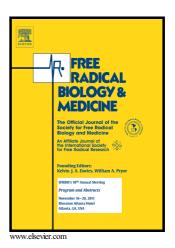
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ACCEPTED MANUSCRIPT

Peroxiredoxin 2 deficiency accelerates age-related ovarian failure through the reactive oxygen species-mediated JNK pathway in mice

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Abstract

Reactive oxygen species (ROS) produced in biological reactions have been shown to contribute to ovarian aging. Peroxiredoxin 2 (Prx2) is an antioxidant enzyme that protects cells by scavenging ROS; however, its effect on age-related, oxidative stress-associated ovarian failure has not been reported. Here, we investigated its role in age-related ovarian dysfunction and 4-vinylcyclohexene diepoxide (VCD)-induced premature ovarian failure using Prx2-deficient mice. Compared to those in wildtype (WT) mice, serum levels of anti-Müllerian hormone, 17β -estradiol, and progesterone and numbers of follicles and corpora lutea were significantly lower in 18-month-old $Prx2^{-/-}$ mice. Moreover, levels of Bax, cytochrome c, cleaved caspase-3, and phosphorylated JNK proteins were higher and numbers of apoptotic (terminal deoxynucleotidyl transferase dUTP nick end labeling-positive) cells were considerably greater in 18-month-old $Prx2^{-/-}$ ovaries than WT ovaries.

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