Reviewer's report

Title: Disruption of Focal Adhesion Kinase and p53 Interaction with Small Molecule Compound R2 Reactivated p53 and Blocked Tumor Growth.

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Reviewer: Juan Martinez

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Comments to:

Disruption of Focal Adhesion Kinase and p53 Interaction with Small Molecule Compound R2 Reactivated p53 and Blocked Tumor Growth.
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The paper by Golubovskaya et al describes the structural modeling of the polyproline region of p53 involved in the interaction with the FERM domain of FAK. The p53 heptapeptide known to bind to FAK was docked into the known structure of the FERM domain, thus defining the cavity in which this heptapeptide fits. The authors, then, screened “in silico” more than 200,000 compounds from the NCI database, docking them into the FAK-p53 interaction surface, and finding a family of compounds they named “roslins” that fit into this cavity. Among them, the R2 compound was the one that provided the best binding to FAK, disruption of the FAK-p53 interaction and reduction of the cancer cell viability in a p53-dependent fashion. Furthermore, the authors demonstrated that R2 compound is able to functionally disrupt the interaction of p53 with FAK, increasing the transactivation of p53 target genes and, as a result, enhancing its tumor suppressor activity in vitro and in vivo.

After improving the overall quality of the figures and addressing other questions that were unclear in the previous version of the paper, now the paper is suitable for publication in BMC Cancer.

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.

Declaration of competing interests:

I declare that I have no competing interests