Reviewer's report

Title: MicroRNA-21 inhibitor sensitizes human glioblastoma cells U251 (PTEN-mutant) and LN229 (PTEN-wild type) to taxol

Version: 2 Date: 16 October 2009

Reviewer: Hideo Baba

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Major Compulsory Revisions

Substantial data indicate that miR-21 regulates multiple genes associated with cancer cell proliferation, apoptosis, and invasiveness in several solid cancers including GBM. Thus, miR-21 can become a target to enhance the chemotherapeutic effect in cancer therapy.

In this study, Authors demonstrated that miR-21 blockage could increase the chemosensitivity to taxol. The miR-21 inhibitor might interrupt the activity of EGFR pathways. So, it is very interesting that a combination of the miR-21 inhibitor and taxol could be an effective therapeutic strategy for suppressing the growth of GBM.

However there are some questions and problems to be solved as follows.

1) Though there are some reports that PTEN is a target gene of miR-21, PTEN has no binding site for miR-21. It is expected that the effect of the miR-21 inhibitor for taxol is unchanged regardless of the presence of the mutation of PTEN.

   What kind of mechanism of miR-21 regulating PTEN may exist?

2) In figure 4, PTEN seems to be regulated by miR-21 inhibitor, and to regulate EGFR pathways as a result. What is the mechanism for control of EGFR.

3) How do you explain the effect of the miR-21 inhibitor and taxol is different from additively and synergistically?

4) In figure 6, the legends and the scale are not correct. In addition, there are some graphs in which the unit is not described.

Level of interest: An article whose findings are important to those with closely related research interests

Quality of written English: Acceptable

Statistical review: Yes, but I do not feel adequately qualified to assess the statistics.