For reviewer 1:

Major comments

1- As Amona et al (1) indicated, it has been difficult to prove the hypothesis that autophagy degrades intracellular pathogens, since all individual components of the autophagic machinery have not been identified although, authors indicated that the number of CFU were higher in mir-30d mimic and 3-MA groups, and lower in mir-30d inhibitor and autophagy activators (starvation, or Rapamycin) groups at all time point during a 60-h experiment, but there is stil a question arising from a reference by Chu YT. (page 16, at the end of part Mir-30d increases intracellular survival of H. pylori in AGS cells through inhibition of autophagy)

In Chu YT et al's study, they indicated following findings. "**H. pylori could invade the epithelial cells and multiply within double-layer vesicles either on the plasma membrane or in the cytoplasm and It is generally accepted that coccoid forms of H. pylori are viable but are not culturable. H. pylori is spiral in nature, but it transforms into a coccoid form under stress conditions**." Authors may have been missed these not culturable H. pylori in their study. Chu YT et al also indicated that "**many coccoid forms of H. pylori were found on the membrane of the infected AGS cells, and the ratio of coccoid to bacillary forms increased as infection proceeded. Several intracellular bacteria have developed different mechanisms to evade the autophagic surveillance in macrophages. The autophagic vesicles are induced, but their maturation intoautophagolysosomes is arrested or delayed**." Therefore, it is not certain to be killing of H. pylori strains inside the autophagic vesicles. My comment is to add this mechanisms in their reference 10 to their discussion part as indicated above. (**Chu YT, Wang YH, Wu JJ, Lei HY. Invasion and Multiplication of Helicobacter pylori in Gastric Epithelial Cells and Implications for Antibiotic Resistance. Infection And Immunity 2010; 78:4157-4165**)

1.Amano A, Nakagawa I, Yoshimori T.Autophagy in innate immunity against intracellular bacteria.J Biochem. 2006 Aug;140(2):161-6.

Answer: Thanks for the comment. We have add the mechanisms in our reference 10 to discussion part as indicated above and highlight it.

2. In discussion part, page 17, second paragraph, authors indicated that "**In this case, induction of autophagy would seem to be beneficial for cancer prevention**." However, It seems that autophagy also promotes cancer by enabling survival of cancer cells under starvation condition (**as authors reported in their previous study. and as other researchers reported**). I suggest, the role of autophagy, itself in cancer development is also important but sometimes promotes cancer development. They have to add this information given in their previous published manuscript.

Answer: Thanks for the comment. Yes, the role of autophagy in cancer development is important, sometimes it promotes cancer development. We have added this information to **discussion part** of this manuscript and highlight it.

3- There are some important references that were not used by authors. For example, the reference related with prostate cancer and miR-30d includes very important data for miR-30d and cancer development, authors may use this reference to support their hypothesis " **Kobayashi N, Uemura H, Nagahama K, Okudela K, Furuya M, Ino Y, Ito Y, Hirano H, Inayama Y, Aoki I, Nagashima Y, Kubota Y, Ishiguro H.Identification of miR-30d as a novel prognostic maker of prostate cancer.Oncotarget. 2012 Nov;3(11):1455-71**.".

Answer: Yes, we have added this reference to the manuscript to support our hypothesis(reference 37)

Minor Comments

1- Summary: The first and second sentences of the aim is not necessary and can be omitted.

Answer: Thanks. The 2 sentences in summary have been omitted.

2- Summary: Authors indicated "we can concluded that" but they may say "we suggested" or "we concluded".

Answer: Thanks. We have corrected this sentence

3- Summary: Last sentence "We suggested that enhanced autophagy by mir-30d inhibtor have a protective potential against Helicobacter pylori-related gastric cancer.

Must be modify as "We suggested that enhanced autophagy by mir-30d **inhibitor may** have a protective potential against Helicobacter pylori-related gastric cancer."

Answer: Thanks. We have corrected this sentence

4- Key words: miR-30d; this is the true word but in the text they used mir-30d, it must be corrected in entire text.

Answer: Thanks. We have corrected this word in entire text.

5. INTRODUCTION; This section is too long and some revisions must be; In the first paragraph, some of the sentences may be excluded from the text. My suggestion is **"Gastric cancer is the second most frequent cause of cancer-related death in the world and almost two-thirds of the cases occur in Asian countries, especially China and Japan[2,3]. The prognosis of gastric cancer is generally rather poor, and therefore, prevention is a better choice than cure for patients with gastric cancer."**

Answer: Thanks for the comment. We have corrected this paragraph following your instruction and highlight it.

6. INTRODUCTION; second paragraph the first sentence" **Helicobacter pylori is one of the most common human bacterial pathogens, and infection causes all kinds of gastric diseases, including gastritis, peptic ulcer and gastric cancer**[6]." can be excluded from the text.

Answer: Thanks for the comment. We have excluded this sentence from the manuscrpt.

7. INTRODUCTION; This long paragraph **"However, H. pylori infection has been proven difficult to cure despite intensive antibiotic treatment. One possible reason for the relatively high resistance to antimicrobial therapy is the ability of H. pylori to reside inside host cells[8,9]. Although considered by most as an extracellular pathogen, H. pylori can invade both gastric epithelial cells and immunocytes to some extent. The intracellular survival of H. pylori has been implicated in its ability to persist in the stomach, evade host immune responses**

and resist eradication by membrane-impermeable antibiotics[10]. Up to now, the precise mechanisms by which H. pylori exploits host cell machineries for intracellular survival are poorly understood. " may be shortened as **"One possible hypothesis for the relatively high resistance to therapy may be the ability of H. pylori to reside inside host cells[8,9]. Although considered as an extracellular pathogen, the intracellular survival of H. pylori in both gastric epithelial cells and immunocytes has been implicated in its ability to persist in the stomach, evade host immune responses and resist eradication by membrane-impermeable antibiotics[10]. Up to now, the precise mechanisms by which H. pylori exploits host cell machineries for intracellular survival are poorly understood."**

Answer: Thanks for the comment. We have corrected this part following your instruction and highlight it.

8. **INTRODUCTION, Again very long paragraph, "**Autophagy is an evolutionarily conserved process for delivering cellular materials and organelles to lysosome for degradation, and releasing them to the cytoplasm for recycle use [11,12]. This process is initiated by enclosing materials destined for degradation within double-membraned vacuoles, called autophagosome, and followed by fusion with lysosomes for promoting degradation of the luminal content. Autophagy is present in cells at a low basal level and can be induced when cells are undergoing environmental stress. This process helps to maintain cellular homeostasis and prevents organism from damaging and diseases. Autophagy is also regarded as one of the innate immunity effectors against intracellular bacterial infection[13]. For instance, Streptococcus pyogenes (Group A Streptococcus; GAS)-specific autophagy appears to selectively sequester and eliminate bacteria, which is distinct from nonselective canonical autophagy [14]. When the innate defense system recognizes invasive bacterial pathogens and their infection processes, autophagic proteins act as cytosolic sensors to rapidly launch the autophagic pathway [15]. However, many intracellular bacterial pathogens deploy highly evolved mechanisms to evade autophagic recognition, manipulate the autophagic pathway, and remodel the autophagosomal compartment for their own benefit [16]. Over the last decade, several research groups have independently reported that infection by H. pylori can

induce macroautophagy [10.17-19]. H. plori has been reported also to evade the autophagic machinery by downregulating the expression of autophagic proteins[19].The subversion of, or subjection to, host autophagic machinery by H. pylori adds further complexity to the already multifaceted pathogenesis of this bacterium." may be shorten as "**Autophagy, present in cells at a low basal level is an evolutionarily conserved process for delivering cellular materials and organelles to lysosome for degradation within double-membraned vacuoles, called autophagosome [11,12]. Autophagy is also regarded as one of the innate immunity effectors against intracellular bacterial infection (For example,Streptococcus pyogenes (Group A Streptococcus; GAS)-specific autophagy) [13,14]. Autophagic proteins act as cytosolic sensors to rapidly launch the autophagic pathway when the innate defense system recognizes invasive bacterial pathogens [15]. However, many intracellular pathogens use highly evolved mechanisms to evade autophagic recognition, manipulate the autophagic pathway, and remodel the autophagosomal compartment for their own benefit [16]. Over the last decade, several groups have reported that infection by H. pylori can induce macroautophagy and H. pylori may also evade the autophagic machinery by downregulating the expression of autophagic proteins [10.17-19]**."

Answer: Thanks for the comment and suggestion. We have corrected this part following your instruction and highlight it.

9. MATERIALS AND METHODS; "psicheck2 reporter vectors" must be corrected as "**The psiCHECK-2 Vector**"

Answer: Yes, we have corrected this part following your instruction and highlight it.

10. MATERIALS AND METHODS; green fluorescent protein must be specified for The GFP-LC3 as green fluorescent protein (GFP)-LC3.

Answer: Yes, we have corrected this part following your instruction and highlight it.

11. MATERIALS AND METHODS; ***Antibodies and reagents*** obtained from Cell Signaling must be explained as obtained from Cell Signaling Technology (CST).

Answer: Yes, we have corrected this part following your instruction and highlight it.

12. Countries of companies must be indicated inside paranthesiss for purchased materials [for example, RPMI1640 (Cellgro), FBS; (Invitrogen) (Stemcell Technologies)].

Answer: Yes, we have corrected this part following your instruction and highlight it.

13. MATERIALS AND METHODS; "The human gastric cancer cell line AGS was cultured" AGS must be specified as "**AGS cells (a human gastric adenocarcinoma cell-line) were obtained from the American Type Culture Collection (Rockville, MD, USA)**," or from other company but the purchased company's name must be specified.

Answer: Yes, we have corrected this part following your instruction and highlight it.

14. MATERIALS AND METHODS; "the cell line GES-1" in the same way, the company's name must be specified as " **was cultured human gastric mucosal epithelial cell line GES-1 (Purchased from Cell bank of Xiangya Medical School, Central South University, China)**"

Answer: Yes, we have corrected this part following your instruction and highlight it.

15. MATERIALS AND METHODS; "26695(700392) was obtained from ATCC" ATCC must be specified as "**American Type Culture Collection (Rockville, MD, USA)".**

Answer: Yes, we have corrected this part following your instruction and highlight it.

16. MATERIALS AND METHODS; (Invitrogen) mus be TRIzol reagent **(Invitrogen, USA).**

Answer: Yes, we have corrected this part following your instruction and highlight it.

17. MATERIALS AND METHODS; (P<0.05 vs.control mimic transfected cells) must be2- (P<0.05, mimics versus control).

Answer: Yes, we have corrected this part following your instruction and highlight it.

18. MATERIALS AND METHODS; (P<0.05 vs. without H. pylori infected cells) must be (P<0.05, H. pylori infected cells versus without H. pylori infected cells).

Answer: Yes, we have corrected this part following your instruction and highlight it.

19. MATERIALS AND METHODS; "with H. pylori compared to **no infected cells** at 24h after H. pylori infection," must be "with H. pylori compared to **non-infected cells** at 24h after H. pylori infection,".

Answer: Yes, we have corrected this part following your instruction and highlight it.

20. MATERIALS AND METHODS; (**P<0.05, vs.control mimic transfected cells, Fig. 2C**) must be (**P<0.05, mimics versus control, Fig. 2C**).

Answer: Yes, we have corrected this part following your instruction and highlight it.

21. MATERIALS AND METHODS; (**P<0.05, vs.control mimic transfected cells, Fig. 2D**) must be (**P<0.05, mimics versus control, Fig. 2D**).

Answer: Yes, we have corrected this part following your instruction and highlight it.

22. MATERIALS AND METHODS; "For loss of function experiments, we using mir-30d inhibitor blocked endogenous mir-30d expression in the above cell lines(AGS and GES-1), then infected with H. pylori." must be revised and I suggest ""For loss of function experiments, **and endogenous mir-30d expression was blocked by mir-30d inhibitor i**n the above cell lines(AGS and GES-1), then infected with H. pylori.

Answer: Thansk for the comment and suggestion. we have corrected this sentence following your instruction and highlight it.

23. MATERIALS AND METHODS; (**P<0.05, vs.control oligos transfected cells**) must be (**P<0.05, oligos transfected cells versus control**).

Answer: Yes, we have corrected this part following your instruction and highlight it.

24. MATERIALS AND METHODS; (**P<0.05, vs.control oligos transfected cells, Fig. 3C**) must be (**P<0.05, oligos transfected cells versus control, Fig. 3C**).

Answer: Yes, we have corrected this part following your instruction and highlight it.

30. **MATERIALS AND METHODS;** (**P<0.05, vs.control oligos transfected cells, Fig. 3D**). must be (**P<0.05, oligos transfected cells versus control, Fig. 3D**).

Answer: Yes, we have corrected this part following your instruction and highlight it.

25. MATERIALS AND METHODS; "we want to demonstrated" must be "**we want to demonstrate**".

Answer: Yes, we have corrected this sentence following your instruction and highlight it.

26. MATERIALS AND METHODS; "**MicRNAs are known to regulate gene**" must be "**MiRNAs are known to regulate gene**"

Answer: Yes, we have corrected this word following your instruction and highlight it.

27. MATERIALS AND METHODS; "**MicRNA post-transcriptionally regulates**" must be "**MiRNA post-transcriptionally regulates**".

Answer: Yes, we have corrected this word following your instruction and highlight it.

28. DISCUSSION; "**Epidemiological, clinical, and animal studies have established a central role for H. pylori in gastric carcinogenesis and provided insights into the mechanisms and biologic relationships between bacterial infection, host genetics, nutrition, and environmental factors[6]**." Unnecessary, may be omitted to shorten the discussion.

Answer: You are right , this paragraph has been deleted from discussion

29. DISCUSSION; "**Basal autophagy plays a critical role in maintaining cellular homeostasis and genomic integrity by degrading aged or malfunctioning organelles and damaged or misfolded proteins. Meanwhile, autophagy also plays a complicated role in tumorigenesis and treatment responsiveness. It can be tumor-suppressing during the early stages of tumorigenesis, as reduced autophagy**

is found in tumor cells and may be associated with malignant transformation[34-36]." may be shortened as "**Autophagy may be tumor-suppressing during the early stages of tumorigenesis, as reduced autophagy is found in tumor cells and may be associated with malignant transformation[34-36].**".

Answer: Thanks for the comment.This paragraph has been shortened following your instrction and highlight it.

30. DISCUSSION; "**In this case, induction of autophagy would seem to be beneficial for cancer prevention**" may be revised as "**In this case, induction of autophagy would seem to be beneficial by indirectly for cancer prevention**,".

Answer: Yes, we have corrected this word following your instruction and highlight it.

31. DISCUSSION; "**regulats** autophagy process by directly targeting multiple genes of the autophagy pathway[29]." must be "**regulates** autophagy process by directly targeting multiple genes of the autophagy pathway[29]."

Answer: Yes, we have corrected this word following your instruction and highlight it.

32. DISCUSSION; "**Meanwhile, the expression of mir-30d is upregulated in both two cells after H. pylori infection; This event appears to be unique to H. pylori infection since infection with other pathogens,(E. coli DH5α and O157:H7) or treatment with autophagy modulators, e.g., rapamycin and 3-methyladenine do not have any effect on mir-30b expression[19].**" You can't say this because the first sentence was emphasizing mir-30d but the second sentence was giving mir-30b. On the other hand, this sentences may be revised as T**he expression of mir-30d is upregulated in both two cells after H. pylori infection in our experiments; This event appears to be unique to H. pylori infection but must be repeated with other pathogens in order to prove this hypothesis. In a similiar way, infection with other pathogens,(E. coli DH5α and O157:H7) or treatment with autophagy modulators,**

e.g., rapamycin and 3-methyladenine do not have any effect on mir-30b expression[19].

Answer: Thanks for the comments. We have corrected this paragraph following your instruction and highlight it.

33. DISCUSSION; "To explore the role of mir-30d in autophagy process during H. pylori infection, mir-30d mimic was transfected into AGS and GES-1 cell lines for 24h, then infected with H. pylori, we found that autophagy process were upregulated(increased LC3B-II expression) in both two cell lines during H. pylori infection, but upregulated mir-30d significantly inhibited this process; However, when we using mir-30d inhibitor blocked endogenous mir-30d expression in the above cell lines, the autophagy process induced by H. pylori infection was obviously increased by down regulation of mir-30d. We also confirmed mir-30d represses autophagy process by directly targeting multiple core genes of autophagy pathway in gastric epithelial cells, including ATG2B, ATG5, ATG12, BECN1 and BNIP3L. At last, a gentamicin protection assay indicate that inhibition of autophagy increases the intracellular survival of H. pylori in AGS cells." This is a repeat and explained by the authors many times in the results section, therefore this part must be shortened with a few sentences. I suggest as "**We found that autophagy were upregulated in both two cell lines by transfection with mir-30d mimic during H. pylori infection, but upregulated mir-30d significantly inhibited this process. However, mir-30d expression was blocked by mir-30d inhibitor and the autophagy was obviously increased by down regulation of mir-30d. Mir-30d also represses the autophagy process by directly targeting multiple core genes (ATG2B, ATG5, ATG12, BECN1 and BNIP3L). A gentamicin protection assay indicated that inhibition of autophagy increases the intracellular survival of H. pylori in AGS cells**."

Answer: Thanks for the comments. We have corrected this paragraph following your instruction and highlight it.

34- DISCUSSION; "The close relationship between H. pylori infection and the gastric cancer indicate logical points for preventive approaches[30]. Optimal

approaches will have to be tailored to local communities, and should include strategies for primary prevention, to interrupt transmission; secondary prevention, to identify individuals with infection and treat with H. pylori eradication therapy; and tertiary prevention, to identify individuals with propensity for disease progression or with early disease for timely curative multimodality treatment [41,42]." This part is not related with the study and may be revised as"**Preventive measures for gastric cancer must include tertiary prevention and effective treatment of H. pylori infections**".

Answer: Thanks. We have corrected this paragraph following your instruction and highlight it.

35- Fig1 "in H. **pylori.** infected cells" must be without dot as "in *H. **pylori*** infected cells".

36- "**We suggested that enhanced autophagy by mir-30d** inhibtor **have a protective potential against Helicobacter pylori-related gastric cancer**. " must be "**We suggested that enhanced autophagy by mir-30d** inhibitor **have a protective potential against Helicobacter pylori-related gastric cancer**. "

Answer: Yes, we have corrected this paragraph following your instruction and highlight it.

37- "by enhanced autophagy using mir-30d **inhibtor**." must be "by enhanced autophagy using mir-30d **inhibitor**."

Answer: Yes, we have corrected this word.

38- "after mir-30d **inhibito** transfection(P<0.05, vs.control oligos transfected cells), same result also found in GES-1 cells (P<0.05, vs.control oligos transfected cells)." must be "after mir-30d **inhibitor** transfection(P<0.05, vs.control oligos transfected cells), same result also found in GES-1 cells (P<0.05, vs.control oligos transfected cells).".

Answer: Yes, we have corrected this sentence following your instruction and highlight it.

39. H. pylori must be italic *H. pylori* throughout the entire text.

Answer: Yes, we have corrected this word in entire text.

40. In Discussion, page 19, second paragraph; "**However, Secondary prevention**" must be "**However, secondary prevention**"

Answer: Yes, we have corrected this sentence following your instruction and highlight it.

For reviewer 2:

Title: Yes, it reflects in the best way the topic and content of the study

(1) Abstract:

In methods, it is needless to mention the procedures to evaluate that mir-30d represses (suppresses) the expression levels of multiple core proteins in the autophagy pathway in gastric epithelial cells.

Answer: Yes, we have corrected this sentence following your instruction and highlight it.

In conclusion, the phrase "Our findings may provide a novel mechanism for elucidating persistent H. pylori infection appears to provide a promising target for gastric cancer prevention. We suggested that enhanced autophagy by mir-30d inhibtor have a protective potential against Helicobacter pylori-related gastric cancer". It sounds very adventurous to mention this even when much remains to be known on mir RNA mediated autophagy inhibition mechanism since it was not only mir-30d has been observed to have the capability to inhibit H. pylori induced autophagy. And so, to mention it as a protective potential against Helicobacter pylori-related gastric cancer is still too early, since mir-30b is also capable of inhibiting autophagy (Tang B et al 2012).

Answer: Thanks for the comment. We have corrected this phrase following your instruction and highlight it.

(2) Text body:

There are some type errors in some words.

Background:

a) In this part, all the paragraph should be summarized because it seems very dense.

"Gastric cancer is the third most common cancer among males and fifth most common among females [1]. In 2002, 700,000 deaths were recorded, making it the second most frequent cause of cancer-re¬lated death in the world and almost two-thirds of the cases occur in Asian countries, especially China and Japan [2,3]. The incidence of gastric cancer is declining in developed countries, but the global burden is rising and is projected to top 1.1 million cases per year in 2010, mostly due to cases occurring in developing countries [4]. Although current major therapies, including surgery and chemotherapy, have been widely used, the prognosis of gastric cancer is generally rather poor, with 5-year relative survival below 30% in most countries [5]. It suggests us (This suggests) that prevention is better than cure for patients with gastric cancer. *Helicobacter pylori* is one of the most common human bacterial pathogens, and infection causes all kinds of gastric diseases, including gastritis, peptic ulcer and gastric cancer [6]. This bacterium was designated a class I carcinogen by the International Agency for Research on Cancer in 1994 due to its strong correlation with gastric cancer in humans [7]."

Answer: Thanks for the comment. We have corrected this paragraph following your instruction and highlight it.

b) In accordance with your previous study, you know more gens (ATG2B, ATG5, ATG12, BECN1, and BNIP3L). Why did you only consider two in this part? Moreover, in materials and methods as well as in results ATG2B, ATG5, ATG12, BECN1, and BNIP3L were involved. This part sholud be restructured because it creates confusion "We hypothesized that mir-30d could downregulate key autophagy genes expression, such as BECN1 and ATG12 to inhibit autophagy

response to H. pylori invading into gastric epithelial cells, resulting in increased H. pylori intracellular survival."

Answer: Thanks for the comment. We have corrected this part following your instruction and highlight it.

c) In the background, it would be good to consider a justification of the study i.e. the importance of the study, what is not known or new and its contributions, and make more emphasis on your previous study.

Answer: Yes, we have corrected this part following your instruction and highlight it.

Methods:

a) Why did you carry out gentamicin protection assay only on AGS cells and not in GES-1?

Answer: We carried out gentamicin protection assay on both AGS cells and GES-1 cells. The results from GES-1 cells was not so significantly different comparing with that from AGS.

b) In the part that said, "Autophagy increased in AGS and GES-1 cell lines in response to H. pylori infection", to evaluate the efficient flow of autophagy. Did you use some inhibitors of autophagy? in order to know if H. pylori induced autophagy could eliminate intracellular bacteria.

Answer: No, we did not use any inhibitor of autophagy. We used only non-infected as control and also autophagy activators (starvation. Rapamycin) in this experiment. .

c) You used only 26695 strain? Perhaps, the type of strain influences in the induction of autophagy and/or in the induction of mir 30d expression.

Answer: We used only 26695 strain in this experiment. Yes. It is possible that the type of strain may influences the induction of autophagy and/or in the induction of mir 30d expression It is interesting to pursue the experiment.

d) What importance has the use of AGS vs. GES cell lines?

Answer: AGS cells is a human gastric adenocarcinoma cell-line, and GES-1 is a human gastric mucosal epithelial cell line. These two cell lines can be the represent in early stage of gastric cancer.

Results:

a) In the figures 1, 2 and 3, H. pylori is written as "Polyri". This should be corrected.

Answer: Yes, we have corrected this word.

b) In the figure 2B, why in the case of AGS cells in the presence of mir-30d, LCB3-II was observed? What is the explanation of this?

In the experiment, we focused on the ratio of LCB3-II/LCB3-I more than the absolute number for LCB3-I. Although LCB3-II was observed in the presence of mir-30d, the expression of LCB3-I increased more significant in AGS cells. The ratio of LCB3-II/LCB3-I decreased and then it proved a phenotype of repressed autophagy.

Disscusion:

a) Don´t discuss the importance that although mir 30d and mir 30b inhibit autophagy. The target gens of thesemir RNA are ATG12 and BECN 1 which are common for both, but BNIP3L, ATG5 and ATG2 are only for mir 30d. Can this have any meaning?

Answer: Thanks for the comment. We have corrected this part following your instruction and highlight it.

b) Phrases in the discussion as:

"We suggested that enhanced autophagy by mir-30d inhibtor have a protective potential against Helicobacter pylori-related gastric cancer."

"Although the mechanism of H. pylori infection persistence remains to be further determined, the present study provided a new approach for secondary prevention of gastric cancer: eradication of Helicobacter pylori by enhanced autophagy using mir-30d inhibtor." Can not be affirmed or suggested without first checking an

Inverse correlation between mir30d and ATG12/BECN1/BNIP3L/ATG5/ATG2 expression in H. pylori-positive human samples.

The results no provide sufficient experimental evidence or data to draw these comments or conclusions. However this study establishes a basis necessary for future evaluation of mir30d in H. pylori infections.

Answer: Thanks. We have corrected this part following your instruction and highlight it.

b) Sometimes, there are parts in the discussion that sound like description of results. Should you take this into account

Answer: Yes, we have corrected this part and highlight it.