Reviewer's report

Title: Pim1 promotes human prostate cancer cell tumorigenicity and c-MYC transcriptional activity

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Reviewer: guenter schneider

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Kim et al. investigated the oncogenic function of the serine/threonine kinase PIM1 in several prostate cancer model systems in vitro and in vivo. The main finding is that Pim1 expression enhances the in vitro and in vivo tumorigenic potential of prostate cancer cell lines LNCaP and DU145. Furthermore, the authors provide some data, that c-myc is a downstream effector of PIM1. This is an interesting and important study validating oncogenic PIM1 function in prostate cancer. Nevertheless, the proposed PIM1-c-myc pathway is not completely shown at the experimental level and therefore, the manuscript needs some revision.

1) Upregulation of c-myc at the protein level in PIM1 transfected LNCaP cells is not visible in the western blot provided. Therefore the statement “was further increased” should be excluded.

2) The authors must provide evidence for the conclusion that c-myc is an important PIM1 effector in the models investigated. Is a myc siRNA or the myc inhibitor 10058-F4 reducing colony formation in LNCaP and DU145 cells? Furthermore, increased c-myc transcriptional activity is only shown in PIM1 expressing RWPE1 cells. Is c-myc more active in PIM1 expressing LNCaP and DU145 cells? Also the PIM1/c-myc target genes, which were defined in an elegant way in RWPE1-mycER cells, should be validated in LNCaP and DU145 cells. Are some of the target genes indeed upregulated at the mRNA level in PIM1 expressing LNCaP and DU145 cells and is this upregulation responsive towards a c-myc siRNA or the myc inhibitor?

3) Figure 2D, which is described in the text is not included in the figures.

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