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Reviewer 1

This is well presented report, providing the evidence for the lack of relationship between Hp infection and NAFLD. Considering that Hp has been blamed for most of maladies affecting the human race, the data provided are clearly going against the grain. However,to improve the flow, the Introduction should be judiciously shortened by eliminating the redundancies. Also, in the abstract, under AIMS, the word "discover" should be changed to "determine". Otherwise, good job !

We thank the reviewer for the positive comments. The Introduction has been shortened to improve the flow as suggested. Also, the word "discover" has been changed to "determine".

Reviewer 2

This study aimed to investigate whether Helicobacter pylori (Hp) infection confers a higher risk of Nonalcoholic fatty liver disease (NAFLD).

Major comments 1. Comparison of continuous variables should be made by using the Mann-Whitney U test and not the Student's t test.

We thank the reviewer for this advice. We have changed the Statistics section to inform the readers that continuous variables were compared through the Mann-Whitney U test. Also, Table 1 and the Results section have been changed so that these reflect the change in Statistical analysis.

2. Discussion is very long and should be shortened.

We agree that the Discussion is too long. Though we have attempted to shorten the Discussion, adding the changes suggested by the reviewers has lengthened it. We are sorry that we could not comply with the reviewer's suggestion.

3. Is not clear how multivariable analysis of the risk factors for both NAFLD scores was performed. What was the cut-off p-value of factors from univariate to enter multivariate analysis?

A cut-off value of 0.05 was set. However, as we mentioned in the text, variables already associated with the formula such as Factors included in the HSI or NAFLD-LFS formula, such as sex, body mass index, diabetes, insulin, or metabolic syndrome components, were excluded from the analysis.

Minor comments 1. Continuous variables should be better expressed as median (IQR).

We thank the Reviewer for this excellent point. Continous variables have been expressed as

median in Table 1 and the Results section. The Methods section has been changed to reflect the change in statistical modality.

2. Data regarding virulence factors, such as the cytotoxin-associated gene A (CagA) and vacuolating cytotoxin A (VacA), of H. pylori strains are missing. This is a major limitation of the study.

Unfortunately, virulence factors could not be examined as this was a retrospective study involving individuals who underwent routine health check-up. This has been noted as a limitation in the Discussion section on page 13, lines 17-20 as below.

Fourth, though virulence factors of *H. pylori* such as *cagA* and *VacA* genes may have affected the degree of NAFLD, we could not investigate virulence factors as this was a retrospective study involving individuals who underwent routine health check-up.

3. There are a few grammatical errors that should be corrected (eg. Table 1. Clinicodemographic)

Thank you for pointing out that grammatical errors are in the manuscript. These have been corrected.

Reviewer 3

The manuscript is well designed and studied an interesting association between H. Pylori infection and NAFLD.

1.A major drawback is diagnosis of NAFLD, Authors did not use ultrasound or biospy to confirm diagnosis of NAFLD..It is better to more explain the accuracy of these two applied methods (HSI and NAFLD-LFS) for diagnosis of NAFLD in discussion.....

We thank the Reviewer for this excellent point. We had mentioned this as a limitation in the manuscript. However, as per the reviewer's suggestion, we have discussed the accuracy of these methods in the Discussion section on page 13, lines 10-12 as below.

However, though these formula may be limited in differentiating the degree of NAFLD[39,40], studies have reported that they are fairly robust in discriminating NAFLD with an AUROC of 0.80[24,40,41].

2.It is noteworthy to asses insulin resistance by HOMA-IR index and it association with H.pylori infection Since Insulin resistance is a mechanism for NAFLD Authors can refer and cite these articles: -Helicobacter pylori infection as a risk factor for insulin resistance.Dig Dis Sci. 2009 Sep;54(9):1966-70. doi: 10.1007/s10620-008-0557-7. -Insulin resistance and metabolic syndrome: is Helicobacter pylori criminal?Minerva Gastroenterol Dietol. 2011 Dec;57(4):379-85. Review. -The continuous story of

Helicobacter pylori infection and insulin resistance: this time in Japan.Helicobacter. 2010 Apr;15(2):160; author reply 161. doi: 10.1111/j.1523-5378.2009.00741.x

We thank the reviewer for pointing out these excellent references. These have been added to our articles as citations.

Reviewer 4

Determination of NAFLD was based on two noninvasive steatosis indices such as the hepatic steatosis index and NAFLD liver fat score. These two steatosis biomarkers are confounded by fibrosis and inflammation,and do not accurately quantify steatosis(Alimentary Pharmcology and Therapeutics 2014;vol.40:1209-1222/PLOs one 2014;vol.9:e94059);this may limit their clinical utility.

This has also been pointed out by Reviewer 3. We had acknowledged the limitations of HSI and NAFLD-LFS in the Discussion section. However, we further examined the accuracy of these two scoring formula in the Discussion section as per the suggestion of Reviewer 3 on page 13, lines 10-12.

However, though these formula may be limited in differentiating the degree of NAFLD[39,40], studies have reported that they are fairly robust in discriminating NAFLD with an AUROC of 0.80[24,40,41].

Furthermore, secondary causes of steatosis, including some hepatic virus infections (hepatitis non B and non C, cytomegalovirus, and Epstein-Barr virus), autoimmune hepatitis, metabolic liver disease, α-1-antitrypsin deficiency, cystic fibrosis, Wilson's disease, hemochromatosis, and celiac disease)were not excluded.

Secondary causes of steatosis as mentioned by the Reviewer are rare or nonexistent in Korea. Therefore we believe that our study has merit as the inclusion of the mentioned diseases would most likely not have affected our study. This has been added as a limitation in the Discussion section on page 13, lines 12-17 as below.

Third, we did not account for secondary causes of steatosis such as some hepatic viral infections, autoimmune hepatitis, Wilson's disease, α-1-antitrypsin deficiency, cystic fibrosis, hemochromatosis, and celiac disease. However, as these are either very rare in the Korean population[37-42], we believe that the inadvertent inclusion of these diseases would not have affected our study results.