Exposure of airway smooth muscle cells to cigarette smoke extract

Marc B. Hershenson and Yutein Chung
University of Michigan Medical School, Ann Arbor, Michigan

Submitted 13 June 2014; accepted in final form 16 July 2014

TO THE EDITOR: We read with interest the article by Aravamudan and colleagues (1) on cigarette smoke-induced mitochondrial fragmentation and dysfunction in human airway smooth muscle. As explained in a recent Perspectives (4), mitochondria are at the center of a reactive oxygen species-dependent pathway regulating cellular cytotoxic responses. The study by Aravamudan et al. demonstrates that cigarette smoke could have powerful effects on airway smooth muscle function.

However, I have a naive question: Does cigarette smoke have direct effects on airway smooth muscle? Reactive oxygen species in cigarette smoke may cause direct damage of airway epithelial cells, but they are not capable of diffusing through plasma membranes (2). It is conceivable that lipid-soluble components of the cigarette smoke particulate phase, such as phenolic compounds, aldehydes, and polycyclic aromatic hydrocarbons, could pass through the membranes of cells (3), eventually reaching the smooth muscle via the circulation. But is this the same as directly exposing airway smooth muscle cells to cigarette smoke extract in culture? In this context, wouldn’t the concentration of cigarette smoke lipid-soluble components be much higher than those achieved by smoking in vivo? Might such studies overestimate the effect of cigarette smoke on airway smooth muscle biology?

This line of research may benefit from using specific components of the cigarette smoke particulate phase, rather than undefined extracts. Concentrations of these components normally found in the circulation of smokers, rather than cigarette smoke itself, might be more physiological. Better still, investigators bathing airway smooth muscle cells in cigarette smoke extract might try to confirm their findings by examining the airway smooth muscle of animals or humans exposed to cigarette smoke in vivo. Finally, whether or not cigarette smoke directly affects airway smooth muscle, all workers in the field should consider whether exposures and infections of airway smooth muscle cells in culture truly reflect the actual conditions encountered inside a living organism.

REFERENCES


Address for reprint requests and other correspondence: M. B. Hershenson, Univ. of Michigan Medical School, 1150 W. Medical Center Dr., Rm. 3570 MSRB II, Ann Arbor, MI 48109-5688 (e-mail: mhershen@umich.edu).