Iran J Ped Hematol Oncol. 2026, Vol. 16, No. 3, 961-968

Original Article

Mean Platelet Volume and Neutrophil-to-Lymphocyte Ratio in Pediatric Gastritis: Relation to Helicobacter pylori Infection

Mahbubeh Erfanipour[1] MD, Mohammadreza Esmaeili Dooki[2] MD, Hossein-Ali Nikbakht[3] PhD, Hassan Mahmoodi Nesheli[4] MD, Mohammad Pornasrolah[5] PhD, Maryam Nikpour[6] PhD, Sanaz Mehrabani [7*] MD

[1] Student Research Committee, Babol University of Medical Sciences, Babol, Iran.
[2] Professor of Pediatric Gastroenterology, Non-communicable Pediatric Diseases Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, IR Iran
[3] Assistant Professor, Non-communicable Pediatric Diseases Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, IR Iran
[4] Associate Professor of Pediatric Hematology & Oncology, Non-communicable Pediatric Diseases Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, IR Iran
[5] Clinical Research Development Unit of Amirkola Children's Hospital, Babol University of Medical Sciences, Babol, IR Iran
[6] Non-Communicable Pediatric Diseases Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, I.R.Iran
[7*] Associate Professor of Pediatrics Gastroenterology, Non-Communicable Pediatric Diseases Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, Iran
*Corresponding Author: Dr. Sanaz Mehrabani Associate Professor of Pediatrics Gastroenterology, Non-Communicable Pediatric Diseases Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, Iran. Email: mehrabanisanaz@yahoo.com. ORCID ID: 0000-0002-2062-0448.

Received: November 15 2025;
Accepted: January 5, 2026

Abstract

Background: Helicobacter pylori (*H. pylori)* is a primary contributor to chronic gastritis in children, leading to both local and systemic inflammatory responses. Although endoscopic and histopathologic evaluations are the diagnostic standards, they are invasive and costly. Recently, hematologic indices such as the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV) have been proposed as simple, accessible markers of systemic inflammation. However, previous studies assessing their association with *H. pylori* infection in pediatric gastritis have reported inconsistent findings. Therefore, the aim of this study was the assessment of MPV and NLR in pediatric gastritis and relation to helicobacter pylori Infection.

Materials and Methods: This cross-sectional study was conducted on children aged 1–17 years with gastrointestinal symptoms were candidates for endoscopy at the Gastroenterology Clinic of Amirkola Children's Hospital. Gastric tissue samples were collected via biopsy and examined histopathologically, and H. pylori infection was confirmed using Giemsa staining. In addition, complete blood count (CBC) tests were performed for NLR and MPV measurements.

Results: The study included 126 children (mean age 10.03 ± 2.51 years). H. pylori infection was significantly associated with both endoscopic ($p = 0.001$) and histopathologic gastritis ($p = 0.011$). No significant differences were observed in NLR or MPV between H. pylori–positive and H. pylori–negative groups ($p = 0.990$ and $p = 0.459$, respectively). Similarly, NLR and MPV did not differ significantly according to the presence of histopathologic ($p = 0.874$ and $p = 0.891$, respectively) or endoscopic gastritis ($p = 0.667$ and $p = 0.103$).

Conclusion: *H. pylori* infection was significantly associated with endoscopic and histopathologic gastritis in children, highlighting its central role in pediatric gastritis. However, NLR and MPV showed no significant differences according to *H. pylori* infection or gastritis status, indicating limited diagnostic utility of these hematologic markers.

Keywords: Gastritis, Helicobacter pylori, Mean Platelet Volume, Neutrophil-to-lymphocyte ratio

[DOI: 10.18502/ijpho.v16i3.21753]

Introduction

Gastritis refers to an inflammatory process affecting the gastric mucosal lining, leading to injury and disruption of the mucus-protective barrier (1, 2). The condition may develop acutely with sudden and severe manifestations or progress chronically over time. Although the etiology of gastritis remains unclear in many cases, infection with Helicobacter pylori (H. pylori) is recognized as the most established and well-documented cause (3,4).

A variety of factors have been associated with the onset of gastritis, including excessive gastric acid secretion, autoimmune reactions, parasitic and viral infections, granulomatous disorders such as Crohn's disease and sarcoidosis, mechanical trauma, malnutrition, prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs), and H. pylori infection itself (4).

Among these causes, H. pylori remains the predominant agent worldwide and plays a key role in the development of chronic gastritis, gastroduodenal ulcers, and gastric cancer (5, 6). In developing countries, up to 80% of children are infected, whereas infection rates are much lower in industrialized regions (7, 8). The prevalence is particularly high among low socioeconomic groups and in areas with poor hygiene and household crowding (9). H. pylori is a Gram-negative, microaerophilic bacterium that inhabits the gastric antrum and elicits both local and systemic inflammatory responses characterized by infiltration of neutrophils, macrophages, and lymphocytes into the mucosa (10-12).

This cellular infiltration and the associated cytokine release promote chronic mucosal inflammation and tissue injury. The severity of histopathologic changes tends to vary by age, and H. pylori–associated gastritis in children is generally milder than in adults (10–13).

In recent years, hematologic indices have gained attention as markers of systemic inflammation. The neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV) are inexpensive, easily obtainable indicators reflecting immune activation and inflammatory status (1,9,10). NLR represents the balance between neutrophil-driven inflammation and lymphocyte-mediated regulation, whereas MPV reflects platelet size and activation, which increase during inflammatory responses.

Given that both NLR and MPV reflect systemic inflammatory activity, evaluating their association with H. pylori infection and histopathologic gastritis may provide valuable insights into the inflammatory burden and immune response patterns in affected children.

Material and Methods

Type of Study

This cross-sectional study was conducted in 2021 to investigate Helicobacter pylori–associated gastritis in children aged 1–17 years presenting with chronic abdominal pain. Participants were referred to the Gastroenterology Clinic of Amirkola Children's Hospital.

Inclusion and Exclusion Criteria

Eligible children exhibited persistent gastrointestinal symptoms suggestive of H. pylori infection, including abdominal pain, nausea, vomiting, and heartburn, lasting for at least two months. Written parental consent was obtained prior to enrollment.

Exclusion criteria included

- History of hematologic disorders (e.g., thrombocytopenia, leukemia, lymphoma, autoimmune destruction of red blood cells, or thalassemia)
- Parasitic or other infections
- Exposure to caustic substances
- Concurrent infection within the preceding two to three weeks
- Incomplete medical records

Sample Size and Sampling Method

Using standard formulas for diagnostic studies (15), as defined by the study objectives and previously reported 93%, sensitivity, sample size was calculated (16). Assuming a 95% confidence level, a 20% expected prevalence of gastritis among children, and a 0.10 margin of error, the study required at least 126 patients. Participants were selected through a non-probability convenience sampling approach.

[DOI: 10.18502/ijpho.v16i3.21753]

Data Collection

Chronic abdominal pain was defined as a minimum of three episodes. interfering with daily activities and persisting for one month or longer, suggestive of H. pylori–related gastritis (17). All eligible children underwent detailed history taking and physical examination by a pediatric gastroenterologist. Children presenting with alarm features—such as dysphagia, odynophagia, weight loss, anorexia, anemia, or persistent abdominal pain—were further evaluated for possible peptic disease. Persistent symptoms that resulted in school absence or hospitalization also prompted diagnostic testing, including Complete Blood Count (CBC) and upper endoscopy (18).

Hematological Assessment

Hematological evaluation was done on fasting blood from each child in a seated position by a trained laboratory technician. Blood samples were collected in EDTA-containing tubes, gently mixed, and analyzed using an automated cell counter (Sysmex K-1000 Hematology Analyzer) to determine the neutrophil-to-lymphocyte ratio (NLR) and other hematological indices.

Endoscopic Procedure

All patients underwent diagnostic upper endoscopy following 6–8 hours of fasting. An intravenous line was established, and sedation was administered using ketamine and propofol according to body weight. Endoscopic examinations were performed using an Olympus GIF-30.

gastroscope (Japan) by two experienced gastroenterologists in agreement. Endoscopic findings indicative of H. pylori infection included erythema, mucosal erosions, loss of rugal folds, visible vessels, diffuse nodularity, and superficial or deep gastric ulcers (19, 20).

Biopsy and Histopathological Evaluation

During each endoscopic procedure, three biopsy samples were collected from the esophagus, gastric antrum, and duodenum. The specimens were preserved in 10% formalin and sent to the laboratory for histopathological analysis. Gastritis was confirmed based on histologic features, including glandular atrophy, granulomatous inflammation, eosinophilic or lymphocytic infiltration, and metaplasia. Antral biopsies were additionally stained with Giemsa to detect H. pylori, which appeared as spiral-shaped bacteria under microscopy (21).

Statistical analysis

The data were entered into SPSS (version 23). Non-parametric tests such as the Mann-Whitney U test were used due to the data failing to conform to the normality assumptions underlying parametric methods. In addition, Chi-Square test was also used. P-value <0.05 was assumed significant.

Results

A total of 126 participants were included. The mean age of the children was 10.03 ± 2.51 years, and 67 (53.2%) of them were boys.

Gastritis was diagnosed histopathologically in 42 patients (33.3%), endoscopically in 85 patients (67.5%), and Helicobacter pylori infection was detected in 58 patients (46%).

[DOI: 10.18502/ijpho.v16i3.21753]

Table I presents the comparison of NLR and MPV between H. pylori–positive and H. pylori–negative patients.

Table I: Clinical and laboratory features of patients with ALL

Variables	H-pylori	Number	Median (IQR)	Mean Rank	U	Z	P-value*
NLR	Negative	68	1.37(1.45)	63.54	1969.500	-.012	0.990
	Positive	58	1.45 (1.40)	63.46			
MPV	Negative	68	8.85 (1.2)	65.72	1821.000	-.740	0.459
	Positive	58	8.6 (0.8)	60.90			

***Mann-Whitney Test**

For NLR, the mean rank was 63.54 in the H. pylori–negative group and 63.46 in the positive group, with no statistically significant difference between the two groups (U = 1969.500, Z = –0.012, p = 0.990).
Similarly, for MPV, the mean rank in the H. pylori–negative group (65.72) was higher than that in the positive group (60.90), but this difference was not statistically significant (U = 1821.000, Z = –0.740, p = 0.459).
Therefore, there was no significant difference in NLR or MPV levels between H. pylori–positive and negative individuals.

Table II presents the comparison of NLR and MPV between patients with and without endoscopic evidence of gastritis.

Table II: Comparison between NLR and MPV in patients with and without endoscopic evidence of gastritis

Variables	Gastritis. endoscopy	Number	Median (IQR)	Mean Rank	U	Z	P-value*
NLR	Negative	41	1.4 (1.25)	61.49	1660.000	-.430	0.667
	Positive	85	1.4 (1.6)	64.47			
MPV	Negative	41	8.6 (0.9)	55.88	1430.000	.103	0.103
	Positive	85	8.9 (1.3)	67.18			

***Mann-Whitney Test**

No significant difference in the mean rank of NLR or MPV was seen between patients with and without endoscopic gastritis (p > 0.05). Although the gastritis-positive group had higher mean ranks for both variables, these differences were not statistically significant (Table II).

Table III presents the comparison of NLR and MPV between patients with and without pathological evidence of gastritis

Table III: Comparison of NLR and MPV between patients with and without pathological evidence of gastritis

Variables	Pathological gastritis	Number	Median (IQR)	Mean Rank	U	Z	P-value*
NLR	Negative	84	1.4 (1.3)	63.86	1733.500	-.158	0.874
	Positive	42	1.25 (1.6)	62.77			
MPV	Negative	84	8.8 (1)	63.82	1737.500	-.137	0.891
	Positive	42	8.6 (1.4)	62.87			

***Mann-Whitney Test**

According to these findings, there were no significant differences in the mean rank of NLR or MPV values between patients with and

[DOI: 10.18502/ijpho.v16i3.21753]

without pathological evidence of gastritis ($p > 0.05$). The mean ranks were similar across groups, indicating that gastritis status based on pathology did not influence NLR or MPV levels (Table III).

Table IV: Association between H. pylori infection and endoscopic or pathological findings of gastritis

Variables	H pylori Number		Total Number	P-value*
	No	Yes		
Gastritis. Endoscopy				
No	31	10	41	0.001
Yes	37	48	85	
Gastritis. Histopathology				
No	52	32	84	0.011
Yes	16	26	42	

*** Chi-square test**

A significant association was observed between H. pylori infection and endoscopic evidence of gastritis ($p = 0.001$). Similarly, there was a significant relationship between H. pylori infection and pathological gastritis ($p = 0.011$). These results indicate that gastritis, both endoscopic and histopathologic, was significantly more frequent among H. pylori–positive patients (Table IV).

Table V: Frequency of symptoms 7 and 30 days before in the studied children

Variables		Before 7 days		Before 30 days	
		Frequency	Percent	Frequency	Percent
Vomiting	**Yes**	114	90.5	106	84.1
	No	12	9.5	20	15.9
Regurgitation	**Yes**	55	43.7	79	62.7
	No	71	56.3	47	37.3
Chest pain	**Yes**	62	49.2	77	61. 1
	No	64	50.8	49	38.9
Epigastric pain	**Yes**	98	77.8	94	74.6
	No	28	22.2	32	25.4
Swallowing disorder	**Yes**	25	19.8	40	31.7
	No	101	80.2	86	68.3
Nausea	**Yes**	95	75.4	95	75.4
	No	31	24.6	28	22.2
Belching	**Yes**	48	38.1	66	52.4
	No	78	61.9	60.0	47.6
Return of food into the mouth	**Yes**	29	23.0	52	41.3
	No	97	77.0	74	58.7
Epigastria pain after eating	**Yes**	109	86.5	111	88.1
	No	17	13.5	15	11.9

In some cases, data were missing (Table V). In terms of the severity classification of gastritis in 58 children with H. pylori positive, 50 children (86.2%) had mild gastritis, 4 children (6.9%) had moderate gastritis, and 4 children (6.9%) had severe gastritis.

[DOI: 10.18502/ijpho.v16i3.21753]

Discussion

In our study, we found no significant difference in NLR values between H. pylori–positive and –negative groups. Similarly, Boyuk et al. reported that NLR measurements did not differ significantly between H. pylori–negative and –positive groups (22), aligning with our findings

Similarly, Săsăran et al. reported that children with H. pylori–induced gastritis exhibited elevated PLR and NLR values; however, these increases were not statistically significant. They concluded that gastritis does not significantly influence MPV, PLR, or NLR, which is consistent with our findings (23).

They concluded that gastritis does not significantly influence MPV, PLR, or NLR, which is consistent with our findings." In the study by Melit et al., which examined NLR in children with H. pylori–associated gastritis, a slight but statistically insignificant increase in NLR was observed—again consistent with our findings (24).

In the present study, there was no significant difference in MPV values between H. pylori–positive and –negative groups. This suggests that platelet activation, reflected by MPV, may not be directly influenced by the presence of H. pylori infection in children with gastritis. Our results are consistent with those of Ozgur Yeniova et al. (25) and Sahin et al. (26), who also found no significant correlation between MPV and H. pylori–related gastritis.

Taken together, these findings suggest that systemic inflammatory markers such as NLR and platelet indices like MPV may not reliably reflect the local gastric inflammation caused by H. pylori in pediatric patients. It is possible that the inflammatory response to H. pylori infection in children is more localized and less likely to trigger systemic hematological changes detectable in peripheral blood.

We also did not find any relationship between NLR and MPV in children with gastritis. This suggests that variations in the inflammatory response reflected by NLR are not associated with changes in MPV in this population. In contrast, Wu et al. reported that NLR and PLR levels were significantly increased, whereas MPV values were decreased in patients with gout compared with healthy controls (27). This discrepancy may indicate that the relationship between systemic inflammation and platelet indices differs depending on the underlying disease and its inflammatory characteristics.

Our findings showed that H. pylori infection was significantly associated with both endoscopic and histopathologic gastritis, indicating that the presence of the bacterium contributes to both visible mucosal changes and microscopic inflammatory alterations. Similarly, Dosma et al. reported a significant relationship between H. pylori infection and histopathological parameters, including acute and chronic inflammatory cell infiltration, suggesting that H. pylori plays a direct role in triggering and sustaining gastric mucosal inflammation. Moreover, they observed that bacterial load correlated with the severity of inflammation ($p < 0.001$), supporting the concept that higher levels of colonization intensify the host's inflammatory response (28).

This concordance reinforces the pathogenic role of H. pylori in inducing both macroscopic and microscopic gastric mucosal injury and highlights the importance of bacterial density as a determinant of inflammatory severity.

The limitations of this study include the lack of follow-up of the children after H. pylori treatment. Additionally, the study was conducted in a single-center, cross-sectional study. Furthermore, another limitation of this study is the lack of multivariate analysis. Factors such as age, sex, and gastritis severity could affect hematologic indices, but the sample size was insufficient for reliable regression modeling. Further research with larger sample sizes is recommended to control for these confounders.

Conclusion

Although Helicobacter pylori infection was significantly associated with both endoscopic and histopathologic gastritis, no statistically significant relationship was found between inflammatory hematologic indices—including NLR and MPV—

[DOI: 10.18502/ijpho.v16i3.21753]

and the presence of H. pylori infection or gastritis in pediatric patients. These findings suggest that, unlike in some adult populations, systemic inflammatory markers such as NLR and MPV may not reliably reflect the local gastric inflammatory response induced by H. pylori in children.

Therefore, while endoscopic and histopathologic assessments remain the gold standards for diagnosing H. pylori–related gastritis, NLR and MPV appear to have limited diagnostic or predictive value in this context. Further studies are recommended to explore whether other noninvasive biomarkers could more reliably indicate gastric inflammation in pediatric patients.

Availability of Data

The datasets analyzed during the current study are available from the corresponding author on reasonable request.

Ethical Considerations

This study received approval from the Ethics Committee of Babol University of Medical Sciences under the code MUBABOL.HRI.REC.1402.043

Acknowledgements

We are grateful to the Clinical Research Development Unit of Amirkola Children's Hospital, the Research Council and Non communicable Pediatric Diseases Research Center, Health Research Institute of Babol University of Medical Sciences and Neonatal and NICU ward for their support and cooperation with study.

Authors' Contributions

SM and MED oversaw the study process. HN, ME and MN conducted the data analysis and HMN and MP developed the manuscript. All the authors reviewed the final manuscript and approved it for submission.

Funding

None.

Conflict of Interest

None.

References

1.Erfanipour M, Dooki ME, Nikbakht HA, Nesheli HM, Pornasrolah M, Nikpour M, Mehrabani S. Diagnostic value of neutrophil to lymphocyte ratio in identifying gastritis in children with chronic abdominal pain in northern Iran. BMC Re Notes 2025; 18(1):68-72.

2.Ashtari S, Pourhoseingholi MA, Molaei M, Zali M. Prevalence of Helicobacter pylori and Intestinal Metaplasia in consecutive gastritis patients; over the period of 7 years. Govaresh 2016; 21(4):230-237.

3. Mladenova I. Clinical relevance of Helicobacter pylori infection. J Clin Med 2021; 10(16):3473.

4.Rugge M, Fassan M, Pizzi M, Zorzetto V, Maddalo G, Realdon S, et al. Autoimmune gastritis: histology phenotype and OLGA staging. AP&T 2012; 35(12):1460-6.

5. Harris PR, Wright SW, Serrano C, Riera F, Duarte I, Torres J, et al. Helicobacter pylori gastritis in children is associated with a regulatory T-cell response. Gastroenterology 2008;134(2):491-499.

6. Suerbaum S, Michetti P. Helicobacter pylori infection. N Engl J Med 2002; 347:1175–1186.

7.Torres J, Pérez-Pérez G, Goodman KJ, et al. A comprehensive review of the natural history of Helicobacter pylori infection in children. Arch Med Res 2000; 31:431–469.

8. Rowland M, Daly L, Vaughan M. Age-specific incidence of Helicobacter pylori. Gastroenterology 2006; 130:65–72.

9.Agin M. The relationship between mean platelet volume and platelet levels of children with Helicobacter pylori and gastritis. Gastroenterol. Rev 2019; 14 (3):1-9.

10. Gulcu M. Association of Severity of Helicobacter pylori Infection with Peripheral Blood Neutrophil to Lymphocyte Ratio and Mean Platelet Volume. Euroasian J Hepatogastroenterol 2017; 7(1):11–16.

11.Kusters JG, van Vliet AHM, Kuipers EJ.

[DOI: 10.18502/ijpho.v16i3.21753]

Pathogenesis of Helicobacter pylori infection. Clin Microbiol Rev 2006; 19 (3):449–490.

12.Ruggiero P. Helicobacter pylori and inflammation. Curr Pharm Des 2010;16(38):4225–4236.

13. Guiraldes E, Pena A, Duarte I, et al. Nature and extent of gastric lesions in symptomatic Chilean children with Helicobacter pylori associated gastritis. Acta Paediatr 2002; 91:139–144.

14. Harris PR, Godoy A, Arenillas S, et al. CagA antibodies as a marker of virulence in Chilean patients with Helicobacter pylori infection. J Pediatr Gastroenterol Nutr 2003; 37:596–602.

15.Hajian-Tilaki K. Sample size estimation in diagnostic test studies of biomedical informatics. J Biomed Inform 2014; 48:193-204.

16.Farah R, Khamisy-Farah R. Association of neutrophil to lymphocyte ratio with presence and severity of gastritis due to Helicobacter pylori infection. J Clin Lab Anal 2014; 28(3):219-223.

17.Thapar N, Benninga MA, Crowell MD, Di Lorenzo C, Mack I, Nurko S, et al. Paediatric functional abdominal pain disorders. Nat Rev Dis Primers 2020; 6(1):89.

18.Squires RH, Jr., Colletti RB. Indications for pediatric gastrointestinal endoscopy: a medical position statement of the North American Society for Pediatric Gastroenterology and Nutrition. J Pediatr Gastroenterol Nutr 1996; 23(2):107-10.

19. Rugge M, Correa P, Di Mario F, El-Omar E, Fiocca R, Geboes K, et al. OLGA staging for gastritis: a tutorial. Dig Liver Dis 2008; 40(8):650-658.

20. Rugge M, Fassan M, Pizzi M, Zorzetto V, Maddalo G, Realdon S, et al. Autoimmune gastritis: histology phenotype and OLGA staging. AP&T 2012; 35(12):1460-1406.

21. Pennelli G, Grillo F, Galuppini F, Ingravallo G, Pilozzi E, Rugge M, et al. Gastritis: update on etiological features and histological practical approach. Pathologica 2020; 112(3):153-165.

22. Boyuk B, Saydan D, Mavis O, Erman H. Evaluation of helicobacter pylori infection, neutrophil–lymphocyte ratio and platelet–lymphocyte ratio in dyspeptic patients. Gastroenterol Insights 2020; 11(1):2-9.

Koc S, Gedikli MA. The role of neutrophil-lymphocyte ratio and platelet-lym phocyte ratios in predicting H pylori positivity and severity in patients with chronic gastritis. Cumhuriyet Med J 2022; 44(1):87–91.

23. Săsăran MO, Meliţ LE, Mocan S, Ghiga DV, Dobru ED. Pediatric gastritis and its impact on hematologic parameters. Medicine 2020; 99(35): 3-9.

24. Melit LE, Marginean MO, Mocan S, Marginean CO. The usefulness of inflammatory biomarkers in diagnosing child and adolescent's gastritis: STROBE compliant article. Medicine 2019; 98(26): e16188-e16192.

25.Turk Ozgur, Ozkececi T, Badak B, Bal A. Evaluation of mean platelet volume as a predictor of gastric disorders. ACES 2015; 5:1-9.

26.Sahin Y, Gubur O, Tekingunduz E. Relationship between the severity of Helicobacter pylori infection and neutrophil and lymphocyte ratio and mean platelet volume in children. De Pediatría. 2020:188-192.

27.Wu H, Zhou H, Chen P. Correlation of neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and mean platelet volume (MPV) with gout activity: a monocentric and retrospective study. Medicine 2022; 101(35): e30242-44.

28. Domșa AM, Lupușoru R, Gheban D, Șerban R, Borzan CM. Helicobacter pylori gastritis in children—the link between endoscopy and histology. J Clin Med 2020; 9(3):784-788.

[DOI: 10.18502/ijpho.v16i3.21753]