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reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms. Corrigendum: Oxymatrine attenuates osteoclastogenesis via modulation of ROS-mediated SREBP2 signaling and counteracts ovariectomy-induced osteoporosis

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In the original article, there were mistakes in Figures 1, 3 as published. The scale bars of TRAP staining images in Figures 1D, 3J were incorrect. Furthermore, we applied a mismatched picture for Figure 1F. The corrected Figures 1, 3 appear below.

The authors apologize for these errors and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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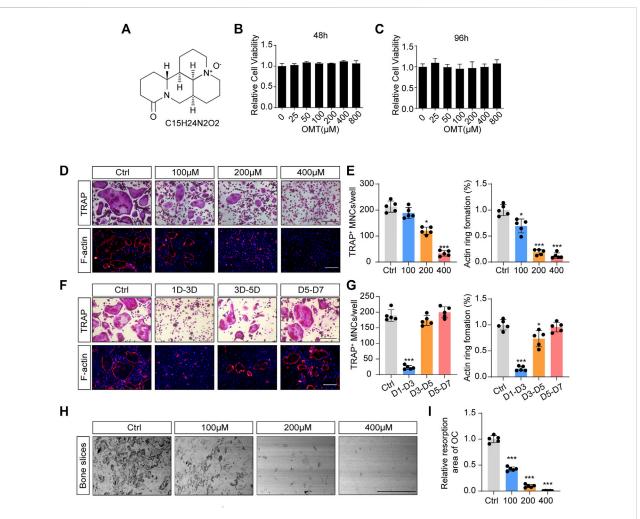


FIGURE 1

OMT inhibits RANKL-induced osteoclast formation and activity *in vitro*. (A) The chemical structure and formula of OMT. (B,C) Cell viability of OMT-treated BMMs at 48 and 96 h (D) BMMs were stimulated by 30 ng/mL M-CSF and 50 ng/mL RANKL, and treated with indicated concentrations of OMT for 5 days. Representative images of TRAP staining and F-actin staining were shown. Scale bar = 200 μ m. (E) Quantification of TRAP-positive multinuclear cells and F-actin ring formation rate. (F) BMMs were stimulated with 30 ng/mL M-CSF and 50 ng/mL RANKL for 7 days, and treated with 200 μ M OMT for the indicated days. TRAP staining and F-actin ring staining were performed. Scale bar = 200 μ m. (G) Quantification of TRAP-positive multinuclear cells and F-actin ring formation rate. (H) Representative images of bone resorption pits. Scale bar = 500 μ m. (I) Quantification of resorption pit area in each group. Data were presented as means \pm SD of 5 independent experiments. *p < 0.05, ***p < 0.001.

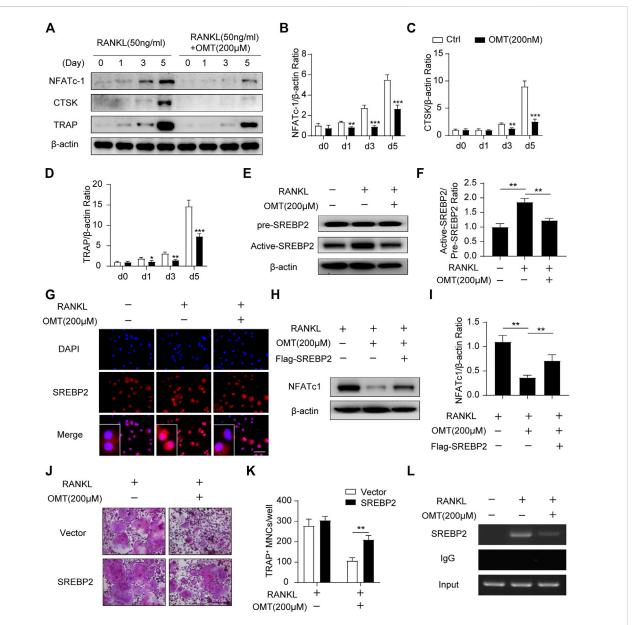


FIGURE 3

OMT attenuates SREBP2 activity and downstream NFATc1 expression during osteoclastogenesis. (A) BMMs were stimulated with RANKL, with or without 200 μ M OMT for 0, 1, 3, 5 days, the expression of NFATc1, CTSK and TRAP was tested by western blots. (B–D) Quantification of the ratios of band intensity of NFATc1, TRAP, and CTSK relative to β -actin (n = 3 per group). (E) BMMs were treated with RANKL and 200 μ M OMT as indicated, western blot was used to detect the level of pre-SREBP2 and active-SREBP2. (F) Quantification of active-SREBP2/pre-SREBP2 ratio (n = 3 per group). (G) RAW264.7 cells were treated with RANKL and OMT as indicated, immunofluorescence assay was performed to detect SREBP2 translocation. Scale bar = 100 μ m. (H) BMMs were transfected with Flag-SREBP2 plasmid or empty vector, then treated with RANKL and OMT as indicated. Representative images of TRAP staining were shown. Scale bar = 200 μ m. (K) Quantification of TRAP-positive multinuclear cells per well (n = 5 per group). (L) ChIP assay was performed on BMMs, treated with RANKL and OMT as indicated. Data were presented as means \pm SD. *p < 0.05, **p < 0.01, ***p < 0.001.

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