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A REVIEW THE RELATIONSHIP BETWEEN CELIAC DISEASE AND LMW GS IN WHEAT OF TURKEY

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ABSTRACT

Gluten enteropathy (Celiac Disease) is an autoimmune and food-borne proximal small bowel disease that develops as a permanent intolerance to gluten-like cereal proteins. Our country is 67 683 CD according to recent data from the Ministry of Health Information System, Sivas and Tokat, respectively; 272, 394 live in the CD. Gluten enteropathy; hypokalemia, hypomagnesemia, hypocalcemia, hypoproteinemia, severe diarrhea resulting in lack of iron and folic acid can lead to the clinical picture characterized by dehydration, and metabolic changes. In a range of clinical gluten or gluten-like proteins take by mouth, combinations of HLA molecules resulting gliadin peptides with the small intestinal mucosa occurs. The strong humoral and cytotoxic cellular response of IgA to glutamine-forming gliadin occurs and the gliadin molecule is toxic. It is important to detect antigliadin antibody, anti-tissue transglutaminase antibody and/or anti-endomicin antibody in the celiac epithelium. Levels of these antibodies decline with a strict gluten-free diet or are expected to disappear completely. Glyadines in wheat varieties; sulfur-rich (alpha, beta, gamma) and sulfur-poor (omega) as they fall into two groups. In this study, some bread wheat cultivars were investigated with *Lmw Gs* of alpha, beta, gamma gliadins which are harmful to celiac disease and especially bread wheat cultivars with less alpha and beta gliadin bands were detected.

The identification of wheat cultivars with fewer gliadin bands is important for breeders to develop wheat gliadin free from celiac disease.

KEYWORDS: Celiac, Gliadin, Intestinal, Clinical Case, Molecular Weight

I. INTRODUCTION

Celiac disease; is an autoimmune enteropathy known as persistent intolerance to gluten-like proteins, caused by inherited and environmental factors (1,2). It is estimated that approximately 0.6-1% of the world population exists (2,3). Acquired and innate immune response in etiopathogenesis is accused. In the acquired immune response, the tissue transglutaminase enzyme (TTG) commonly found in our bodies is active. tTG deamines by loading gluten peptides (-) and allows the binding of deacetylated gluten peptides to the DQ2 and DQ8 proteins of HLA on the antigen presenting cell surface. As a result, the T helper cells become active and produce cytokines. Eventually, the inflammation takes place and in response, the plasma cells in the intestinal tissue secrete anti-gliadin and anti-endomysial antibodies and autoimmune antibodies, TTGs. In the natural immune response, intraepithelial CD8 + is the natural response associated with cytotoxic T lymphocytes. Gluten peptides cause IL-15 overproduction, in particular. In response, NK-like properties are imparted to T-cells by increasing expression of NK receptors. Eventually, the activated T-cells come to intestinal epithelial cells and they attack and cause intestinal damage (6,8). A gluten-free diet is the basic principle in treatment, gluten intake 50 mg/day is sufficient for mucosal damage (9). Many studies have attempted to elicit non-HLA-based factors by identifying genetic factors and polymorphisms that affect the formation of the disease in diverse populations. Although there have been 12 non-HLA-based CELIAC loci so far identified, most of them are still controversial and open to investigate the exact relationship with celiac disease (10).

Cereals, which are continuously improving in quality in the direction of increasing demands, are essential in meeting consumer needs in terms of nutrition. Grain types in the world include wheat; in terms of planting area and production amount (11). In this context, the quality of the product which has a wide range of production is

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also important for the consumer. The amount of protein in wheat and the quality of protein and gluten are important in terms of the quality of the product to be produced. Gluten quality in bread wheat; It is known that the effect of high molecular weight gluten subunits (HMW-GS) is high in product volume, visco-elasticity, and dough (12,13). After the determination of the relation of HMW-GS bands with dough and bread quality in bread wheat varieties, important studies have been carried out by genetic structure, protein density, functional associations and protein potential development (14,15,16).

In this context, they have also been supported by different breeds of barley, such as barley, to determine their chromosomal location. Hor3 locus located on chromosome 5 in barley is associated with the structurally wheat HMW-GS (14,17). Genes that control the endosperm proteins in bread wheat; HMW, LMW, and gliadin are separated into three parts. HMW-GS controlling genes; On the long arms of 1A, 1B, 1D chromosomes, LMW controlling genes are located on the short arms. The genes controlling gliadins are; 6A, 6B, 6D in the short arms of the chromosomes and in the C and D subgroups of the LMW-GS (14, 18). A plurality of common wheat HMW-GS in the endosperm, and due to the great heterogeneity in the structure of LMW-GS was characterized further (18). Gluten; gliadins and glutenins are divided into two sub-fractions. Glutenins are attached to their subunits by di-sulfide bonds and are in polymer form (19). Gliadins are toxic, especially for celiac disease individuals (alpha, beta, gamma). Amino acids are synthesized from the corresponding chromosomes (genes that control particular gliadin) is extremely important for human nutrition and celiac individuals. Recently, a computer modeling system has been used to accurately determine the HMW-GS and LMW-GS molecule properties.

Researchers working on determining exact positions of various cross-links have reported that they have not yet identified relevant disulfide-linked peptides (20,21,22). The computer modeling system will help identify protein concentrations, identify proteins synthesized from primers in genomes, and explain the linkage preferences of S-S bonds between subunits (22).

II. METHODOLOGY

Taking clinical gluten or gluten-like proteins results in a strong humoral and cytotoxic cellular response to IgA-induced glutathione-forming gliadin, and the gliadin molecule is toxic. It is important to detect antigliadin, anti-tissue transglutaminase and/or anti-endomicin antibodies in the presence of GE. With a tight gluten-free diet, these antibody levels are expected to fall or disappear completely. GE is confronted not only with findings of the gastrointestinal system but also with renal, endocrine, metabolic, neurological and psychological problems. Since most patients can be diagnosed late, these problems cause serious mortality and morbidity. Early diagnosis and life-long diet are very important in terms of disease progression.

III. RESULTS

-While GE is a disease until diagnosis, it comes out as a life style after diagnosis.
-It is known that, in addition to alpha, beta, gamma gliadins, which are known to have toxic effects in bread wheat varieties, C and D subunits of LMW-GS bands are caused by gluten enteropathy.

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