

RESEARCH ARTICLE

Assessment of Zonulin in patients with Celiac Diseases

Huda Najah Abd ^{1,*}, Adhraa Baqir Hassan ², Ahmed abdalhadi ³

¹ Department of Department of Biology, Faculty of Science University of Kufa, Najaf, Iraq.

² Professor in Department of Department of Biology, Faculty of Science University of Kufa, Najaf, Iraq.

³ Professor in Faculty of Medicine University of Jabir ibn Hayyan, Najaf, Iraq.

*Corresponding author: Huda Najah Abd, hudan.albozkeer @student.uokufa.edu.iq

ABSTRACT

Background: Celiac disease (CD), also known as gluten-sensitive enteropathy, is a chronic autoimmune disorder caused by gluten consumption. It causes nutritional malabsorption due to small intestinal villi inflammation and atrophy. The immune-mediated response mostly engages both innate and adaptive immune systems, resulting in a systemic disease that may impact several organs outside the gastrointestinal tract. Zonulin is a key physiological regulator of intercellular tight junctions in the gastrointestinal epithelium and plays an important role in intestinal permeability. It has attracted considerable interest because of its involvement in autoimmune diseases such as celiac disease (CeD), where dysregulation of the zonulin pathway may contribute to disease pathogenesis.

Materials and methods: The research included 60 randomly chosen Celiac disease (CeD) patients (22 men and 38 women). Between April and September 2025, it was done. Patients and controls are 1-18 years old.

The consultant doctors diagnosed Celiac Disease. The name, gender, age, weight, height and place of living, the patients have been obtained through a questionnaire. Participants with other diseases were excluded from the current investigation.

A healthy control group consisting of 30 apparently control participants (13 males and 17 females) was included. Their ages were comparable to those of the patients.

Results: A substantial increase ($P \leq 0.05$) in Zonulin levels was observed in celiac disease patients compared to control groups and at different age groups. There is a substantial negative connection ($P \leq 0.05$) between Zonulin levels (ng/ml) and BMI in Celiac disease patients. A substantial increase ($P \leq 0.05$) in Zonulin levels was observed in Celiac disease patients by gender. Results indicate a substantial rise ($P \leq 0.05$) in Zonulin levels in Celiac disease patients based on location, with a significant drop ($P \leq 0.05$) in rural groups compared to urban groups.

Conclusion: The results showed that zonulin levels were significantly higher in female patients with celiac disease than in male patients. Overall, zonulin levels were significantly higher in patients with celiac disease than in healthy controls. These findings suggest that elevated serum zonulin levels may be associated with celiac disease and may reflect alterations in intestinal barrier permeability.

Keyword: Celiac disease, Zonulin, Gluten

ARTICLE INFO

Received: 16 June 2026

Accepted: 25 August 2026

Available online: 15 September 2026

COPYRIGHT

Copyright © 2026 by author(s).

Applied Chemical Engineering is published by Arts and Science Press Pte. Ltd. This work is licensed under the Creative Commons Attribution-NonCommercial 4.0 International License (CC BY 4.0).

<https://creativecommons.org/licenses/by/4.0/>

1. Introduction

Celiac disease (CeD) represents a long-term, immune-associated intestinal disorder caused from gluten-containing foods in susceptible genetically individuals, such as those with the HLA-DQ2 or HLA-DQ8 haplotypes (Rodríguez-Martín, *et al*, 2020)¹. Gluten-containing cereals including wheat, barley, and rye cause an aberrant T-cell-

mediated immune response that damages the small intestine to cause villous atrophy, crypt hyperplasia, and intraepithelial lymphocytosis. (Smithson, *et al*,2021)².

CeD affects approximately 1% of the population, though prevalence varies by geographic region and remains underestimated due to asymptomatic or atypical presentations (Topa, *et al* .,2021)³. This has driven research into novel biomarkers such as cytokine profiles, gluten immunogenic peptides (GIPs), and circulating microRNAs, which could facilitate earlier detection, monitor dietary compliance, and predict disease progression without invasive testing (Mir, *et al* .,2022)⁴. Zonulin is a physiological regulator of intercellular tight junctions (TJs) in the intestinal epithelium, which is central to the control of intestinal permeability. It has recently become the focus of much interest because of its implication in a number of autoimmune and inflammatory diseases, most prominently celiac disease, Zonulin is a physiological regulator of intercellular tight junctions (TJs) in the intestinal epithelium and plays a central role in controlling intestinal permeability, it has attracted considerable attention because of its involvement in several autoimmune and inflammatory diseases, particularly celiac disease (CeD), in celiac disease, dysregulation of the zonulin pathway may impair TJ integrity, allowing gluten-derived peptides to cross the epithelial barrier through the paracellular pathway and trigger inappropriate immune responses (Mohammadi-Kordkhayli *et al.*, 2024)⁶. Zonulin is a 47-kDa protein identified as pre-haptoglobin 2 (pre-HP2), and it is secreted primarily by the small intestine, liver, and adipose tissue. It functions as a regulator of paracellular transport by reversibly modulating tight junctions between epithelial cells (Heickman *et al.*, 2020)⁵. In individuals with celiac disease, elevated zonulin levels may reflect impaired epithelial barrier function and increased intestinal permeability. However, zonulin activity is not inherently pathological; rather, excessive or dysregulated zonulin signaling may contribute to increased intestinal permeability in gluten-related disorders (Mohammadi-Kordkhayli *et al.*, 2024)⁶.

2. Materials and Methods

The research included 60 randomly chosen Celiac disease (CeD) patients (22 men and 38 women). Between April and September 2025, it was done. Patients and controls are 1-18 years old.

The consultant doctors diagnosed Celiac Disease. The name, gender, age, weight, height and place of living. the patients have been obtained through a questionnaire. All diseases were excluded from the current investigation.

As a healthy group, 30 apparently control participants (13 males and 17 females) were involved. Their ages were similar to those of the patients.

2.1. Collection of blood samples

Five milliliters of venous blood was collected from each patient and control subject using a sterile needle and syringe. The blood samples were allowed to clot at room temperature for 10 minutes and were then centrifuged at 4,000 rpm for 10 minutes. The serum was subsequently separated and transferred into new Eppendorf tubes.

2.2. Principle

The assay was based on an enzyme-linked immunosorbent test assay (ELISA). An anti- human Zonulin antibody was pre-coated on the wells of microplate. When Zonulin from the sample is introduced, it attaches itself to the antibodies that have been coated on the wells. After that, a human Zonulin antibody that has been biotinylated is introduced and binds to the Zonulin in the sample. After that, the biotinylated Zonulin antibody is bound by streptavidin-HRP. Unbound streptavidin-HRP is removed during a washing phase after incubation. After adding the substrate solution, color appears according to the quantity of human Zonulin. An acidic stop solution is added to halt the reaction, and absorbance is measured at 450 nm.

2.3. Statistical Analysis

Data were statistically analysed using the Statistical Package for the Social Sciences (SPSS) version 26 . Results are given as mean $\pm$ standard error (Mean $\pm$ SE). The differences between patients with celiac disease and the control group were analyzed using the independent samples t-test, as well as one-way analysis of variance (ANOVA) for comparisons among several groups, like age categories. A p-value of ≤ 0.05 was statistically significant. And Pearson's correlation coefficient and linear regression analysis were used to assess relationships between biomarkers and physiological parameters.

3. Results

The results in figure (1) show a significant increase ($P \leq 0.05$) in Zonulin level in Celiac disease patients (n=60) ($239,333 \pm 10.992$) in comparing with control groups (n= 30) (107.389 ± 1.136).

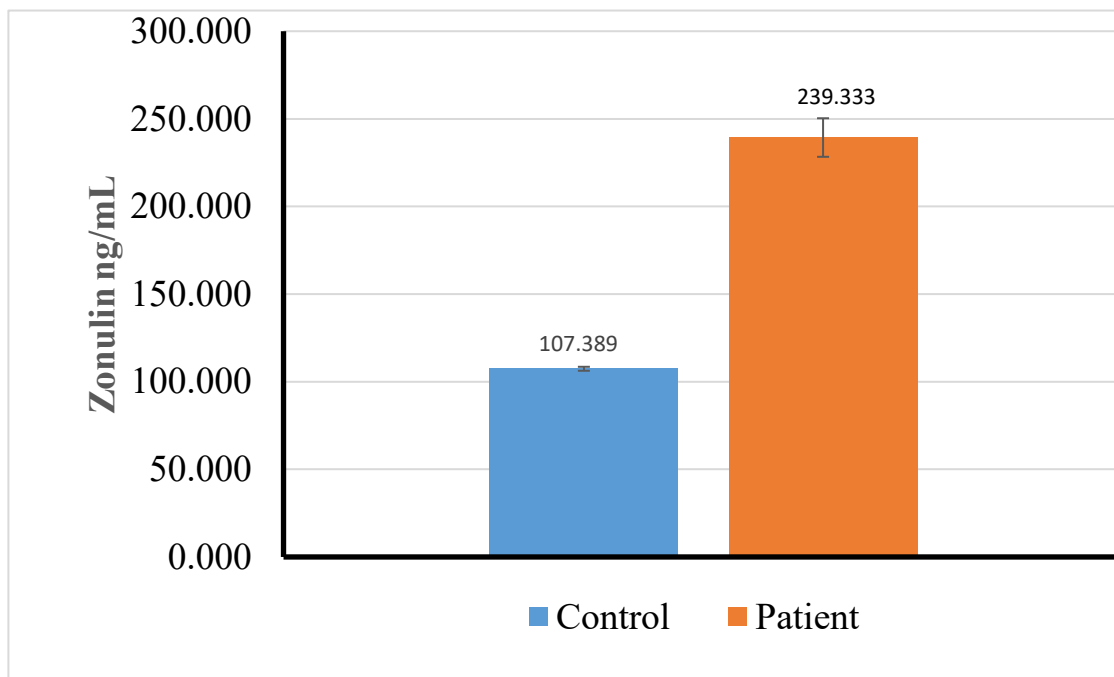


Figure 1. Zonulin level (ng/ml) in Celiac disease patients and control groups

* means significant difference at ($P \leq 0.05$) (Means $\pm$ SE)

The results in Figure (2) indicates a significant increase ($P \leq 0.05$) in Zonulin levels in Celiac disease patients at ages 1-6, 7-12, and 13-18 (37.754 ± 5.779 , 239.892 ± 5.714 , and 140.354 ± 5.843) compared to control groups (106.761 ± 1.682 , 108.117 ± 2.186 , and 107.288 ± 2.186). Also the results show significant increase ($P \leq 0.05$) at age (1-6) y (106.761 ± 1.682) in compared with age (7-12y and 13-18y) (108.117 ± 2.186 and 107.288 ± 2.186) respectively.

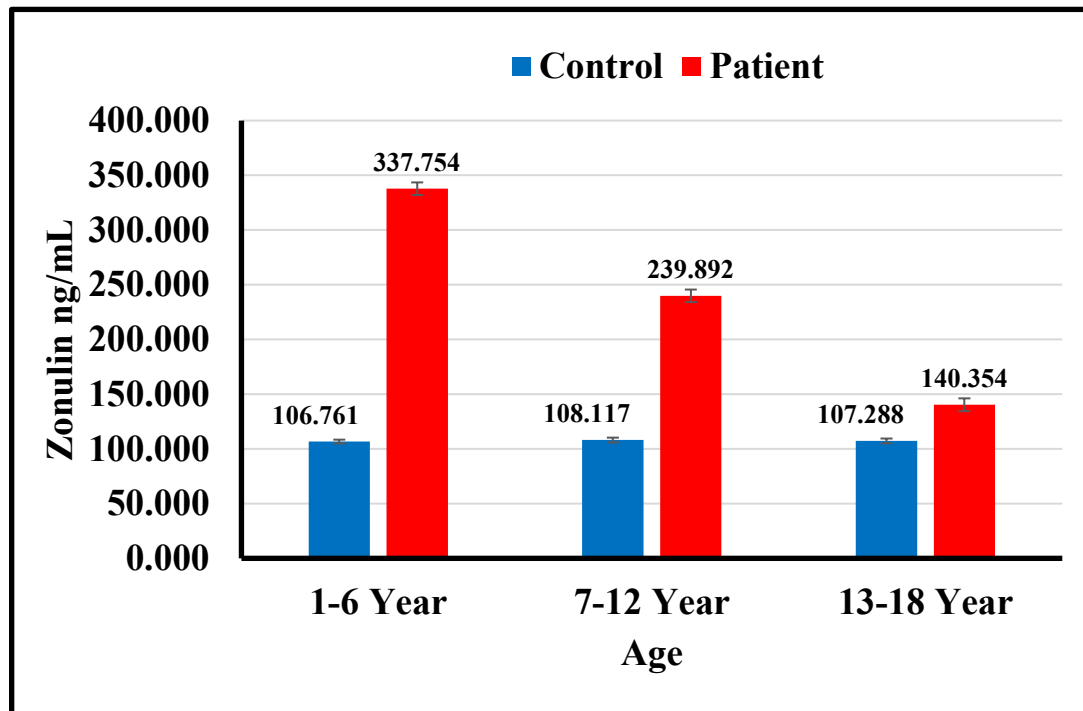


Figure 2. Zonulin level (ng/ml) in Celiac disease patients and control groups according to age.

* means significant difference at ($P \leq 0.05$) (Means $\pm$ SE)

The results in figure (3) show a significant increase ($P \leq 0.05$) in patients with different Sex (7-12y and 13-18y) respectively compared with control groups (7-12y and 13-18y) respectively. Also the results show a significant increase ($P \leq 0.05$) in females (42.222) in compared with males (24.444).

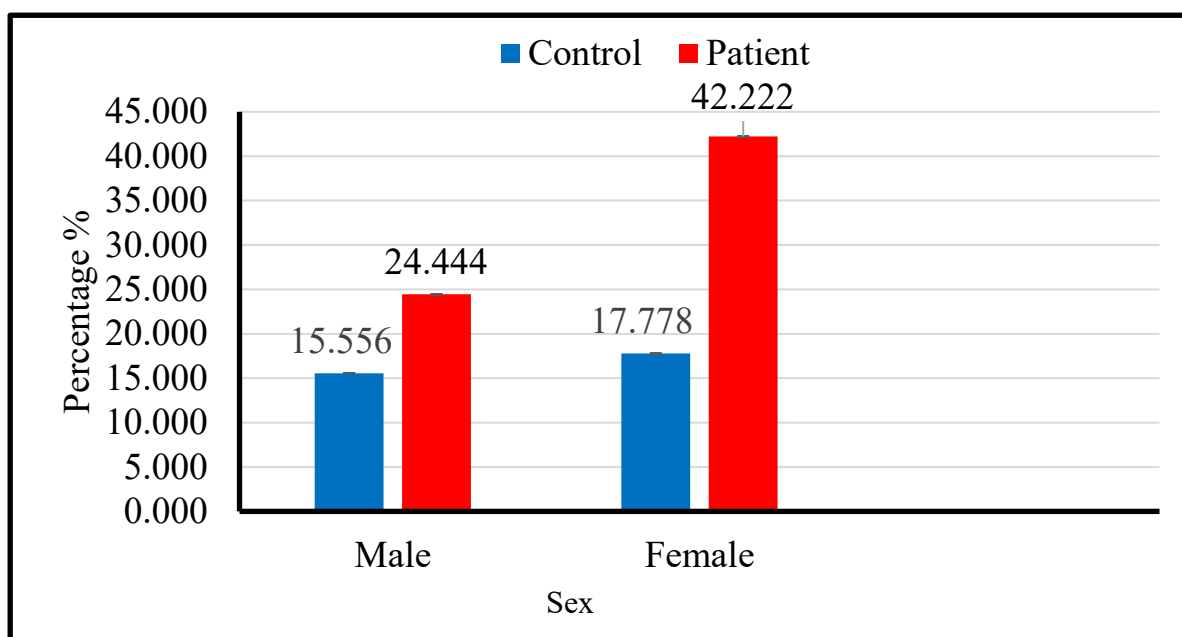


Figure 3. Zonulin level (ng/ml) in Celiac disease patients and control groups according to sex.

* means significant difference at ($P \leq 0.05$) (Means $\pm$ SE)

Figure (4) reveals a significant decrease ($P \leq 0.05$) in patients aged 1-6, 7-12, and 13-18 years (13.286 ± 0.684 , 15.622 ± 0.410 , and 15.588 ± 0.347) compared to control groups (19.966 ± 0.393 , 21.044 ± 0.694 , 21.177 ± 0.359).

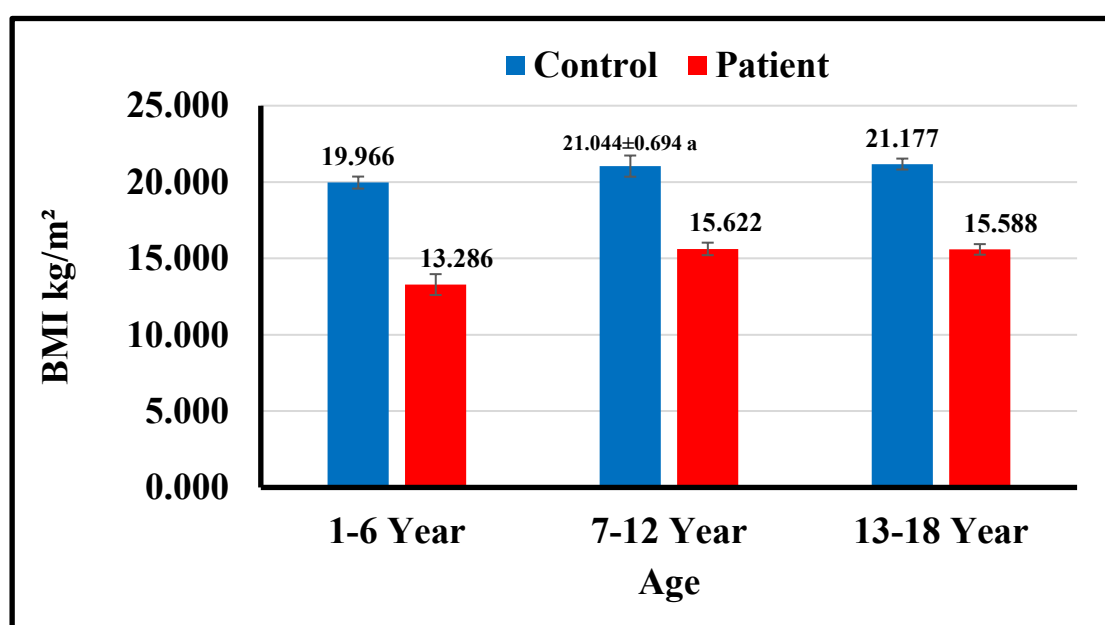


Figure 4. Zonulin level (ng/ml) in Celiac disease patients and control groups according to BMI

The findings in figure (5) revealed a significant increase ($P \leq 0.05$) in Celiac disease patients in place of living (43.333 , 23.333) compared with control groups (17.778 , 15.556). Also the results show a significant decrease ($P \leq 0.05$) at rural groups (23.333) compared with urban groups (43.778).

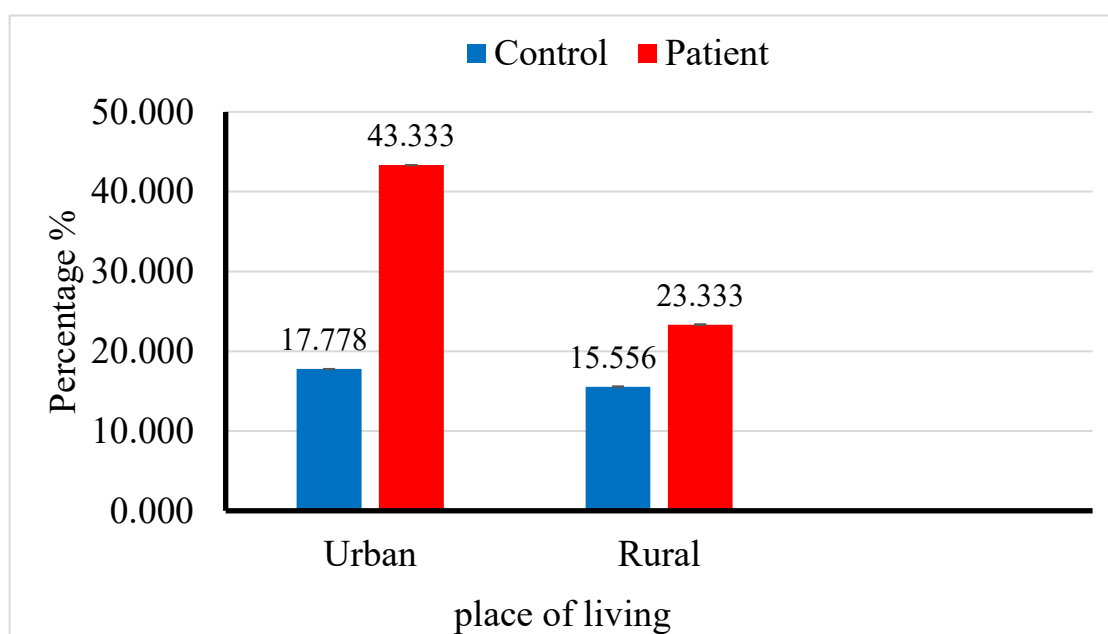


Figure 5. Zonulin level (ng/ml) in Celiac disease patients and control groups according to place of living.

* means significant difference at ($P \leq 0.05$) (Means $\pm$ SE)

4. Discussion

Figure (1) indicates a substantial increase ($P < 0.05$) in Zonulin levels in Celiac disease patients compared to the control group. This suggests that intestinal permeability contributes to celiac disease. Elevation of zonulin is consistent with disturbance of intestinal epithelial tight junctions permitting the transit of gluten-derived peptides, notably gliadin, into the submucosa (Fasano, 2020; Ohlsson *et al.*, 2020)⁸⁻¹⁰. This enhanced permeability promotes immune activation and inflammation, resulting in the mucosal damage (eg, villous atrophy) reflected by celiac disease (Lebwohl *et al.*, 2021; Sapone *et al.*, 2021)⁹⁻¹¹. This finding correlates well with more recent reports about the role of gliadin and zonulin in celiac disease. Fasano (2020)⁸ reported that zonulin is a key regulator of intestinal barrier function and is implicated in many autoimmune diseases, including celiac disease. Barbara *et al.* (2021) also reported an increase in the concentration of zonulin in untreated coeliac patients, which was decreased after 6 months of a gluten-free diet. This supports the current results and suggests that zonulin may be a potential useful biomarker reflecting disease activity. Furthermore, Geller *et al.* (2025)²⁴ emphasises its possible clinical application and links this to the extent of mucosal damage, further mechanistic studies support a biological basis for these findings. Macura *et al.* (2024) demonstrated that gliadin triggers the release of zonulin through specific cellular pathways, leading to increased intestinal permeability. This is in line with the current findings where the high levels of zonulin may suggest continued exposure to gluten and hence barrier function. Moreover, recent studies indicate that gut microbiota is also involved in the expression of zonulin. For example, Leeming *et al.* (2023)²⁵ showed that a gut dysbiosis might drive this intestinal permeability. Therefore, the increased plasma zonulin levels found in this study may be indicative of a synergistic action by dietary gluten, immune stimulation and gut dysbiosis.

The Zonulin levels are significantly increased ($P \leq 0.05$) in Celiac disease patients aged 1-6, 7-12 and 13-18 as compared to control groups (Figure 2). Recent research have connected intestinal permeability to celiac disease initiation and progression. The CD-GEMM cohort found that serum zonulin levels may rise before celiac disease autoimmunity in genetically at-risk children, suggesting that barrier dysfunction may precede or accompany disease onset rather than be a secondary consequence of mucosal injury (DaFonte *et al.*, 2024)¹³. This supports the current finding, especially the significant elevation observed in the 1–6 year group, where early immune activation and immature intestinal barrier regulation may amplify zonulin release. Recent

reviews and experimental evidence also support the present findings and suggest that exposure to gliadin can stimulate release of zonulin, leading to disassembly of tight junctions and increased intestinal permeability. This mechanism allows for increased translocation of luminal antigens possibly supporting innate and adaptive immune responses in celiac disease (Caio *et al.*, 2019; Fasano, 2020)¹⁴⁻¹². The higher levels of zonulin in patients compared to controls may reflect the active interplay between gluten exposure, epithelial barrier disruption, and immune-mediated mucosal inflammation. Recent data in pediatrics further suggest that environmental exposures including antibiotic use, composition of gut microbiota, infections, and dietary patterns may influence levels of zonulin, which may vary by age (DaFonte *et al.*, 2024)¹³. Thus, the age gradient seen in the current study may reflect both biological maturation and environmental modulation of intestinal permeability. However, not all studies fully agree with the use of zonulin as a reliable diagnostic biomarker.

Figure 3 reveals a significant increase ($P \leq 0.05$) in patients aged 7-12y and 13-18y with different sexes compared to control groups. Recent research confirm this view. King *et al.* (2020)¹⁵ and Singh *et al.* (2022)¹⁶ reported that celiac disease is significantly more common in females. The higher prevalence is often explained by sex differences in the regulation of the immune system, with females displaying stronger humoral and cellular immune responses. Moreover, recent data suggest that sex hormones play an important role in the modulation of immune responses in celiac disease. Estrogen has been shown to enhance immune activation and antibody production, which may contribute to the higher biomarker levels observed in female patients (Zanini *et al.*, 2022)¹⁸. In agreement with these findings, a recent large-scale epidemiological study by Lebwohl *et al.* (2018)⁹ confirmed that females not only have a higher risk of developing celiac disease but also tend to present with more pronounced serological and clinical abnormalities. Similarly, Bai & Ciacci (2017) reported that female patients often exhibit more severe nutrient deficiencies and extraintestinal manifestations, which may contribute to the elevated physiological and biomarker values observed. Some studies have reported less pronounced sex differences. Green *et al.* (2015)²⁶ suggested that while celiac disease is more frequently diagnosed in females, the severity of the disease does not always differ significantly between sexes when controlled for factors such as age and duration of disease.

Figure (4) indicates a substantial reduction ($P \leq 0.05$) in patients aged 1-6, 7-12, and 13-18 compared to control groups. The present findings are in strong agreement with recent study published over the last decade that consistently identifies growth impairment and low BMI as common features of celiac disease, particularly in untreated patients. Children with celiac disease classically present with malnutrition and low BMI due to chronic malabsorption and intestinal damage (Caio *et al.*, 2019)¹⁴. Also, more recent evidence by Mearin *et al.* (2022)¹⁹ confirmed that children with untreated celiac disease are often characterized by loss of BMI and growth retardation and emphasized that nutritional deficits are among the first and most sensitive signs of the disease. Also, a recent clinical study of Auricchio *et al.* (2023)²² showed that the BMI is significantly lower in new celiac patients than in healthy controls, especially in the pediatric populations, and that this difference is closely related to the degree of damage of the intestinal villi. Recent epidemiological studies suggest that a subgroup of celiac patients may have normal or even increased BMI, especially in developed countries. As per Lebwohl *et al.* (2018)²⁰ and Norsa *et al.* (2022)²⁷, overweight and obesity are increasingly recognised in patients with celiac disease, challenging the traditional view of the disease being associated only with malnutrition. This difference could be due to early diagnosis, increased awareness and widespread adoption of gluten-free diets, which could lead to normalisation or even weight gain in some patients.

Celiac disease patients had a substantial increase ($P \geq 0.05$) in their location of residence compared to control groups (figure 5). The study found a substantial reduction ($P \leq 0.05$) in rural groups compared to urban groups. Urban people are more likely to have celiac disease due to more knowledge, better health care, and more advanced diagnostics. Lebwohl *et al.* (2018) reported that celiac disease prevalence seems more common in developed and urbanized areas, at least in part, due to better detection and screening practices. Likewise,

King *et al.* (2020) pointed out that the lifestyle and dietary changes associated with urbanization have a prominent role in the epidemiology of autoimmune diseases, including celiac disease. In addition, environmental factors such as hygiene, microbiome composition, and early-life exposures have been implicated in modulating immune responses. Caio *et al.* (2019) suggest that dietary patterns and antibiotic exposure are more common in urban areas and can modify the gut microbiota and consequently influence the onset and progression of celiac disease. Bai *et al.* (2023) propose that rural environmental exposures may have a role in immune regulation and reduction of autoimmune activation. Some studies report conflicting findings. Recent epidemiological studies indicate that the apparent lower prevalence of celiac disease in rural populations may not reflect a true difference in disease occurrence but rather underdiagnosis or limited screening. Rubio-Tapia *et al.* (2023)²⁸ and subsequent updates emphasize that celiac disease is often underrecognized in less developed or rural regions due to limited diagnostic testing. Singh *et al.* (2018)²⁹ noted that when systematic screening is applied, the prevalence of celiac disease in rural areas approaches that of urban populations.

5. Conclusions

In the current investigation, serum zonulin levels were significantly higher in patients with celiac disease than in healthy controls, with higher levels observed in female patients than in male patients. These findings suggest that altered zonulin levels may be associated with celiac disease and may reflect changes in intestinal barrier permeability. Further studies are needed to determine the diagnostic and clinical significance of zonulin in celiac disease.

Acknowledgments

I would like to thank all the patients who participated in our study.

References:

1. Rodríguez-Martín, L., Vaquero, L., & Vivas, S. (2020). New celiac disease biomarkers. *Revista Española de Enfermedades Digestivas*, 112, 7217. <https://doi.org/10.17235/reed.2020.7217/2020>.
2. Smithson, G., Siegelman, J., Oki, T., Maxwell, J., & Leffler, D. (2021). The evolving landscape of biomarkers in celiac disease: Leading the way to clinical development. *Frontiers in Immunology*, 12, 665756.
3. Topa, M., Sanders, D., & Elli, L. (2022). Biomarkers for the diagnosis and monitoring of celiac disease: can you count on me? *Current Opinion in Gastroenterology*, 38, 263–269.
4. Mir, B., Majeed, T., Singh, A., Rajput, M., Kumar, A., & Chauhan, A. (2022). Emerging biomarkers for screening and management of celiac disease. *BioMed Research International*, 2022, 2756242. <https://doi.org/10.1155/2022/2756242>.
5. Heckman, L. K. W., DeBoer, M., & Fasano, A. (2020). Zonulin as a potential putative biomarker of risk for shared type 1 diabetes and celiac disease autoimmunity. *Diabetes/Metabolism Research and Reviews*, 36(5), e3309.
6. Mohammadi-Kordkhayli, M., Mousavi, M. J., Camara-Lemarroy, C. R., Noorbakhsh, F., & Saboor-Yaraghi, A. (2024). Elucidating the significance of zonulin in the pathogenesis of chronic inflammatory disorders: Emphasis on intestinal barrier function and tight junction regulation. *Current Medicinal Chemistry*, 32(30), 6547 – 6562.
7. Rabiee, R., Mahdavi, R., & Nikniaz, Z. (2024). Serum zonulin level as a novel approach in diagnosis and follow-up of patients with celiac disease. *Nutrition Clinique et Métabolisme*. Volume 38, Issue 1, March 2024, Pages 36-43. <https://doi.org/10.1016/j.nupar.2023.11.002>
8. Fasano, A. (2020). All disease begins in the (leaky) gut: Role of zonulin-mediated gut permeability in the pathogenesis of some chronic inflammatory diseases. *F1000Research*, 9, 69.
9. Lebowitz, B., Sanders, D. S., & Green, P. H. R. (2021). Coeliac disease. *The Lancet*, 391(10115), 70–81. [https://doi.org/10.1016/S0140-6736\(21\)00763-0](https://doi.org/10.1016/S0140-6736(21)00763-0)
10. Ohlsson, B., Orho-Melander, M., & Nilsson, P. M. (2020). Higher levels of serum zonulin may rather be associated with increased risk of obesity and hyperlipidemia than with gastrointestinal symptoms. *International Journal of Molecular Sciences*, 21(14), 4828. <https://doi.org/10.3390/ijms21144828>
11. Sapone, A., Bai, J. C., Ciacci, C., Dolinsek, J., Green, P. H. R., Hadjivassiliou, M.

12. Fasano, A. (2021). Spectrum of gluten-related disorders: Consensus on new nomenclature and classification. *BMC Medicine*, 10, 13.
13. DaFonte, T. M., Valitutti, F., Kenyon, V., Locascio, J., Montuori, M., Francavilla, R., Passaro, T., Crocco, M., Norsa, L., Piemontese, P., Baldassarre, M., Fasano, A., Leonard, M. M., & CD-GEMM Study Group. (2024). *Zonulin as a biomarker for the development of celiac disease*. *Pediatrics*, 153(1), e2023063050. <https://doi.org/10.1542/peds.2023-063050>
14. Caio, G., Volta, U., Sapone, A., Leffler, D. A., De Giorgio, R., Catassi, C., & Fasano, A. (2019). *Celiac disease: A comprehensive current review*. *BMC Medicine*, 17, 142.
15. King, J. A., Jeong, J., Underwood, F. E., Quan, J., Panaccione, N., Windsor, J. W., Coward, S., deBruyn, J., Ronskley, P. E., Shaheen, A.-A., Quan, H., & Kaplan, G. G. (2020). *Incidence of celiac disease is increasing over time: A systematic review and meta-analysis*. *The American Journal of Gastroenterology*, 115(4), 507–525.
16. Singh, P., Arora, S., Lal, S., Strand, T. A., & Makharia, G. K. (2022). *Risk of celiac disease in the first- and second-degree relatives of patients with celiac disease: A systematic review and meta-analysis*. *Clinical Gastroenterology and Hepatology*, 20(3), e514–e532.
17. Mearin, M. L., Agardh, D., Antunes, H., Al-Toma, A., Auricchio, R., Castillejo, G., Cellier, C., Dolinsek, J., Donat, E., Dupont, C., Fuchs, V., Koletzko, S., Korponay-Szabó, I., Kramna, L., Langendam, M., Mummert, E., Polanco, I., Riznik, P., Romanos, J., ... Husby, S. (2022). *ESPGHAN position paper on management and follow-up of children and adolescents with celiac disease*. *Journal of Pediatric Gastroenterology and Nutrition*, 75(3), 369–386.
18. Zanini, B., Villanacci, V., De Leo, L., Lanzarotto, F., Ricci, C., Lanzini, A., & Caselani, F. (2022). *Sex-related differences in celiac disease*. *Nutrients*, 14(4), 789.
19. Mearin, M. L., Agardh, D., Antunes, H., Al-Toma, A., Auricchio, R., Castillejo, G., Cellier, C., Dolinsek, J., Donat, E., Dupont, C., Fuchs, V., Koletzko, S., Korponay-Szabó, I., Kramna, L., Langendam, M., Mummert, E., Polanco, I., Riznik, P., Romanos, J., ... Husby, S. (2022). *ESPGHAN position paper on management and follow-up of children and adolescents with celiac disease*. *Journal of Pediatric Gastroenterology and Nutrition*, 75(3), 369–386.
20. Lebowitz, B., Sanders, D. S., & Green, P. H. R. (2018). *Coeliac disease*. *The Lancet*, 391(10115), 70–81. [https://doi.org/10.1016/S0140-6736\(17\)31796-8](https://doi.org/10.1016/S0140-6736(17)31796-8)
21. Bai, J. C., & Ciacci, C. (2017). *World Gastroenterology Organisation global guidelines: Celiac disease February 2017*. *Journal of Clinical Gastroenterology*, 51(9), 755–768.
22. Auricchio, R., Mandile, R., Del Vecchio, M. R., Scapaticci, S., Galatola, M., Maglio, M., & Troncone, R. (2023). *Growth and nutritional status in children with celiac disease*. *Nutrients*, 15(2), 412.
23. Barbara, G., Barbaro, M. R., Fuschi, D., Palombo, M., Falangone, F., Cremon, C., Marasco, G., & Stanghellini, V. (2021). *Inflammatory and microbiota-related regulation of the intestinal epithelial barrier*. *Frontiers in Nutrition*, 8, 718356.
24. Geller, S., et al. (2025). *Markers of enterocyte damage in celiac disease in children*. *Frontiers in Pediatrics*.
25. Leeming, E. R., Johnson, A. J., Spector, T. D., & Le Roy, C. I. (2023). *The role of the gut microbiome in intestinal permeability and human health*. *Gut Microbes*, 15(1), 2156816.
26. Green, P. H. R., Lebowitz, B., & Greywoode, R. (2015). *Celiac disease*. *Journal of Allergy and Clinical Immunology*, 135(5), 1099–1106.
27. Norsa, L., Shamir, R., Zevit, N., Verduci, E., Hartman, C., Ghisleni, D., Riva, E., Giovannini, M., & Scaramuzza, A. E. (2022). *Overweight in celiac disease: Prevalence, clinical characteristics, and management*. *Nutrients*, 14(7), 1434.
28. Rubio-Tapia, A., Hill, I. D., Semrad, C., Kelly, C. P., Calderwood, A. H., & Murray, J. A. (2023). *American College of Gastroenterology guidelines update: Diagnosis and management of celiac disease*. *The American Journal of Gastroenterology*, 118(1), 59–76.
29. Singh, P., Arora, A., Strand, T. A., Leffler, D. A., Catassi, C., Green, P. H. R., Kelly, C. P., Ahuja, V., & Makharia, G. K. (2018). *Global prevalence of celiac disease: Systematic review and meta-analysis*. *Clinical Gastroenterology and Hepatology*, 16(6), 823–836.e2.