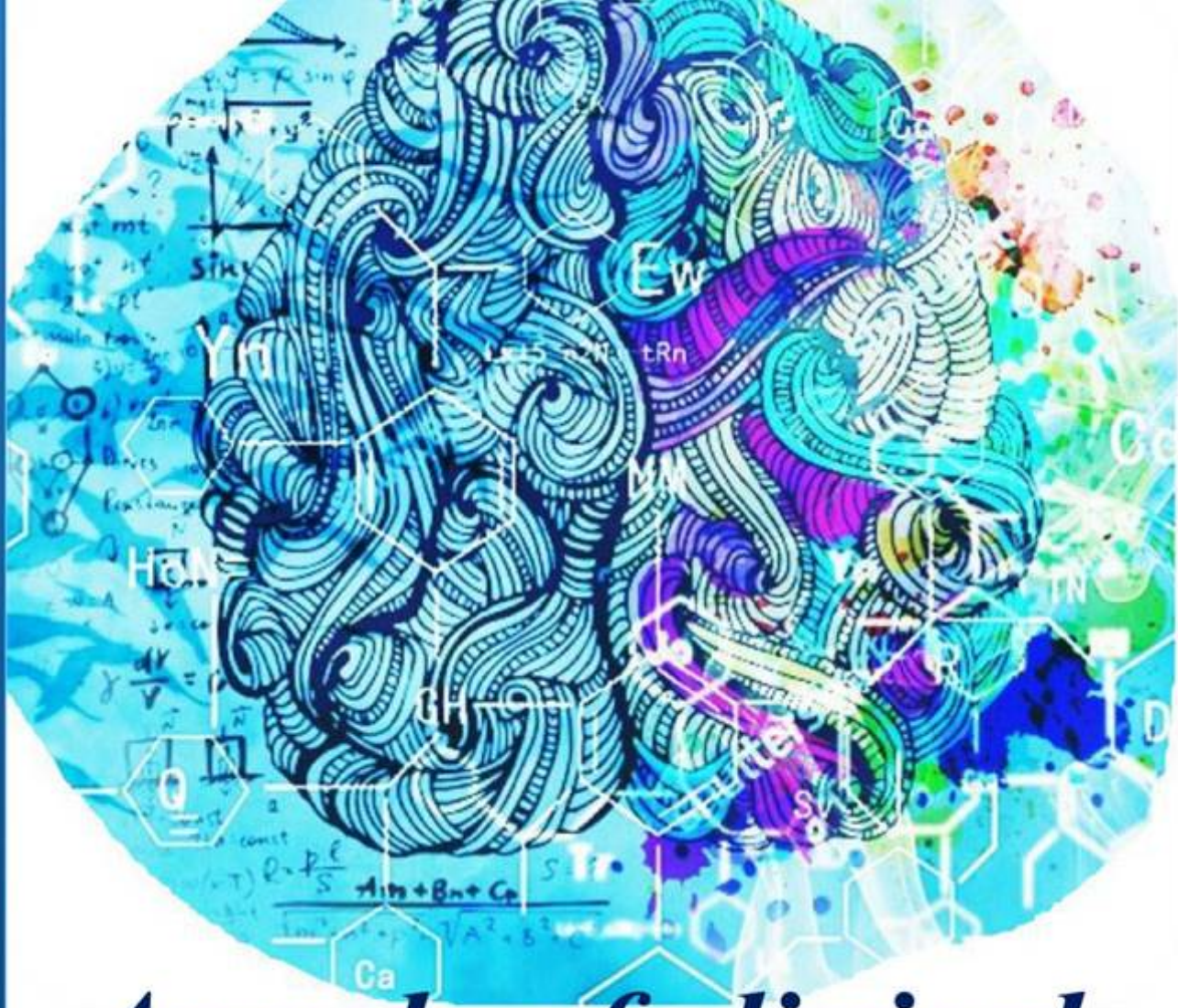
ISSN: 3030-3877



# Annals of clinical disciplines

VOLUME 1, ISSUE 3

2024

https://tadqiqot.uz/index.php/spjacd

БУХОРО ДАВЛАТ ТИББИЁТ ИНСТИТУТИ

ISSN 0000-0000

DOI Journal 10.26739/0000-0000

# ANNALS OF CLINICAL DISCIPLINE

1 ЖИЛД, 3 СОН

# АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН

ТОМ 1, НОМЕР 3

# КЛИНИК ФАНЛАР ЙИЛНОМАСИ

VOLUME 1, ISSUE 3

ТОШКЕНТ-2024

# ANNALS OF CLINICAL DISCIPLINE

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН | КЛИНИК ФАНЛАР ЙИЛНОМАСИ

№3 (2024) DOI http://dx.doi.org/10.26739/0000-0000-2024-3





© **Page Maker | Верстка | Саҳифаловчи:** Хуршид Мирзахмедов

**О журнале**
Журнал зарегистрирован в Агентство информации и массовых коммуникаций при Администрации Президента Республики Узбекистан
№ С-239963 от 14 марта 2024 года



Адрес редакции: Республика Узбекистан, 200114, г. Бухара, ул. Гиждуван, 23
Телефон: +998(65)2230050
Сайт: https://tadqiqot.uz/index.php/spjacd
e-mail: abumkur14@gmail.com

# МУНДАРИЖА | СОДЕРЖАНИЕ | CONTENT

SCIENTIFIC AND PRACTICAL JOURNAL
ANNALS OF CLINICAL DISCIPLINE
АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН
КЛИНИК ФАНЛАР ЙИЛНОМАСИ
ISSN: 0000-0000
www.tadqiqot.uz
BUKHARA STATE MEDICAL INSTITUTE

**UDC 616.33-002.27**

**Yuldashova R.U.,**
**Jarilkasinova G.J.**
Bukhara State Medical Institute, Bukhara, Uzbekistan

# POSSIBILITY OF ASSESSING IRON DEFICIENCY IN HELICOBACTER PYLORI INFECTION

**http://dx.doi.org/10.5281/zenodo.13932406**

**SUMMARY**

H. pylori infection is a significant factor influencing the development and severity of iron deficiency conditions, including IDA. Based on the results of the assessment of the correlation, it can be concluded that the level of antibodies to H. Pylori can indirectly judge the presence and severity of iron deficiency in the body in the absence of data on the level of ferritin, serum iron and other indicators.

**Key words:** helicobacteriosis, iron deficiency anemia, antibodies.

---

**Юлдашова Р.У.,**
**Жарылкасынова Г.Ж.**
Бухарский государственный медицинский институт, Бухара, Узбекистан

# ВОЗМОЖНОСТЬ ОЦЕНКИ ДЕФИЦИТА ЖЕЛЕЗА ПРИ ИНФЕКЦИИ HELICOBACTER PYLORI

**АННОТАЦИЯ**

Инфекция H. pylori является значимым фактором, влияющим на развитие и тяжесть железодефицитных состояний, в том числе ЖДА. По результатам оценки корреляции можно сделать вывод, что уровень антител к H. Pylori может косвенно судить о наличии и выраженности дефицита железа в организме при отсутствии данных об уровне ферритина, сывороточного железа и других показателях.

**Ключевые слова:** хеликобактериоз, железодефицитная анемия, антитела

---

**Yuldashova R.U.,**
**Jarilkasinova G.J.**
Buxoro davlat tibbiyot instituti, Buxoro, O‘zbekiston

# HELICOBACTER PYLORI INFEKTSIYASIDA TEMIR TANQISLIGINI BAHOLASH IMKONIYATI

**ANNOTATSIYA**

H. pylori infeksiyasi temir tanqisligi, shu jumladan temir tanqisligi holatlarining rivojlanishi va og‘irligiga ta’sir qiluvchi muhim omil hisoblanadi. Korrelyatsiyani baholash

natijalariga ko‘ra, xulosa qilish mumkinki, ferritin, zardobdagi temir va boshqa ko‘rsatkichlar darajasi haqida ma’lumotlar mavjud bo‘lmaganda, H. Pylori ga qarshi antitanachalar darajasi organizmda temir tanqisligining mavjudligi va yaqqolligi haqida bilvosita fikr yuritishi mumkin.

**Kalit so'zlar:** helikobakterioz, temir tanqisligi anemiyasi, antitelalar.

---

**Relevance.** Iron deficiency is one of the most common diseases in the world. According to World Health Organization, the prevalence of such a manifestation of iron deficiency as anemia is 32.9% [5]. In Europe, there are about 14% of cases of iron deficiency anemia per 100,000 populations [15]. Iron deficiency leads not only to the development of anemia, but also to the development of such symptoms that worsen the quality of life, such as: weakness, increased fatigue, impaired attention, anxiety, forgetfulness, irritability, morning headaches, fainting, dizziness, increased susceptibility to infections, changes skin and mucous membranes, dyspeptic disorders [6].

One of the most important extra gastroduodenal manifestations of H. Pylori infection is hematological diseases. A significant association between H. pylori and the development of idiopathic thrombocytopenic purpura (ITP), iron deficiency anemia (IDA) and B 12 deficiency anemia has now been recognized. The role of H. Pylori in the pathogenesis of IDA has been proven in experimental models. M. Burns et al. [1] reported that laboratory mice infected with H. pylori had significantly lower levels of ferritin, erythrocytes, hematocrit and hemoglobin.

Most researches on the relationship between iron deficiency and H. pylori include patients with anemia, and only a few studies include patients with latent iron deficiency. In this regard, there are no reliable data on the frequency of latent and pre-latent iron deficiency in patients infected with H. pylori.

According to the results of many epidemiological studies, one of the risk factors for iron deficiency is the presence of H. pylori infection [10]. H. pylori infection leads to a decrease in ferritin levels, regardless of gender, age, and other risk factors for iron deficiency, increasing the risk of anemia in this category of patients [1,5]. The presence of H. pylori infection also affects the effectiveness of iron therapy; most researches show an increase in the effectiveness of IDA therapy with successful eradication of H. pylori. With successful eradication, the use of iron preparations led to an increase in the level of ferritin, hemoglobin, normalization of MCV and MCH.

The mechanisms by which H. pylori infection influences the development of iron deficiency remain unclear. Several studies have proposed various biological mechanisms by which H. pylori infection can cause iron depletion in the patient's body. One of these mechanisms is the effect on inflammation caused by Helicobacter pylori infection, on the hepcidin-ferroportin mechanism of regulation of iron metabolism. An increase in hepcidin levels reduces the absorption of iron from the intestine. Studies have been published showing that hepcidin is elevated in patients with H. pylori infection, increasing as an acute phase marker in response to inflammation in the gastric mucosa, leading to a pathology known as “anemia of inflammation or chronic disease.” At the same time, eradication of H. Pylori leads to a decrease in the level of hepcidin, which increases the effectiveness of iron deficiency therapy in this group of patients.

When considering the treatment of ID and IDA in patients with H. pylori infection, it is important to note that eradication therapy is as important as the administration of iron supplements. The studies described above examined the relationship between eradication therapy and the resolution of ID and IDA. In many cases, after successful eradication therapy, the levels of hemoglobin, ferritin, and also transferrin increased. In addition, the levels of TIBC and hepcidin in patients after successful eradication therapy decreased. Based on this, it can be assumed that in the treatment of ID and IDA in patients suffering from H. pylori infection, eradication therapy can lead to the resolution of ID and IDA. Also, the control of H. pylori infection over time in patients after successful eradication therapy may prevent the development of ID and IDA.

When considering iron therapy, it is necessary to take into account comorbidities that can lead to the development of ID and IDA. In some cases, treatment of the underlying disease may lead to resolution of ID and IDA. Iron treatment should be started immediately, even in the absence of anemia, especially if patients develop characteristic symptoms [11, 13].

A systematic review of the efficacy of iron supplementation in non-anemic individuals with iron deficiency showed that treatment (of any type) increased hemoglobin and ferritin levels and reduced patients' feelings of fatigue, but did not improve exercise performance, nor did it maximize oxygen saturation [9].

The choice of form of iron and route of administration largely depends on the presence and degree of anemia, reversibility of the underlying cause of ID and IDA, clinical status (age, sex, prolonged or recent onset), and in some cases patient preference. Oral iron preparations such as ferrous sulfate, fumarate, and ferrous gluconate remain the mainstay of therapy for absolute iron deficiency.

Thereby, the purpose of these guidelines is to study the correlation between the level of antibodies to H. Pylori and indicators of iron metabolism in the body of patients with IDA.

The relationship between H. pylori and IDA has been convincingly proven in numerous studies. Current international and national guidelines advise H. pylori eradication in patients with unexplained IDA, which was also confirmed by our study [13,14].

A meta-analysis including 16 different studies including a total of 956 patients showed that eradication therapy in combination with prostate therapy led to a statistically significant increase in hemoglobin levels by 1.48 times, serum iron by 1.15 times and ferritin levels by 1.48 times. 84 times compared with pancreatic therapy alone. All this allowed international experts studying H. pylori infection in 2010 to include IDA of unknown etiology as an additional indication for eradication therapy [15,16,17].

The results of the analysis of 17 thousand cases showed the prevalence of IDA in 5.5% of patients with H. pylori infection compared with 5.2% in the group with negative results of the study for the bacterium. The relative risk of IDA in patients with H. pylori was 1.19. There was also an increased risk of developing moderate to severe IDA in patients with H. pylori, in whom the relative risk ratio was 1.39 [18,19].

The results of this research are consistent with the results of foreign studies, which showed that the H. pylori bacterium is able to secrete special chelate complexes that have an affinity for ferric iron, which helps them absorb iron for their life. It is known that this microorganism synthesizes proteins that contain ferric iron, that is, they directly need it [18,19,20].

Table 1 presents the results of studying the morphological properties of erythrocytes in patients in the study groups.

Table 1

**Morphological characteristics of erythrocytes (M±m)**

| Indicator | Main group(n=40) | Control group(n=40) |
|---|---|---|
| Hemoglobin | 96,76±9,23 | 93,31±7,72 |
| Red blood cells | 4,38±0,1 | 4,4±0,3 |
| Average erythrocyte volume (MCV) | 79,3±1,5 | 82,4±1,1 |
| Number of patients with reduced MCV | 36 (90%) | 35 (87,5%) |
| Mean erythrocyte hemoglobin (MCH) - pkg | 28,1±1,2 | 32,2±1,1 |
| Number of patients with reduced MCH | 30 (75%) | 28 (70%) |
| Mean erythrocyte hemoglobin concentration (MCHC) -g/dl | 35,4±1,1 | 37,1±0,9 |
| Number of patients with reduced MCHC | 24 (60%) | 22 (55%) |
| Microcyticanemiafactor (MAF) | 9,8±1,1 | 11,3±0,8 |
| Number of patients with reduced MAF | 38 (95%) | 39 (97,5%) |

Table 2 presents the results of the study of iron metabolism in the study groups.

Table 2

**Indicators of iron metabolism in the studied groups**

| Indicator | Main group (n=40) | Control group (n=40) |
|---|---|---|
| Serum iron (µmol/l) | 8,82±1,31 | 9,42±0,4 |
| TIBC (µmol/l) | 67,1±2,7 | 68,1±1,5 |
| Transferrin (g/l) | 4,1±0,7 | 4,7±1,1 |
| Ferritin (mcg/ml) | 157,4±31,2 | 120±23,5 |

Figure 1 shows the distribution of patients by ferritin level.

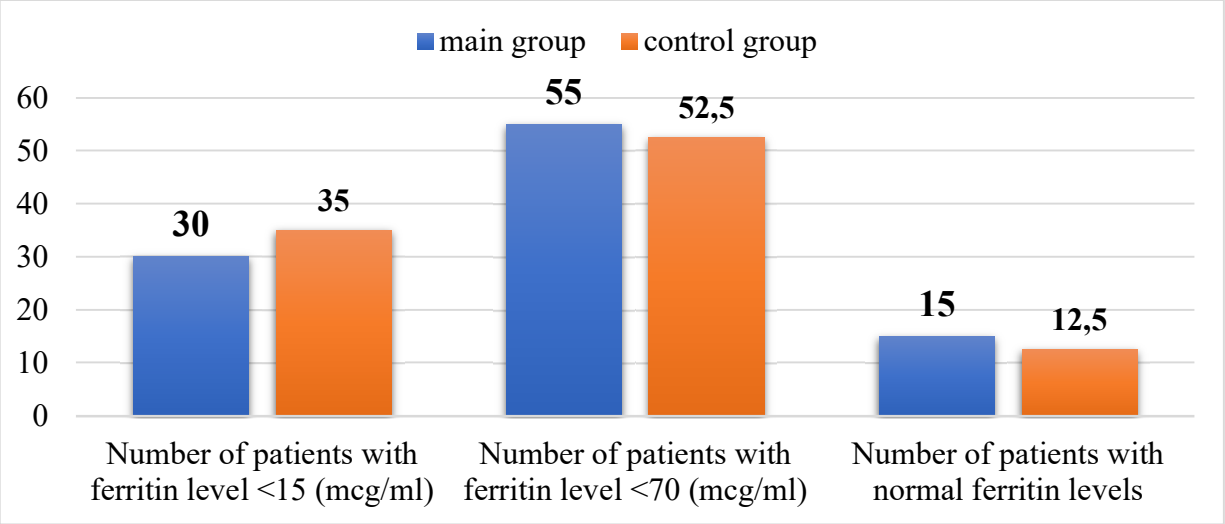
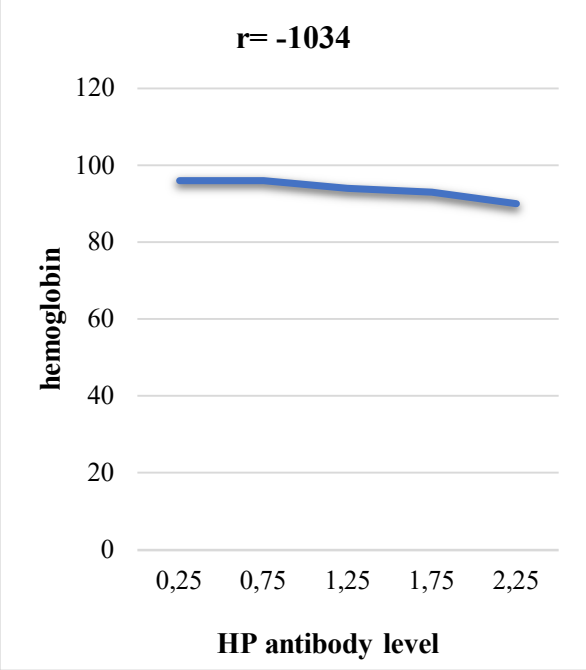
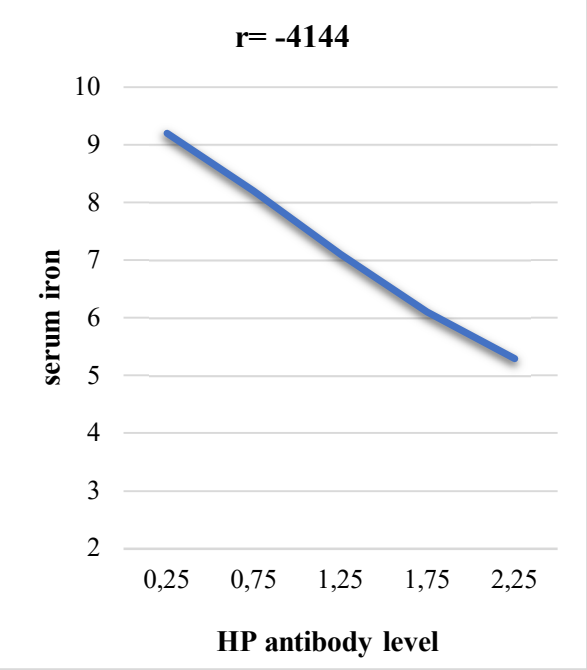



Figure 1. Distribution of patients by ferritin level

To clarify the relationship between iron metabolism parameters in patients with HP infection, a correlation analysis was carried out. The data reflecting the correlation relationships are presented in Figure 2.

Significant correlations were found between indicators of iron metabolism - iron in blood serum and ferritin, as well as the level of antibodies to H. pylori. The established correlation relationship was a confirmation of the reliability of the results of the studies obtained.



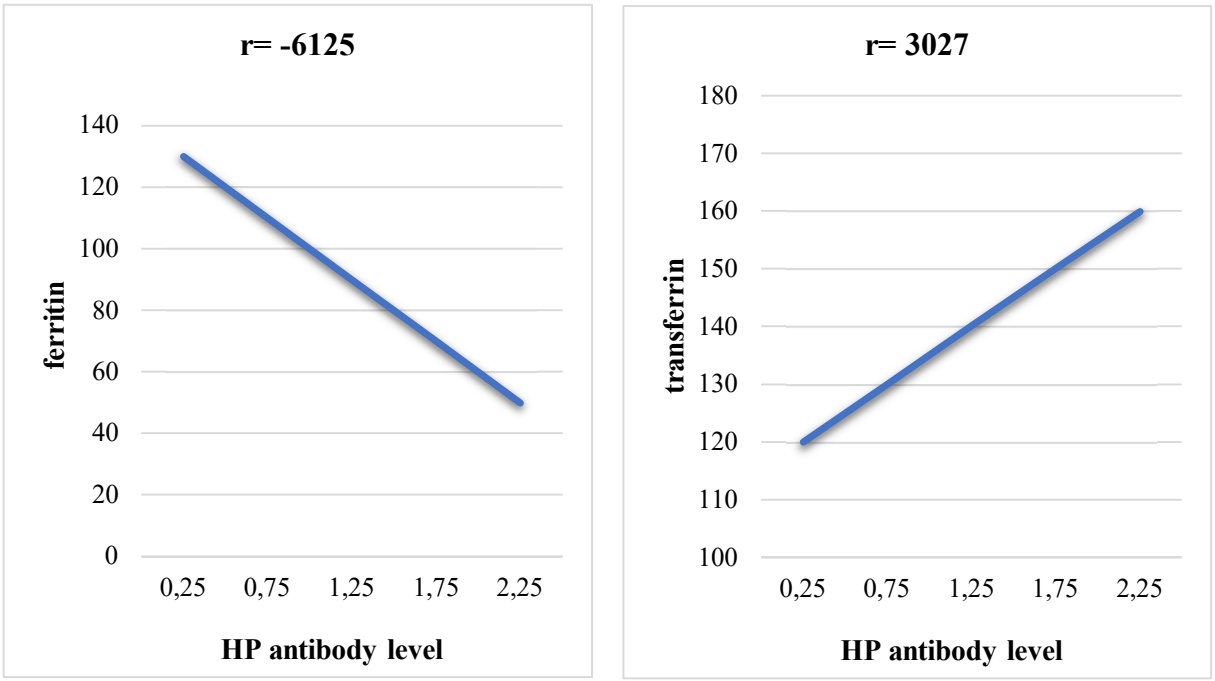



Figure 2. The results of the analysis of the correlation between the indicators of iron metabolism in the body and the level of antibodies to H. pylori

**Conclusions.** Thus, the research showed that H. pylori infection is a significant factor influencing the development and severity of iron deficiency conditions, including IDA. Based on the obtained results on the assessment of the correlation, it can be concluded that the level of antibodies to H. pylori can indirectly judge the presence and severity of iron deficiency in the body in the absence of data on the level of ferritin, serum iron and other indicators.

Based on the shown medical, social and economic efficiency, as well as its accessibility and simplicity, the proposed method can be recommended for implementation in wide medical practice.

## References

1. Burns, M. Helicobacter pylori infection induces anemia, depletes serum iron storage, and alters local iron-related and adult brain gene expression in male INS-GAS mice / M. Burns, S. Muthupalani, Zh. Ge et al. // PLoS One. – 2015. – Vol. 10. – № 11. – e0142630. – doi: 10.1371/journal.pone.0142630.

2. Emiralioglu, N. An insight into the relationships between prohepcidin, iron deficiency anemia, and interleukin-6 values in pediatric Helicobacter pylori gastritis / N. Emiralioglu, I. Yenicesu, S. Sari et al. // European Journal of Pediatrics. – 2015. – Vol. 174. – № 7. – P. 903–910.

3. Muhsen, K. & Cohen, D. Helicobacter pylori Infection and Anemia. Am J Trop Med Hyg 89, 398–398 (2013).

4. Mei-Yan Xu, Bing Cao, Bao-Shi Yuan, Jian Yin1, Lan Liu & Qing-Bin Lu / Association of anemia with Helicobacter pylori infection: a retrospective study // Scientific REPORTS |7: 13434 wwww.nature.com/scientific reports

5. Xia, W., Zhang, X., Wang, J., Sun, C. & Wu, L. Survey of anaemia and Helicobacter pylori infection in adolescent girls in Suihua, China and enhancement of iron intervention effects by H. pylori eradication. Br J Nutr 108,357–362 (2012).

6. Hu, Y. et al. Study on the anemia status of Chinese urban residents in 2010–2012. Chin J Prev Med 50, 213–216 (2016).

7. Afifi RAR, Ali DK, Shaheen IAM. A localized case control study of extra gastric manifestations of Helicobacter pylori infection in children. Indian J Pediatr 2011; 78:418–22; PMID: 21165719; http:// dx.doi.org/1 0.1007/s12098–010–0308–6

8. Queiroz DMM, Harris PR, Sanderson IR, Windle HJ, Walker MM, Rocha AMC, Rocha GA, Carvalho SD, Bittencourt PF, de Castro LP, et al. Iron status and Helicobacter pylori infection in symptomatic children: an international multi-centered study. PLoS One 2013; 8:e68833; PMID: 23861946; http://dx.doi. org/10.1371/journal.pone.0068833

9. Yuan W, Li Y, Yang K, Ma B, Guan Q, Wang D, et al. Iron deficiency anemia in Helicobacter pylori infection: meta-analysis of randomized controlled trials. Scand J Gastroenterol. 2010 Jun; 45(6):665–676.

10. Qu XH, Huang XL, Xiong P, Zhu CY, Huang YL, Lu LG, et al. Does Helicobacter pylori infection play a role in iron deficiency anemia? A meta-analysis World J Gastroenterol. 2010 Feb 21; 16(7):886–896.

11. Huang X, Qu X, Yan W, Huang Y, Cai M, Hu B, et al. Iron deficiency anaemia can be improved after eradication of Helicobacter pylori. Postgrad Med J. 2010 May; 86(1015):272–278.

12. Zhang ZF, Yang N, Zhao G, Zhu L, Zhu Y, Wang LX. Effect of Helicobacter pylori eradication on iron deficiency.Chinese medical journal. 2010 Jul; 123(14):1924–1930.

13. Duclaux-Loras R, Lachaux A. [Helicobacter pylori infection, a classic but often unrecognized cause of iron deficiency anemia in teenagers]. Arch Pediatr 2013; 20(4):395–7.

14. Mubarak, N., Gasim, G. I., Khalafalla, K. E., Ali, N. I. & Adam, I. Helicobacter pylori, anemia, iron deficiency and thrombocytopenia among pregnant women at Khartoum, Sudan. Trans R Soc Trop Med Hyg 108, 380–384 (2014)

15. Cardenas VM, Prieto-Jimenez CA, Mulla ZD, Rivera JO, Dominguez DC, Graham DY, et al. Helicobacter pylori eradication and change in markers of iron stores among non-iron-deficient children in El Paso, Texas: an etiologic intervention study. J PediatrGastroenterolNutr. 2011 Mar; 52(3):326–332.

16. Prentice A M Clinical Implications of New Insights into Hepcidin-Mediated Regulation of Iron Absorption and Metabolism Ann NutrMetab. 2017; 71Suppl 3:40–48.doi:10.1159/000480743.Epub 2017 Dec 22.

17. Girelli D, Ugolini S, Busti F, Marchi G, Castagna A. Modern iron replacement therapy: clinical and pathophysiological insights Int J Hematol. 2018 Jan; 107(1):16–30. Doi: 10.1007/s12185–017–2373–3. Epub 2017 Dec 1.

18. Burns M, Amaya A, Bodi C, Ge Z, Bakthavatchalu V, Ennis K, Wang TC, Georgieff M, Fox JG. Helicobacter pylori infection and low dietary iron alter behavior, induce iron deficiency anemia, and modulate hippocampal gene expression in female C57BL/6 mice. 2017 Mar 29; 12(3): e0173108. doi: 10.1371/journal.pone.0173108.eCollection 2017.

19. Siddique O, Ovalle A, Siddique AS, Moss SF. Helicobacter Pylori Infection: an Update for the Internist in the Age of Increasing Global Antibiotic Resistance. Am J Med. 2018 Jan 15. pii: S0002–9343(18)30013–5. doi: 10.1016/j.amjmed.2017.12.024.

20. Schwarz P, Kübler JA, Strnad P, Müller K, Barth TF, Gerloff A, Feick P, Peyssonnaux C, Vaulont S, Adler G, Kulaksiz H Hepcidin is localised in gastric parietal cells, regulates acid secretion and is induced by Helicobacter pylori infection. Gut. 2012 Feb;61(2):193–201. doi:10.1136/gut.2011.241208. Epub 2011 Jul 13.

БУХОРО ДАВЛАТ ТИББИЁТ
ИНСТИТУТИ

# ANNALS OF CLINICAL DISCIPLINE

1 ЖИЛД, 3 СОН

## АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН

ТОМ 1, НОМЕР 3

## КЛИНИК ФАНЛАР ЙИЛНОМАСИ

VOLUME 1, ISSUE 3

Научно-практический журнал по всем
направлениям медицины
основан в 2024 году
Бухарским государственным
медицинским институтом
Выходит один раз в 3 месяца
Учредитель Бухарский государственный
медицинский институт