Cigarette smoke impacts genes linked to health of heart and lungs

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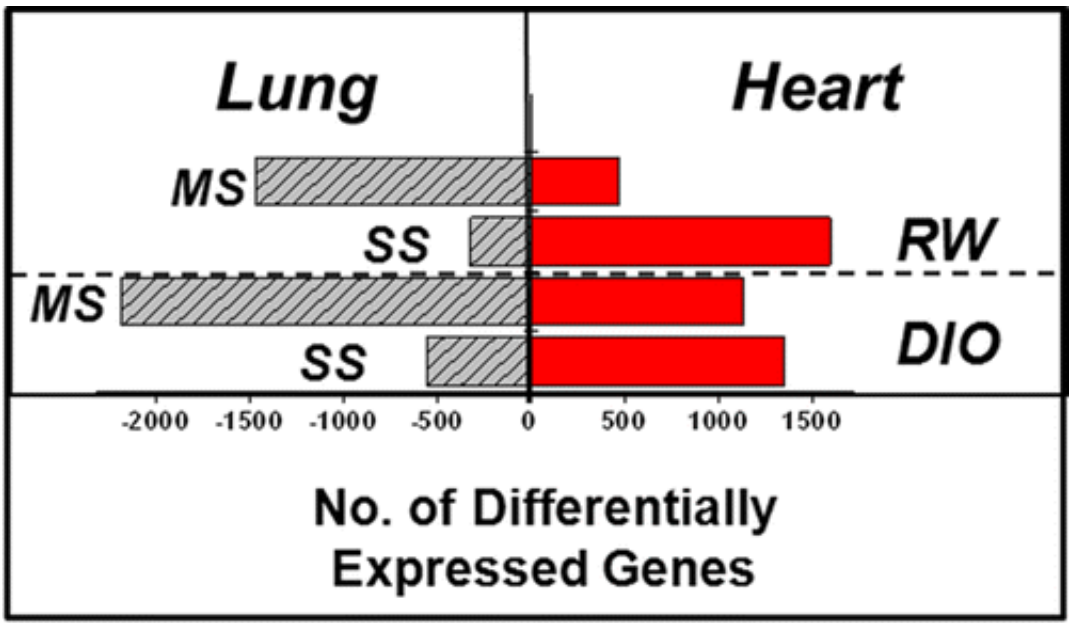

New insights into why obese cigarette smokers experience a high risk of heart disease suggest that cigarette smoke affects the activity of hundreds of key genes that both protect the heart and lungs and expose them to damage. The study, published in ACS' *Chemical Research in Toxicology*, suggests that the effects may be especially profound in obese nonsmokers who inhale "sidesteam smoke" from cigarettes smoldering nearby.

Diana J. Bigelow and colleagues point out that active smoking doubles the risk of heart disease, while second-hand smoke exposure increases this risk by about one-third. They set out to gain more information on why the risks are especially high among people with obesity, using specially fed laboratory mice that are stand-ins for humans in such experiments.

The report describes how mainstream smoke and to a greater extent, sidestream smoke, inhibit the activity of genes that protect the heart and lungs, and activate genes associated with an increased risk of heart disease. Those changes were more profound in obese mice than normal-weight mice. "The present study is the first, to our knowledge, that addresses the in vivo transcriptional response of the heart to cigarette smoke exposure in the setting of high fat diet and obesity, and thus takes a first step toward identifying the molecular basis of adaptive responses that may lead to an increased risk of heart disease in obese smokers," the report states.

More information: "Impaired Transcriptional Response of the Murine Heart to Cigarette Smoke in the Setting of High Fat Diet and Obesity" *Chem. Res. Toxicol.*, Article ASAP DOI: 10.1021/tx400078b

Abstract

Smoking and obesity are each well-established risk factors for cardiovascular heart disease, which together impose earlier onset and greater severity of disease. To identify early signaling events in the response of the heart to cigarette smoke exposure within the setting of obesity, we exposed normal weight and high fat diet-induced obese (DIO) C57BL/6 mice to repeated inhaled doses of mainstream (MS) or sidestream (SS) cigarette smoke administered over a two week period, monitoring effects on both cardiac and pulmonary transcriptomes. MS smoke (250 ?g wet total particulate matter (WTPM)/L, 5 h/day) exposures elicited robust cellular and molecular inflammatory responses

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