CORRIGENDUM

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All-*trans* retinoic acid restored the osteogenic ability of BMP9 in osteosarcoma through the p38 MAPK pathway

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Subsequently to the publication of the above paper, the authors have realized that the images presented in Fig. 1A were selected erroneously (essentially, the images for group 'AdBMP9 +++' were chosen to represent the group 'AdGFP'). A corrected version of Fig. 1, including the correct data for the experiments depicted in Fig. 1A, is shown opposite.

Note that this change does not affect the results or the conclusions reported in this paper, and all the authors agree to this correction. The authors apologize to the Editor and to the readership of the Journal for any inconvenience caused.

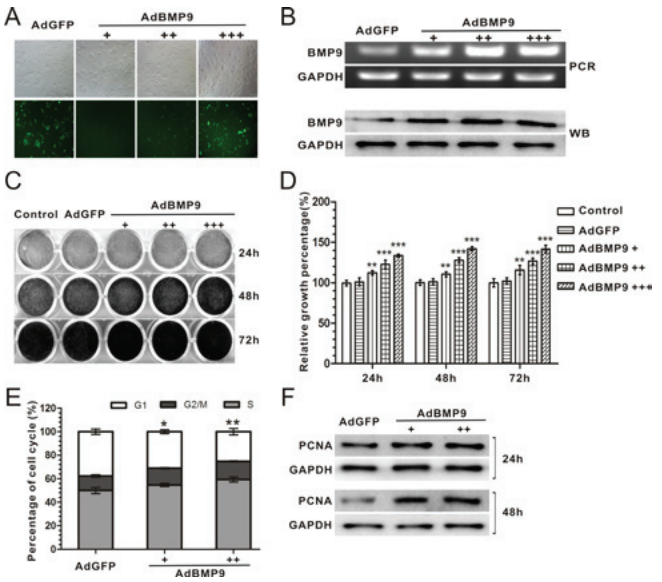

Figure 1. Effect of BMP9 on the proliferation of 143B cells. (A) AdBMP9 effectively transfected into 143B cells. 143B cells were infected with AdBMP9 and AdGFP. The GFP signal was detected under a fluorescence microscope (x100) 24 h after infection. (B) AdBMP9 effectively increased the expression of BMP9 24 h after infection, as detected by semi-quantitative PCR (sqPCR) (top) and western blotting assay (bottom). (C) Crystal violet assays showed that BMP9 promoted proliferation in 143B cells. (D) Quantitative results of the crystal violet assay in 143B cells (**P<0.01 vs. the control group; ***P<0.001 vs. the control group). (E) Quantitative results of cell cycle assay showed that BMP9 decreased the G1 phase arrest of 143B cells 48 h after infection (*P<0.05 vs. the AdGFP group; **P<0.01 vs. the AdGFP group). (F) Western blotting assays showed that the protein level of PCNA was affected by BMP9 in 143B cells. GAPDH was used as loading control. All assays were performed in triplicate. +, ++ and +++ refer to the infection rates of AdBMP9; PCNA, proliferating cell nuclear antigen.