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Erratum

PFKFB4 promotes angiogenesis via IL-6/STAT5A/P-STAT5 signaling in breast cancer: Erratum

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In the original version of our article, there were 3 errors concerning misused images. Specifically,

- Fig 2D, the WB blots of IκBα and β-actin of T47D cells, β-actin of MDA-MB-231 cells and NF-κB of T47D cells are un-intentionally reused. We repeated the experiment and the correct image is provided below.
- Fig 5C, the WB blots of STAT5B of HUVEC cells treated with MDA-MB-231 CM or T47D CM are un-intentionally reused. We repeated the experiment and the correct image is provided below.
- Fig 6E, the IHC images of IL-6R of DMSO-MCS group and 5-MPN-PFKFB4 group are un-intentionally reused. The correct image is provided below.

The correction will not affect the results and conclusion. The authors apologize for any inconvenience this may have caused.

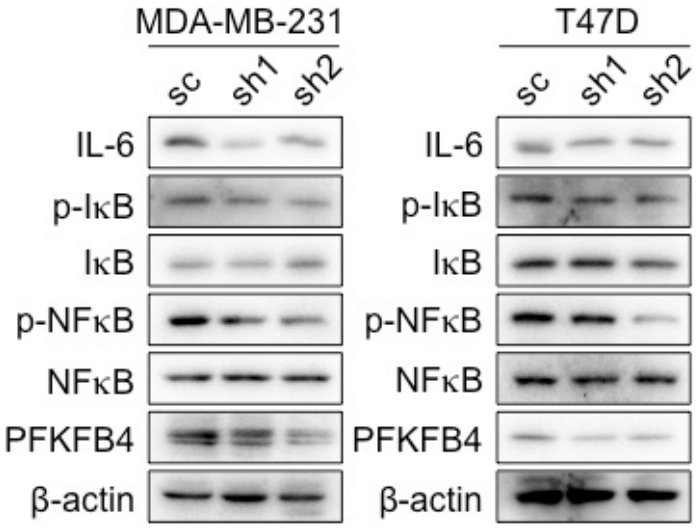

Fig. 2D. The Western blot analysis showed that knocking down of PFKFB4 in MDA-MB-231 and T47D cells decreased IκBα (S36) and NF-κB p65 phosphorylation. β-actin was used as a loading control.

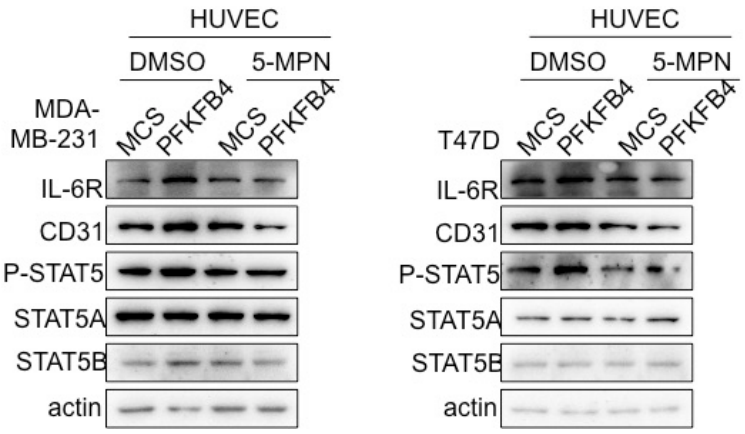

Fig. 5C. The Western blot analysis showed that 5-MPN treatment of MDA-MB-231 and T47D cells diminished PFKFB4-induced STAT5 phosphorylation and STAT5A, IL-6R, CD31 expression in HUVEC cells. β-actin was used as a loading control.

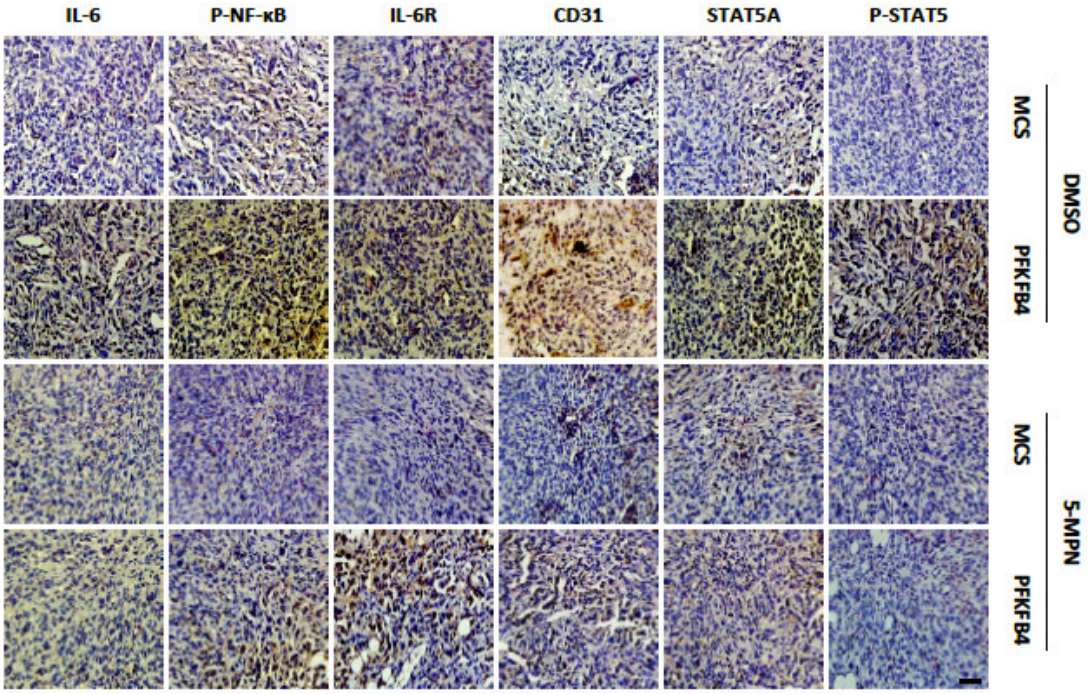

Fig. 6E. The representative micrographs of IL-6, P-NF-κB, IL-6R, CD31, STAT5A, and P-STAT5 immunocytochemical staining in xenograft MDA-MB-231 tumors. Ectopic expression of PFKFB4 increased immunocytochemical staining of above-mentioned molecules versus the MCS group, whereas, 5-MPN treatment inhibited PFKFB4-induced immunocytochemical staining of above-mentioned molecules. Scale bar= 50μm.