Journal for Technology and Science
Homepage : https://journal.antispublisher.com/index.php/IPTEKS
Email : admin@antispublisher.com

e-ISSN : 3047-4337
JOTECH, Vol. 3, No. 4, October 2026
Page 197-204

Evaluating The Effect of Helicobacter Pylori Infection on Iodine Levels and Thyroid Gland Function

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DOI : https://doi.org/10.61796/ipteks.v3i4.619

Sections Info
Article history:
Submitted: June 07, 2026
Final Revised: July 26, 2026
Accepted: August 18, 2026
Published: September 18, 2026
Keywords:
H. pylori
T3
T4
TSH
iodine
thyroid

ABSTRACT

Objective: *The current study aimed to ascertain effect of H. pylori infection on iodine levels and thyroid activity.* ***Method:*** *Present study that included 70 patientssuffering from gastric ulcers as a result of infection with H. pylori. The study also included 70 healthy person as a control group. The ICMA, UniCel® DxI – Beckman Coulter, was used to measure serum TSH, free tetraiodothyronine (T4), and free triiodothyronine (T3). The Sandell–Kolthoff method was used to measure spectrophotometric iodine in the urine. The status of H. pylori was determined using the 13C-(UBT) Test. Forest plot test showed the P-evaluate, OR, and CI for the risk of thyroid disorder among gastric ulcer at each population level (age, sex, T3, T4, TSH and iodine) by Fisher's exact test.* ***Results:*** *Decreased concentration of T3, T4, TSH and iodine in patients (38.08 ng/dL, 3.5µg/dL, 21.95 IU/L and 99.3µg/L respectively), compared to the control (80.77ng/dL, 13.2 µg /dL, 27.53IU/L and 199µg/L respectively). Significant association appeared of T3 (P value=0.034, OR=4.73, CI= 0.16 to 1.18), T4 (P value= 0.045, OR= 1.97, CI= 0.28 to 3.54) and iodine (P value = 0.011, OR= 5.0, CI=0.09 to 0.59).* ***Novelty:*** *Helicobacter pylori infection can lead to damage to iodine levels and thyroid hormones, causing hypothyroidism.*

INTRODUCTION

It is *H. pylori.* the really causes specialist of gastritis in people and as a fundamental figure the basic factor in causing of gastric lymphoma, gastric ulcer, stomach cancer, and mucosa-associated lymphoid tissue type [1]. Moreover, A wide range of extra-gastric issues, including iron deficiency, have been causally linked to infection, constant resistant thrombocytopenic purpura, development hindrance, and Glycosuria [2]. There are a number of important factors that contribute to both hypothyroidism and hyperthyroidism, and may also be linked to the infection the onset of autoimmune thyroid conditions like Graves' disease and Hashimoto's thyroiditis (HT). One intriguing aspect of this connection involves molecular mimicry. It appears that parts of the thyroid and the *H. pylori* antigens may have similarities. This relationship could be an essential part of the underlying mechanisms at work. [3,4,5]. In addition, the association is primarily based on illnesses caused by positive CagA strains, which can be connected to antibodies' cross-reactivity against the protein CagA of bacteria and follicular thyroid cells. [6]. The way this bacteria annihilation prompts diminishing degrees of The presence of thyroid autoantibodies suggests that there may be a connection between infections and autoimmune thyroid disease. This idea opens a door to understanding how our body responds to certain triggers, highlighting the complex interplay between infections and our immune system. It's a reminder of how delicate and intricate our health can be, and how various factors might influence our wellbeing. Exploring this relationship further

can provide insights that are not only fascinating but also incredibly important for those affected by these conditions.[7].

Iodine deficiency remains a concern in many corners of our world, affecting people at all economic development stages. In 2020, around the world, 21 nations have deficient iodine in their weight control plans [8]. The iodine admission is most reduced in Madagascar, It's important to acknowledge that even in Europe, certain nations like Germany and Finland are facing challenges with low iodine intake. This essential nutrient contributes significantly to our health, particularly in the production of hormones T4 and T3 by the thyroid gland, contain iodine, which is a necessary component [9]. These chemicals manage digestion and advance development, improvement, and development, everything being equal, particularly the mind. Tireless iodine lack and in this way low thyroid chemical amalgamation might bring about hypothyroidism [10]. Even though iodine deficiency is a significant global problem, the most effective method for assessing iodine status and the significance of an individual's test results are still up for debate. Studying the role of bacteria infection on the rate the absorption from iodine, which is the energy operating the thyroid gland, still needs to be studied [11]. Therefore, we conducted the present study, , which aims to ascertain how *H. pylori* infection on iodine levels and thyroid activity throughout assessment serum level of T3, T4 and TSH.

MATERIAL AND METHODS

The present investigation is a case-control study included 70 people suffering from gastric ulcers as a result of with *H. pylori* The study also included 70 healthy people as a control group. Samples were abtained from General Teaching Hospital in Al-Diwaniyah, outpatient clinics and laboratories. Patientswith either chronic illness, for example, this connected to digestive system retention change, and immune system disorders were excluded. The rejection standards additionally incorporated the utilization of drugs that kill bacteria and inflammation, PPI2, and the Histamine H2 receptor bad guys as long as 15 days before this work.

Using the ICMA, UniCel® DxI – Beckman Coulter, thyroid stimulating hormone, thyroxine, and free triiodothyronine were measured. The reference values were 34.4 to 5.69 UI/ml, 0.55 to 1.239 ng/dl, and 2.6 to3.8 pg/ ml, respectively. The 13C-Urea Breath Test (IRIS®, Wagner, Analysen Technik Bremen, Germany) was used to determine *H. pylori* status.

Urinary iodine was assessed using the Sandell–Kolthoff method for spectrophotometry. The World Health Organization examined iodine deficiency in their report. (WHO) uses the method of calculating median urinary iodine concentration (UIC) from spot samples for population screening purposes. A median UIC of more than 100 g/L is considered adequate. There are three levels of deficiency: mild (UIC 50–99 g/L), moderate (UIC 20–49 g/l), and severe (UIC 20 g/l). The World Health Organization (WHO) recommends consuming 150 milligrams of iodine daily for adults and 250 milligrams daily for women who are pregnant or breast feeding to ensure adequate iodine status [8].

The analyzed the social sciences for statistical analysis package software version 22.0 (Chicago, IL, USA) with Microsoft excel 2010. Fisher's exact test was used to calculate probability, ratios of odds (OR), and confidence intervals (CI) for demographic and medical variables (age, sex, hormone levels). The variables were considered statistically different if the probability value (*P* value) was less than 0.05. Pearson's linear correlation

was used to measure the strength and nature of the relationship between the studied indicators.

RESULTS

The present investigation is a case-control study included 70 cases of a person suffering from gastric ulcers as a result of having a *H. pylori* infection. The patients ages range from 13 to 67 years, with an average age of 38.5 ± 8.7 years. The majority of the patients were female (56%), as in Table (1). The control group included 70 healthy people whose median age is 36.2 ± 10.2 years, and the percentage of females was equal to the percentage of males. Moreover, we did not find significant differences when comparing the case with the control according to age or gender ($P > 0.05$).

The results of Figure (1) showed a decrease in the concentration of T3, T4, TSH and iodine in patients (38.08 ng/dL, 3.5µg/dL, 21.95 IU/L and 99.3µg/L respectively) compared to the control (80.77ng/dL, 13.2 µg /dL, 27.53IU/L and 199µg/L respectively).

A forest plot depicting the risk's P-value, OR, and CI is shown in Figure 2. of thyroid disorder among gastric ulcer at each population level (age group, sex, T3, T4, TSH and iodine) Fisher's exact test. Significant association appeared of T3 (*P* value=0.034, OR=4.73, CI= 0.16 to 1.18), T4 (*P* value= 0.045, OR= 1.97, CI= 0.28 to 3.54) and iodine (*P* value = 0.011, OR= 5.0, CI=0.09 to 0.59).

To determine the relationships among studied indicators, we use Pearson correlation coefficient (r). The linear correlation showed that age was associated by a weak relationship with males, females and iodine (r= 0.072, 0.047 and 0.111), while it was associated by a strong linear relationship with the TSH was associated with T4, T3 and T4 (r= 0.563, 0.541 and 0.636 respectively). TSH was associated with T4 (r= 0.547) by a strong positive linear relationship, but its relationship was stronger with T3 and iodine (r= 0.607 and 0.704 respectively). However, the strongest positive linear relationships in our study were between T3 and T4 as depicted in Table (2).

The majority of ulcer patients, according to the study's findings, (83%) suffer from mild iodine deficiency, while only two patients suffer from severe iodine deficiency and 10 people suffer from moderate iodine deficiency as shown in Figure (3). We did not find a significant difference in the concentrations of T4 and TSH when distributed according to the degree of iodine deficiency (*P* value >0.05) while a noticeable increase in the concentration of T3 appeared in patients with severe iodine deficiency, which led to the appearance of significant distinctions (P value= 0.051) as shown in Table (3) .Moreover, the concentration of T3 and T4 decreased with low iodine concentration as shown in table (3).

Table 1. Compared control groups and ages and gender for cases.

Properties	Cases	Control	P value
Age range / years	13 – 67	13 – 69	
Mean ± standard deviation	38.5 ± 8.7	36.2 ± 10.2	0.227
Standard error	1.04	1.22	
Gender	**N (%)**	**N (%)**	
Females	39 (56%)	35 (50%)	0.116
Males	31 (44%)	35 (50%)	0.120
Total number	**70**	**70**	

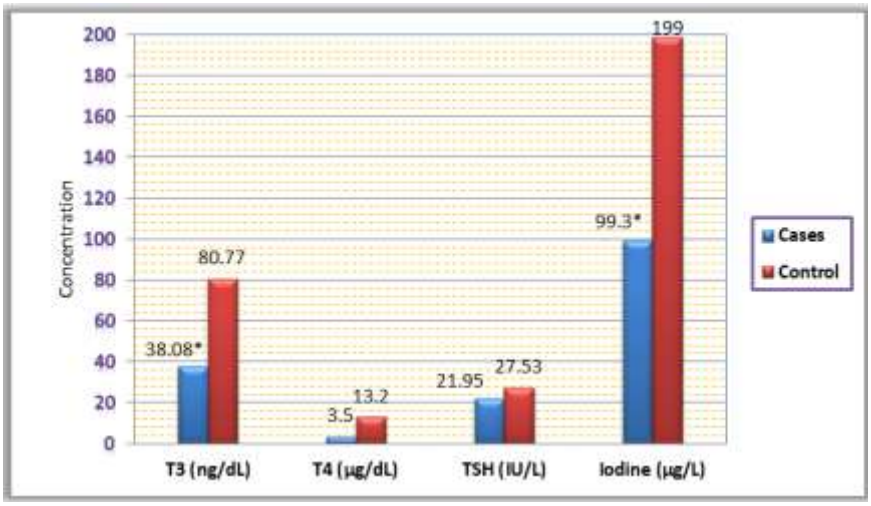

Figure 1. Mean of concentration of T3, T4, TSH and iodine of patients and controls (* significant association in compared with control (*P* value < 0.05).

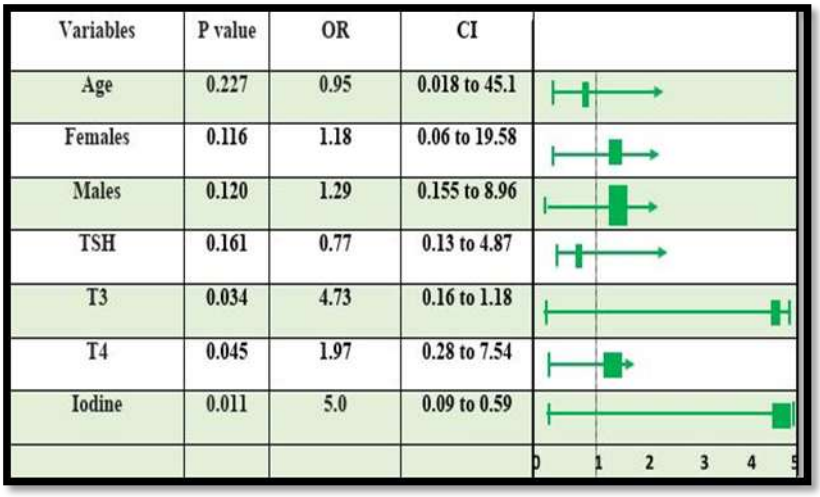

Figure 2. Shows the P-value, (OR) ratio and each demographic level's confidence interval (CI) (age, gender, T3, T4, TSH, and iodine) in our patients are depicted in Figure 2. The OR for each demographic level is depicted in each square, and the square's size indicates precision. The 95% CI for each OR is indicated by the green line intervals and vectors.

Table 2. Pearson correlation coefficient (r) among studied parameters.

Variables	Age	Males	Females	TSH	T3	T4	Iodine
Age	1	0.072	0.047	0.563	0.541	0.636	0.111
Males	0.072	1	0.185	0.361	0.673	0.666	0.586
Females	0.047	0.185	1	0.642	0.604	0.711	0.590
TSH	0.563	0.361	0.642	1	0.607	0.547	0.704
T3	0.541	0.673	0.604	0.607	1	0.848	0.599
T4	0.636	0.666	0.711	0.547	0.848	1	0.700
Iodine	0.111	0.586	0.590	0.704	0.599	0.700	1

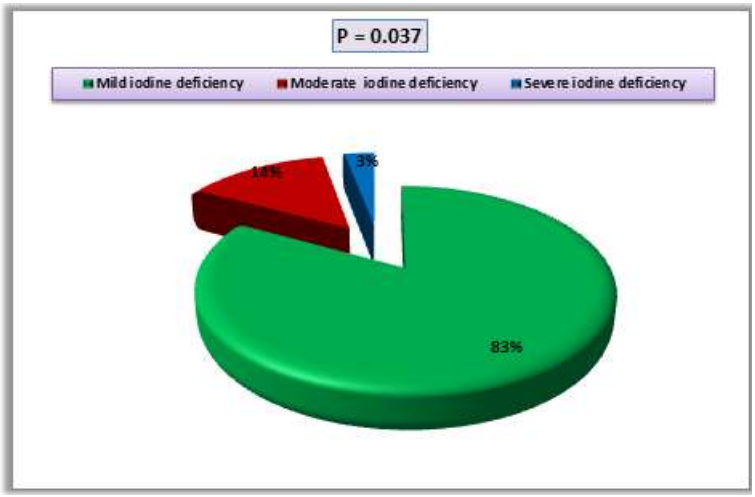

Figure 3. Distribution mean of serum T3, T4 and TSH concentration according to iodine deficiency.

Table 3. Distribution of T3, T4 and TSH according to lack of iodine.

Variables	Mild iodine deficiency	Moderate iodine deficiency	severe deficiency in iodine	P value
	Mean ± SD	Mean ± SD	Mean ± SD	
T3 (ng/dL)	55.6 ± 9.92	46.6 ± 9.74	26.3 ± 12.2	0.015*
T4 (µg/dL)	3.1 ± 0.09	3.8 ± 0.66	3.6 ± 0.81	0.362
TSH (IU/L)	28.7 ± 5.22	18.52 ± 6.51	18.88 ± 4.8	0.121
Total number	58	10	2	

***significant association in compared with control (*P* value < 0.05)**

Discussion

Despite the fact that other studies [14,15] a few studies no correlation was observed between the presence of *H. pylori* and thyroid problems [12,13] have demonstrated a positive correlation. In addition, we demonstrated a direct connection, independent of hormonal status, between *H. pylori* infection and hypothyroidism [15]. The consequences of our review showed that stomach ulcers that subsequent from infection with *H. pylori* cause a diminishing in iodine levels and T3,T4 and TSH. In a previous study, anti-*H. pylori* IgG Graves' disease and Hashimoto's thyroiditis patients had higher levels and breath test results than controls [16,17]. Atrophic autoimmune thyroiditis clearly has a higher prevalence of H. pylori infection, which impairs gastric secretory function. Microsomal antibodies and anti-*H. pylori* IgG levels strongly correlate, suggesting that autoimmune atrophic thyroiditis may be caused by *H. pylori* antigens or that autoimmune function in autoimmune atrophic thyroiditis may make it more likely that H. pylori infection occurs. [16]. Bassi and his coworkers demonstrated that the striking correlation between H. pylori and Cag-A in ATDs could be explained by the distinct expression of adhesion molecules in the gastric mucosa. [17].

There has been hypothesized that HP's pathogenetic role in the onset of ATDs may be significantly influenced by viral and bacterial infections. Most of the time, GD patients have antibodies that are strong against some bacteria, and many gram-positive and gram-negative bacteria have antigen structures like TSH-binding protein. [17]. Additionally, the Cag-A positive strains of *H. pylori* share some nucleotide sequence similarities with the TPO sequence. After *H. pylori* was eradicated, a positive linear regression between *H.*

pylori-Abs titers and microsomal autoantibodies was found, and these antibodies significantly decreased [18]. As a result, the onset of autoantibodies against the H+K+-ATPase in gastric autoimmunity may be triggered by inter-antibody cross-reactivity produced by *H. pylori* infections and antibodies produced against thyroid antigen structures [19]. In addition, the observation that *H. pylori* infection typically begins in childhood and the increased prevalence of the bacterium in GD upon first diagnosis suggest that the bacterium may have been present prior to the autoimmune disease's onset. Larizza *et al.*, [20] proposed that the human leukocyte DRB1*0301 antigen-carrying *H. pylori* infection can cause or exacerbate GD in susceptible young patients. The authors also suggested that these "at high risk" children might avoid GD if H. pylori was eradicated. The authors found a correlation between ATD disease and *H. pylori* positivity, but only if the genetic autoimmunity marker HLA-DRB1*0301 was present. [20].

This study found that *H. pylori* had a significant effect on iodine levels, which is in line with previous research by Simões and his colleagues who found no evidence that H. pylori infection affected serum zinc, copper, or calcium status. Iodine, phosphorus, and magnesium status studies are justified [21]. It is a necessary component that enables thyroid T4 and T3 production. Iodine is a minor component in water and soil that is consumed in a few synthetic structures [21]. Most types of iodine are diminished to iodide in the stomach. Because the stomach and duodenum absorb almost all iodide, *H. pylori*-caused peptic ulcers in the stomach or intestine may lower the body's iodine levels [22]. Thyroid autonomy that persists later in life despite normal iodine intake was observed in a previously iodine insufficient population in a longitudinal study, despite the current being in an adequate iodine state. This suggests that thyroid autonomy is caused by low iodine intake at a young age [23]. Regarding Obregon *et al.*'s findings, showed that iodine inadequacy portrayed by low serum T4, typical or marginally raised T3, and as an outcome of the last option, ordinary TSH [24,25].

CONCLUSION

Fundamental Finding: The results showed that stomach ulcers resulting from infection with H. pylori caused a decrease in iodine levels and T3,T4 and TSH, especially T3, compared to healthy people in the control group. **Implication:** This indicates that H. pylori infection may be one of the reasons of thyroid disorders as hypothyroidism, and that treating this infection may reduce the risks of thyroid diseases. **Limitation:** The study does not clarify the role of iodine deficiency on other dysfunction in different parts of the body when ulcers occur. **Future Research:** In the end, we need other studies that clarify the role of iodine deficiency on other dysfunction in different parts of the body when ulcers occur.

References

[1] C. Varon, L. Azzi-Martin, S. Khalid, L. Seeneevassen , A. Ménard, P. Spuul. Helicobacters and cancer, not only gastric cancer?. Semin Cancer Biol. 2022;86(Pt 2):1138-1154.

[2] Z. Ju, L. Shen, M. Zhou, J. Luo, Z. Yu, Can Qu, R.Lei, M. Lei. Ruixue Huang Helicobacter pylori and Alzheimer's Disease-Related Metabolic Dysfunction: Activation of TLR4/Myd88 Inflammation Pathway from p53 Perspective and a Case Study of Low-Dose Radiation Intervention. ACS Chem Neurosci. 2022 Apr 6;13(7):1065-1081. Silva IN, Marçal LV, Queiroz DMM. Helicobacter pylori Infection

Is Associated With Thyroid Dysfunction in Children With Congenital Hypothyroidism. Front Pediatr. 2022;10:875232. Published 2022 Jun 21.

[3] M. Z. Z. Jamil Sr , S. Salman , M. Akhtar , S. Iqbal , A. Bhalli, H. Farooq. Determining the Association Between Helicobacter pylori Infection and Treatment-Refractory Hypothyroidism. Cureus. 2022 Jan 17;14(1):e21316.

[4] O.J. Mirgoli, V. Ramjas , S. Munugoti , H.Silverstein, F. Malik, A. Salem , F. Cassavell , A. Atoot.An Unusual Cause of Refractory Hypothyroidism. Cureus. 2022 Mar 26;14(3):e23522.

[5] Y. Park, T.J. Kim[1], H. Lee , H. Yoo, I. Sohn , Y. W.Min, B.Min , J. H. Lee , P. Rhee , J. J. Kim. Eradication of Helicobacter pylori infection decreases risk for dyslipidemia: A cohort study. Helicobacter. 2021 Apr;26(2):e12783.

[6] J. Astl , I. Sterzl. Activation of Helicobacter pylori causes either autoimmune thyroid diseases or carcinogenesis in the digestive tract. Physiol Res. 2015;64(Suppl 2):S291-S301.

[7] WHO, Assessment of Iodine Deficiency Disorders and Monitoring Their Elimination: A Guide for Programme Managers, WHO, Geneva, Switzerland, 3rd edition, 2007, https://apps.who.int/iris/bitstream/handle/10665/43781/9789241595827_eng.pdf.

[8] C.M.Dayan, andV. Panicker . Interpretation of thyroid function tests and their relationship to iodine nutrition: changes in TSH, free T4, and free T3 resulting from iodine deficiency and iodine excess, Comprehensive Handbook of Iodine: Nutritional, Biochemical, Pathological and Therapeutic Aspects, Academic Press, Boston, MA, USA, pp. 47–54, 2009.

[9] J.H.Lazarus. Relevance of iodine nutrition to health in the 21st century. Minerva Med. 2017;108(2):114-115.

[11] M.B.Zimmermann and M. Andersson. Global Endocrinology: Global perspectives in endocrinology: coverage of iodized salt programs and iodine status in 2020. Eur J Endocrinol. 2021;185(1):R13-R21.

[12] K.X.Papamichael, G.Papaioannou, H.Karga, A.Roussos, G.J.Mantzaris. Helicobacter pylori infection and endocrine disorders: is there a link? World J Gastroenterol. 2009;15:2701–2707 .

[13] N.Figura, G.Di Cairano, F.Lorè, E.Guarino, A.Gragnoli, D.Cataldo, R.Giannace, D.Vaira, L. Bianciardi, S.Kristodhullu, C.Lenzi, V.Torricelli, G.Orlandini, C.Gennari. The infection by Helicobacter pylori strains expressing CagA is highly prevalent in women with autoimmune thyroid disorders. J Physiol Pharmacol. 1999 Dec;50(5):817-26.

[14] P.A.Tomasi, M.P.Dore, G.Fanciulli, F.Sanciu, G.Realdi, G.Delitala. Is there anything to the reported association between Helicobacter pylori infection and autoimmune thyroiditis? Dig Dis Sci. 2005;50:385–388.

[15] F.Franceschi, M.A.Satta, M.C.Mentella, R.Penland, M.Candelli, R.L.Grillo, D.Leo, L.Fini, E.C. Nista, I.A.Cazzato, et al. Helicobacter pylori infection in patients with Hashimoto's thyroiditis. Helicobacter. 2004;9:369.

[16] D.A.De Luis, C.Varela, H.de La Calle, R.Cantón, C.M.de Argila, A.L.San Roman, D.Boixeda. Helicobacter pylori infection is markedly increased in patients with autoimmune atrophic thyroiditis. J Clin Gastroenterol. 1998 Jun;26(4):259-63.

[17] F. Ma , W. Zhao, M. Kudo, K. Aoki, J. Misumi. Inhibition of vacuolation toxin activity of Helicobacter pylori by iodine, nitrite and potentiation by sodium chloride, sterigmatocystin and fluoride. Toxicol In Vitro. 2002; 16:531–537.

[18] V.Bassi, G.Marino, A.Iengo, O.Fattoruso, C.Santinelli. Autoimmune thyroid diseases and Helicobacter pylori: the correlation is present only in Graves's disease. World J Gastroenterol. 2012 Mar 14;18(10):1093-7.
[19] Y. Hou, W.Sun , C.Zhang , T.Wang , X.Guo , L. Wu , L.Qin ,T. Liu. Meta-analysis of the correlation between Helicobacter pylori infection and autoimmune thyroid diseases. Oncotarget. 2017; 8: 115691-115700.
[20] I.N.Silva, L.V.Marçal, D.M.M.Queiroz. Helicobacter pylori Infection Is Associated With Thyroid Dysfunction in Children With Congenital Hypothyroidism. Front Pediatr. 2022 Jun 21;10:875232.
[21] D.Larizza, V. Calcaterra , M.Martinetti , R.Negrini, A.De Silvestri, M.Cisternino, A.M.Iannone, E.Solcia. Helicobacter pylori infection and autoimmune thyroid disease in young patients: the disadvantage of carrying the human leukocyte antigen-DRB1*0301 allele. J Clin Endocrinol Metab. 2006;91:176–179.
[22] A.M.Simões, D.Araújo, P.M.Teixeira, H.Antunes. Effect of Helicobacter pylori Infection on Macromineral and Trace Element Status-Systematic Review and Meta-Analysis. J Pediatr Gastroenterol Nutr. 2022;75(5):661-665.
[23] H.R. Chung. Iodine and thyroid function. Ann Pediatr Endocrinol Metab. 2014 Mar;19(1):8-12.
[24] A.C.Van de Ven, R.T.Netea-Maier, H.A.Ross, van Herwaarden TA, Holewijn S, de Graaf J, Kiemeney BL, van Tienoven D, Wetzels JF, Smit JW, Sweep FC, Hermus AR, den Heijer M. Longitudinal trends in thyroid function in relation to iodine intake: ongoing changes of thyroid function despite adequate current iodine status. Eur J Endocrinol. 2013 Nov 22;170(1):49-54.
[25] M.J.Obregon, Escobar del Rey F, Morreale de Escobar G. The effects of iodine deficiency on thyroid hormone deiodination. Thyroid. 2005 Aug;15(8):917-29.

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