Pathogenic Mechanisms in Chronic Obstructive Pulmonary Disease Due to Biomass Smoke Exposure [Mecanismos patogénicos en la enfermedad pulmonar obstructiva crónica causada por exposición a humo de biomasa] Silva R.

Oyarzún M.

Olloquequi J.

Chronic obstructive pulmonary disease (COPD) mortality and morbidity have increased significantly worldwide in recent decades. Although cigarette smoke is still considered the main risk factor for the development of the disease, estimates suggest that between 25% and 33% of COPD patients are non-smokers. Among the factors that may increase the risk of developing COPD, biomass smoke has been proposed as one of the most important, affecting especially women and children in developing countries. Despite the epidemiological evidence linking exposure to biomass smoke with adverse health effects, the specific cellular and molecular mechanisms by which this pollutant can be harmful for the respiratory and cardiovascular systems remain unclear. In this article we review the main pathogenic mechanisms proposed to date that make biomass smoke one of the major risk factors for COPD. © 2014 SEPAR.

Biomass smoke

Chronic obstructive pulmonary disease

Genotoxicity

Inflammation

Pathogenesis

biomass

biomass smoke

chronic obstructive lung disease

developing country

environmental exposure

mortality
pathogenesis
risk factor
Short Survey
smoke
smoking
age
high risk population
inflammation
molecular mechanics
oxidative stress
pathogenicity
pollutant
sex
adult
adverse effects
air pollutant
animal
child
complication
cooking
drug effects
environmental exposure
female

human

morbidity

gas
heating
indoor air pollution
inflammation
lung burn
male
metabolism
mouse
particulate matter
pregnancy
prenatal exposure
Pulmonary Disease, Chronic Obstructive
rural population
secretion (process)
smoke
ascorbic acid
glutathione
interleukin 6
interleukin 8
reactive oxygen metabolite
superoxide dismutase
tobacco smoke
tumor necrosis factor
air pollutant
autacoid
cytokine

gas
inflammasome
particulate matter
smoke
transient receptor potential channel
Adult
Air Pollutants
Air Pollution, Indoor
Animals
Biomass
Child
Cooking
Cytokines
Environmental Exposure
Female
Gases
Heating
Humans
Inflammasomes
Inflammation
Inflammation Mediators
Male
Mice
Oxidative Stress
Particulate Matter
Pregnancy

Prenatal Exposure Delayed Effects

Pulmonary Disease, Chronic Obstructive

Rural Population

Smoke

Smoke Inhalation Injury

Transient Receptor Potential Channels