Reviewer's report

Title: Cytosolic galectin-7 impairs p53 functions and induces chemoresistance in breast cancer cells

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Reviewer: Pankaj Singh

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In present manuscript authors have studied the role of a cell surface glycan binding protein galectin-7, in chemoresistance of breast cancer cells. Authors have shown that galectin-7 overexpression leads to increased chemoresistance in breast cancer cells that is mediated through impaired translocation of p53 from cytosol to nucleus. They also demonstrate that mutant galectin-7 (R74S) gets accumulated to cytosol and impairs p53 translocation to nucleus by directly interaction with it. Impaired p53 function leads to reduced expression of p21 protein which is a downstream target of p53.

Major compulsory revision:
1- As authors claim that increased cytosolic galectin-7 leads to increased resistance to chemotherapy, they should compare the chemosenstivity of different cells which they have represented in Figure 1D.

Minor essential revision:
2- In Figure 3A, galectin-7 level in whole cell extract should be represented. In same Figure, Beta-tubulin blot should be repeated.
3- In result section authors have written that (page 6, last paragraph) inhibition of p53 co-related with decrease in p21 protein expression and CDKN1A mRNA (Figure 6, B-C), but in figure only p21 has been presented.
4- In results section, page 5, line 127-129 needs to be reframed.

Discretionary revisions
5- Effect of Galectin-7 and its mutant expression on cell growth should be studied, which will ensure that it just inhibits apoptosis without affecting cell proliferation.
6- Authors should study the half-life of cytosolic p53 protein in presence and absence of galectin-7 wild type as well as mutant form.

Level of interest: An article of importance in its field

Quality of written English: Needs some language corrections before being published
**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.