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# Association Between Fecal GIP Concentrations and Tissue Transglutaminase in Celiac Disease Patients

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## Abstract

**Introduction.** Celiac disease (CD) is an autoimmune disorder triggered by gluten ingestion in genetically predisposed individuals. Gluten immunogenic peptides (GIP) in feces and tissue transglutaminase (tTG) are key biomarkers for monitoring gluten intake and immune response, respectively. Despite the increasing use of GIP for assessing gluten-free diet (GFD) adherence, its correlation with tTG remains unclear. Understanding their relationship could enhance CD monitoring.

**Aim.** To evaluate fecal GIP concentrations in CD patients, examine their correlation with tTG levels, and assess the utility of combining these biomarkers for CD management.

**Methods.** This cross-sectional study included 60 CD patients adhering to a GFD and 10 healthy controls. Fecal and serum GIP levels were quantified using ELISA tests, and tTG concentrations were measured. Statistical analyses included Mann-Whitney U tests for group comparisons and Spearman's rank correlation for assessing relationships between biomarkers.

**Results.** Median fecal GIP concentration in CD patients was significantly lower (39.0 ng/g) compared to controls (474.2 ng/g; $p<0.001$), confirming GFD adherence. Similarly, serum GIP was lower in the CD group ($p<0.001$). No significant correlation was found between GIP and tTG levels (Rho=0.114, $p=0.387$), indicating they measure distinct aspects of CD activity.

**Conclusion.** This study specifically evaluated fecal GIP concentrations in patients with celiac disease and their correlation with tTG levels. Our findings indicate no significant correlation, demonstrating that these biomarkers assess different aspects of disease activity. This study confirms the sensitive nature of GIP for detecting gluten intake and tTG's role in reflecting immune response and mucosal damage. Hence, the integrated use of these biomarkers, as suggested by our results, can improve the management and monitoring of celiac disease, providing a more precise assessment of dietary adherence and immune activity.

## Introduction

Celiac disease (CD) is a chronic autoimmune disorder triggered by the ingestion of gluten - a protein found in wheat, rye, and barley. The disease occurs in genetically predisposed individuals and is mediated by tissue transglutaminase (tTG), a ubiquitous enzyme that serves as the primary autoantigen in CD. The resulting immune response leads to structural damage in the small intestine, characterized by villous atrophy, crypt hyperplasia, and infiltration of intraepithelial lymphocytes, disrupting nutrient absorption and contributing to a range of gastrointestinal and systemic symptoms (1,2).

The prevalence of CD has increased significantly over recent decades and is now estimated at 1–2% globally (3). This rise is largely attributed to enhanced diagnostic capabilities, including the availability of highly sensitive and specific serological tests such as those detecting tTG antibodies. Improved screening has also facilitated the identification of subclinical cases, even among elderly populations (4). Environmental and dietary factors, including increased gluten consumption (up to 20 g/day in certain populations) and changes in gluten quality due to agricultural innovations, are thought to contribute to this trend (5). Moreover, the "hygiene hypothesis" suggests that reduced exposure to pathogens in industrialized societies has led to a dysregulated immune response, further increasing the prevalence of autoimmune conditions like CD (6).

The mainstay of CD management is strict, lifelong adherence to a gluten-free diet (GFD), which alleviates symptoms, promotes intestinal healing, and reduces the risk of complications such as refractory CD and small intestinal lymphoma (7). However, maintaining adherence remains challenging due to the ubiquitous presence of gluten in processed foods and the social and economic burdens associated with dietary restrictions. The availability of gluten-free products has increased dramatically over the past five years, with such products now widely accessible in major supermarkets, health food stores, and online retailers (8). Nonetheless, these products remain significantly more expensive than their gluten-containing counterparts (9,10).

The assessment of GFD compliance traditionally relies on self-reports, dietary interviews, serological tests (e.g., anti-tTG antibodies), or small bowel biopsies. However, these methods have significant limitations. Self-reports are often unreliable due to intentional or unintentional inaccuracies, while serological markers correlate poorly with mucosal healing (10). Small bowel biopsies, although the gold standard for assessing mucosal recovery, are invasive and not routinely performed, particularly in asymptomatic patients who show clinical improvement (11,12).

Gluten immunogenic peptides (GIP) are a promising new biomarker for monitoring dietary adherence. These peptides, derived from immunotoxic fragments of gluten such as the α-gliadin-33-mer, are resistant to enzymatic digestion and are excreted intact in stool or urine. Their detection directly reflects gluten ingestion, providing an objective measure of dietary transgressions (13,14).

Research has shown that fecal GIP concentrations can remain detectable for up to four days after gluten ingestion, making them a highly sensitive indicator of recent dietary lapses (13). For example, one study demonstrated that 30% of CD patients on a GFD for at least one year had detectable fecal GIP, indicating dietary noncompliance. In comparison, serological tests identified dietary infractions in only 18% of patients, underscoring the superior sensitivity of GIP detection (14).

Despite these advances, the relationship between fecal GIP concentrations and traditional markers of CD activity, such as tTG, remains unclear. Investigating this correlation is critical to understanding how GIP testing can complement existing diagnostic tools in CD management.

## Aim

This study aims to:

- Assess fecal GIP concentrations in CD patients.
- Analyze the relationship between fecal GIP levels and tTG concentrations.

## Methods

### Study Design

This observational, cross-sectional study was conducted at the Clinical Nutrition Outpatient Department of the Clinical Hospital Center Zagreb over a 12-month period. The study involved a total of 70 participants, including 60 adult patients diagnosed with celiac disease and a control group of 10 healthy volunteers who eat food containing gluten.

### Participants

The inclusion criteria required participants to be over 18 years of age, have a confirmed diagnosis of celiac disease, and provide written informed consent. Exclusion criteria encompassed individuals under 18 years of age, as well as those diagnosed with gastrointestinal diseases such as diverticulitis, enterocolitis, or ischemic colitis. Additionally, patients with liver dysfunction (e.g., cirrhosis or active hepatitis), chronic kidney disease, severe hypertension, coronary artery disease, or peripheral arterial disease were excluded. Other exclusion criteria included hematological, malignant, or autoimmune disorders, as well as pregnancy.

### Procedure and Instruments

Demographic and anthropometric data were gathered through a structured questionnaire, medical records, and direct measurements. Blood samples were analyzed for complete blood count and routine biochemical parameters. Fecal and urinary gluten immunogenic peptides were detected using the iVYLISA GIP ELISA test (Biomedal SL).

### Ethics

The study protocol was approved by the Ethics Committee of the Clinical Hospital Center Zagreb (Approval No. 02/21 AG, Class: 8.1-19/153-2). All participants provided written informed consent prior to enrolment, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

### Statistics

Data were presented in tables and figures. The normality of data distribution was evaluated using the Kolmogorov-Smirnov test, which revealed that most continuous variables did not follow a normal distribution. As a result, nonparametric tests were used in further analyses. Differences between continuous variables in independent groups were analyzed using the Mann-Whitney U test, and significant results were displayed with Box-and-Whisker plots. Fisher's exact test was employed for categorical data. Spearman's rank correlation coefficient (Rho) was used for correlational analyses.

A significance level of $p<0.05$ was used for all analyses. Statistical analyses were performed using MedCalc® Statistical Software version 20.022 (MedCalc Software Ltd, Ostend, Belgium; https://www.medcalc.org; 2021).

Power analysis for Fisher's exact test was based on pilot study results, estimating a 100% positive GIP biomarker detection rate among non-compliant participants and a 30% detection rate among compliant participants. For a power of 90% and a significance level ($\alpha$) of 0.05, with a case-to-control ratio of 6:1, a minimum of 35 participants was required (30 in the study group and 5 in the control group). Power analysis was performed using MedCalc® Statistical Software version 20.022.

## Results

Descriptive statistics of anthropometric indicators for the included celiac disease patients (N=60) are presented in Table 1.

Table 1. **Descriptive statistics of anthropometric indicators for the included celiac disease patients (N=60)**

| | Arithmetic Mean | Standard Deviation | Min | Max | Centile | | |
|---|---|---|---|---|---|---|---|
| | | | | | 25. | Median | 75. |
| Age | 45.52 | 12.91 | 19.00 | 77.00 | 3725 | 44.00 | 55.00 |
| Body Mass (kg) | 67.68 | 15.34 | 45.60 | 128.50 | 57.55 | 62.70 | 75.80 |
| Height (cm) | 168.56 | 8.93 | 143.00 | 188.00 | 163.00 | 169.00 | 174.00 |
| Body Mass Index (BMI) (kg/m²) | 23.73 | 4.40 | 18.10 | 37.80 | 20.80 | 22.60 | 25.75 |
| Lean Mass (kg) | 47.74 | 9.86 | 37.30 | 82.90 | 41.85 | 44.50 | 49.50 |
| Muscle Mass (kg) | 45.32 | 9.39 | 35.40 | 78.80 | 39.75 | 42.20 | 47.00 |
| Bone Mass (kg) | 2.42 | 0.47 | 1.90 | 4.10 | 2.10 | 2.30 | 2.50 |
| Basal Metabolic Rate (BMR) (kcal) | 1429.37 | 295.14 | 1111.00 | 2556.00 | 1260.00 | 1367.00 | 1458.00 |
| Visceral Fat Index | 5.54 | 3.70 | 1.00 | 18.00 | 3.00 | 5.00 | 7.00 |

The median Body Mass Index (BMI) of our study participants, which was recorded at 22.6 kg/m² with an interquartile range of 20.8 to 25.8, classifies them within the "Normal Weight" category according to the World Health Organization standards. This categorization is defined for BMIs ranging from 18.5 to 24.9 kg/m² (15). The median muscle mass of the participants was 42.2 kg (39.8–47.0), which is within the range for healthy adults, considering variations due to age, gender, and body size. Similarly, the median basal metabolic rate (BMR) of 1367.0 kcal (1260.0–1458.0) is in line with expected values for a population of similar demographic characteristics, factoring in the influences of age, sex, and muscle mass. Both measurements indicate a healthy physiological status among the participants, similar to the median visceral fat index value, which was within the acceptable range (<13) and amounted to 5.0 (3.0–7.0). These results shows that physical condition of the participants does not exhibit deviations that would likely impact the study's outcomes related to celiac disease biomarkers.

Descriptive statistics of biomarkers in celiac disease patients (N=60) are presented in Table 2 and Figures 1 - 3. The median serum GIP concentration was 0.78 (0.78–0.78) /mL, while the median fecal GIP concentration was 39.0 (39.0–39.0) /g. The median tissue transglutaminase (tTg) concentration was 5.20 (0.85–14.70). Serum GIP values showed a perfect correlation with fecal GIP values (Rho=1.000, $p<0.001$), allowing these biomarker concentrations to be treated as a single variable.

Table 3 presents an analysis of differences in the distribution of GIP biomarker results between the study and control groups, treated as categorical variables (positive vs. negative findings). Positive results were defined as any detectable GIP concentrations using the applied analytical method. The control group, consisting of participants not adhering to a gluten-free diet, exhibited a significantly higher frequency of positive results.

These analyses reveal significantly higher GIP biomarker concentrations in participants from the control group, who were not on a gluten-free diet ($p<0.001$). This finding supports the conclusion that the study group, comprising celiac disease patients, adhered to a gluten-free dietary regimen, unlike the control group (Table 4).

Table 2. **Descriptive statistics of biomarkers in celiac disease patients (N=60)**

| | Arithmetic Mean | Standard Deviation | Min | Max | Centile | | |
|---|---|---|---|---|---|---|---|
| | | | | | 25. | Median | 75. |
| GIP/mL (dil 10) | 1.74 | 2.27 | 0.78 | 9.95 | 0.78 | 0.78 | 0.78 |
| GIP/g in stool | 86.96 | 113.43 | 39.00 | 497.70 | 39.00 | 39.00 | 39.00 |
| tTg (IgA) | 111.12 | 634.52 | 0.85 | 4861.10 | 0.85 | 5.20 | 14.70 |

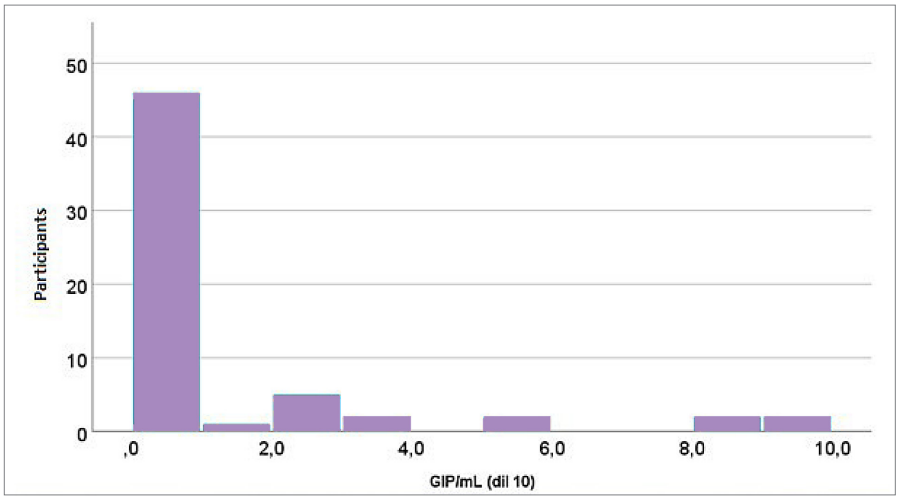


Figure 1. **Distribution of measured GIP concentrations (GIP/mL, dilution 10) in the study group (celiac disease patients)**

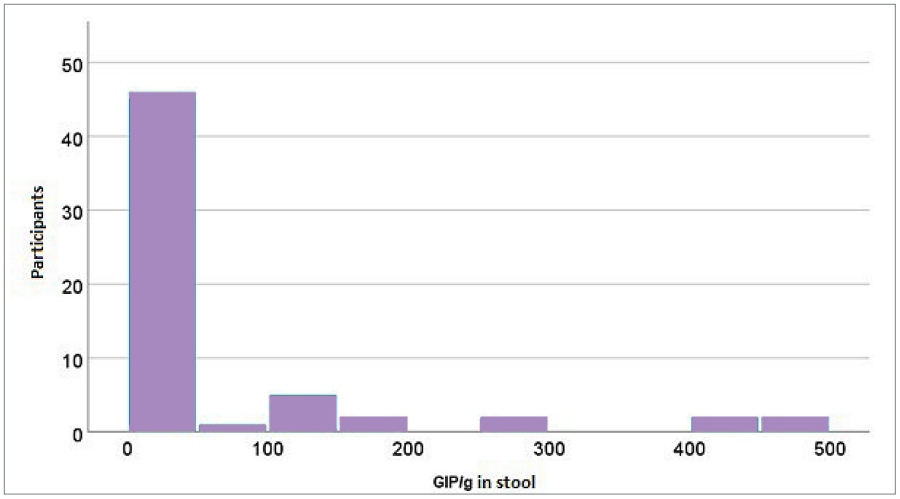


Figure 2. **Distribution of measured GIP concentrations (GIP/g) in fecal samples from the study group (celiac disease patients)**

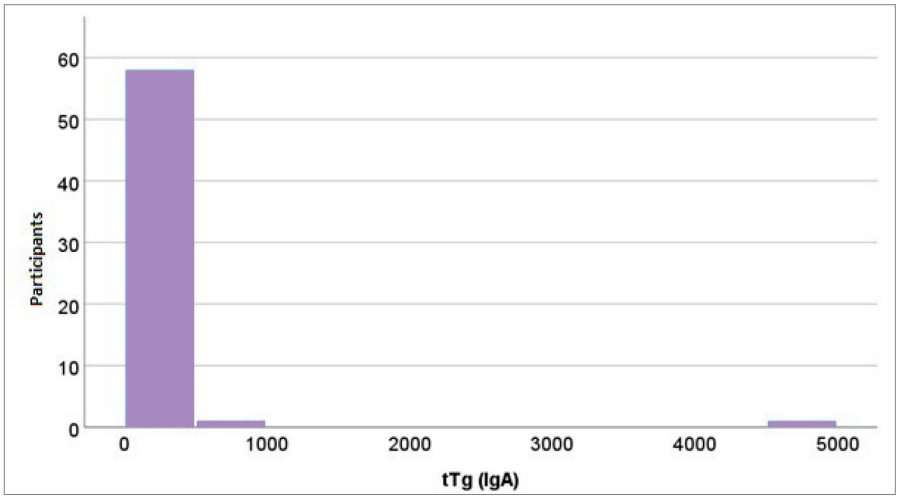


Figure 3. **Distribution of measured tissue transglutaminase concentrations in the study group (celiac disease patients)**

Table 3. **Analysis of differences in GIP biomarker distribution between the study and control groups: Fisher's exact test**

| | | Groups | | | | *p* |
|---|---|---|---|---|---|---|
| | | Study group | | Control | | |
| | | N | % | N | % | |
| GIP / mL (dil 10) categories | Normal Level | 47 | 78.3 | 0 | 0 | <0.001 |
| | Elevated | 13 | 21.7 | 10 | 100 | |
| GIP / g in stool categories | Normal Level | 47 | 78.3 | 0 | 0 | <0.001 |
| | Elevated | 13 | 21.7 | 10 | 100 | |

Table 4. **Analysis of differences in GIP serum and GIP fecal concentrations between the study and control groups**

| Groups | | Min | Max | Centile | | | Mann-Whitney U | Z | *p* |
|---|---|---|---|---|---|---|---|---|---|
| | | | | 25. | Median | 75. | | | |
| GIP / mL (dil 10) | Study group | 0.78 | 9.95 | 0.78 | 0.78 | 0.78 | 21.000 | -5.533 | <0.001 |
| | Control | 4.39 | 14.22 | 7.80 | 9.49 | 13.16 | | | |
| GIP / g in stool | Study group | 39.00 | 497.70 | 39.00 | 39.00 | 39.00 | | | |
| | Control | 219.43 | 711.23 | 390.38 | 474.20 | 658.32 | | | |

The correlation between GIP biomarker concentrations and tissue transglutaminase levels using Spearman's rank correlation coefficient (Rho) indicate no significant correlation between GIP concentrations and tissue transglutaminase (Rho=0.114, *p*=0.387).

## Discussion

The results of this study demonstrate that the median fecal concentrations of gluten immunogenic peptides in celiac disease patients were significantly lower compared to the control group not adhering to a gluten-free diet (Table 5). Similarly, serum GIP concentrations were also significantly lower in the study group (Table 4), confirming the adherence of the study group to the gluten-free diet. These findings underscore the high sensitivity of GIP as a biomarker for detecting gluten intake in individuals with CD. Previous studies have confirmed that GIP is a reliable tool for quantitative monitoring of dietary infractions, even with occasional gluten exposure, surpassing traditional monitoring methods such as serological tests (14-17).

The association between fecal GIP concentrations and tissue transglutaminase was examined using Spearman's rank correlation coefficient (Table 6). The results indicated no significant correlation between these two biomarkers, suggesting that GIP and tTG measure different aspects of disease activity. GIP directly reflects gluten intake, while tTG represents the immune response and potential mucosal damage. These findings are in line with prior studies showing that tTG does not reflect occasional dietary infractions, whereas GIP enables precise detection of actual gluten ingestion, including its effects on intestinal mucosa (13,18,19). Furthermore, studies have confirmed a significant association between fecal GIP detection and future histological changes, highlighting GIP as an extremely valuable biomarker in clinical practice (14,17).

The graphical representation of GIP concentration distributions in serum and stool (Figures 1 and 2) further supports these findings, showing highly uniform values in patients adhering to the GFD and wide variability in the control group. This uniformity among adherent patients corroborates findings demonstrating the high specificity of GIP in detecting gluten in-

take, as consistently observed in studies across various age groups (14,17). For example, research has shown that younger children (<3 years old), under strict parental dietary supervision, exhibit lower GIP levels compared to adolescents, where dietary nonadherence is more common. Conversely, the distribution of tTG (Figure 3) reveals significant variability in this marker among patients but no clear association with GIP concentrations. This confirms that tTG better reflects long-term immune activation, while GIP accurately measures recent gluten exposure (16).

These results highlight the potential complementarity of GIP and tTG in CD monitoring. GIP offers an immediate assessment of gluten intake, while tTG, particularly when combined with other indicators, aids in evaluating immune responses and chronic disease activity. The combination of these two biomarkers could enable more accurate and comprehensive monitoring of CD patients, as supported by studies emphasizing the benefits of integrating both methods into clinical practice (17,19).

Further studies with larger and more diverse cohorts are needed to validate these findings and establish standardized thresholds for GIP and tTG concentrations in clinical settings. Longitudinal research should also explore the relationship between GIP levels, tTG concentrations, and clinical outcomes, such as symptom severity and mucosal healing, to refine their combined use in CD monitoring.

## Limitations

This study has several limitations. The sample size may restrict the generalizability of the findings, particularly when evaluating the variability of GIP and tTG biomarkers across different subgroups. Additionally, while GIP is highly sensitive to recent gluten intake, its short detection window may not capture long-term dietary adherence, unlike tTG, which reflects cumulative immune response. This temporal difference may explain the lack of significant correlation between the two biomarkers. Variability in dietary habits, including inadvertent gluten consumption, could have influenced GIP levels and affected the interpretation of results.

## Conclusion

This study rigorously evaluated fecal GIP concentrations in celiac disease patients and examined their correlation with serum tTG levels. Our findings reveal no significant correlation between these biomarkers, indicating they assess different dimensions of celiac disease pathology. This study confirms GIP's efficacy in detecting gluten intake and tTG's capacity to reflect immune response and mucosal damage. Integrating these biomarkers can thus enhance the accuracy of dietary compliance assessments and improve the monitoring of immune activity in the management of celiac disease. These findings suggest the potential for developing more nuanced strategies that utilize both biomarkers to tailor patient management more effectively.

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# POVEZANOST KONCENTRACIJE FEKALNOG GIP-A S TKIVNOM TRANSGLUTAMINAZOM KOD BOLESNIKA S CELIJAKIJOM


## Sažetak

**Uvod.** Celijakija je autoimuni poremećaj uzrokovan unosom glutena kod genski predisponiranih osoba. Glutenski imunogeni peptidi (GIP) u stolici i tkivna transglutaminaza (tTG) ključni su biomarkeri za praćenje unosa glutena i imunosnog odgovora. Unatoč sve češćoj primjeni GIP-a za procjenu pridržavanja bezglutenske prehrane (GFD), njihova povezanost s tTG-om nije u potpunosti razjašnjena. Bolje razumijevanje ovog odnosa moglo bi unaprijediti praćenje bolesnika s celijakijom.

**Cilj.** Procijeniti koncentracije fekalnog GIP-a kod bolesnika s celijakijom, ispitati njihovu povezanost s razinama tTG-a te procijeniti korisnost kombiniranja ovih biomarkera za upravljanje celijakijom.

**Metode.** Ovo presječno istraživanje uključilo je 60 bolesnika s celijakijom na GFD-u i 10 zdravih kontrola. Koncentracije fekalnih i serumskih GIP-a kvantificirane su testom ELISA, dok su razine tTG-a mjerene serološki. Za statističke analize primijenjeni su Mann-Whitneyjev U-test za usporedbu skupina te Spearmanov koeficijent korelacije za procjenu odnosa između biomarkera.

**Rezultati.** Medijan koncentracije fekalnog GIP-a kod bolesnika s celijakijom bio je značajno niži (39,0 ng/g) u usporedbi s kontrolnom skupinom (474,2 ng/g; $p<0,001$), što potvrđuje pridržavanje BGP-a. Slično tome, serumski GIP bio je niži kod bolesnika s celijakijom ($p<0,001$). Nije utvrđena značajna povezanost između razina GIP-a i tTG-a ($\rho=0,114$, $p=0,387$), što ukazuje na to da mjere različite aspekte aktivnosti bolesti.

**Zaključak.** Ova studija posebno je procjenjivala koncentracije fekalnog GIP-a u bolesnika s celijakijom i njihovu korelaciju s razinama tTG-a. Naši rezultati ne pokazuju značajnu korelaciju, što ukazuje na to da ovi biomarkeri procjenjuju različite aspekte aktivnosti bolesti. Studija potvrđuje osjetljivu prirodu GIP-a za detekciju unosa glutena i ulogu tTG-a u reflektiranju imunosnog odgovora i oštećenja sluznice. Integrirana upotreba ovih biomarkera može poboljšati upravljanje i praćenje celijakije, omogućujući precizniju procjenu pridržavanja prehrane i imunosne aktivnosti.


**Ključne riječi:** celijakija, bezglutenska prehrana, transglutaminaze