Reviewer's report

Title: MiR-125b promotes proliferation and migration of type II endometrial carcinoma cells through targeting TP53INP1 tumor suppressor in vitro and in vivo

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Jing et. al. MiR-125b promotes proliferation and migration of type II endometrial carcinoma cells through targeting TP53INP1 tumor suppressor in vitro and in vivo.

Jing et. al. reported over-expression of miR-125b in KLE and AN3CA (type II and ER- EC cells) compared to ishikawa and RL95-2 (type I and ER+ EC cells) by qRT-PCR and northern blotting. Using same cells lines, the authors demonstrated that over-expression of miR-125b promoted the growth of type I EC cells in vitro and in vivo, and promoted cell migration using an in vitro migration assay. Similarly, knocking down miR-125b expression decreased the in vitro proliferation and the migration capability of type II EC cells in vitro. The authors then used qRT-PCR, western blotting, and luciferase-based assay and demonstrated TP53INP1 as a direct target for miR-125b in EC cells.

Overall this is a good study as it described an oncogenic role for miR-125b in EC cancer cell lines, which is consistent with previous studies reporting an oncogenic role for this microRNA in other human cancers. Over-expression of miR-125b in advanced EC tissue has been described before (Ref# 23). Nonetheless, and as stated by the authors, it was previously shown by many studies that miR-125b is an oncogenic microRNA in several human cancers. The authors also claimed TP53INP1 as a novel target for miR-125b, but a previous study (Genes Dev. 2009 Apr 1;23(7):862-76. Epub 2009 Mar 17. MicroRNA-125b is a novel negative regulator of p53. Le MT, Teh C, Shyh-Chang N, Xie H, Zhou B, Korzh V, Lodish HF, Lim B.) reported regulation of several proteins in the TP53 pathway (including TP53INP1) by miR-125b. In this study, the authors had confirmed TP53INP1 as a target for miR-125b using additional assays such as western blotting and luciferase assay. Therefore, the novelty in this work relates to reporting miR-125b as an oncogenic microRNA in type II EC.

Major Compulsory Revisions

1. Is the phenotype observed in the experiments in figures 2,3,4, and 6 mediated by TP53INP1? The authors can perform either rescue experiments (to reverse the phenotype observed when overexpressing miR-125b) or at least to knockdown the expression of TP53INP1 in EC I cells to see if this would mimic the effects of miR-125b overexpression.

2. What is the expression level of TP53INP1 in EC I vs EC II primary tissues? Is there inverse relationship between the expression of TP53INP1 and miR-125b?
3. The authors referred to their previous microRNA microarray in type I and type II EC cells. Are those data published? If not, is it possible to include at least the miR-125b expression data to confirm the clinical relevance of the findings in primary cancer specimens.

Minor Essential Revisions
1. The authors claim ref#22 as their own work, but this is not true?
2. In Fig 2C and 2D, why is the doubling time different even for the untreated cells?

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Statistical review:** No, the manuscript does not need to be seen by a statistician.