

Séminaire conjoint des Services de Pneumologie et
d'Immuno-allergologie

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Salle de séminaire 2, BH-08 CHUV

**Asbestos and Silica: old toxics for the lungs,
new mechanisms**



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The inhalation of airborne pollutants, such as asbestos or silica, is linked to inflammation of the lung, fibrosis, and lung cancer. How the presence of pathogenic dust is recognized and how chronic inflammatory diseases are triggered are poorly understood. Here, we show that asbestos and silica are sensed by the Nalp3 inflammasome, whose subsequent activation leads to interleukin-1 β secretion. Inflammasome activation is triggered by reactive oxygen species, which are generated by a NADPH oxidase upon particle phagocytosis. (NADPH is the reduced form of nicotinamide adenine dinucleotide phosphate.) In a model of asbestos inhalation, Nalp3^{-/-} mice showed diminished recruitment of inflammatory cells to the lungs, paralleled by lower cytokine production. Our findings implicate the Nalp3 inflammasome in particulate matter-related pulmonary diseases and support its role as a major proinflammatory "danger" receptor.

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