Response to reviewers' comments:
We highly appreciate the reviewers' insightful and helpful comments on our manuscript. The response is now highlighted in red characters.

Response to reviewer 1:

Reply: Thanks for your high praise.

Response to reviewer 2:

1. Figure 1 does not describe clearly whether the data was obtained from the cell lines or from tumor tissues derived from patients. The sample size that was used to gather this data was also not described
   Reply: Thanks for your review. Exosomes were isolated from NOZ cell lines. The specific isolation process is described in "Separation and screening of exosomes".
2. To describe intracellular miR-182 as endogenous and exosomal miR-182 as exogenous is not appropriate. Exogenous tends to refer to materials that originate from outside the body. Therefore, exosomal miR-182 are still considered as endogenous as it is derived from host cells.
   Reply: Thank you for your review. Endogenous has been revised to exosomal miR-182.
3. In the paragraph on miR-182 targeted inhibition of RECK expression, miR-182 was wrongly described as miR-195
   Reply: Thanks for your review. The mistake has been modified.
4. There are multiple typographical errors that need to be corrected. It is recommended that a language expert be engaged to proofread the final manuscript
   Reply: Thanks for you review. The mistake has been modified.

Response to reviewer 3:

1. In the introduction,"Moreover, RECK exerts its influence on tumor progression through the regulation of miRNA." But all the following examples demonstrated that miRNAs exert influence on tumor progression through regulating RECK.
   Thanks for your review.
   Reply: Thanks for your review, we have already revised the sentence to "Moreover, the influence of RECK on tumor progression was regulated by miRNA".
2. Although numerous studies have elucidated the molecular mechanism of miR-182 and RECK in various cancers, yet the very role of exosomal miR-182 and RECK in GC remains unclear." The paper is about miR-182 and RECK, but the well-known or established molecular mechanism about the relationship between miR-182 and RECK was not elucidated in the introduction
   Reply: Thank you for your review. We have modified it to "Although many a study has respectively clarified the molecular mechanism of miR-182 and RECK in a variety of cancers, the co-role of exosomal miR-182 combined with RECK in GC remains unclear".
3. In the materials and methods, (Sample Collection) "The collected tissues were

sectioned and stored in liquid nitrogen at -80 °C for testing" "the supernatant was collected and placed in -80℃ liquid nitrogen to be measured." Were the collected tissues stored in liquid nitrogen or at -80 °C?

Reply: Thanks for your review. It should be -80 ° C instead of -80 °C liquid nitrogen and we have already modified it.

4. In methods, the expression "Cells were hydrolyzed with trypsin to make cell suspension." is not appropriate.

   Reply: Thanks for your review. We have revised it to "The adherent cells were digested with trypsin and the supernatant was obtained by 1103 xg centrifugation for 1 min, and then added to the cell medium to resuspend the cells".

5. About the apoptosis assay, the author didn't mention the details in the paper, such as whether any reagent was used or not?

   Reply: Thank you for your review. In this paper, flow cytometry and Western blot were used to detect the apoptosis of apoptosis-related proteins Caspase 3/Caspase 9/Bax/Bcl2. See "Western blot" and "flow cytometry" for detailed methods

6. Page 11, Result "Compared with normal human blood samples, serum miR-182 level was significantly elevated in patients with GC (Figure 2B). " "Figure 2. B: Exogenous miR-182 was highly expressed in patients with GC. On the image, "exosomal miRNA182" The expression is not consistent. "Here, we compared the expression of exogenous/endogenous miR-182 in different NM phases and analyzed the correlation between miR-182 and clinical features" This author used "exogenous and endogenous" to distinguish "cell or tissue" derived and "exosome "derived miR-182, which is confusing.

   Reply: Thank you for your review. The Exogenous miR-182 has uniformly modified to exosomal miRNA182.

7. Page 12 "Exosomal miR-182 was associated with GC metastasis" Based on figure 3 data, both cellular and exosomal miR-182 increased in cancer as compared to normal tissue.

   Reply: Thank you for your review. This suggests that both exosomal miR-182 and self-encoded miR-182 are involved in GC metastasis, and we have modified title as "miR-182 is involved in GC metastasis".

8. Page 13, in rescue assay, "Exosomal miR-182, exosomal miR-182+RECK overexpression vector were added to GC cells for the conduction of corresponding MTT, Western blot and Transwell experiments." Is synthesized miR-182 or exosome used in this assay? If only synthesized miR-182 was used in this assay, this experiment may only prove miR-182 relevant to cancer, not exosomal miR-182 specifically

   Reply: Thank you for your review. The rescue experiment was intended to show that upregulation of RECK could counteract the changes caused by exosomal miR-182. For the source of exosomal miR-182 here, see "Separation and screening of exosomes". No miR-182 overexpression vector was introduced in the rescue experiment.