (IIT) Kharagpur, and graduate from CFTRI, Mysore. She has over 10 years of research experience in the field of food technology at both academic and industrial levels covering various topics but mostly focused on product development. She has hands-on experience of working with gluten-free products in the past, which has been very helpful while editing this book. She has authored many research articles, chapters, and books covering various topics of food technology.

Madhuresh Dwivedi, MTech, PhD, is an assistant professor in the Department of Food Process Engineering at the National Institute of Technology (NIT) Rourkela. Dr. Dwivedi obtained his BTech degree in agricultural engineering from the College of Agricultural Engineering, Jabalpur India, in the year 2010, MTech in food process engineering from the Indian Institute of Technology (IIT) Kharagpur in the year 2012, and PhD (2015) from the Department of Food Process Engineering, Indian Institute of Technology (IIT) Kharagpur.

Chapter 1
Current Advances in Celiac Disease: Consequences and Improvement Strategies

Chitrangada Das Mukhopadhyay

Abstract Celiac is a chronic enteric disease resulted from an abnormal immune response to gluten proteins in patients having a certain genomic constitution. Tissue transglutaminase enzyme 2 converts the glutamine residues of gluten peptides by deamidation reaction into glutamic acid, which binds to human leukocyte antigen (HLA)-DQ2 or -DQ8 molecules and subsequently evokes T cell responses leading to small intestine inflammation. These events lead to the typical symptoms associated with celiac disease. Also, wheat proteins are rich in proline content and are resistant to human pancreatic and gastric enzymes. Different peptidases from microbial and fungal sources can degrade these incompletely digested peptides. While following a gluten-free diet is the best preventive strategy, a combination therapy by using proteases or carboxypeptidases from microbial sources for gluten detoxification or treatment with tissue transglutaminase inhibitors may also be a good option. Intestinal epithelial cell lines (Caco-2) may be used as *in vitro* model to study trans/paracellular permeability, distortion of intercellular tight junction protein viz., occludin, and ZO-1, and rearrangement of actin filaments.

Keywords Celiac · Tissue transglutaminase · Gluten · Enzyme therapy

1.1 Introduction

Celiac disease is an autoimmune disease caused by an intolerance to gluten proteins (Dewar et al., 2003; Simón et al., 2017; Lo et al., 2003; Cellier et al., 2000; Clemente et al., 2003). This disease develops in genetically predisposed persons, who upon ingestion of gluten-rich food such as products of wheat, rye, barley, etc (Lo et al., 2003). The pathological symptoms include a flattened mucosal layer of small intestinal with uncontrolled epithelial cell proliferation, presence of lymphocytes,

C. D. Mukhopadhyay (✉)
Centre for Healthcare Science and Technology, Indian Institute of Engineering Science and Technology, Shibpur, Howrah, West Bengal, India

N. Singh Deora et al. (eds.), *Challenges and Potential Solutions in Gluten Free Product Development*, Food Engineering Series,
https://doi.org/10.1007/978-3-030-88697-4_1

cryptic hyperplasia, and lesser differentiation of enterocytes (Cellier et al., 2000). This leads to impaired absorptive function of the small intestine. Gluten contains a very high amount of proline (15%) and glutamine (35%) . In celiac patients, this gluten is resistant to normal luminal digestion and becomes toxic to the intestine. The antigenic property of some epitopes on gluten and their role in disease progression has been identified. Any structural alteration to this antigenic potential of the toxic protein may lead to a novel therapeutic pathway. It is now established that induction of the deamidating activity of tissue transglutaminase (tTG) triggers the pathogenic immune responses. Gliadin peptide, a part of gluten protein is a good substrate of tTG. So, enzymatic degradation of the antigenic epitope in toxic peptides coupled with inhibition of tTG activity may become an alternative treatment strategy for CD patients.

Many of the patients with celiac disease are asymptomatic or have mild symptoms. With the advanced diagnostic systems more silent cases are reported which shows that the prevalence of this disease is much more than thought earlier. This autoimmune disease is highly prevalent in Western countries (1% of the total population in European countries) but also diagnosed to be a serious disorder and increasing importance in Northern India. A stringent gluten-free diet (GFD) can help to overcome this enteropathy, however cross-contamination, improper labeling contributes to the immune response in patients. An oral dietary supplement may restore immunological tolerance and represent the ideal cure for celiac disease. In this connection enzymatic hydrolysis of α- gliadin of gluten by proteolytic and amylolytic enzymes coupled with small-molecule inhibition of tTG should be an efficient therapeutic approach (Lorand & Graham, 2003). In this chapter, an overview of the disease, its pathological complexities, detection method, prevention, and therapeutic interventions have been discussed in detail.

1.2 Overview of the Disease

Microscopic analysis of the sections of the inner mucosal layer of human small intestinal from a normal person shows several villi with one layer of columnar epithelial cells having basal nuclei, intraepithelial lymphocytes (IEL), and plasma cells in the lamina propria of the small intestine generally with villous to crypt ratio of 1:5.

On the contrary, the sections of the specimen from the CD patients are characterized by complete loss of villi, plane mucosal layer, having ridges and of the crypt openings onto the luminal surface, appearance of abnormal squamous epithelial cells, an increase in the number of lymphocytes and plasma cells infiltrated in the intraepithelial area and lamina propria, increase in mitoses of crypts. Loss of villi causes decreased surface area for absorption of nutrients in the small intestine and patients to suffer from diarrhea, severe weight loss, iron deficiency led anemia, malnutrition (Clemente et al., 2003; Molberg et al., 2003; Shewry et al., 1992; Lorand & Graham, 2003; Solid, 2000).

1.3 Gluten and Gluten-Free Diet (GFD)

Wheat contains 8–17% protein based on its breeding variety and environmental makeup. The water-insoluble protein fraction of wheat protein constitutes a viscoelastic mass, called gluten. Gluten is a complex protein made up of polymeric and monomeric subunits and constitutes approximately 78–85% of the total wheat protein rich in proline and glutamine. Gluten is responsible for water absorption capacity, baking quality, viscosity, cohesiveness, and elasticity of the dough (Han et al., 2013). Gluten is classified into two main fractions viz., gliadin, which is soluble in aqueous alcohol, and glutenins which are insoluble in the same. Gliadins are monomeric proteins (28–55 KDa) and further subdivided as α/β-, γ- and ω-type based on their primary structure. Glutenins have high (67–88 KDa) or low molecular weight (32–35 KDa) peptides connected by intermolecular disulfide bonds (Khosla, 2017). Gluten proteins are also strongly connected by hydrogen bonds, ionic bonds, and hydrophobic bonds which confer structural and physical properties of the gluten. Glutenins provide elasticity, and gliadins provide viscosity to the gluten complex (Deora, 2017; Daum et al., 1999; Moss et al. 1996; Schuppan et al., 2009; Han et al., 2013). The gliadins having high glutamine and proline content and humans lack endopeptidases to cleave intermolecular bonds between them. The incompletely digested gliadin are immunogenic to CD patients.

1.4 Clinical Symptoms and Possible Mechanism of Villous Atrophy

CD patients manifest mild to severe intestinal inflammation characterized by an increase in intraepithelial lymphocytes, infiltration of mononuclear cells from the subepithelial layer, increase in epithelial mitosis causing total villous atrophy and crypt hyperplasia. Loss of villi is the histopathologic hallmark of celiac disease. This may be due to degradation of intracellular tight junction proteins, caspase-mediated apoptosis, and defect in epithelial regeneration (Kelly et al. 2015; Butterworth & Louis, 2019; De et al., 2017; Palejwala & Watson, 2000). Briefly, the sequence of events starts with the ingestion of gluten proteins by the persons having impaired genetic makeup. HLA-DQ2 and DQ8 heterodimers react with incompletely digested gluten peptides and are presented to CD4+ T cells in the small intestine (Ciccocioppo et al., 2002; Miura et al., 2005; Marsh & Crowe, 1995; Lionetti & Catassi, 2011; Gujral et al. 2012; Kelly et al. 2015). The incompletely digested gluten peptides cross the epithelium by conventional trans paracellular transport and reach the subepithelial region. Activation of macrophages and dendritic cells increased production of IFN I and II and several cytokines in the small intestine subsequently initiates humoral immunity. The release of matrix metalloproteinases can also damage the intestinal mucosa.

1.5 Factors Causing Celiac Disease

1.5.1 Environmental Factors

This disease is stimulated by the consumption of gluten – a protein found in wheat endosperm- or of related proteins found in other grains like rye and barley (Fernandez-Jimenez et al., 2014; Hollon et al., 2015). Abundant gluten protein acts as an environmental trigger. Removal of gluten from daily diet prevents the autoimmune process and again may be restored by the slightest consumption of gluten. Intestinal tight junctions come apart and allow a large amount of incompletely digested gluten into the small intestine. An increase in tissue transglutaminase (tTG) activity acts as an autoantigen. Oats, rice, maize, sorghum, and millet do not activate the celiac disease. The high proline-containing proteins escape proteolytic digestion in the human intestine (Gujral et al., 2012; Moss et al. 1996; Arzu, 2010; Dickson et al., 2006).

1.5.2 Genetic Factors and HLA Class II Genes

CD has a strong association with HLA class II genes on DQ locus (Karell et al., 2003) for the phenotypic expression of disease. HLA-DQ2 or -DQ8 heterodimers are relatively common in white populations. Persons homozygous for DR3 results only DQ2 molecules; but if they are DR3(DR17)/17 heterozygous then 50% of the DQ molecules are DQ2, and, in those who are heterozygous for DR5/7 or DR3 (DR17)/ other, 25% of the DQ molecules are DQ2 (Margaritte et al., 2004; Ehrmann et al., 2003; Augustin et al., 2005). Celiac disease is prevalent in DR3 homozygous or DR3/17 heterozygous genetic makeup.

1.5.3 Binding of DQ2 to Gluten Peptides

DQ2 molecules have active sites for –ve charges residues. They favor left-handed poly-proline II helical configuration, similar to that of gliadins. It has been estimated that there are approximately 50 aa in wheat, 60 aa in the rye, and 35 aa in barley that have the potential to bind with DQ2 or DQ8 and considered to be the candidate sequences for activating celiac disease (Kim et al., 2004).

1.6 Importance of tTG

Human (tTG) is an allosterically regulated enzyme involved in cell signaling blood clotting, wound healing, extracellular matrix formation, and cell adhesion and motility. tTG causes sequence-specific deamidation of dietary gluten peptides in the small intestines of CD patients (Marsh & Crowe, 1995). They are found in intracellular as well as extracellular environments of many organs. The catalytically active form of tTG2 or "open" form can crosslink specific g-glutaminyl and 3-lysine residues on proteins in the presence of calcium and absence of GTP or GDP and forms proteolytically resistant covalent bonds. In the reverse condition, TG2 assumes an inactive/ "closed" form. Human tTG also possesses Ca^{2+}-binding sites, N-terminal fibronectin association site, and guanosine triphosphate hydrolysis site. In celiac sprue, peptides derived from dietary gluten are deamidated by TG2 and enhances their affinity towards MHC, and induces humoral immunity. Inhibition of TG2 activity may be a suitable target for CD therapy.

1.6.1 Modification of Gluten by tTG

The presence of serum antibodies to tissue transglutaminase (tTG) in CD patients confirms its autoimmune response. tTG is the main autoantigen among the antibodies produced by gluten antigen called endomysial antibodies (EMA) (Dieterich et al., 1997). tTG favors the formation of isopeptide bonds between the γ-carboxamide group of glutamine residue of a peptide to the ε-amino group of a lysine residue or other biogenic molecules viz., putrescine, spermidine, spermine, and histamine in low pH. Gluten acts as an exogenous trigger for the production of tTG-specific autoantibodies in CD patients and also generates other antigenic epitopes by cross-linking of gliadin peptides to itself and/or to other peptides leading to autoimmunity.

1.6.2 Role of Intraepithelial Lymphocytes and Muscular Pericytes

An increase in intraepithelial lymphocytes is a characteristic feature of celiac disease. Interleukin (IL)-15 plays a major role and is up-regulated by epithelial cells and cells in the lamina propria in CD patients (Karell et al., 2003; Margaritte-Jeannin et al., 2004; Myrsky et al., 2008; Naluai et al., 2001; Ploski et al., 1993; Louka et al., 2002 ; Mazzarella et al., 2003; Kim et al., 2004; Singh et al., 1995; Liu et al., 2002).

1.6.3 Auto Antibody-Mediated Disease Pathogenesis

The gluten-induced disease-specific autoantibodies might constitute in CD pathogenesis. Angiogenesis commences with the formation of the endothelial tube (Kale et al., 2005; Lorand, 2007). Then mesenchymal cells accumulate surrounding the endothelial tubes and later differentiates into vascular smooth muscle cells or pericytes. Coeliac disease-specific autoantibodies hinder angiogenesis. CD-specific autoantibodies inhibit angiogenesis and disorganize the actin cytoskeleton. Cytoskeletal disarrangement inhibits cell migration, thus the entire vasculature network seen in the small-intestinal mucosa shows abnormality. Overexpression of TG2 in cells leads to reduced cell migration. The autoantibody deposits have been identified around the blood vascular system of the liver causing mild liver in CD patients. Auto-antibodies in the blood vessels of the brain of CD patients suffer from neurological problems and neuroblast apoptosis (Cervio et al., 2007; Pinkas et al., 2007; Lai et al., 2010; Lai et al., 2008; Andringa et al., 2004)

1.7 Detection of Celiac Disease

The diagnosis of CD is very important because delayed diagnosis adversely affects the health and quality of life of patients. The diagnosis of CD requires accurate, sensitive, simple, specific, and cheaper as well-as non-invasive analytical tools (Sollid & Lundin, 2009; Stamnaes et al., 2010; Dieterich et al., 1997; Farre, 2014). CD-specific autoantibody detection in patient serum and saliva may be done using electrochemical, optic-fiber, piezoelectric biosensors, and POC finger-prick methods. CD-specific volatile organic compounds (VOCs) in urine and faeces may also be used as a sample for detection. The most common methods of celiac disease detection are discussed here.

Blood tests are considered the most important. This includes detection of total IgA, tTG IgA, and EMA IgA at first. A biopsy of the small intestine can confirm the findings of the blood test. This is done by endoscopy of the small intestine which is another most reliable detection method. Because the symptoms of celiac disease can be varied, it is often undiagnosed or misdiagnosed. Detection of CD-specific antibodies in blood serum may require additional testing, preferably a DNA test for an accurate diagnosis. Genetic testing to check the presence of DQ2 or DQ8 genes are necessary to diagnose CD.

1.8 Prevention of Celiac Disease

1.8.1 Adherence to GFD

The best option to get rid of CD is adherence to a gluten-free diet life-long. However complete removal of gluten from the daily diet is difficult. Several other grains like maize and rice, soya beans, and starch sources are recommended for a gluten-free diet. Some other options for GFD include tapioca, sorghum, carob, buckwheat, and millet. The main problem related to a gluten-free diet is the poor taste of gluten-free products. Additionally, improper food labels, cross-contamination, and lack of awareness are also related to low compliance. GFD draws social and financial limitations creating a huge impact on family life, workplace. travelling etc (Lerner et al., 2017; Donaldson et al., 2015; Farre, 2014, Guandalini and Rose, 2012, Lequin, 2005). Also, immunological issues are not influenced by diet. The use of a "natural gluten-free diet" offers the greatest compliance and the lowest risk of nutritional imbalance.

1.8.2 Problems in Maintaining a Strict GFD

Gluten is extensively used in the food industry because of its unique viscoelastic properties discussed earlier in this chapter. CD patients are challenged with several issues like insufficient information about the disease, food contamination, and improper food labeling on the packaged food items (Myrsky et al., 2008; Brusca, 2015; Guandalini & Rose, 2012; Hall et al., 2009). Gluten is also present in ice-creams, sweets, confectionary foods, spreads and seasonings, beer, soups and sauces, malted beverages, and many more which need to be avoided. The contamination of food with gluten can occur: during milling, preparation of commercial food products, harvesting, storage, and packaging of grain bags by the farmers (Cervio et al., 2007; Farre, 2017; Lerner et al., 2017). A team approach, including patients, family, physicians, and dietician is required to manage CD patients. After the diagnosis is made, patients should go for nutritional assessment, meal planning, diet education, and counseling with the social and emotional adaptation to the GF lifestyle (García-Manzanares & Lucendo, 2011; Saturni et al., 2010; See & Murray, 2006).

1.8.3 Non-availability of GF (Gluten-Free) Food Products

The number of patients with CD in India is low, thus the commercial production of GFD is very limited. Production of mainly flour and biscuits are being produced but quality control is not done (Green & Cellier, 2007; Di Sabatino & Corazza, 2009;

Zarkadas et al., 2006; Lee et al., 2007). Non-availability of GFD outside their home environment restricts their travel, occupation, and profession. GF food items are considerably more expensive than regular gluten-containing food (J.G. Donaldson, 2015; Butler, 2015; Lequin, 2005; Guandalini et al., 2005; Olsson et al., 2008).

1.9 Counselling of the CD Patients Undergoing GFD

1.9.1 Patient Education and Awareness

The management of CD patients involves education of the patients and their families about the disease and dietary restrictions, timely visits, consistent supervision, and guidance of the nutritionist are important. Also, the disease status should be monitored regularly (Hall et al., 2009; García-Manzanares & Lucendo, 2011; Saturni et al., 2010; Stevens & Rashid, 2008).

1.9.2 CD Management and Counselling by Dieticians

The dietary counselor should have sufficient knowledge about GF food and food products. Prescribing GFD and a specific well-balanced diet is necessary (Stevens & Rashid, 2008; Olsson et al., 2008). The dietician is the most qualified health care professional to provide nutrition therapy. They have extensive academic and practical experience including in-depth knowledge of nutrition, nutritional needs, nutrition composition, and food preparation information, and educational factors that affect the food and nutrition behavior of people (Nasr et al., 2012; Niewinski, 2008).

1.9.3 Celiac Disease Support Groups

Celiac disease support groups provide a platform for patients to discuss their problems amongst themselves and learn from each other, provides information about GF products and their availability, but also can act as an advocacy for gluten labeling and other issues to the Government and regulatory bodies (Green & Cellier, 2007; Di Sabatino & Corazza, 2009; Zarkadas et al., 2006; Lee et al., 2007). Furthermore, it has been observed that the adherence to a GFD increases when individuals are members of a patient support group (Addolorato et al., 2004; Silvester & Rashid, 2010; Leffler et al., 2008 ; Catassi et al., 2007).