

PWPI Promotes the Malignant Phenotypes of Lung Cancer Cells by Interacting with DVL2 and Merlin [Corrigendum]

Wei L, Li P, Luo Y, et al. *Onco Targets Ther.* 2020;13:10025—10037.

DVL2”, and the last panel in part E is missing a label. The correct Figure 4 is shown below.

The authors have advised Figure 4 on page 10033 is incorrect due to errors at the time of figure assembly. Part A, second panel, the label “DVL2, p-DVL2” should read “p-DVL2,

The authors apologize for these errors and advise they do not affect the results of the paper.

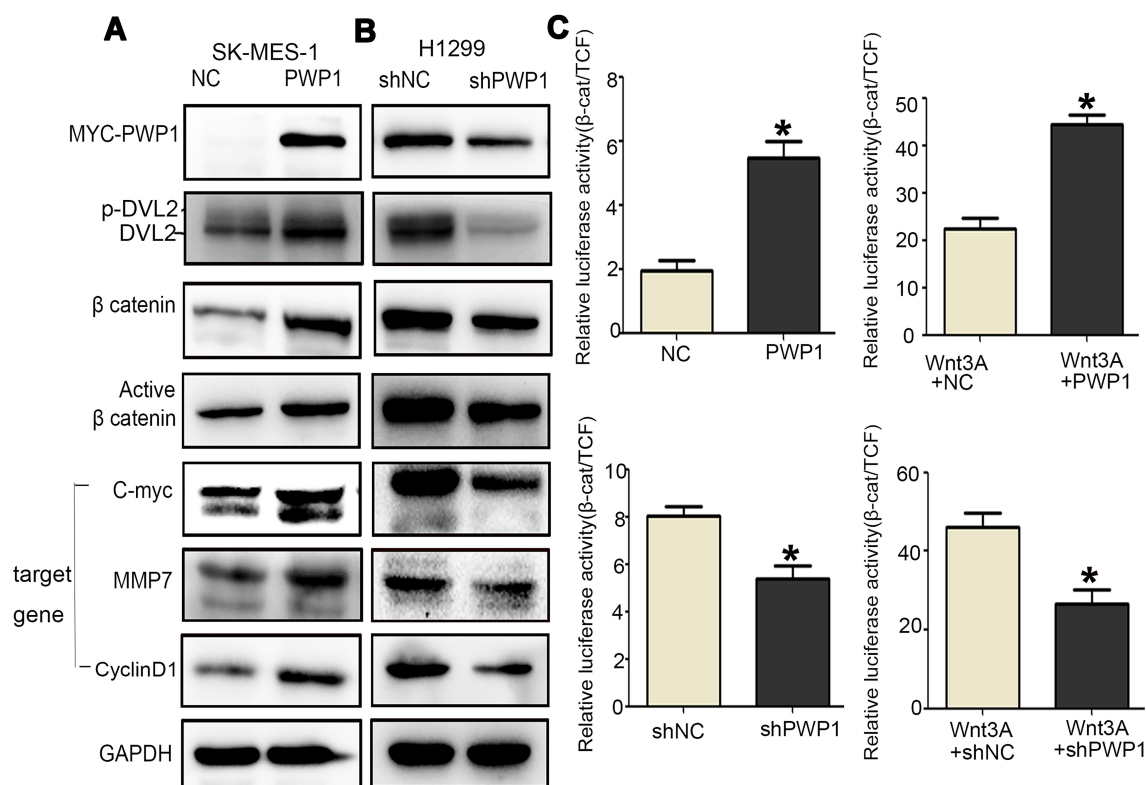


Figure 4 Continued.

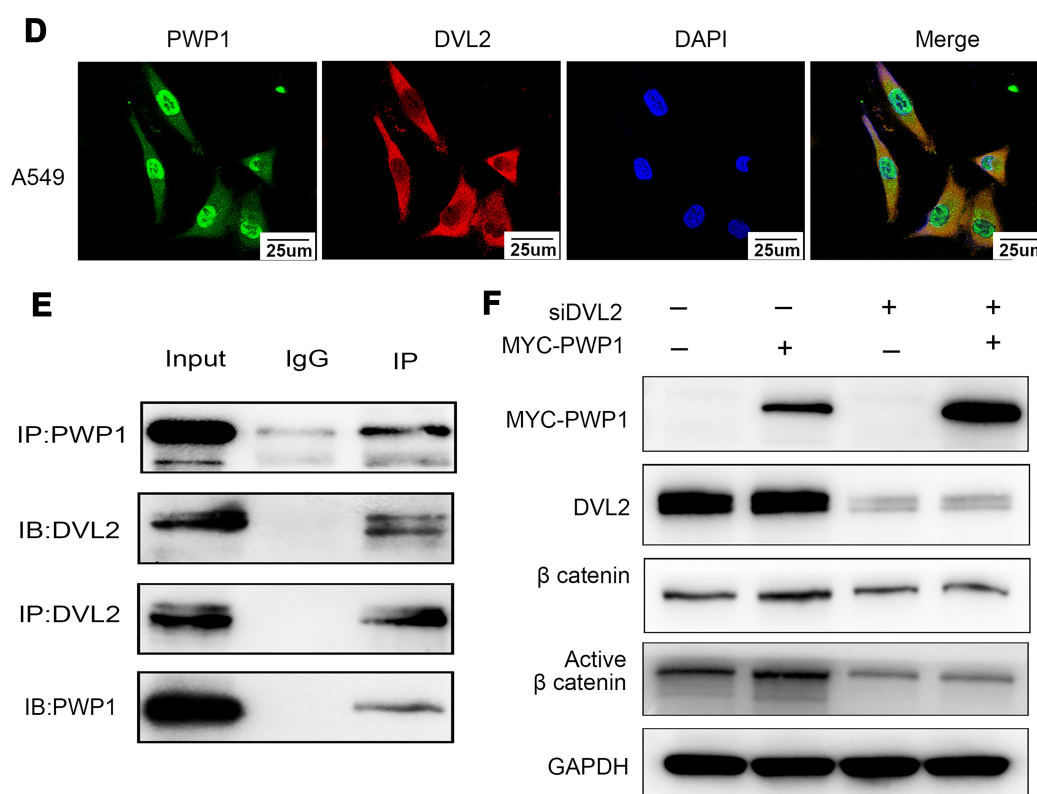


Figure 4 PWP1 activates the Wnt signaling pathway by interaction with DVL2. After transfection of PWP1 into SK-MES-1 cell line, wb showed that the phosphorylation of DVL2 and Active β -catenin were upregulated, and the expression levels of target genes of Wnt pathway, C-myc, Cyclin D1 and MMP7 were upregulated (**A**). After knocking down of PWP1 into H1299 cell line, wb showed that the phosphorylation of DVL2 and Active β -catenin were downregulated, and the expression levels of target genes of Wnt pathway, C-myc, Cyclin D1 and MMP7 were downregulated (**B**). GAPDH serves as a loading control. The grey value was measured using Image software. Luciferase gene-reporter assays showed that PWP1 could activate the Wnt pathway, after knocking down PWP1, the Wnt pathway would be inhibited (**C**). Columns: mean numbers, Bar: SD. (* $P < 0.05$). PWP1 and DVL2 co-localized in the cytoplasm (**D**, magnification 600 \times). Co-ip testing confirmed the interaction between PWP1 and DVL2 (**E**). Transfection of PWP1 and knocking down DVL2 would offset the effects of PWP1 on the Wnt pathway (**F**). Results are shown from three independent experiments.

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