Sleep apnea thickens blood vessels, increases heart disease risk

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Obstructive sleep apnea, or periodic interruptions in breathing throughout the night, thickens sufferers' blood vessels. Moreover, it increases the risk of several forms of heart and vascular disease.

Emory researchers have identified the enzyme NADPH oxidase as important for the effects obstructive sleep apnea has on blood vessels in the lung.

The results are published in the May 1 issue of the *American Journal of Respiratory Cell and Molecular Biology*. C. Michael Hart, professor of medicine at Emory University School of Medicine and Atlanta Veterans Affairs Medical Center, is senior author.

Obstructive sleep apnea is thought to affect one in every 50 women and one in every 25 men in the United States. Standard treatment involves a mechanical application of air pressure. Anything that blunts sleep apnea's effects on blood vessel physiology could reduce its impact on disease risk, Hart says.

Cyclically depriving mice of oxygen - researchers call this "chronic intermittent hypoxia" -- in a way that simulates obstructive sleep apnea gives them pulmonary hypertension. Pulmonary hypertension, which can be life threatening, is a condition in which the right side of the heart has trouble pumping blood because of resistance in the lung's blood vessels.

Chronic intermittent hypoxia forces the blood vessels in the lung to

make more NADPH oxidase, Hart and his colleagues found. Mice that lack NADPH oxidase are immune to hypoxia's effects.

NADPH oxidase is a helpful enzyme because it is responsible for making superoxide, a reactive free radical that the immune system uses to kill bacteria. But superoxide also interferes with nitric oxide, a signal that allows blood vessels to relax.

Humans with mutations in genes for NADPH oxidase have recurrent bacterial infections because their ability to fight the bacteria is weakened. Thus Hart says inhibiting the NADPH oxidase enzyme in the entire body may be harmful, and he favors an indirect intervention.

"We think that strategies to lower NADPH oxidase expression induced by hypoxia may be useful in preventing hypoxia-induced pulmonary hypertension," says Hart.

More information: "The role of NADPH oxidase in chronic intermittent hypoxia-induced pulmonary hypertension in mice." Nisbet R.E, et al. *Am J Respir Cell Mol Biol*. 40: 601-9 (2009).

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