Comments of the reviewer and point-by-point responses:

We thank the reviewer for the helpful comments that have improved our manuscript. Please find below our point-to-point responses to the specific comments and the denotation of the changes made in the manuscript accordingly.

Reviewer: 1 (02822816)

Comments to the Author

1. There are few grammar/syntax/spelling mistakes that need corrections (e.g. introduction, last paragraph"moreover, the canceration tendency..").

We are grateful to the reviewer for the suggestions to improve our work. We had revised "Moreover, the canceration tendency…" to "Moreover, the cancerization tendency…" in this sentence.

2. Introduction section: second paragraph: has anything to do with H pylori infection in children? If not, the entire paragraph including references 3-8 should be deleted.

Method section: too many details regarding cell culture, qRT-PCR, flow cytometry, luciferase assay, Western blot etc. I suggest that many of these could be mentioned as previously described associated with references.

Statistical analysis: please, mention if analysis was carried out by a specialist statistician.

Results need to be concisely and most of the Figures with their A,B, C, D, E, F G. H, I, J as well as a Table 1 could be deleted, as their presentation in text is enough.

Please make clear the novelty of your study, strength and limitations.

Introduction section: we have revised this part in the second paragraph as "*H. pylori* infection is regarded as the class I carcinogen[3]. Normally, *H. pylori*-induced gastritis could lead to gastric ulcer, which is the major precancerous lesion if without treatment. Although mainly residing in stomach, *H. pylori* display a strong ability of acid resistance. As a pathogen, *H. pylori* could attack and damage the mucosa of digestive tract by recruiting and activating neutrophils[4], inducing abnormal expression of key proteins[5] and microRNAs (miRNAs)[6], and releasing cytotoxic substances [7]. A previous study showed that *H. pylori* infection accounted for 6% of children with duodenitis[8]. Moreover, Gimiga *et al* found that gastritis and duodenitis were contributed to half of children with upper gastrointestinal bleeding, and 36.89% of participants were diagnosed with

H. pylori infection[9]. These findings suggested a relatedly high prevalence of children with *H. pylori* infection in digestive system."

Method section: We had revised the methodologies concisely.

Statistical analysis: The statistical analysis was supervised by a biostatistician from department of medical statistics, and we had uploaded the certificate.

To make readers better understand the results of the corresponding figures, it is necessary to mark A, B, C… in the results.

The novelty is that we found the potential target and underlying mechanism of miR-32-5p-induced intestinal epithelial cell injury of pediatric enteritis induced by *H. pylori*. The idea of this research was from the outpatients of our hospital and clinical studies abroad, and some children with enteritis were accompanied by *H. pylori* infection. The results provided a potential theoretical basis for better understanding the clinical problems. We thought the limitation of this research is the lack of *in vivo* experiments. Therefore, our group planned to further investigate this research in rat models.

Reviewer: 2 (00607640)
Comments to the Author
The topic "MicroRNA-32-5p aggravates intestinal epithelial cell injury of pediatric enteritis induced by Helicobacter pylori." is interesting and the author's made a major assertion that the aberrant expression of miR-32-5p plays crucial role in H. pylori–related pediatric enteritis, and TAK1-p38 pathway is involved in it. The conclusion is insightful, and could be helpful in clinic study.

We all show much respect to the reviewer's comment.

Reviewer: 3 (00504545)
Comments to the Author
This is a very interesting study demonstrating the important patogenic role of the miR32-5p in the production of chronic enteritis secondary to helicobacter pylori infection in children actuating through the target of the SMAD6 and actuvating the system by the TAK1-p38 inflammatory cascade. The experimental work has been well designed and performed and the conclusions are very clear demosntrated I want to congratulate the authors for your excellent contribution.

We are grateful for your reviewing.

Reviewer: 4 (00503405)

Comments to the Author

In this original article the authors demonstrated the harmful role of aberrant miR-35-5p in H. pylori-infected intestinal epithelial cells. They found that SMAD6 is the downstream of miR-32-5p and exerts an opposite role, moreover, miR-32-5p/SMAD6 contributes to TGF-β1-TAK1-p38 cascade in intestinal epithelial cells under H. pylori infection. The title, the abstract and the key words are all correct. The manuscript adequately describes the background, present status and significance of the study. The described methods are also adequate, a third part can reproduce the experiments by using the descriptions. Their findings are new, and of great clinical importance since the new aspects of HP pathogenesis are highlighted and a deeper insight of the pathogenesis and complex interaction of the bacteriumm and the host is possible now. The tables, figures are all representative. The used biostatistical methods are also correct and adequate. The used referece list is up to date. I suggest to accept the manuscript for publication in WJG.

Thank you for your reviewing.