Author's response to reviews

Title: Loss of miR-133a expression associated with poor survival of breast cancer and restoration of miR-133a expression inhibited breast cancer cell growth and invasion

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Author's response to reviews: see over
December 27, 2011

Re: re-submission of our manuscript (#MS: 8203712595721335)

Dear Dr. Chap:

Many thanks for your email and the constructive comments on our revised manuscript, 8203712595721335. We have made all possible changes to satisfy the reviewers. However, we couldn’t add any xenograft data due to limitation of the time and we also strongly believe, it will not increase the value of the manuscript (we discussed it briefly in discussion section of the revised manuscript according to your advise). Furthermore, our ex vivo data confirmed a previous study and demonstrated that the lost miR-133a expression was associated with lymph node metastasis, high clinical stages, and shorter survival of these breast cancer patients. If an in vivo experiment is required for justification of publication, it will be the nude mouse metastasis assay with stably inducible miR-133a-expressed cell sublines, which will take long time, but it may not get the data we really want because of the difference between human and rodent. Therefore, I will really appreciate it, if you agree with us. Thank you very much for your supports.

The changes in the text are underlined in red. Thank you again for your consideration.

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Our responses to the reviewers’ comments

Reviewer: Hailong Wu

As I mentioned in my last comments, I think xenograft model is required. Apparently, the authors want to connect mir-133a mediated impairment on breast cancer migration and invasion in vitro with mir-133a related relapse-free survival, so at least they need demonstrate that mir-133a over-expression can suppress breast cancer invasion in vivo. in vitro data only is not enough to make this work published in BMC cancer.

We thank the reviewer for this constructive suggestion. However, nude mouse
xenograft model takes longer time and costly. Most importantly, such an animal experiment requires stable gene transfection, which may generate selection bias for resistant cell sublines, unless to use an inducible vector. Addition of xenograft assay will not mechanistically answer the question of the role of mir-133a in silence of FSCN1 in breast cancer. Nevertheless, the conclusion of our current ex vivo data is clear and justified, although there is a need to be further verified in different patient populations before use of miR-133a as a biomarker for predication of breast cancer invasion and metastasis in clinic.