Reviewer's report

Title: Overexpressed E2F1 in human gastric carcinoma is associated with anti-cancer drug resistance

Version: 2 Date: 21 October 2014

Reviewer: Johann Rotheneder

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Major Compulsory Revisions:

1. The surprising finding in the original manuscript that overexpression of E2F1 results in lower rather than in higher p73 (Tap73) levels has been revised now. The new Figure 3A shows the expected upregulation of p73 by E2F1.

But the new data have not found their way into the discussion and conclusion sections:

In the “Discussion” section (page 12 lane 395) it is stated:
In our result, E2F1-overexpression lentiviral vector induced ZEB1 and ZEB2 expression up-regulation and down-regulation of TAp73 and GAX.

In the “Conclusion” section (page 13 lane 404) it is stated:
We conclude that up-regulation of E2F1 promotes the development of multidrug resistant in gastric carcinoma via inhibition of apoptosis related gene expression (TAp73 and GAX).

This must be corrected.

2. The revised finding that E2F1 overexpression does stimulate p73 contradicts the finding that E2F1 decreased the percentage of apoptotic cells. This must be discussed.

Minor Essential Revisions:

In the “Results” section (page 10 lane 318) it is stated:
To better understand the function of E2F1, we performed a yeast two-hybrid screen using E2F1 as the bait and identified MRP as a MRP-interacting protein.

I assume this should mean: MRP as an E2F1-interacting protein

Discretionary Revisions:

Although it is still my opinion that the role of E2F1 in tumorigenesis should be described more thoroughly it can be argued (as the authors do) that this not essential for the paper.
Level of interest: An article whose findings are important to those with closely related research interests

Quality of written English: Acceptable

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

Declaration of competing interests:
I declare that I have no competing interests