Biomass smoke as a risk factor for chronic obstructive pulmonary disease: Effects on innate immunity

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Chronic obstructive pulmonary disease (COPD), a major cause of mortality and morbidity worldwide, is considered an archetypical disease of innate immunity, where inhaled particles and gases trigger an inflammatory response, favoring tissue proliferation in small airways and tissue destruction in lung parenchyma, in addition to the recruitment of immune cells to these compartments. Although cigarette smoking is still considered the main risk factor for developing COPD, the trend of proposing biomass smoke (BS) exposure as a principal risk factor is gaining importance, as around 3 billion people worldwide are exposed to this pollutant daily. A considerable amount of evidence has shown the potential of BS as an enhancer of lung inflammation. However, an impairment of some innate immune responses after BS exposure has also been described. Regarding the mechanisms by which biomass smoke alters the innate immune responses, three main classes of cell surface receptors - the TLRs, the scavenger receptors and the transient receptor potential channels - have shown the ability to transduce signals initiated after BS exposure. This article is an updated and comprehensive review of the immunomodulatory effects described after the interaction of BS components with these receptors. © SAGE Publications.

Biomass smoke

chronic obstructive pulmonary disease

innate immunity

lung inflammation

Toll-like receptors

scavenger receptor

tobacco smoke

toll like receptor

transient receptor potential channel
air pollutant
irritant agent
smoke
biomass
chemical composition
chronic obstructive lung disease
exposure
host resistance
human
immunomodulation
innate immunity
particulate matter
pneumonia
Review
risk factor
smoke
toxicity
adverse effects
air pollutant
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Risk Factors
Smoke
Smoking