

# Block estrogen to treat lung disease

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The strongest epidemiological risk factor for many forms of pulmonary arterial hypertension (PAH) is female gender. Increased estrogen makes females three times more likely to develop PAH, a chronic disease that eventually leads to right heart failure and death.

Mutations in the receptor BMPR2 cause a heritable form of PAH, and BMPR2 [mutant mice](#) spontaneously develop the disease. Estrogen increases the penetrance (the percentage of mice that develop PAH) and the severity of the disease.

James West, Ph.D., and colleagues treated BMPR2 mutant mice with the estrogen inhibitors anastrozole, fulvestrant and tamoxifen. They found that estrogen inhibition both prevented and treated PAH in BMPR2 mutant mice, associated with a reduction in metabolic defects including oxidized lipid formation and insulin resistance.

The findings, reported in the *European Respiratory Journal*, suggest that estrogen inhibition may improve pulmonary vascular disease by correcting [metabolic defects](#). Based on these studies, clinical trials of estrogen inhibition in human PAH have recently been approved and funded by the National Institutes of Health (NIH).

**More information:** Xinping Chen et al. Oestrogen inhibition reverses pulmonary arterial hypertension and associated metabolic defects, *European Respiratory Journal* (2017). [DOI: 10.1183/13993003.02337-2016](#)

Provided by Vanderbilt University

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