J Bone Miner Metab. 2014 Sep;32(5):494-504. doi: 10.1007/s00774-013-0530-1. Epub 2013 Nov 7.

Treatment with hydrogen molecules prevents RANKL-induced osteoclast differentiation associated with inhibition of ROS formation and inactivation of MAPK, AKT and NF-kappa B pathways in murine RAW264.7 cells

Dong-Zhu Li [1], Qing-Xiang Zhang, Xiao-Xian Dong, Huai-Dong Li, Xin Ma

Affiliations

PMID: 24196871 DOI: 10.1007/s00774-013-0530-1

Abstract

The bone protective effects of the hydrogen molecule (H2) have been demonstrated in several osteoporosis models while the underlying molecular mechanism has remained unclear. Osteoclast differentiation is an important factor related to the pathogenesis of bone-loss related diseases. In this work, we evaluated the effects of incubation with H2 on receptor activator of NFκB ligand (RANKL)-induced osteoclast differentiation. We found that treatment with H2 prevented RANKL-induced osteoclast differentiation in RAW264.7 cells and BMMs. Treatment with H2 inhibits the ability to form resorption pits of BMMs stimulated by RANKL. Treatment with H2 reduced mRNA levels of osteoclast-specific markers including tartrate resistant acid phosphatase, calcitonin receptor, cathepsin K, metalloproteinase-9, carbonic anhydrase typeII, and vacuolar-type H(+)-ATPase. Treatment with H2 decreased intracellular reactive oxygen species (ROS) formation, suppressed NADPH oxidase activity, down-regulated Rac1 activity and Nox1 expression, reduced mitochondrial ROS formation, and enhanced nuclear factor E2-related factor 2 nuclear translocation and heme oxygenase-1 activity. In addition, treatment with H2 suppressed RANKL-induced expression of nuclear factor of activated T cells c1 and c-Fos. Furthermore, treatment with H2 suppressed NF-κB activation and reduced phosphorylation of p38, extracellular signal-regulated kinase, c-Jun-N-terminal kinase, and protein kinases B (AKT) stimulated with RANKL. In conclusion, hydrogen molecules prevented RANKL-induced osteoclast differentiation associated with inhibition of reactive oxygen species formation and inactivation of NF-κB, mitogen-activated protein kinase and AKT pathways.

Related information

GEO Profiles
Gene
Gene (GeneRIF)
MedGen
Nucleotide (RefSeq)
Nucleotide (RefSeq)
Nucleotide (Weighted)
Protein (RefSeq)
Protein (Weighted)
PubChem Compound (MeSH Keyword)
Taxonomy via GenBank
UniGene

LinkOut - more resources

Full Text Sources

Springer

Other Literature Sources

The Lens - Patent Citations

scite Smart Citations

Research Materials

NCI CPTC Antibody Characterization Program

Miscellaneous

NCI CPTAC Assay Portal