

Bone loss associated with increased production of ROS

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Bone is constantly being broken down and remodeled. Osteoporosis results when bone resorption outpaces bone regeneration. Production of reactive oxygen species, a form of oxidative stress, has been predicted to promote bone loss, but a source of reactive oxygen is unknown.

In this issue of the *Journal of Clinical Investigation*, Katrin Schröder and colleagues at Goethe-University identify a relationship between NADPH oxidase 4 (NOX4), an enzyme that promotes [reactive oxygen species](#) formation, and [bone](#) resorption. In a mouse model of osteoporosis, genetic disruption or drug-induced loss of NOX4 protected the mice from bone loss.

Additionally, the authors identify a small nuclear polymorphism in *NOX4* in human patients that associated with increased bone turnover. Together, these data suggest treatments targeting NOX4 activity may benefit osteoporosis patients.

More information: NADPH oxidase 4 limits bone mass by promoting osteoclastogenesis, *J Clin Invest.* [DOI: 10.1172/JCI67603](https://doi.org/10.1172/JCI67603)

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