Beyond the Gluten-Free Diet: Emerging Diagnostics and Therapies in Celiac Disease”

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Abstract

Celiac disease (CD) is a chronic, immune-mediated enteropathy precipitated by dietary gluten in genetically susceptible individuals. Historically managed through strict adherence to a lifelong gluten-free diet (GFD), CD treatment faces limitations due to inadvertent gluten exposure and challenges with full compliance. Recent research has advanced the therapeutic landscape beyond dietary measures, exploring pharmacologic interventions, immunomodulatory strategies, AI-enhanced diagnostics, and personalized approaches. Larazotide acetate, a tight-junction regulator, has demonstrated efficacy in phase II clinical trials by attenuating gluten-induced gastrointestinal symptoms, although its larger phase III evaluation was discontinued due to underpowered endpoints (Leffler et al., 2012; BeyondCeliac.org, 2022). Innovations such as inverse vaccines—digital nanoparticle-based agents—have shown promise in inducing immune tolerance, with early human trials indicating protection against gluten-mediated mucosal injury (NY Post, 2025). Engineered regulatory T cells (eTregs) that express gluten-specific T-cell receptors have achieved antigen-specific immunosuppression in preclinical HLA-DQ2.5 mouse models, opening the door to cell-based therapies (Wikipedia, 2025). Concurrently, artificial intelligence (AI) platforms analyzing duodenal biopsies have achieved pathologist-level accuracy (>95%) within minutes, offering potential for more timely and consistent diagnosis (Wei et al., 2019; The Guardian, 2025). Complementary efforts in microbiome modulation and epigenetics are also underway to understand and possibly prevent disease activation. Future priorities include conducting large-scale, prospective clinical trials to validate these therapies, integrating genomic risk profiling and AI diagnostics into routine care, and expanding access through population-tailored screening and equitable global implementation. Together, these

multidimensional strategies promise to transform CD management from a diet-exclusive model to one of precision medicine and therapeutic versatility.

Keywords: Gluten-free Diet (GFD), Larazotide Acetate, Histopathology, Microbiome Modulation

Introduction

Celiac disease (CD) is a chronic, immune-mediated disorder triggered by the ingestion of gluten in genetically predisposed individuals. It leads to inflammation and damage of the small intestine's mucosal lining, resulting in malabsorption of nutrients and a spectrum of gastrointestinal and extra-intestinal manifestations. The global prevalence of CD varies, with seroprevalence estimated at **1.4%** and biopsy-confirmed prevalence at **0.7%**. However, a significant proportion of cases remain undiagnosed, particularly in regions with limited access to healthcare.

The pathogenesis of CD involves a complex interplay between genetic susceptibility and environmental factors. The majority of individuals with CD possess the **HLA-DQ2** or **HLA-DQ8** alleles, which are essential for the development of the disease. Environmental triggers, such as the timing and amount of gluten introduction during infancy, gastrointestinal infections, and alterations in the gut microbiota, may influence disease onset and progression [1-2].

Clinically, CD presents with a wide range of symptoms, from classic gastrointestinal complaints like diarrhea and abdominal pain to extra-intestinal manifestations such as anemia, osteoporosis, infertility, and neurological disorders. Notably, many individuals remain asymptomatic or present with atypical symptoms, leading to under diagnosis [2].Diagnosis of CD is based on a combination of serological tests, histological examination of duodenal biopsies, and the presence of specific genetic markers. The gold standard for diagnosis remains the demonstration of villous atrophy on biopsy specimens obtained from the duodenum. Serological markers, including anti-tissue transglutaminase (tTG) and anti-endomysial antibodies (EMA), are commonly used as initial screening tools.

The only effective treatment for CD is a strict, lifelong gluten-free diet (GFD), which leads to mucosal healing and symptom resolution in the majority of patients. Adherence to the GFD is

crucial, as even small amounts of gluten can trigger an immune response and cause intestinal damage.

Recent advancements in artificial intelligence (AI) have shown promise in improving the diagnosis of CD. AI algorithms trained on large datasets of duodenal biopsy images have demonstrated the ability to accurately classify CD and differentiate it from other gastrointestinal disorders, potentially reducing diagnostic time and improving accuracy [3].

Global Prevalence

In Europe, CD is relatively common, with prevalence rates ranging from **0.6% to 1.2%**. For instance, Finland reports a prevalence of **2%**, while Germany has a rate of **0.3%**. The United States has a prevalence of approximately **0.75%**, with higher rates observed in individuals with gastrointestinal symptoms or a family history of CD. In Asia, the prevalence of CD is generally lower but varies significantly across countries. In South Asia, India has a prevalence of **0.7%**, with higher rates in the northern regions compared to the south. Southeast Asian countries like Vietnam and Malaysia report seroprevalence rates of **1%** and **1.2%**, respectively. In East Asia, China shows a prevalence of **2.19%** among adolescents and young adults, while Japan has a lower prevalence compared to other parts of Asia (Anywhere Gluten Free, 2024).The Middle East and North Africa exhibit varying prevalence rates. Saudi Arabia reports a prevalence ranging from **1.5% to 3%**, while Iran has a seroprevalence of **3%** and a biopsy-confirmed prevalence of **2%**. Other countries in the region, such as Lebanon, Turkey, and Egypt, have reported prevalence rates between **0.5% and 1.5%** [4-5].

Data on CD prevalence in Sub-Saharan Africa are limited. However, studies suggest that CD is less common in this region compared to Europe and North America. The lower prevalence may be attributed to genetic factors, dietary patterns, and limited diagnostic facilities (Ziberna et al., CD affects both genders, but studies indicate a higher prevalence in females. For example, in Europe, the prevalence is **0.6% in females** compared to **0.4% in males**. In the United States, the prevalence is approximately **0.75%**, with higher rates observed in individuals with gastrointestinal symptoms or a family history of CD.

Children are more frequently diagnosed with CD than adults. In Europe, the prevalence in children is approximately **0.9%**, while in adults, it is about **0.5%**. The higher prevalence in children may be due to more frequent screening and the manifestation of symptoms at an earlier age [6].

Genetics and Pathogenesis of Celiac Disease

Genetic Factors

Celiac disease (CD) is a complex autoimmune disorder with a strong genetic component. The primary genetic risk factors are associated with the human leukocyte antigen (HLA) class II molecules, specifically HLA-DQ2 and HLA-DQ8.

- **HLA-DQ2**: Approximately 95% of individuals with CD express HLA-DQ2, which is encoded by the DQA1*05 and DQB1*02 alleles. This allele is often inherited in a cis-configuration, known as DQ2.5, and is strongly associated with CD susceptibility.
- **HLA-DQ8**: The remaining 5% of CD patients express HLA-DQ8, encoded by the DQA1*03 and DQB1*03:02 alleles. This allele is also associated with CD but to a lesser extent than HLA-DQ2.

While these HLA alleles are necessary for the development of CD, they are not sufficient on their own. Approximately 30% of the general population carries HLA-DQ2 or HLA-DQ8 alleles, yet only a small percentage develops CD, indicating the involvement of other genetic and environmental factors.

Non-HLA Genetic Factors

Beyond HLA genes, several non-HLA loci have been identified that contribute to CD susceptibility. Genome-wide association studies (GWAS) have pinpointed over 40 additional risk loci, including genes involved in immune regulation, epithelial cell function, and tissue repair [7-8].

These loci include genes such as IL2, IL21, CTLA4, and SH2B3, which play roles in T-cell signaling, immune tolerance, and inflammation. The presence of specific variants in these genes can modulate the immune response to gluten and influence the risk of developing CD [9-10].

Pathogenesis

The pathogenesis of CD involves a complex interplay between genetic susceptibility, environmental factors (primarily gluten ingestion), and immune system dysregulation.

1. **Gluten Ingestion and Processing**: In genetically predisposed individuals, the ingestion of gluten leads to the release of gliadin peptides in the small intestine. These peptides are resistant to enzymatic digestion and can cross the intestinal epithelium [11-12].
2. **Deamidation by Tissue Transglutaminase (tTG)**: Once inside the intestinal mucosa, gliadin peptides are modified by the enzyme tTG, which deamidates specific glutamine residues. This modification increases the affinity of the peptides for HLA-DQ2 and HLA-DQ8 molecules on antigen-presenting cells (APCs).
3. **Activation of T-helper Cells**: The deamidated peptides bind to HLA-DQ2/DQ8 molecules on APCs, leading to the activation of gluten-specific CD4+ T-helper cells. These activated T-cells release pro-inflammatory cytokines, such as interferon-gamma (IFN-γ) and interleukin-21 (IL-21), which contribute to the inflammatory response in the intestinal mucosa.
4. **Intestinal Damage**: The inflammatory cytokines promote the recruitment and activation of intraepithelial lymphocytes (IELs) and B-cells, leading to the production of anti-tTG antibodies. These immune responses result in villous atrophy, crypt hyperplasia, and increased intestinal permeability, hallmark features of CD.
5. **Chronic Inflammation and Mucosal Healing**: Persistent gluten exposure in individuals with CD leads to chronic inflammation and ongoing damage to the intestinal mucosa. The only effective treatment is a strict, lifelong gluten-free diet, which can lead to mucosal healing and symptom resolution in the majority of patients[13-17].

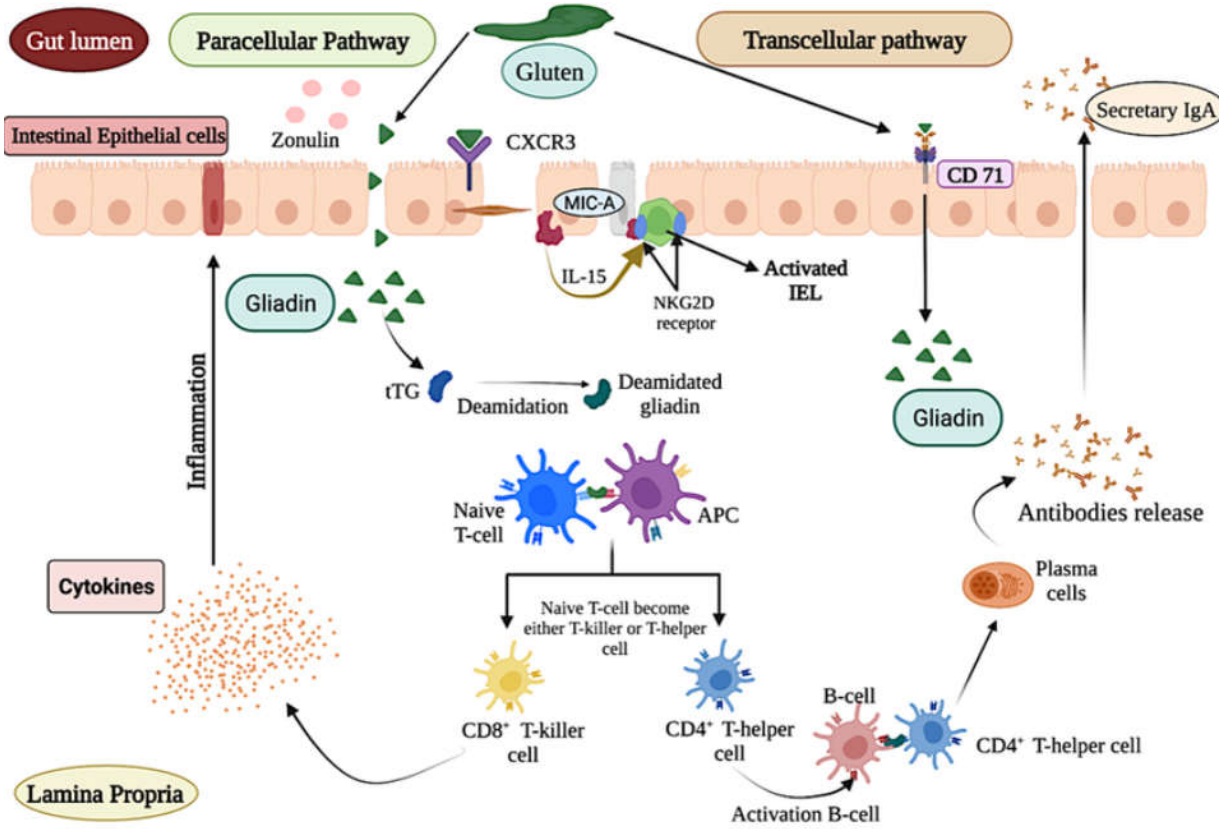

Fig. Pathogenesis of Celiac Disease

Diagnosis of Celiac Disease

Celiac disease (CD) is a chronic autoimmune disorder characterized by an inappropriate immune response to dietary gluten in genetically predisposed individuals. Diagnosing CD can be challenging due to its diverse clinical manifestations and the overlap with other gastrointestinal disorders. A comprehensive diagnostic approach combining clinical evaluation, serological testing, histological examination, and, when necessary, genetic testing is essential for accurate diagnosis [18-20].

Clinical Evaluation

The initial step in diagnosing CD involves a thorough clinical assessment, including a detailed patient history and physical examination. Symptoms of CD can vary widely, ranging from gastrointestinal manifestations such as diarrhea, abdominal pain, and bloating, to extra-intestinal symptoms like fatigue, anemia, and dermatitis herpetiformis. A high index of suspicion is crucial, especially in individuals with risk factors such as a family history of CD or associated autoimmune conditions.

Serological Testing

Serological tests serve as the first-line diagnostic tool for CD. The most commonly used antibodies include:

- **Anti-tissue transglutaminase (tTG) IgA**: This test has high sensitivity and specificity for CD and is considered the most reliable initial screening test.
- **Anti-endomysial antibodies (EMA) IgA**: While highly specific, the EMA test is more labor-intensive and less widely available than the tTG test.
- **Deamidated gliadin peptide (DGP) antibodies IgA and IgG**: These are particularly useful in patients with IgA deficiency or those who are young, as tTG and EMA tests may be less reliable in these populations. It is important that patients do not initiate a gluten-free diet before testing, as this can lead to false-negative results [21-24].

Histological Examination

If serological tests are positive, the next step is a duodenal biopsy to assess for villous atrophy, crypt hyperplasia, and increased intraepithelial lymphocytes, which are indicative of CD. The Modified Marsh criteria are commonly used to grade the histological findings.

In certain cases, particularly in children with high levels of tTG antibodies, a biopsy may be unnecessary if the clinical presentation is typical and other causes are excluded.

Genetic Testing

Genetic testing for HLA-DQ2 and HLA-DQ8 alleles can support the diagnosis of CD. While the presence of these alleles is necessary for the development of CD, their absence makes the diagnosis highly unlikely [25-26].

Management of Celiac Disease

Celiac disease (CD) is a chronic autoimmune disorder characterized by inflammation and damage to the small intestine upon ingestion of gluten in genetically predisposed individuals. The cornerstone of managing CD is the strict and lifelong adherence to a gluten-free diet (GFD). This approach not only alleviates symptoms but also promotes mucosal healing and prevents long-term complications. However, effective management extends beyond dietary modifications and encompasses nutritional support, monitoring, and, in certain cases, pharmacological interventions [27-28].

1. Gluten-Free Diet (GFD)

The primary treatment for CD is the elimination of all sources of gluten, a protein found in wheat, barley, and rye. Adherence to a GFD leads to the resolution of symptoms and mucosal healing in the majority of patients. It is essential to avoid even trace amounts of gluten, as inadvertent ingestion can trigger immune responses and mucosal damage. Patients should be educated on reading food labels, identifying hidden sources of gluten, and preventing cross-contamination in food preparation areas. Consultation with a dietitian specializing in CD is recommended to ensure nutritional adequacy and adherence to the GFD [29].

2. Nutritional Support and Monitoring

Individuals with CD are at risk for various nutritional deficiencies due to malabsorption, including deficiencies in iron, folate, vitamin B12, calcium, vitamin D, and fiber. Therefore,

regular monitoring of nutritional status is crucial. Supplementation may be necessary to correct deficiencies, and dietary counseling should focus on incorporating nutrient-dense, naturally gluten-free foods. Fortified gluten-free products can help meet the recommended intake of essential nutrients. Additionally, patients should be monitored for potential complications such as osteoporosis, anemia, and growth disturbances in children [30].

3. Monitoring and Follow-Up

Regular follow-up is essential to assess adherence to the GFD, monitor for persistent or recurrent symptoms, and detect complications. This includes clinical evaluation, serological testing for celiac-specific antibodies, and, if necessary, repeat duodenal biopsies to assess mucosal healing. In cases of non-response or relapse, a thorough review of the dietary history, potential sources of gluten contamination, and evaluation for other conditions such as refractory CD or enteropathy-associated T-cell lymphoma is warranted [31].

4. Pharmacological Interventions

While a GFD remains the cornerstone of CD management, pharmacological treatments are being explored, particularly for patients with refractory CD or those who cannot maintain a strict GFD. Larazotide acetate, an investigational drug, has shown promise in clinical trials by modulating intestinal permeability and reducing the immune response to gluten. However, it is not yet approved for routine clinical use. Other therapeutic agents, including corticosteroids and immunosuppressive drugs, may be considered in specific cases but are not standard treatments [32].

5. Patient Education and Support

Effective management of CD requires comprehensive patient education and support. Patients should be informed about the nature of the disease, the importance of strict adherence to a GFD, and strategies to avoid gluten exposure. Support groups and counseling can provide emotional support and practical advice for coping with the challenges of living with CD. Family involvement is also crucial, as they play a significant role in meal preparation and ensuring a gluten-free environment [33].

Future Directions in Celiac Disease Research and Management

Celiac disease (CD) is a chronic autoimmune disorder triggered by the ingestion of gluten in genetically predisposed individuals. Currently, the only effective treatment is a strict, lifelong gluten-free diet (GFD). However, this approach presents challenges due to accidental gluten exposure and dietary adherence issues. Recent advancements in research are paving the way for novel therapeutic strategies and improved diagnostic tools.

1. Pharmacological Therapies

While the GFD remains the cornerstone of CD management, pharmacological interventions are being explored to complement dietary restrictions. These therapies aim to mitigate immune responses to gluten and promote mucosal healing.

- **Larazotide Acetate**: This investigational drug acts as a tight junction regulator, preventing the intestinal permeability induced by gluten peptides. Clinical trials have demonstrated its potential to reduce symptoms and intestinal damage associated with gluten exposure.
- **IMU-856**: Developed by Immunic, this small-molecule drug promotes gut renewal without suppressing the immune system. Early-stage trials have shown it to be safe and effective in improving nutrient absorption and reducing symptoms in CD patients.
- **Inverse Vaccines**: A novel approach involves "inverse vaccines," which retrain the immune system to tolerate gluten. These vaccines use synthetic nanoparticles to mimic natural cell death, instructing the immune system not to react to gluten. Early human trials have shown promising results in preventing gut damage after gluten ingestion.

2. Diagnostic Innovations

Advancements in diagnostic technologies are enhancing the accuracy and efficiency of CD detection

- **Artificial Intelligence (AI) in Histopathology**: Researchers at the University of Cambridge have developed an AI tool that analyzes duodenal biopsy images to diagnose

CD. Trained on over 4,000 images, the AI demonstrated accuracy comparable to pathologists, significantly reducing diagnosis time.

- **Deep Learning Models**: A deep learning approach has been employed to automate the detection of CD in duodenal biopsy slides. The model achieved high accuracy in identifying CD, normal tissue, and nonspecific duodenitis, offering a promising tool for pathologists.

3. Microbiome and Epigenetics

The gut microbiome and epigenetic factors are increasingly recognized for their roles in CD pathogenesis.

- **Microbiome Modulation**: Research is exploring how specific microbial compositions influence gluten metabolism and immune responses. Strategies like probiotics, prebiotics, and fecal microbiota transplantation are being investigated to restore a balanced microbiome and promote tolerance.
- **Epigenetic Modifications**: Environmental factors such as diet and stress can lead to epigenetic changes that affect immune system regulation. Understanding these modifications may provide insights into disease onset and potential therapeutic targets.

4. Personalized Medicine

Advancements in genomics and predictive analytics are facilitating personalized approaches to CD management.

- **Genetic Profiling**: Identifying genetic risk factors can help predict disease susceptibility and inform individualized treatment plans. Predictive genomics tools are being developed to assess lifetime risk and guide clinical decisions.
- **Tailored Therapies**: Personalized medicine approaches aim to customize treatments based on individual genetic, environmental, and lifestyle factors, optimizing efficacy and minimizing adverse effects.

5. Global Health Initiatives

Efforts to improve CD awareness and diagnosis are essential, especially in regions with limited healthcare resources.

- **Educational Programs**: Increasing awareness among healthcare providers and the public about CD symptoms and risk factors can lead to earlier diagnosis and better management.
- **Screening Initiatives**: Implementing screening programs in at-risk populations can facilitate early detection and intervention, reducing the burden of undiagnosed CD [34-37].

Conclusion:

Celiac disease is a multifaceted autoimmune disorder presenting diverse clinical profiles. Advances in diagnostics (serology, AI pathology), strict GFD adherence, and emerging therapeutics (larazotide, enzymes, eTregs, biologics) promise improved outcomes. Key future priorities include large-scale prospective trials, population-tailored screening, technology-enabled diagnostics, and equitable global care. The next decade in celiac disease care is poised for disruption. Pharmacologic agents offer a buffer against dietary slips, cellular therapies hold promise for long-term immune resetting, AI is revolutionizing diagnostic speed and precision, and microbiome-epigenetic research is charting new territory. However, these advances must be validated through rigorously designed Phase III trials, accessible digital infrastructure, and inclusive public health policies to benefit **all** individuals affected by celiac disease.

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