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Title:

Erratum to: Wnt Signaling Inhibits Osteoclast Differentiation by Activating Canonical and Noncanonical cAMP/PKA Pathways: Wnt INHIBITS OC DIFFERENTIATION BY CANONICAL/ NONCANONICAL PATHWAYS (Journal of Bone and Mineral Research, (2016), 31, 1, (65-75), 10.1002/jbmr.2599)

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Erratum Wnt Signaling Inhibits Osteoclast Differentiation by Activating Canonical and Noncanonical cAMP/PKA Pathways

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A reader of the JBMR informed the editors about concern regarding two figures in the article by Weivoda et al. The authors wish to acknowledge that an error was made in [1] where the figure legend for Figure 1 is incomplete, and Figure 2 was inadvertently reported incorrectly. The figure legends below show the corrections in bold text

In the legend for Figure 1 (below), the description of the experimental sample for 1E is incorrect in that the serum starvation part of the protocol was omitted. Because of this error, it can be concluded that they are separate experiments from 2A. This figure legend has been corrected below.

The incorrect bands were used in figure 2E due to an inadvertent error. The original data were provided to the Editors for review, and the the corrected Figure 2E is shown below as well as the corrected Figure 2 caption.

These corrections do not change the outcome of the paper and the authors regret any confusion and inconvenience this may have caused the readers of JBMR.

This is the author manuscript accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1002/jbmr.3740](https://doi.org/10.1002/jbmr.3740).

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Figures

Figure 1: Wnt receptors are expressed in the osteoclast lineage and Wnt signaling in early osteoclasts suppresses osteoclast differentiation. (A) Bone marrow-derived osteoclast precursors were harvested (D0) or cultured with RANKL and M-CSF to obtain osteoclasts on day 4 (D4) and protein lysates were examined for Wnt receptor expression. (B,C) Cells were fixed on day 4, TRAP stained, and quantitated as detailed in the Methods section. (B) Osteoclast precursors were treated with vehicle (VEH) or the indicated Wnt3a concentration throughout differentiation culture. (C) Osteoclast precursors were cultured with either vehicle or 100ng/ml Wnt3a beginning on the indicated day of culture. (D) Osteoclast precursors were cultured with either vehicle or 100ng/ml Wnt5a beginning on the indicated day of culture. (E) Osteoclast precursors were serum starved for 60 minutes, and treated with vehicle (Veh) or 100ng/ml Wnt3a for 15 minutes. 40 mg of cell lysate was analyzed by western blotting for phosphorylated NFATc1 and tubulin. (F) Osteoclast precursors were cultured for 6 hours with RANKL and M-CSF with either vehicle (Veh) or 100ng/ml Wnt3a prior to nuclear isolation and extraction. 5 mg of nuclear extract was probed by western blotting for total NFATc1 and lamin. (G) Osteoclast precursors were cultured as in (E). 40 mg of cell lysate was analyzed by western blotting for total NFATc1 and tubulin. (H) Osteoclast precursors differentiated for the indicated time were serum starved for 60 minutes, and treated with vehicle (Veh) or 100ng/ml Wnt3a for 15 minutes. 40 mg of cell lysate was analyzed by western blotting for phosphorylated NFATc1 and tubulin. * $p < 0.05$ compared to vehicle treatment (B) or * $p < .01$ compared to vehicle treatment (C).

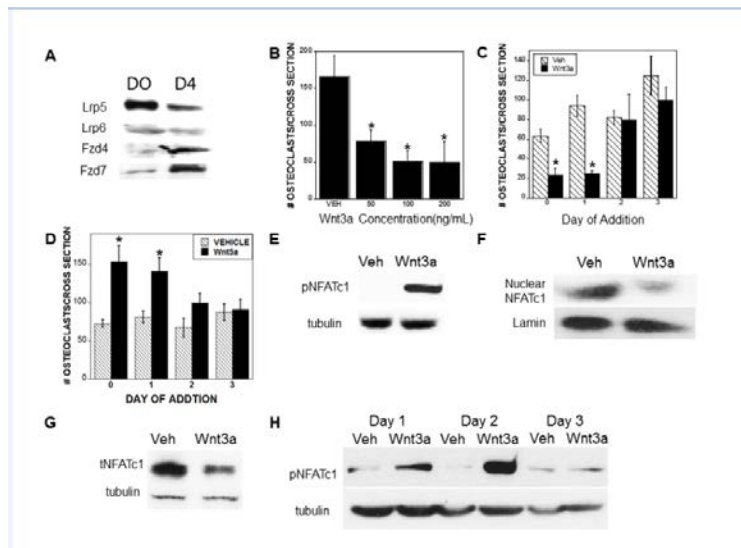
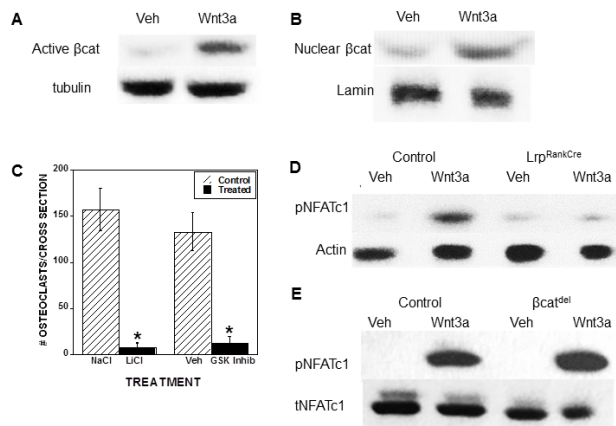


Figure 2. Wnt3a activates canonical Wnt signaling in osteoclast precursors to suppress osteoclast differentiation. (A) Osteoclast precursors were serum starved for 60 minutes, and treated with vehicle (Veh) or 100ng/ml Wnt3a for 15 minutes (samples from the experiment described in Figure 1E). 40 mg of cell lysate was analyzed by western blotting for active β -catenin and tubulin. (B) Osteoclast precursors were cultured with vehicle or 100 ng/ml Wnt3a treatment for 6 hours prior to nuclear isolation and extractions. 5 mg of nuclear extract was probed by western blotting for total β -catenin and actin. (C) Osteoclast precursors were cultured with either sodium chloride (NaCl) (salt control), Lithium Chloride (LiCl), vehicle DMSO (Veh, GSK Inhib control), or a GSK3- β inhibitor (GSK Inhib) throughout culture. Cells were fixed on day 4, TRAP stained, and quantitated as detailed in the Methods section. Results are the ratio of Wnt3a treated to vehicle treated osteoclast numbers. * $p < 0.05$ compared to vehicle or control treatment. (D, E) Osteoclast precursors from control littermate or *LrpRankCre* mice (D) or β cat flox osteoclast precursors treated with control or Cre-adenovirus (MOI=100). (E) were cultured 15 minutes with RANKL and M-CSF and either vehicle (Veh) or 100 ng/ml Wnt3a. 40 mg of cell lysate was analyzed by western blotting for phosphorylated and total NFATc1.



Reference

1. Megan M Weivoda, Ming Ruan, Christine M Hachfeld, Larry Pederson, Alan Howe, Rachel A Davey, Jeffrey D Zajac, Yasuhiro Kobayashi, Bart O Williams, Jennifer J Westendorf, Sundeep Khosla, Merry Jo Oursler. Wnt Signaling Inhibits Osteoclast Differentiation by Activating Canonical and Noncanonical cAMP/PKA Pathways. *J Bone Miner Res.* 2016;31(1):65-75. DOI: 10.1002/jbmr.2599