

Lung disease may increase risk of insulin resistance, diabetes, mouse study suggests

30 September 2015

Numerous studies have identified obesity and poor diet as risk factors for insulin resistance and diabetes. A new study adds another risk factor to the list: inflammatory lung disease. Published in the *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology*, the study reports that inflammation in the lungs is enough to induce the body-wide inflammation that can lead to insulin resistance.

Individuals with inflammatory lung diseases, such as asthma and pneumonia, frequently have high blood glucose (sugar) levels and show insulin resistance. However, these individuals often also have conditions such as obesity or steroid treatment that predispose them to risk factors of diabetes. Researchers at Vanderbilt University sought to determine if having only lung disease could increase the likelihood of developing diabetes risk factors.

The researchers observed that mice with [airway inflammation](#) also developed inflammation in the liver and other organs. Although [insulin signaling](#) in these mice was normal, insulin was less effective in controlling blood sugar: Insulin did not suppress glucose production by the liver, and [insulin action](#) was moderately impaired in other organs. The mice had signs of insulin resistance, demonstrating that inflammation in the lungs can contribute to insulin resistance and high blood sugar, according to the researchers. Therapies that reduce lung inflammation to treat lung injuries may have the additional benefit of lowering the risk of body-wide inflammation and insulin resistance, the researchers wrote.

The study "NF- κ B-dependent airway inflammation triggers systemic [insulin resistance](#)" is published ahead of print in the *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology*.

More information: "NF- κ B Dependent Airway

Inflammation Triggers Systemic Insulin Resistance." *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology* Published 16 September 2015 Vol. no. , [DOI: 10.1152/ajpregu.00442.2014](#)

Provided by American Physiological Society

APA citation: Lung disease may increase risk of insulin resistance, diabetes, mouse study suggests (2015, September 30) retrieved 15 October 2022 from <https://medicalxpress.com/news/2015-09-lung-disease-insulin-resistance-diabetes.html>

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