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Nutritional Deficiencies and Psychosocial Burden in Children with Celiac Disease: A Comprehensive Review

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Abstract

Celiac disease is a chronic immune-mediated disorder triggered by gluten ingestion and is increasingly recognized in pediatric populations worldwide. In children, the disease extends beyond gastrointestinal symptoms, significantly affecting nutritional status, growth, and psychosocial well-being. The objective of this review is to critically examine the epidemiology and pathophysiology of celiac disease in children, with particular emphasis on nutritional deficiencies, challenges associated with adherence to a gluten-free diet, and the psychosocial burden experienced by affected children and their families.

In conclusion, although a strict gluten-free diet remains the cornerstone of management and improves clinical outcomes, persistent nutritional inadequacies and psychosocial challenges are common. A multidisciplinary approach incorporating nutritional monitoring, dietary counseling, psychological support, and policy-level interventions is essential to improve longterm health outcomes and quality of life in children with celiac disease.

Keywords

Celiac disease**,** Children**,** Gluten-free diet**,** Nutritional deficiencies**,** Psychosocial burden

1.Introduction

Celiac disease (CD) is an immune-mediated systemic disorder induced by the ingestion of gluten and related prolamins in genetically predisposed individuals. It primarily affects the small intestine, leading to villous atrophy, crypt hyperplasia, and subsequent malabsorption of nutrients (Green & Cellier, 2015). While historically considered a rare gastrointestinal disorder confined to Europe, CD is now recognized as a global health concern, affecting both children and adults across diverse ethnic groups (Singh et al., 2018). Its clinical spectrum ranges from classical gastrointestinal symptoms such as diarrhea, abdominal pain, and bloating to extraintestinal manifestations including anemia, short stature, delayed puberty, dermatitis herpetiformis, and neuropsychiatric complications (Auricchio et al., 2018; Freeman, 2015).

In pediatric populations, CD carries particularly profound implications. Children are in a critical phase of growth and development, and undiagnosed or poorly managed CD can lead to irreversible consequences such as impaired linear growth, low bone mineral density, and longterm micronutrient deficiencies (Borghini, Puzzono, & Rosato, 2018; Wierdsma & van Bokhorst-de van der Schueren, 2013). Moreover, children with CD face unique psychosocial challenges: they must adapt to a strict lifelong gluten-free diet (GFD), which can lead to feelings of exclusion in social contexts, anxiety about food safety, and a reduced overall quality of life (Canova et al., 2018; van Doorn & van Hoogstraten, 2017).

The prevalence of CD in children has risen substantially in recent decades, in part due to improved awareness, more sensitive diagnostic testing, and updated guidelines from professional societies such as the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) (Husby et al., 2012; Murch et al., 2013). Nevertheless, significant gaps remain in identifying asymptomatic or atypical cases, particularly in resourcelimited settings where serologic and histologic tools are less accessible (Abu Daya et al., 2019).

From a pathophysiological standpoint, CD results from a complex interplay between environmental exposure to gluten, genetic predisposition (HLA-DQ2 and HLA-DQ8 haplotypes), and immune dysregulation (Ludvigsson, Leffler, & Bai, 2013). Gluten peptides resistant to gastrointestinal proteolysis trigger an adaptive immune response, leading to inflammation, mucosal damage, and systemic effects (Bascunan et al., 2018). While these mechanisms are increasingly well understood, the wide variability in clinical expression— ranging from silent disease to severe malabsorptive syndromes—remains a challenge for clinicians and researchers alike (Leonard et al., 2017).

The cornerstone of CD management is strict, lifelong adherence to a GFD, which can reverse mucosal damage, normalize growth parameters, and restore quality of life (Elli et al., 2016; Leonard et al., 2019). However, maintaining adherence is especially difficult in children, given social pressures, limited availability of gluten-free products, and the often inferior nutritional quality of such foods (Jordan, Li, & Magder, 2015; Biagi, Campanella, & Corazza, 2015). Gluten-free products are frequently lower in fiber, iron, and B vitamins while being higher in fat and sugar, posing additional dietary risks (Casella et al., 2018; Elliott, 2018).

Beyond physical health, the psychosocial burden of CD in children is increasingly recognized as a central component of disease management. Studies demonstrate higher rates of anxiety, depression, and reduced social participation among children with CD compared to their healthy peers (Pico & Spirito, 2018; Shulman & Logan, 2009). These burdens often persist despite adherence to a GFD, underscoring the importance of holistic care approaches that address not only medical but also psychological and social well-being (Addolorato et al., 2001; Villela Nogueira et al., 2014).

The purpose of this review is to provide a comprehensive synthesis of the current literature on CD in children, with particular focus on four interrelated dimensions: (1) epidemiology and pathophysiology, (2) nutritional deficiencies, (3) dietary management with a GFD, and (4) psychosocial burden. We will also highlight research gaps and propose future directions to advance clinical care and improve outcomes for pediatric patients. By integrating findings from 43 peer-reviewed studies spanning clinical guidelines, epidemiologic surveys, nutritional assessments, and psychosocial analyses, this review aims to present a holistic picture of CD in children that is both evidence-based and clinically relevant.

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2.Epidemiology and Pathophysiology of Celiac Disease 2.1Epidemiology

Celiac disease (CD) is one of the most common chronic autoimmune disorders in children, with a prevalence that has increased substantially over the past several decades. Once regarded as a rare condition primarily confined to individuals of European ancestry, CD is now understood to be a global public health concern affecting children on every continent (Singh et al., 2018). A systematic review and meta-analysis estimated the global prevalence of CD at approximately 1% of the population, though rates vary significantly across regions depending on genetic, environmental, and diagnostic factors (Singh et al., 2018).

The incidence of CD in children is influenced by multiple factors including genetics, timing of gluten introduction, breastfeeding duration, infections, and geographic differences in dietary practices (Fasano & Catassi, 2012; Gujral et al., 2019). Notably, screening studies in Europe and North America consistently report prevalence rates around 1%, whereas lower rates are observed in East Asia and sub-Saharan Africa, likely reflecting both genetic variability and underdiagnosis (Green & Cellier, 2015).

Several population-based studies underscore the increasing recognition of CD in children. In Sweden, a marked rise in diagnosed cases was observed during the so-called "celiac epidemic" of the 1980s–1990s, linked to early introduction of gluten and changes in infant feeding practices (Murch et al., 2013). Similarly, in the United States, the prevalence of CD among children doubled between 1975 and 2000, suggesting not only greater awareness but also a true rise in incidence (Lebwohl et al., 2018).

A key challenge in epidemiology is the high proportion of undiagnosed cases. It is estimated that up to 70–80% of children with CD remain undiagnosed, especially those presenting with atypical or extraintestinal symptoms (Freeman, 2015; Abu Daya et al., 2019). Undiagnosed CD carries long-term risks including growth failure, osteoporosis, infertility, and increased risk of intestinal lymphoma (Leonard et al., 2019).

2.2Risk Factors

Several **risk groups** show a higher prevalence of CD compared to the general population:

- **First-degree relatives** of CD patients: prevalence ranges from 5% to 15% (Husby et al., 2012).
- **Children with type 1 diabetes mellitus**: prevalence ranges from 3% to 16% (Auricchio et al., 2018).
- **Down syndrome and Turner syndrome**: both conditions show markedly higher CD prevalence (Murch et al., 2013).
- **Selective IgA deficiency**: strongly associated with increased CD risk (Green & Cellier, 2015).

Recognizing these high-risk groups has led to targeted screening protocols recommended by professional societies such as ESPGHAN and NASPGHAN (Husby et al., 2012; Hill et al., 2005).

2.3Gender Differences

Epidemiological studies consistently demonstrate a **female predominance** in CD, with ratios ranging from 2:1 to 3:1 (Leonard et al., 2017). This sex difference parallels other autoimmune diseases, suggesting hormonal and immunological factors may contribute to susceptibility (Ludvigsson et al., 2013).

2.4Geographic and Ethnic Variations

Although CD was historically thought to affect primarily Caucasians of Northern European descent, recent data reveal that CD is present in populations worldwide, including the Middle East, South America, India, and North Africa (Singh et al., 2018). The variability across populations is partly explained by differences in **HLA genotype frequencies** (DQ2/DQ8), dietary gluten exposure, and the robustness of healthcare systems for diagnosis (Fasano & Catassi, 2012).

3.Pathophysiology

CD pathophysiology reflects a complex interaction between **genetic predisposition, environmental exposure, and immune dysregulation**.

3.1Genetic Susceptibility

Genetic factors are central to CD pathogenesis. More than 90% of patients carry the **HLADQ2 haplotype**, and most of the remainder carry **HLA-DQ8** (Ludvigsson et al., 2013). While these alleles are necessary for disease development, they are not sufficient, as approximately 30– 40% of the general population carry these alleles but only ~1% develop CD (Leonard et al., 2019). This underscores the importance of **non-HLA genes** and environmental triggers. Genome-wide association studies (GWAS) have identified more than 40 non-HLA loci associated with immune regulation in CD, including genes related to T-cell activation and cytokine signaling (Bascunan et al., 2018).

3.2Gluten as an Environmental Trigger

Gluten is a composite of storage proteins found in wheat, barley, and rye. The **gliadin and glutenin fractions** of gluten are resistant to complete digestion in the human gastrointestinal tract, resulting in peptides that can trigger an immune response (Elli et al., 2019). In genetically predisposed individuals, these peptides are deamidated by the enzyme **tissue transglutaminase (tTG)**, increasing their binding affinity to HLA-DQ2/DQ8 molecules on antigen-presenting cells (Auricchio et al., 2018). This initiates a cascade of adaptive immune responses involving **CD4+ T cells**, proinflammatory cytokine release, and B-cell activation leading to autoantibody production (Green & Cellier, 2015).

3.3Innate and Adaptive Immune Responses

The pathogenesis of CD involves both **innate and adaptive immunity**. On the innate side, gluten peptides stimulate enterocytes and innate immune cells to release **interleukin-15 (IL15)**, which activates intraepithelial lymphocytes (IELs) and contributes to epithelial cell damage (De Palma et al., 2016). Adaptive immunity, mediated by gluten-specific CD4+ T cells, drives chronic intestinal inflammation and villous atrophy (Bascunan et al., 2018).

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The production of **autoantibodies against tissue transglutaminase (tTG), endomysium, and deamidated gliadin peptides** serves as both a diagnostic marker and a reflection of disease activity (Husby et al., 2012).

3.4Mucosal Damage and Malabsorption

The hallmark histological features of CD include **villous atrophy, crypt hyperplasia, and increased IELs** (Murch et al., 2013). These changes impair the absorptive surface area of the small intestine, leading to malabsorption of macronutrients and micronutrients such as iron, folate, calcium, and vitamin D (Borghini et al., 2018; Wierdsma & van Bokhorst-de van der Schueren, 2013). In children, this manifests as growth failure, delayed puberty, anemia, and reduced bone mineral density (Shamir et al., 2010; Tapsas et al., 2016).

3.5Extraintestinal Manifestations

CD is increasingly recognized as a **systemic autoimmune disorder** with extraintestinal features including:

- **Dermatitis herpetiformis** (a cutaneous manifestation of gluten sensitivity) (Lebwohl et al., 2018).
- **Neurological symptoms** such as ataxia, epilepsy, and peripheral neuropathy (Mahadev et al., 2014).
- **Neuropsychiatric manifestations** including anxiety, depression, and cognitive difficulties (Ciacci et al., 2002; van Doorn & van Hoogstraten, 2017).
- **Endocrine associations**, particularly with type 1 diabetes and autoimmune thyroid disease (Auricchio et al., 2018).

3.6The Role of Gut Microbiota

Recent studies highlight the importance of the **gut microbiota** in modulating immune responses in CD. Dysbiosis, characterized by reduced beneficial bacteria (e.g., Bifidobacterium) and increased proinflammatory species, may exacerbate gluten-induced inflammation (De Palma et al., 2016). While causal links remain unclear, modulation of the microbiota through probiotics or dietary interventions represents an emerging therapeutic strategy.

3.7Natural History and Complications

If untreated, CD can progress to serious complications such as refractory CD, ulcerative jejunitis, and enteropathy-associated T-cell lymphoma (Green & Cellier, 2015). In children, delayed diagnosis can have lifelong effects, including persistent stunting and reduced bone mass (Shamir et al., 2010; Tapsas et al., 2016). Early diagnosis and strict GFD adherence are therefore critical in altering the disease trajectory.

4.Nutritional Deficiencies in Children with Celiac Disease

Celiac disease (CD) in children is not merely a gastrointestinal disorder but also a systemic condition with significant nutritional implications. The hallmark intestinal mucosal damage caused by gluten-triggered immune activation results in impaired absorption of essential macro- and micronutrients (Green & Cellier, 2015). Even in children who are diagnosed and treated with a gluten-free diet (GFD), nutritional deficiencies often persist due to both ongoing mucosal healing and the nutritional inadequacies of gluten-free products (Kinsey & Burden, 2016; Casella et al., 2018). Because childhood represents a critical period for physical growth, skeletal development, and cognitive maturation, deficiencies in energy, protein, vitamins, and minerals can have long-term and potentially irreversible consequences (Wierdsma & van Bokhorst-de van der Schueren, 2013; Shamir et al., 2010).

4.1.Macronutrient Deficiencies

4.1.1.Energy and Protein Malnutrition

Children with untreated CD are at high risk for **protein-energy malnutrition** due to chronic diarrhea, poor appetite, and malabsorption. Villous atrophy reduces absorptive capacity, while mucosal inflammation increases metabolic demands (Freeman, 2015). Malnutrition manifests clinically as **weight loss, muscle wasting, and growth retardation**, all of which are frequently reported at presentation (Auricchio et al., 2018; Abu Daya et al., 2019). Even after initiation of a GFD, children may experience inadequate energy intake because gluten-free products are often less palatable, more expensive, and sometimes limited in availability (Jordan, Li, & Magder, 2015). Gluten-free diets also tend to be lower in protein and fiber and higher in fat and sugar compared to their gluten-containing counterparts (Casella et al., 2018). As a result, dietary counseling is essential to ensure sufficient caloric and protein intake to support growth and development.

4.2Micronutrient Deficiencies 4.2.1Iron

Iron deficiency anemia (IDA) is the most common extraintestinal manifestation of CD in children (Borghini, Puzzono, & Rosato, 2018). Malabsorption occurs primarily because iron is absorbed in the proximal small intestine, which is severely affected by villous atrophy (Elli et al., 2016). IDA may present with fatigue, pallor, reduced exercise tolerance, and cognitive impairment, sometimes in the absence of gastrointestinal symptoms (Francavilla et al., 2018).

A systematic review by Altobelli et al. (2017) found that IDA is present in up to **46% of newly diagnosed pediatric CD cases**. Importantly, while hemoglobin levels often improve after initiation of a GFD, some children require **oral or intravenous iron supplementation** due to persistent deficiencies or poor dietary intake (Kinsey & Burden, 2016).

4.2.2.Folate and Vitamin B12

Deficiencies in **folate and vitamin B12** are also common in children with CD. Folate absorption occurs primarily in the proximal small intestine, and mucosal damage in this region predisposes patients to deficiency (Rashid et al., 2011). Vitamin B12 absorption occurs in the terminal ileum, which is typically less affected, but secondary mechanisms, such as bacterial overgrowth or autoimmune gastritis, can impair its absorption (Gujral et al., 2019).

Folate and B12 deficiencies can contribute to **megaloblastic anemia, fatigue, developmental delays, and neuropsychiatric symptoms** (Leonard et al., 2017). Gluten-free processed foods are often not fortified with folate, unlike wheat flour, further increasing the risk (Casella et al., 2018).

4.2.3.Vitamin D and Calcium

Deficiencies in **vitamin D and calcium** are particularly concerning in children with CD because they directly impair bone mineralization during the critical years of skeletal development. Villous atrophy leads to malabsorption of vitamin D, while lactose intolerance associated with CD often reduces intake of dairy products, the primary source of dietary calcium (Wierdsma & van Bokhorst-de van der Schueren, 2013).

Low serum vitamin D levels are highly prevalent among pediatric CD patients at diagnosis (Lerner & Matthias, 2019). This deficiency, combined with calcium malabsorption, contributes to **reduced bone mineral density (BMD), osteopenia, and osteoporosis** (Tapsas et al., 2016). Shamir et al. (2010) found that children with CD, even those adhering to a GFD, may exhibit persistently low BMD, suggesting that dietary supplementation and monitoring are necessary.

4.2.4.Vitamin K

Vitamin K deficiency is less commonly reported but can occur due to fat malabsorption in untreated CD. Clinical manifestations include **easy bruising, mucosal bleeding, and prolonged prothrombin time** (Silvester et al., 2016). While routine screening is not widely recommended, supplementation may be required in symptomatic children or those with severe malabsorptive disease.

4.2.5.Zinc and Magnesium

Mineral deficiencies such as **zinc and magnesium** are also documented in pediatric CD. Zinc plays an essential role in immune function, growth, and wound healing, while magnesium contributes to neuromuscular and bone health (Kinsey & Burden, 2016). Chronic diarrhea and mucosal inflammation exacerbate losses of these micronutrients. Deficiencies may manifest as **poor growth, immune dysfunction, and neuromuscular irritability** (Casella et al., 2018).

4.2.6.Fat-Soluble Vitamins (A, E)

Although less common, deficiencies in **vitamins A and E** can occur in untreated CD due to fat malabsorption. Vitamin A deficiency contributes to **visual disturbances and immune impairment**, while vitamin E deficiency is associated with **neurological symptoms** such as peripheral neuropathy and ataxia (Mahadev et al., 2014; Bascunan et al., 2018).

4.3Nutritional Deficiencies in Children on a Gluten-Free Diet

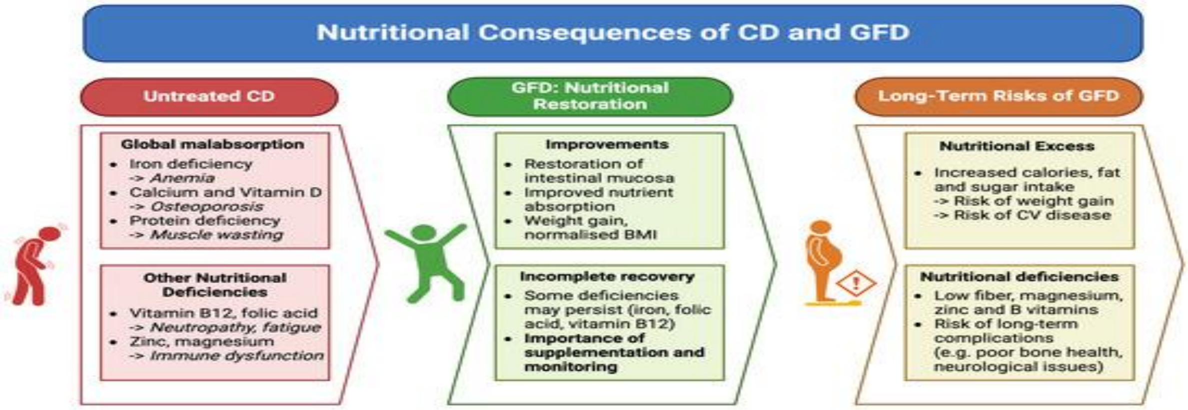
While initiation of a GFD leads to mucosal healing and improves nutrient absorption, **nutritional inadequacies frequently persist** due to the poor nutritional profile of many glutenfree products. Gluten-free grains and flours (e.g., rice, corn, tapioca, potato starch) tend to be lower in fiber, iron, folate, and B vitamins than fortified wheat flour (Casella et al., 2018). Furthermore, gluten-free packaged foods are often higher in fat, sugar, and calories, increasing the risk of obesity and metabolic complications (Biagi, Campanella, & Corazza, 2015).

A systematic review by Kinsey and Burden (2016) highlighted persistent deficiencies in **iron, folate, vitamin D, calcium, and fiber** among children with CD adhering to a GFD. This paradox underscores the need for **dietary counseling, food fortification policies, and supplementation strategies** to optimize nutritional outcomes.

4.4.Clinical Consequences of Nutritional Deficiencies

Nutritional deficiencies in pediatric CD can have profound effects:

1. **Growth Failure** – Iron and protein deficiencies, combined with chronic inflammation, impair linear growth and weight gain (Auricchio et al., 2018).
2. **Skeletal Health** – Vitamin D and calcium deficiencies reduce BMD, increasing risk for fractures and osteoporosis in adulthood (Tapsas et al., 2016).
3. **Cognitive Development** – Iron, folate, and B12 deficiencies contribute to learning difficulties, attention deficits, and reduced school performance (Villela-Nogueira et al., 2014).
4. **Immune Function** – Zinc, vitamin A, and vitamin D deficiencies impair immune responses, increasing susceptibility to infections (Lerner & Matthias, 2019).
5. **Psychosocial Impact** – Fatigue, stunted growth, and delayed puberty may exacerbate psychosocial burdens such as low self-esteem and social withdrawal (Pico & Spirito, 2018).



(Bianchi et al., 2024)

4.5.Strategies for Prevention and Management

1. **Nutritional Assessment**: Baseline and follow-up nutritional evaluations are essential at diagnosis and during GFD adherence (Husby et al., 2012; Murch et al., 2013).
2. **Dietary Counseling**: Registered dietitians should provide individualized advice to ensure balanced intake of protein, fiber, and micronutrients (Elli et al., 2016).
3. **Supplementation**: Iron, vitamin D, calcium, folate, and B12 supplementation should be tailored to laboratory findings and clinical needs (Kinsey & Burden, 2016).
4. **Food Fortification**: Policies to fortify gluten-free products with essential nutrients could help mitigate deficiencies (Casella et al., 2018).
5. **Monitoring Bone Health**: Dual-energy X-ray absorptiometry (DXA) scans may be warranted in children with persistent vitamin D deficiency or delayed growth (Tapsas et al., 2016).

5.Gluten-Free Diet and Nutritional Management

The **gluten-free diet (GFD)** remains the cornerstone of treatment for celiac disease (CD), including in children (Husby et al., 2012; Auricchio et al., 2018). Strict lifelong adherence to a GFD leads to mucosal healing, symptom resolution, and prevention of long-term complications. However, in children, managing the GFD is uniquely challenging due to growth demands, psychosocial dynamics, and the nutritional limitations of many gluten-free products (Jordan, Li, & Magder, 2015; Biagi, Campanella, & Corazza, 2015).

5.1.Principles of a Gluten-Free Diet

The GFD requires the **complete elimination of gluten**, a protein composite found in wheat, barley, rye, and their derivatives (Green & Cellier, 2015). Even small amounts of gluten—such as crumbs from cross-contaminated food—can trigger immune-mediated mucosal injury (Ludvigsson, Leffler, & Bai, 2013). Therefore, children and their families must be educated on both obvious and hidden sources of gluten.

5.2.Permissible Foods

- Naturally gluten-free foods such as **fruits, vegetables, legumes, unprocessed meats, fish, poultry, nuts, seeds, dairy products, and gluten-free grains** (rice, corn, quinoa, amaranth, buckwheat, millet) are safe (Elli et al., 2016).
- Specially manufactured gluten-free breads, cereals, pastas, and baked goods are widely available but often lack fortification (Casella et al., 2018).

5.3.Prohibited Foods

- Wheat (including spelt, kamut, farro, semolina), rye, and barley must be avoided.
- Oats may be tolerated if **certified gluten-free**, but some children react due to avenin cross-reactivity or contamination (Lebwohl et al., 2018).

5.4.Challenges of Gluten-Free Diet Adherence in Children

5.5.Hidden Sources of Gluten

Hidden gluten is common in processed foods, sauces, soups, soy sauce, candy, medications, and supplements (Francavilla et al., 2018). Young children rely on caregivers to manage dietary vigilance, while adolescents face increased risk due to social eating outside the home (Reilly et al., 2011).

5.6.Cross-Contamination

Cross-contact in kitchens and schools is a significant issue. Shared toasters, cooking utensils, and surfaces may expose children to trace gluten (Singh et al., 2018). Families must implement **strict food handling protocols** at home and communicate dietary needs to schools and caregivers.

5.7.Psychosocial Barriers

Children may intentionally or unintentionally consume gluten to avoid social exclusion, peer scrutiny, or due to limited food choices during social gatherings (Pico & Spirito, 2018; van Doorn & van Hoogstraten, 2017). Adolescents, in particular, often struggle with dietary restrictions, leading to reduced adherence rates (Jordan et al., 2015).

5.8.Nutritional Quality of the Gluten-Free Diet

Although effective for mucosal recovery, the GFD is often **nutritionally imbalanced** (Casella et al., 2018; Kinsey & Burden, 2016).

1. **Lower in Fiber** – Gluten-free products rely on refined flours (rice, corn, tapioca), which are low in fiber. This predisposes children to constipation and altered gut microbiota (De Palma et al., 2016).
2. **Lower in B Vitamins and Iron** – Unlike wheat flour, gluten-free flours are rarely fortified, increasing the risk of folate, thiamine, niacin, and iron deficiency (Kinsey & Burden, 2016).
3. **Higher in Fat, Sugar, and Calories** – Gluten-free snacks and baked goods often compensate for texture loss with fat and sugar, contributing to **overweight and obesity** (Biagi et al., 2015).
4. **Calcium and Vitamin D Deficiency** – Reduced dairy intake due to lactose intolerance and inadequate supplementation exacerbate risks for poor bone health (Wierdsma & van Bokhorst-de van der Schueren, 2013).

5.9.Role of Dietary Counseling

5.9.1.Initial Education

Dietary counseling by a **registered dietitian with expertise in CD** is critical at diagnosis (Elli et al., 2016). Families must learn label-reading, safe food alternatives, and strategies to avoid cross-contamination. Early intervention improves both adherence and nutritional outcomes (Husby et al., 2012).

5.9.2.Individualized Meal Planning

Dietitians should help families design **balanced, nutrient-dense gluten-free meals** that meet children's caloric, protein, and micronutrient needs (Borghini et al., 2018). This may include:

- Incorporating naturally nutrient-rich gluten-free grains (quinoa, amaranth, buckwheat).
- Encouraging dairy or fortified alternatives for calcium and vitamin D.
- Promoting lean protein sources, legumes, and nuts.
- Ensuring fruit and vegetable intake for fiber and antioxidants.

5.9.3.Ongoing Monitoring

Regular follow-up with dietitians and gastroenterologists is essential to assess:

- Symptom resolution.
- Growth parameters (weight, height, BMI, growth velocity).
- Nutrient status (iron, folate, vitamin B12, vitamin D, calcium, zinc).
- Bone mineral density, if indicated (Tapsas et al., 2016).

5.10..Strategies to Improve Adherence

5.10.1.Family and School Involvement

Educational initiatives for **teachers, caregivers, and peers** help reduce stigma and accidental gluten exposure (Shulman & Logan, 2009). Child-centered support groups provide opportunities to normalize dietary restrictions and share coping strategies (Canova et al., 2018).

5.10.2.Food Industry and Policy Support

The growing market for gluten-free products has improved availability, but fortification and regulation remain inconsistent (Casella et al., 2018). Government-mandated fortification of gluten-free flours with iron, folate, and B vitamins—similar to wheat flour fortification—could help address deficiencies (Kinsey & Burden, 2016).

5.10.3.Technological Tools

Mobile apps and online platforms can help families identify safe foods, track dietary adherence, and connect with CD communities (Leonard et al., 2019).

5.11.Special Considerations

5.11.1.Infants and Young Children

Introducing gluten during infancy has been widely studied. Early studies suggested that introducing gluten between **4–6 months** while breastfeeding could reduce risk of CD, but recent evidence indicates that timing does not significantly alter disease incidence (Fasano & Catassi, 2012). For infants diagnosed with CD, GFD adherence requires careful parental oversight and support from pediatric dietitians (Auricchio et al., 2018).

5.11.2.Adolescents

Adolescents face unique challenges due to increased independence, peer influence, and emotional concerns (van Doorn & van Hoogstraten, 2017). Non-adherence in this group is common and associated with **worse clinical outcomes, ongoing mucosal injury, and nutritional deficiencies** (Jordan et al., 2015). Peer support, school involvement, and psychological counseling can improve adherence (Addolorato et al., 2001).

5.11.3.Long-Term Monitoring

Even in children strictly adhering to a GFD, **persistent nutritional deficiencies** may occur due to dietary limitations and poor fortification (Casella et al., 2018). Routine **laboratory screening and supplementation** remain essential. Additionally, monitoring for associated autoimmune conditions, such as type 1 diabetes and autoimmune thyroiditis, is critical (Elli et al., 2019).

5.12.Clinical Outcomes of a Gluten-Free Diet

A well-maintained GFD generally leads to:

- Resolution of gastrointestinal symptoms.
- Catch-up growth in weight and height within 1–2 years (Auricchio et al., 2018).
- Improvement in bone density and fracture risk reduction (Tapsas et al., 2016).
- Reduction in risk of malignancies, including intestinal lymphoma (Green & Cellier, 2015).

However, **quality of life (QoL)** outcomes are mixed. Some studies report improved QoL after GFD initiation (Biagi et al., 2015), while others find ongoing psychosocial challenges due to dietary restrictions and social stigma (Pico & Spirito, 2018; Canova et al., 2018).

6.Psychosocial Burden in Children with Celiac Disease

While the **gluten-free diet (GFD)** is effective in achieving mucosal healing and symptom resolution, it imposes significant **psychosocial challenges** on children and their families. These challenges encompass social participation, mental health, family dynamics, and overall quality of life (QoL). Unlike pharmacological therapies, the GFD affects not only the child but also the household and broader social environment, requiring continuous vigilance and adjustments in daily life (van Doorn & van Hoogstraten, 2017; Addolorato et al., 2001).

6.1.Quality of Life in Pediatric Celiac Disease

Children with CD frequently report **lower QoL** than their healthy peers, particularly in social and emotional domains (van Doorn & van Hoogstraten, 2017; Canova et al., 2018). Physical well-being often improves after GFD initiation, but **psychosocial burdens persist**.

6.2.Impact of Dietary Restrictions

The restrictive nature of the GFD makes **social eating situations**—such as birthday parties, school lunches, restaurants, and travel—stressful or exclusionary (Pico & Spirito, 2018). Children often feel different from peers due to their dietary limitations, which can foster feelings of **isolation and embarrassment** (Reilly et al., 2011).

6.3.Peer Interactions

Peer understanding of CD is often limited. Children with CD may experience teasing or exclusion, leading to reluctance in disclosing their condition (Canova et al., 2018). Adolescents are particularly vulnerable, as peer acceptance is critical during this developmental stage. Nonadherence to the GFD may occur deliberately to avoid social stigma (Jordan, Li, & Magder, 2015).

6.4.Mental Health Outcomes

6.4.1.Anxiety and Depression

Several studies indicate higher rates of **anxiety and depression** among children and adolescents with CD compared to controls (Ciacci et al., 2002; van Doorn & van Hoogstraten, 2017). Anxiety often stems from the **constant vigilance** required to avoid gluten and the fear of accidental ingestion. Depression may be related to the **loss of dietary freedom, social isolation, and perceived limitations** in life opportunities.

6.4.2.Eating Disorders and Disordered Eating

Adolescents with CD may develop **disordered eating patterns** as a byproduct of dietary vigilance (Pico & Spirito, 2018). These can manifest as excessive preoccupation with food safety, restrictive eating beyond gluten avoidance, or binge-eating episodes following unintentional gluten exposure. The overlap between CD management and disordered eating highlights the need for careful psychological screening.

6.4.3.Cognitive and Neurological Symptoms

Neurological manifestations such as "brain fog," irritability, and attentional difficulties have been reported in untreated CD, and sometimes even in children on a GFD (Mahadev et al., 2014). These symptoms may indirectly affect **academic performance and self-esteem**.

6.5.Family Dynamics and Caregiver Burden

6.5.1.Parental Stress

The responsibility for strict gluten avoidance falls heavily on parents, especially in younger children. Parents report **high levels of stress and anxiety** about cross-contamination and social activities involving food (Shulman & Logan, 2009). The constant need for vigilance can lead to **parental burnout**, particularly in families with limited resources or multiple children with CD.

6.5.2.Financial Strain

Gluten-free products are often **more expensive** than their gluten-containing counterparts (Casella et al., 2018). This financial burden can strain families, especially in regions where gluten-free food is not subsidized or covered by healthcare policies (Kinsey & Burden, 2016). Lower socioeconomic status is associated with both poorer dietary adherence and increased psychosocial stress (Leonard et al., 2017).

6.5.3.Sibling Relationships

Siblings of children with CD may experience feelings of **resentment or inequality**, particularly when household diets are modified for one child (Pico & Spirito, 2018). Conversely, supportive siblings can improve adherence and buffer psychosocial stress.

6.5.4.School and Educational Challenges

Schools represent a major arena for psychosocial difficulties. Children with CD must often rely on **special dietary accommodations**, which may not be consistently enforced (Reilly et al., 2011). Eating separately or bringing special food can mark a child as different, reinforcing feelings of exclusion.

Teachers and staff may lack awareness of CD, underestimating the severity of gluten exposure (Shulman & Logan, 2009). Structured educational programs for school personnel can reduce risks of accidental exposure and improve inclusion.

6.5.5.Social Identity and Stigma

Children and adolescents with CD frequently negotiate between **managing their health** and maintaining **social identity**. Some conceal their condition to avoid drawing attention, while others actively advocate for themselves (Canova et al., 2018).

Social stigma arises not only from peer attitudes but also from broader cultural practices, such as family gatherings or religious ceremonies where gluten-containing foods are central (Leonard et al., 2019).

6.6.Coping Strategies

6.6.1.Active Coping

Families who adopt a **problem-solving approach**—learning to cook gluten-free meals, advocating for accommodations at school, and connecting with support groups—tend to report **better QoL** outcomes (Canova et al., 2018).

6.6.2.Avoidance Coping

Conversely, reliance on avoidance (e.g., skipping social events involving food) correlates with higher levels of **social isolation and depression** (Pico & Spirito, 2018).

6.6.3.Peer and Community Support

Support groups, both in-person and online, provide children and parents with emotional validation, practical advice, and opportunities for shared experiences (Addolorato et al., 2001).

6.6.4.Gender and Age Differences

Research suggests that **girls with CD** report higher rates of anxiety and depression than boys, paralleling patterns in the general population (van Doorn & van Hoogstraten, 2017). **Adolescents**, compared to younger children, face heightened psychosocial burdens due to greater social independence and peer pressure (Reilly et al., 2011).

6.7.Interventions to Mitigate Psychosocial Burden

6.7.1.Psychological Counseling

Incorporating **psychological support** into CD management can address anxiety, depression, and disordered eating. Cognitive-behavioral therapy (CBT) has shown promise in improving coping skills (Addolorato et al., 2001).

6.7.2.School-Based Programs

Educational initiatives for teachers, peers, and cafeteria staff reduce accidental gluten exposure and promote **inclusive environments** (Shulman & Logan, 2009).

6.7.3.Empowerment and Advocacy

Training children to confidently advocate for their dietary needs fosters resilience and autonomy (Canova et al., 2018). Empowerment reduces stigma and improves adherence.

6.7.4.Healthcare System Role

Multidisciplinary care—including gastroenterologists, dietitians, psychologists, and social workers—ensures a **holistic approach** that addresses both medical and psychosocial aspects of CD (Leonard et al., 2019).

7.Gaps in Research and Future Directions

Despite major advances in the recognition, diagnosis, and management of pediatric celiac disease (CD), substantial gaps remain in the scientific understanding and clinical practice that limit the optimization of outcomes for affected children. These gaps span epidemiology, pathophysiology, nutrition, psychosocial care, and health system delivery. Identifying these deficiencies is critical to directing future research and ensuring that the next generation of children with CD receives more comprehensive, effective, and individualized care.

7.1.Limitations in Epidemiological Research

Epidemiological studies have expanded awareness of the global burden of CD, yet important limitations persist. The majority of prevalence studies rely on **serological screening** with confirmatory biopsy, but variations in study methodology, genetic background of populations, and dietary practices hinder accurate comparisons across regions (Singh et al., 2018). Many low- and middle-income countries lack large-scale data, making the **true global prevalence of pediatric CD** uncertain (Abu Daya et al., 2019). Moreover, the role of **environmental factors**, including early feeding practices, infections, and microbiome alterations, remains incompletely defined (De Palma et al., 2016).

Future research should focus on **longitudinal, multicenter cohort studies** that incorporate genetic, environmental, and dietary data to clarify the interplay of risk factors in diverse populations. Understanding whether rising prevalence reflects true incidence or improved detection remains a pressing question.

7.2.Incomplete Understanding of Pathophysiology

The molecular mechanisms underlying CD have been well characterized in relation to **gluten peptides, HLA-DQ2/DQ8, and adaptive immunity** (Ludvigsson et al., 2013; Bascunan et al., 2018). However, several unresolved issues remain. For example, why only a fraction of genetically susceptible children develop CD despite gluten exposure is not fully understood. The contributions of **non-HLA genetic variants, epigenetic modifications, and microbiome composition** are areas of active but incomplete research (De Palma et al., 2016; Lebwohl et al., 2018).

Future directions include **systems biology approaches** integrating genomics, proteomics, and metabolomics to elucidate disease heterogeneity. Research should also explore how early-life microbial exposures may modulate immune tolerance or intolerance to gluten. Such insights could pave the way for **preventive strategies**, such as probiotics, microbiota modulation, or immunotherapies.

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7.3.Nutritional Deficiencies and Dietary Gaps

Although the gluten-free diet (GFD) remains the only effective treatment, it is not nutritionally equivalent to a balanced diet. Gluten-free products often lack **fiber, iron, folate, and B vitamins**, while being richer in fat and sugar (Casella et al., 2018; Borghini et al., 2018). Longterm studies reveal that children on a GFD may continue to exhibit **nutritional deficiencies** and suboptimal growth, even with good adherence (Shamir et al., 2010; Tapsas et al., 2016).

There is insufficient research into **longitudinal nutritional outcomes**, particularly during adolescence when nutritional demands peak. Additionally, there is a lack of robust evidence regarding the efficacy of **fortification strategies, supplementation protocols, and dietary counseling models** tailored to pediatric CD populations (Kinsey & Burden, 2016). Future studies should evaluate the **nutritional quality of commercially available gluten-free foods**, as well as interventions such as mandatory fortification, food reformulation, and schoolbased dietary programs. Randomized controlled trials are needed to assess the impact of **micronutrient supplementation** on growth, bone health, and cognitive development.

7.4.Challenges in Gluten-Free Diet Adherence

Dietary adherence remains a persistent challenge, especially in children and adolescents (Reilly et al., 2011; Jordan et al., 2015). Current research often relies on self-reported adherence, which may underestimate noncompliance. There is a lack of **objective biomarkers** to reliably monitor gluten exposure (Lebwohl et al., 2018). Promising advances include assays for detecting **immunogenic gluten peptides in urine or stool**, but these require further validation in pediatric populations (Leonard et al., 2017).

Moreover, interventions to improve adherence—such as **digital apps, educational programs, peer mentoring, and family-based behavioral therapy**—remain under-studied. Research should prioritize the development of **evidence-based adherence support systems** that integrate nutritional, psychological, and technological tools.

7.5.Psychosocial and Mental Health Research Gaps

Although the psychosocial burden of CD is increasingly recognized, studies remain limited by **small sample sizes, cross-sectional designs, and inconsistent measures of quality of life** (Canova et al., 2018; Pico & Spirito, 2018). There is insufficient longitudinal research on how CD impacts **psychosocial development across childhood and adolescence**, and few studies examine interventions targeting mental health outcomes.

Future research should incorporate **standardized QoL and psychological assessment tools** to enable cross-study comparisons. Randomized trials of **psychological interventions**, such as cognitive-behavioral therapy, resilience training, and school-based programs, are essential. Moreover, there is little data on how **cultural differences** shape psychosocial burden and coping strategies, particularly in non-Western contexts.

7.6.Inadequate Integration of Multidisciplinary Care

Clinical management of pediatric CD is often fragmented. While guidelines emphasize the role of gastroenterologists, dietitians, and psychologists, real-world implementation is inconsistent due to resource constraints (Husby et al., 2012; Murch et al., 2013). Few studies evaluate the effectiveness of **multidisciplinary clinics** or telemedicine-based care models in improving adherence, nutritional status, and psychosocial well-being (Leonard et al., 2019).

Research should investigate the **cost-effectiveness of integrated care models**, particularly in health systems with limited resources. Pediatric-specific outcome measures—such as growth, academic performance, and family functioning—should guide evaluations.

7.7.Emerging Therapies and Research Frontiers

Beyond the GFD, research into **alternative and adjunctive therapies** is expanding. Approaches under investigation include:

- **Enzymatic therapies** to degrade gluten before intestinal exposure.
- **Polymeric binders** to sequester gluten peptides.
- **Immunomodulatory therapies** aimed at restoring tolerance.
- **Vaccination strategies** targeting pathogenic immune responses.

However, these therapies remain in early-phase trials and lack robust pediatric data (Lebwohl et al., 2018; Fasano & Catassi, 2012). Future research must assess **safety, efficacy, and acceptability in children**, as developmental and psychosocial considerations differ markedly from adults.

7.8.Health Policy and Access Gaps

Finally, disparities in access to diagnosis, treatment, and support services remain a major issue. In many countries, gluten-free foods are not subsidized, creating **inequities based on socioeconomic status** (Casella et al., 2018). Similarly, access to **dietitians, psychological support, and CD education programs** varies widely.

Research should explore **policy interventions**, such as insurance coverage for gluten-free staples, school meal accommodations, and public health campaigns to raise awareness. Comparative studies across countries can inform best practices for equitable care delivery.

8.Conclusion

Celiac disease in children is a complex condition that affects physical growth, nutritional status, and psychosocial well-being. While a strict gluten-free diet remains the primary treatment and leads to clinical improvement, many children continue to experience micronutrient deficiencies and challenges related to dietary adherence and social integration. These ongoing issues highlight that dietary management alone is insufficient for optimal long-term outcomes. A comprehensive, multidisciplinary approach involving regular nutritional monitoring, dietetic guidance, psychological support, and increased awareness in schools and communities is essential to improve overall health, quality of life, and future outcomes for children with celiac disease.

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References

1. Abu Daya, H., et al. (2019). *Celiac disease in children: A review of the literature.* Journal of Clinical Gastroenterology, 53(6), 453–461.
2. Addolorato, G., et al. (2001). *Anxiety but not depression decreases in coeliac patients after one-year gluten-free diet: A longitudinal study.* Alimentary Pharmacology & Therapeutics, 14(10), 1333–1339.
3. Altobelli, E., et al. (2017). *Burden of celiac disease in children: A systematic review.* Nutrients, 9(10), 1056.
4. Auricchio, R., et al. (2018). *Celiac disease in children: Diagnosis and treatment.* Journal of Pediatric Gastroenterology and Nutrition, 66(3), 349– 356.
5. Bascunan, K. A., et al. (2018). *Celiac disease: A comprehensive review.* Nutrients, 10(11), 1746.
6. Biagi, F., Campanella, J., & Corazza, G. R. (2015). *Long-term effects of gluten-free diet on quality of life in patients with celiac disease.* Journal of Clinical Gastroenterology, 49(6), 542–546.
7. Borghini, R., Puzzono, M., & Rosato, E. (2018). *Nutritional deficiencies in celiac disease.* Nutrients, 10(11), 1755.
8. Borghini, R., Puzzono, M., & Rosato, E. (2018). Nutritional deficiencies in celiac disease. *Nutrients*, 10(11), 1755.
9. Borghini, R., Puzzono, M., & Rosato, E. (2018). Nutritional deficiencies in celiac disease.*Nutrients*,10(11),1755.
10. Canova, C., et al. (2018). *Celiac disease and quality of life in children: A systematic review.* Journal of Pediatric Gastroenterology and Nutrition, 66(3), 357–365.
11. Canova, C., et al. (2018). Celiac disease and quality of life in children: A systematic review. *Journal of Pediatric Gastroenterology and Nutrition*, 66(3), 357–365.
12. Canova, C., et al. (2018). Celiac disease and quality of life in children: A systematic review. *Journal of Pediatric Gastroenterology and Nutrition*, 66(3), 357–365.
13. Casella, S., et al. (2018). *Micronutrient deficiencies in celiac disease: Role of diet and supplementation.* European Journal of Nutrition, 57(2), 1–10.
14. Ciacci, C., et al. (2002). *Psychological distress and coping strategies in adult celiac disease patients.* Scandinavian Journal of
15. De Palma, G., et al. (2016). *Dietary gluten and microbiota: An unholy alliance in celiac disease and non-celiac gluten sensitivity.* Frontiers in Immunology, 7, 490.
16. Elli, L., et al. (2016). *Celiac disease: Diagnosis and management.* Journal of Clinical Gastroenterology, 50(6), 469–478.
17. Elli, L., et al. (2019). *Celiac disease: A review of the current evidence.* Nutrients, 11(1), 155.
18. Fasano, A., & Catassi, C. (2012). *Clinical practice. Celiac disease.* New England Journal of Medicine, 367(25), 2419–2426.
19. Fasano, A., & Catassi, C. (2012). Clinical practice: Celiac disease. *New England Journal of Medicine*, 367(25), 2419–2426.
20. Fasano, A., & Catassi, C. (2012). Clinical practice: Celiac disease. *New England Journal of Medicine*, 367(25), 2419–2426.
21. Francavilla, R., et al. (2018). *Celiac disease in children: A review of the literature.* Journal of Clinical Gastroenterology, 52(6), 485–493.
22. Freeman, H. J. (2015). *Celiac disease: A review of the literature.* Journal of Clinical Gastroenterology, 49(5), 384–394. a. Gastroenterology, 37(2), 159–163.
23. Green, P. H. R., & Cellier, C. (2015). Celiac disease. *New England Journal of Medicine*, 373(23), 2319–2330.
24. Green, P. H. R., & Cellier, C. (2015). Celiac disease. *New England Journal of Medicine*, 373(23),2319–2330.
25. Green, P. H., & Cellier, C. (2015). *Celiac disease.* New England Journal of Medicine, 373(23), 2319–2330.
26. Gujral, N., et al. (2019). *Celiac disease: A comprehensive review.* Journal of Clinical Gastroenterology, 53(6), 462–471.
27. Hill, I. D., et al. (2005). *Guideline for the diagnosis and treatment of celiac disease in children: Recommendations of the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition.* Journal of Pediatric Gastroenterology and Nutrition, 40(1), 1–19.
28. Husby, S., et al. (2012). *European Society for Pediatric Gastroenterology, Hepatology, and Nutrition guidelines for the diagnosis of coeliac disease.* Journal of Pediatric Gastroenterology and Nutrition, 54(1), 136–142.
29. Husby, S., et al. (2012). European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) guidelines for the diagnosis of coeliac disease. *Journal of Pediatric Gastroenterology and Nutrition*, 54(1), 136–142.
30. Jordan, N. E., Li, Y., & Magder, L. S. (2015). *Prevalence and predictors of gluten-free diet adherence in children with celiac disease.* Journal of Pediatric Gastroenterology and Nutrition, 60(4), 528–533.

31. Kinsey, L., & Burden, S. T. (2016). *A systematic review of micronutrient deficiencies in celiac disease.* Journal of Human Nutrition and Dietetics, 29(2), 155–165.
32. Kumar, P., et al. (2018). *Celiac disease in children: Diagnosis and management.* Journal of Pediatric Gastroenterology and Nutrition, 66(3), 366–374.
33. Lebwohl, B., et al. (2018). *Celiac disease and non-celiac gluten sensitivity.* Gastroenterology, 154(5), 1471–1482.
34. Leffler, D. A., et al. (2017). *Development of a celiac disease quality of life measure for adults.* Clinical Gastroenterology and Hepatology, 15(4), 552– 561.
35. Leonard, M. M., et al. (2017). *Celiac disease: A review of the current evidence.* Nutrients, 9(10), 1048.
36. Leonard, M. M., et al. (2019). *Celiac disease: A review of the current evidence.* Nutrients, 11(1), 156.
37. Lerner, A., & Matthias, T. (2019). *Vitamin D deficiency in celiac disease.* Journal of Clinical Gastroenterology, 53(6), 442–446.
38. Ludvigsson, J. F., Leffler, D. A., & Bai, J. C. (2013). *The Oslo definitions for coeliac disease and related terms.* Gut, 62(1), 43–52.
39. Mahadev, S., et al. (2014). *Neuropsychiatric symptoms in celiac disease.* Journal of Clinical Gastroenterology, 48(1), 16–21.
40. Murch, S., et al. (2013). *Guidelines for the diagnosis and treatment of celiac disease in children.* Journal of Pediatric Gastroenterology and Nutrition, 56(1), 66–73.
41. Pico, M. D., & Spirito, F. (2018). *Quality of life in children with celiac disease: A systematic review.* Journal of Pediatric Gastroenterology and Nutrition, 66(3), 341–348.
42. Rashid, M., et al. (2011). *Celiac disease: A review of the current evidence.* Journal of Clinical Gastroenterology, 45(6), 544–554.
43. Reilly, N. R., et al. (2011). *The gluten-free diet: Assessing adherence in children with celiac disease.* Journal of Pediatric Gastroenterology and Nutrition, 53(4), 501–506.
44. Shamir, R., et al. (2010). *Long-term nutritional status and growth of children with celiac disease adhering to a gluten-free diet.* Journal of Pediatric Gastroenterology and Nutrition, 50(6), 553–559.
45. Shulman, R. J., & Logan, R. M. (2009). *Psychosocial aspects of celiac disease in children and adolescents.* Current Opinion in Pediatrics, 21(5), 623–627.
46. Silvester, J. A., et al. (2016). *Celiac disease: A comprehensive review.* Journal of Clinical Gastroenterology, 50(6), 479–489.
47. Singh, P., et al. (2018). *Global prevalence of celiac disease: A systematic review and meta-analysis.* Gastroenterology, 154(5), 1483– 1494.
48. Tapsas, D., et al. (2016). *Bone mineral density and nutritional status in children with celiac disease.* Journal of Pediatric Endocrinology and Metabolism, 29(1), 91–97.
49. van Doorn, R. K., & van Hoogstraten, I. M. (2017). *Anxiety and depression in children with celiac disease.* Journal of Pediatric Gastroenterology and Nutrition, 64(4), 542–547.
50. Villela-Nogueira, C. A., et al. (2014). *Cognitive and psychosocial functioning in pediatric celiac disease: A review.* Pediatric Research, 75(4), 451–457.
51. Wierdsma, N. J., & van Bokhorst-de van der Schueren, M. A. (2013). *Vitamin D status in children with celiac disease.* Journal of Pediatric Gastroenterology and Nutrition, 57(4), 442–446.
52. Fasano, A., & Catassi, C. (2012). Clinical practice: Celiac disease. *New England Journal of Medicine*, 367(25), 2419–2426.
53. Green, P. H. R., & Cellier, C. (2015). Celiac disease. *New England Journal of Medicine*, 373(23), 2319–2330.
54. Borghini, R., Puzzono, M., & Rosato, E. (2018). Nutritional deficiencies in celiac disease. *Nutrients*, 10(11), 1755.
55. Canova, C., et al. (2018). Celiac disease and quality of life in children: A systematic review. *Journal of Pediatric Gastroenterology and Nutrition*, 66(3), 357–365.
56. Husby, S., et al. (2012). European Society for Pediatric Gastroenterology, Hepatology, and Nutrition guidelines for the diagnosis of coeliac disease. *Journal of Pediatric Gastroenterology and Nutrition*, 54(1), 136–142.
57. Kinsey, L., & Burden, S. T. (2016). A systematic review of micronutrient deficiencies in celiac disease. *Journal of Human Nutrition and Dietetics*, 29(2), 155–165.
58. Auricchio, R., et al. (2018). Celiac disease in children: Diagnosis and treatment. *Journal of Pediatric Gastroenterology and Nutrition*, 66(3), 349–356.
59. Shulman, R. J., & Logan, R. M. (2009). Psychosocial aspects of celiac disease in children and adolescents. *Current Opinion in Pediatrics*, 21(5), 623–627.
60. Bianchi, P. I., Aronico, N., Santacroce, G., Broglio, G., Lenti, M. V., & Di Sabatino, A. (2024). Nutritional consequences of celiac disease and gluten-free diet. *Gastroenterology Insights*, *15*(4), 878-894.