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Correlation between immunoglobulin and growth hormone with age and gender in Iraqi celiac disease patients

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Abstract

Background An enteropathy mediated by the autoimmune system is celiac disease (CD) caused by dietary gluten and related polyamines in people with the HLA-DQ2 or HLA-DQ8 haplotype. It presents in a variety of clinical manifestations and is associated with multiple CD-specific antibodies.

Objective To assess the impact of age and gender on growth hormone, IgA, and IgG levels in Iraqi patients with celiac disease.

Methods This study included 100 patients diagnosed with celiac disease (CD) and 50 healthy controls. Patients and controls were stratified by age (<10 years and ≥10 years) and gender (females and males). Serum growth hormone, IgA, and IgG levels were measured in all participants. Growth hormone was analyzed using the Dirui CM-180 apparatus, while IgA and IgG concentrations were determined by ELISA kits.

Results This study found that patients aged <10 years and female patient groups have lower growth hormone levels compared to controls, whereas IgA and IgG levels significantly increased in patients' groups than in the control group. A moderate negative correlation was observed between IgG and growth hormone levels in the patient group.

Conclusion Patients aged ≥10 years had lower growth hormone levels compared to controls. It is lower in female and male patient groups compared to the control group. Furthermore, IgA and IgG levels were higher in the patient group compared to the control group. These results highlight the importance of integrating demographic and serological characteristics to improve diagnosis, risk assessment, and understanding of the pathophysiology of celiac disease.

Keywords celiac disease | growth hormone | age | gender | immunoglobulin

Introduction

Celiac disease (CD) is an autoimmune-mediated enteropathy caused by dietary gluten in genetically sensitive individuals, characterized by a variety of clinical symptoms and CD-specific antibodies (Husby *et al.*, 2020). Classic CD symptoms include diarrhea and bloating, but it also produces extraintestinal manifestations such as short stature (SS), delayed puberty, osteoporosis, dental abnormalities, iron defi-

ciency anemia, infertility, and arthropathy. Notably, children with CD may present atypically with SS as the sole clinical manifestation, with no additional gastrointestinal (GI) symptoms (Sahin, 2021; Almahmoud *et al.*, 2024).

Numerous studies have found that 2–8% of patients with SS develop CD. Due to the high risk, all children with SS should be frequently checked for CD. The three most common causes of CD-related growth retardation are malnutrition, a malfunctioning growth hormone (GH) axis, and a lack

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of GH sensitivity (Troncone & Kosova 2010; Street *et al.*, 2008). In Saudi Arabia, moderate SS affects 11.3% of boys and 1.8% of girls, whereas severe SS affects 10.5% of males and 1.2% of females (Almutairi & Rabbani, 2025). After ruling out endocrine causes, chronic diseases, molecular abnormalities, and chromosomal abnormalities, the frequency of CD in Saudi children was found to be between 9.5% and 10.9% (Al-Jurayyan N *et al.*, 2012).

Recent years have seen significant advances in our understanding of the wide clinical range of CD, establishing it as a common illness affecting people of all ages. As a result, the CD working committee of the European Society for Paediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) updated the CD diagnostic criteria in 2012 (Al-Toma *et al.*, 2025; Al-Toma *et al.*, 2019; Rogalidou & Christodoulou, 2026). The most noteworthy result of these recommendations was that CD could be detected without a biopsy in a narrow subset of pediatric patients, as coeliac enteropathy was virtually invariably present in children with extremely high blood levels of the celiac auto-antibody (Pumar *et al.*, 2025).

The underlying mucosal damage in active CD is caused by a local immune response, with the mucosa extensively infiltrated with IgA-producing cells, the majority of which have antiglutin antibodies (Tinguria, 2024). Some of this IgA is clearly lost to the systemic circulation (Giner-Pérez *et al.*, 2023). When starting a gluten-free diet, the number of IgA-containing cells in the jejunal mucosa falls immediately and recovers to normal after 2–3 months (Balaban *et al.*, 2025). In contrast, a gluten challenge triggers an immunological response, resulting in the reappearance of a high number of IgA-containing cells in the jejunal mucosa, as well as a significant increase in IgA antibodies (Mpakosi *et al.*, 2025).

The three disease-specific biomarkers are serum IgM, IgA, and IgG, which are tissue transglutaminase 2 (TG2)-reactive antibodies (Paoletta *et al.*, 2022). Systemic IgA measurement is still the gold standard and most often used serological test for CD disease. ESPGHAN stated several years ago that if a child's IgA level is more than 10 times the upper limit of normal, a conclusive diagnosis can be made without a biopsy (Litwin *et al.*, 2025). Despite the demonstrated clinical value of these markers, there is a lack of complete data integrating demographic profiles (age and gender) with both endocrine status (GH) and particular immunoglobulin parameters (IgA and IgG) in this specific pediatric group. As a result, the goal of this study was to look into the relationship between demographic factors (age and gender) and serological parameters (growth hormone, IgA, and IgG) in the development of celiac disease, in order to gain insight into their potential role in CD diagnosis, risk assessment, and pathophysiology.

Methods

Ethical approval

The Statement of Helsinki's guiding ethics were followed in the conduct of this investigation. The Al-Qasim Green University Ethics Committee gave its approval on February 17, 2026 (Ethical Certificate Number: qgec/67/2026). Everyone who took part gave their informed consent. Dr. Sabah Al Fat-

lawy performed diagnostics for celiac disease (CD) at the Al Najaf specialized laboratory.

Samples collections

This study employed a matched case-control design, A convenience sampling method was utilized for participant recruitment based on their availability and accessibility. An a priori power analysis was not conducted to determine the sample size beforehand. Consequently, the statistical power of the study was not predefined and is limited by the final size of the convenience sample obtained.

This study included 100 patients diagnosed with celiac disease (CD) by a specialized physician and the completion of all necessary laboratory and clinical investigations and 50 healthy controls. Patients were stratified by age (<10 years: 74 cases; ≥10 years: 26 cases) and gender (65 females; 35 males). Controls were similarly grouped (<10 years: 32 cases; ≥10 years: 18 cases; 25 females; 25 males). They all supplied written consent after receiving a full explanation of the study's objectives.

The control group consisted of fifty healthy blood donors who were fully screened for celiac disease and confirmed to be serologically negative. To minimize confounding, controls were strictly matched with the cases based on age and gender. Furthermore, strict exclusion criteria were applied, ensuring that all selected controls were entirely free from any chronic or autoimmune condition. Blood was taken, put in gel tubes, and spun in a centrifuge. The acquired serum samples from patients and controls were kept in a deep freezer at -20°C to be utilized later basic characteristics of CD patients and control groups.

Growth hormone levels measurement

Serum Growth Hormone (GH) levels were quantified using the Dirui CM-180 automated chemiluminescence immunoassay (CLIA) analyzer (Dirui Industrial Co., Ltd., Changchun, China). The assay was performed strictly according to the manufacturer's specifications. The analytical sensitivity (limit of detection) of the GH kit was [e.g., 0.05 ng/mL] with a high specificity showing no cross-reactivity with other pituitary hormones. System calibration procedures were conducted using multi-point reference calibrators provided by the manufacturer and traceable to international standards (WHO IS [e.g., 98/574]) before each run batch. The established clinical reference range for healthy individuals was defined as [e.g., 0.05–5.0 ng/mL for adults]. Assay precision was confirmed by intra-assay and inter-assay coefficients of variation (CV), which were [e.g., <5.0%] and [e.g., <7.5%], respectively, demonstrating high reproducibility.

IgA and IgG antibodies measurement

Serum total IgA and IgG levels were quantified using commercially available enzyme-linked immunosorbent assay (ELISA) kits [Euroimmun AG, Lübeck, Germany] in accordance with the manufacturer's operational protocols. The optical density (OD) was measured spectrophotometrically at 450 nm using a microplate reader. The optical density (OD) was measured at a wavelength of 450 nm using an ELISA mi-

croplate reader. The results were calculated as a ratio of the OD of the clinical sample to the OD of the provided calibrator, using the following formula: Ratio = [OD of Sample / OD of Calibrator]. For the IgG assay, the clinical sensitivity was 94.4–100% (after day 10 to 21 post-symptom onset) with a specificity of 99.6%. For the IgA assay, the sensitivity ranged from 88–92% during the early phase of infection, with a specificity ranging from 88.4–92.4%. To ensure methodological precision, the reproducibility of the assays was evaluated; the intra-assay variability (repeatability) and inter-assay variability (reproducibility) for both IgA and IgG exhibited coefficients of variation (CV) of <5.8% and <7.9%, respectively, confirming high reliability and technical consistency.

The microwells were coated with pure wheat gliadin. Antibodies in diluted serum quandary to the antigen. Washing the microwells eliminates non-specific serum components. Horseradish peroxidase conjugated anti-human IgA or IgG antibodies distinguish the binding of patient antibodies, resulting in a conjugate/antigen complex. Unbound conjugate is removed by cleaning the microwells. An enzyme substrate hydrolyzes and becomes blue when it is attached to a conjugate. The presence of an acid inhibits the reaction, resulting in a yellow product. This yellow color's intensity is determined photometrically at 450 nm. The number of IgA or IgG antibodies in the initial sample is proportional to the color intensity. A standard curve was created by plotting the average absorbance optical density of each standard against its concentration and absorbance value. Unknown sample concentrations were estimated using interpolation from the standard curve.

Data analysis

GraphPad Prism7 software was used to undertake the statistical analysis. P-value was determined using t-test. The correlation between the variations of IgA, IgG and growth hormone in this study was examined using the Pearson correlation coefficient. The threshold for statistical significance was

set at $P < 0.05$. The means and standard deviation (std.) of all the data were displayed.

Results

Analysis of growth hormone levels revealed a significant reduction in celiac disease (CD) patients compared to controls, particularly among children younger than 10 years and female patients. As shown in **Table 1**, the mean growth hormone concentration in patients under 10 years was markedly lower (0.65 ± 0.81 ng/L) than in age-matched controls (2.35 ± 1.13 ng/L; $p = 0.0001$). Similarly, female patients exhibited a significant decrease (0.73 ± 1.17 ng/L) compared to female controls (2.03 ± 0.61 ng/L; $p = 0.0001$). In contrast, no statistically significant differences were observed in patients aged ≥ 10 years or in male patients when compared with their respective control groups.

Serological analysis demonstrated a consistent elevation of IgA and IgG levels in CD patients across both age categories (**Table 2**). In children younger than 10 years, IgA levels were significantly higher in patients (14.81 ± 0.78) compared to controls (3.38 ± 0.34 ; $p = 0.0001$). A similar pattern was observed in patients aged ≥ 10 years (14.09 ± 1.83 vs. 4.33 ± 0.48 ; $p = 0.0001$). IgG levels also showed a pronounced increase in CD patients, with values of 14.7 ± 0.98 in the < 10 years group and 17.39 ± 1.75 in the ≥ 10 years group, compared to 3.66 ± 0.37 and 2.23 ± 1.22 in controls, respectively ($p = 0.0001$ for both).

These findings indicate that CD patients exhibit a dual pattern of reduced growth hormone and elevated immunoglobulin levels. **Figure 1** illustrates this inverse relationship, showing decreased growth hormone alongside increased IgA and IgG concentrations in the patient group.

Correlation analysis in the control group revealed weak and statistically non-significant associations between growth hormone and immunoglobulin levels. **Figure 2** showed growth hormone a slight positive correlation with IgA

Table 1 Growth hormone level in Iraqi celiac disease patients according to age and gender parameters

Parameters		Celiac disease patients		Control		P-value
		Number of samples	Growth hormone levels (ng/L)	Number of samples	Growth hormone levels (ng/L)	
Age	<10 years	74	$0.65 \pm 0.81^*$	32	2.35 ± 1.13	0.0001
	≥ 10 years	26	1.02 ± 1.35	18	1.17 ± 0.41	Non sig.
Gender	Female	65	$0.73 \pm 1.17^*$	25	2.03 ± 0.61	0.0001
	Male	35	0.87 ± 0.10	25	1.09 ± 0.12	Non sig.

*Significantly different at $P < 0.05$ compared to control group.

Table 2 IgA and IgG parameters in Celiac disease patient according to age group

Immunoglobulin	Age	Celiac disease patients		Control		P-value
		Number of samples	Immunoglobulin levels (mg/dL)	Number of samples	Immunoglobulin levels (mg/dL)	
IgA	<10 years	74	$14.81 \pm 0.78^*$	32	3.38 ± 0.34	0.0001
	≥ 10 years	26	$14.09 \pm 1.83^*$	18	4.33 ± 0.48	0.0001
IgG	<10 years	74	$14.7 \pm 0.98^*$	32	3.66 ± 0.37	0.0001
	≥ 10 years	26	$17.39 \pm 1.75^*$	18	2.23 ± 1.22	0.0001

*Significantly different at $P < 0.05$ compared to control group.

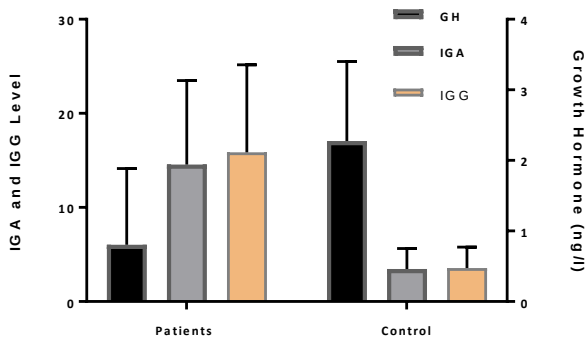


Figure 1 Comparison of growth hormone (GH), IgA, and IgG levels between the patient and control groups. Statistical analysis was performed using the unpaired t-test. Patients (n = 100), Controls (n = 50). (p = 0.0001 for GH, p = 0.0001 for IgA, and p = 0.0001 for IgG) versus the control group.

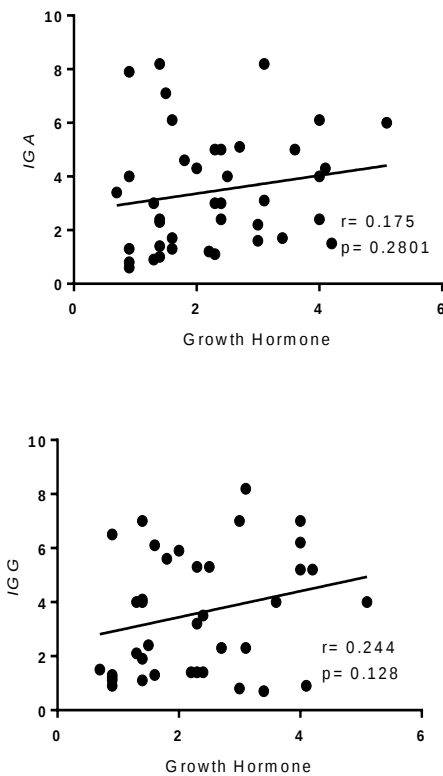


Figure 2 Weak correlations between the variations of IgA and growth hormone and IgG and growth hormone in the control group. Growth hormone showed a positive correlation with IgA (r=0.175, p=0.2801) and IgG (r=0.244, p=0.128).

(r=0.175, p=0.2801) and IgG (r=0.244, p=0.128). These findings indicate that, in healthy individuals, variations in growth hormone do not significantly influence immunoglobulin concentrations, suggesting that the observed alterations in patients with celiac disease are disease-specific rather than physiological.

In contrast, correlation analysis in CD patients revealed distinct patterns compared to controls. As shown in **Figure 3**, growth hormone exhibited a weak and non-significant negative correlation with IgA (r=-0.1835, p=0.0705). In contrast, a moderate and statistically significant negative correlation

was observed between growth hormone and IgG (r=-0.3333, p=0.0008). These findings suggest that reduced growth hormone levels in CD patients are more closely associated with elevated IgG concentrations.

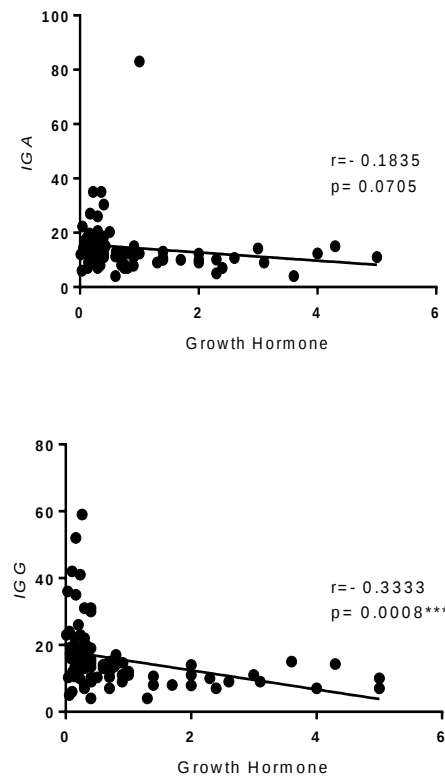


Figure 3 Weak correlation between variations in IgA and growth hormone and a moderate correlation between variations in IgG and growth hormone. Growth hormone has a weak but non-significant negative correlation with IgA (r=-0.1835, p=0.0705) and a moderately significant negative correlation with IgG (r=-0.3333, p=0.0008).

Discussion

This is the first research that compares the symptoms and indicators of Iraqi CD patients by age and gender, as far as we are aware. The number of samples and the consistency of clinical data acquired at the time of diagnosis are the study's strengths. Some of our findings differ from those of other research around the world, which could be attributable to genetic and environmental factors (including nutrition) unique to each nation.

In genetically sensitive individuals, CD is a chronic inflammatory disorder linked with small intestine injury caused by gluten intolerance (Sharma *et al.*, 2020). Several years ago, a lack of GH response to hypoglycaemia was discovered in celiac children following the implementation of a gluten-free diet. A study discovered a reduction in GH in 69% of kids with active celiac disease (Mauro *et al.*, 2018).

The current study's findings were consistent with other studies (Binicier & Tosun, 2020; Jabeen *et al.*, 2022), which discovered that CD is two to three times more common in girls. Women are up to four times as likely to have autoimmune illness as males. Many causes have been offered, including sex hormones, the X-chromosome, microchimerism,

environmental influences, and the microbiome (Kronzer *et al.*, 2021). Also, the present study show a significant increase of IgA and IgG in the patients group than in healthy subjects, these results agreed with (Husby *et al.*, 2020).

In genetically sensitive people who have either the HLA DQ2 (95%) or HLA DQ8 haplotype (5%), as well as several environmental triggers, gluten exposure can cause celiac disease, an autoimmune enteropathy. There are several different intestinal and extraintestinal symptoms of celiac disease. Different physiological systems which include central nervous and endocrine system are involved in extraintestinal symptoms. After iron deficiency anemia, short stature is regarded as the second communal extraintestinal sign (Rogalidou & Christodoulou, 2026).

A little portion of the gluten protein known as gliadin is recognized by the antigliadin antibodies IgG and IgA. Due to their similar sensitivity and specificity, antigliadin IgG and IgA together produced the first trustworthy CD screening test. IgA antibodies had 71% sensitivity and 97% specificity in clinical trials. While the IgG anti-gliadin antibodies are 87% sensitive and 91% specific, the combined IgA and IgG anti-gliadin antibody test has an overall sensitivity of 95% and a specificity of 90% (Saadah, 2020; Eli *et al.*, 2018).

As shown in **Figure 2**, the results demonstrate a non-significant positive association between IgA, IgG, and Growth hormone in the control group, but a substantial negative correlation in the sick group. CD is an autoimmune illness that is commonly associated with other endocrine and non-endocrine autoimmune disorders (Greco *et al.*, 2026). In truth, CD is regarded to be an immune-mediated systemic disease (Sahin, 2021) discovered significant levels of anti-pituitary antibodies in CD children, indicating an autoimmune type of hypophysitis. The pathophysiological reasons causing the lack of catch-up development with a gluten-free diet in certain CD patients are poorly understood. These processes are most likely explained by decreased GH and IGF1 secretion, and these individuals might benefit from GH therapy (Durazzo *et al.*, 2022). Patients with CD and GH deficiency who get GH can reach a final adult height close to their genetic potential (O'Neill *et al.*, 2022).

Conclusion

This study demonstrates that growth hormone was reduce in patients age groups (≤ 10 years) than control. Also, it reduce in female and male patients groups than control group. The confluence of celiac disease with growth hormone insufficiency necessitates a multifaceted therapy. A timely diagnosis and adequate treatment, such as a gluten-free diet and growth hormone replacement therapy, can have a considerable influence on afflicted persons' general well-being and development trajectory. Regular monitoring and communication between gastroenterologists and endocrinologists is required for best results in this scenario.

Furthermore, IgA, and IgG levels were more elevated in patients' group than in the control group. While generalizing our findings to other groups may not be suitable, the insights acquired about the significance of total IgA and IgG levels

may aid in the better implementation of the non-biopsy diagnosis of CD. This practical method should be further addressed, increasing the diagnostic capability of this test.

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Conflict of interest All authors state no conflict of interest in this research

Author contribution ASN: Writing—original draft, writing—review & editing, conceptualization, investigation, methodology; ASA: conceptualization, investigation, and methodology. ASM: provided clinical samples, designed the study, and contributed analytic tools. All authors have read and agreed to the published version of the manuscript.

Availability of data and materials All data are available in the manuscript.

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References

- Al-Jurayyan N NA, Mohamed SH, Al Otaibi HM, Al Issa ST, Omer HG. 2012. Short stature in children: Pattern and frequency in a pediatric clinic, Riyadh, Saudi Arabia. *Sudanese Journal of Paediatrics*, 12(1): 79–86.
- Al-Toma A, Zingone F, Branchi F, Schieppatti A, Malamut G, Canova C, Biagi F, Schumann M. 2025. European Society for the Study of Coeliac Disease 2025 updated guidelines on the diagnosis and management of coeliac disease in adults. Part 1: diagnostic approach. *United European Gastroenterology Journal*, 13(10): 1855–1886. DOI: 10.1002/ueg2.70119.
- Al-Toma A, Volta U, Auricchio R, Castillejo G, Sanders DS, Cellier C, Mulder CJ, Lundin KE. 2019. European Society for the Study of Coeliac Disease (ESsCD) guideline for coeliac disease and other gluten-related disorders. *United European Gastroenterology Journal*, 7(5): 583–613. DOI: 10.1177/2050640619844125.
- Almahmoud E, Alkazemi DUZ, Al-Qabandi W. 2024. Growth Stunting and Nutritional Deficiencies among Children and Adolescents with Celiac Disease in Kuwait: A Case–Control Study. *Children*, 11(9): 1042. DOI: 10.3390/children11091042.
- Almutairi F, Rabbani U. 2025. Assessment and Referral of Patients With Short Stature at Primary Care Buraidah, Saudi Arabia. *Cureus*, 17(6): e62100. DOI: 10.7759/cureus.86197.
- Balaban DV, Enache I, Balaban M, David RA, Vasile AD, Popp A, Jinga M. 2025. Outcomes in Adults with Celiac Disease Following a Gluten-Free Diet. *Journal of Clinical Medicine*, 14(14): 5144. DOI: 10.3390/jcm14145144.
- Binicier OB, Tosun F. 2020. Evaluation of adult celiac disease from a tertiary reference center: a retrospective analysis. *Revista da Associação Médica Brasileira*, 66(1): 55–60. DOI: 10.1590/1806-9282.66.1.55.
- Durazzo M, Ferro A, Brascugli I, Mattivi S, Fagoonee S, Pellicano R. 2022. Extra-intestinal manifestations of celiac disease: what should we know in 2022? *Journal of Clinical Medicine*, 11(1): 258. DOI: 10.3390/jcm11010258.
- Eli ZN, Mustafa NW, Othman GQ. 2018. Evaluation of Diagnostic Efficiency of Anti-reticulin and Anti-gliadin antibody test in Celiac

- Disease. *Polytechnic Journal*, 8(1): 4. DOI: [10.25156/ptj.2018.8.1.143](https://doi.org/10.25156/ptj.2018.8.1.143).
- Giner-Pérez L, Donat E, Sinisterra-Sebastián P, Masip E, Ballester V, Polo B, Ribes-Koninckx C, Roca M. 2023. Study of the immune response in celiac patients with selective IgA deficiency who start a gluten-free diet. *Clinical and Experimental Medicine*, 23(6): 2829–2838. DOI: [10.1007/s10238-023-01040-1](https://doi.org/10.1007/s10238-023-01040-1).
- Greco M, Mirabelli M, Misiti R, Dragone F, Donato A, Casella D, Brunetti A, Foti DP. 2026. Endocrine Autoimmunity and Inflammatory Signatures in Pediatric Celiac Disease: Context-Dependent Patterns. *Diagnostics*, 16(9): 1330. DOI: [10.3390/diagnostics16091330](https://doi.org/10.3390/diagnostics16091330).
- Husby S, Koletzko S, Korponay-Szabó I, Kurppa K, Mearin ML, Ribes-Koninckx C, Shamir R, Troncone R, Auricchio R, Castillejo G. 2020. European society paediatric gastroenterology, hepatology and nutrition guidelines for diagnosing coeliac disease 2020. *Journal of Pediatric Gastroenterology and Nutrition*, 70(1): 141–156. DOI: [10.1097/MPG.0000000000002497](https://doi.org/10.1097/MPG.0000000000002497).
- Jabeen S, Khan AU, Ahmed W, Ahmad MUD, Jafri SA, Bacha U, Ur-Rehman H. 2022. Disease specific symptoms indices in patients with celiac disease—a hardly recognised entity. *Frontiers in Nutrition*, 9: 944449. DOI: [10.3389/fnut.2022.944449](https://doi.org/10.3389/fnut.2022.944449).
- Kronzer VL, Bridges Jr SL, Davis III JM. 2021. Why women have more autoimmune diseases than men: An evolutionary perspective. *Evolutionary Applications*, 14(3): 629–633. DOI: [10.1111/eva.13167](https://doi.org/10.1111/eva.13167).
- Litwin A, Le Thi TG, El-Lababidi N, Kindermann A, Pancheva R, Gerasimidis K, Goncalves CC, Escobar PC, Niseteo T, Ikrath K, Koletzko S. 2025. Celiac disease diagnosis in clinical practice: ESPGHAN quality of care survey from 129 pediatric hospitals across 28 countries. *Journal of Pediatric Gastroenterology and Nutrition*, 81(3): 606–617. DOI: [10.1002/jpn3.70143](https://doi.org/10.1002/jpn3.70143).
- Mauro B, Chiara M, Elena B, Filomena SA, Daniela L, Pietro F, Alberto V. 2019. Management of celiac patients with growth failure. In: Assaad F, Wassmann H, Khodor M (eds). *Pituitary Diseases*. IntechOpen. DOI: [10.5772/intechopen.77129](https://doi.org/10.5772/intechopen.77129).
- Mpakosi A, Kaliouli-Antonopoulou C, Cholevas V, Cholevas S, Tzouvelekis I, Mironidou-Tzouveleki M, Sokou R. 2025. Challenges in the pediatric celiac disease diagnosis: an up-to-date review. *Diagnostics*, 15(18): 2392. DOI: [10.3390/diagnostics15182392](https://doi.org/10.3390/diagnostics15182392).
- O'Neill C, Gangat M, Radovick S. 2022. Growth hormone deficiency. *Endocrines*, 3(4): 736–744. DOI: [10.3390/endocrines3040060](https://doi.org/10.3390/endocrines3040060).
- Paoletta G, Sposito S, Romanelli AM, Caputo I. 2022. Type 2 transglutaminase in coeliac disease: A key player in pathogenesis, diagnosis and therapy. *International Journal of Molecular Sciences*, 23(14): 7513. DOI: [10.3390/ijms23147513](https://doi.org/10.3390/ijms23147513).
- Pumar M, Choo S, Rosenbaum J, Alex G, Ho SS. 2025. No-Biopsy Diagnosis of Coeliac Disease in Children Without Anti-Endomysial IgA Antibody Testing: Combining Anti-Tissue Transglutaminase IgA and Anti-Deamidated Gliadin IgG Antibodies. *Journal of Paediatrics and Child Health*, 61(4): 628–634. DOI: [10.1111/jpc.16801](https://doi.org/10.1111/jpc.16801).
- Rogalidou M, Christodoulou D. 2026. Unraveling the complexity of celiac disease: a narrative review of its multisystem nature. *Medicina*, 62(1): 120. DOI: [10.3390/medicina62010120](https://doi.org/10.3390/medicina62010120).
- Saadah OI. 2020. Short children with impaired growth hormone secretion: Do they have celiac disease?. *Saudi Medical Journal*, 41(1): 68–74. DOI: [10.15537/smj.2020.1.24785](https://doi.org/10.15537/smj.2020.1.24785).
- Sahin Y. 2021. Celiac disease in children: A review of the literature. *World Journal of Clinical Pediatrics*, 10(4): 53–62. DOI: [10.5409/wjcp.v10.i4.53](https://doi.org/10.5409/wjcp.v10.i4.53).
- Sharma N, Bhatia S, Chunduri V, Kaur S, Sharma S, Kapoor P, Garg M. 2020. Pathogenesis of celiac disease and other gluten related disorders in wheat and strategies for mitigating them. *Frontiers in Nutrition*, 7: 6. DOI: [10.3389/fnut.2020.00006](https://doi.org/10.3389/fnut.2020.00006).
- Street ME, Volta C, Ziveri MA, Zannacca C, Banchini G, Viani I, Bernasconi S. 2008. Changes and relationships of IGFS and IGFBPS and cytokines in coeliac disease at diagnosis and on gluten-free diet. *Clinical Endocrinology*, 68(1): 22–28.
- Tinguria M. 2024. Pathology of celiac disease: an insight about etiopathogenesis, pathophysiology and histologic diagnosis. *Medical Research Archives*, 12(9): 5696. DOI: [10.18103/mra.v12i9.5696](https://doi.org/10.18103/mra.v12i9.5696).
- Troncone R, Kosova R. 2010. Short stature and catch-up growth in celiac disease. *Journal of Pediatric Gastroenterology and Nutrition*, 51: S137–S138. DOI: [10.1097/MPG.0b013e3181f1dd66](https://doi.org/10.1097/MPG.0b013e3181f1dd66).