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Review article

Lung diseases associated with hydrocarbon exposure

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ABSTRACT

The human race has been exposed to the potential toxicity of hydrocarbons, whether by the inhaled or ingested route, for thousands of years and the consequent inflammatory reaction in the lungs depends on the degree of exposure, volatility and viscosity of the particular hydrocarbon in question. Heating, lighting, transportation, industry and nature all provide the potential for both inhalation and/or ingestion of hydrocarbons. Some forms, such as those related to petroleum products, e.g. diesel exhaust particles (DEP) and polycyclic aromatic hydrocarbons (PAH) have been shown to cause both malignant and non-malignant respiratory diseases. Accidental ingestion represents another significant exposure risk and we now have increasing evidence that pollutant particles may adsorb allergens to their surface and potentially enhance the allergic response. It seems unlikely that this potential will significantly decrease in the near future and depending on individual socio-economic circumstances, work environment and habitation, the risks of significant lung disease will vary. This review outlines the domestic, outdoor, occupational and natural sources of hydrocarbon exposure and considers the evidence relating to radiological and pathological lung changes in both animals and man. The acute effects of hydrocarbon toxicity are well recognised but the effects of longer term, lower exposure, and the mechanisms of their toxicity, require further research.

Contents

1. Introduction

Hydrocarbons first appeared in the universe at the birth of new stars and planets. They are found in modern-day natural sources

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such as creosote; as a result of incomplete combustion of organic matter; or when organic matter is transformed into oil or coal. Following mankind's discovery of fire as a source of heat and energy, the contribution of hydrocarbons to the atmosphere has been predominantly due to the burning of wood, coal, dung, crop residues, wild fires, and tobacco. Methane and butane gases are examples of the shorter molecular chain hydrocarbons. Petroleum is a fossil fuel (the name is derived from 'rock oil') and is a mixture of many different hydrocarbons which determine their physical characteristics of volatility and viscosity. Strictly speaking, petroleum is synonymous with crude oil but is an all-encompassing term for derived solid, liquid and gaseous hydrocarbons. As industrialisation has progressed, occupations will have exposed workers to a variety of hydrocarbons such as fossil fuels, diesel fumes, kerosene, metal working fluids or wood smoke. Petroleum, coal, and by-products of industrial processing are major sources of hydrocarbons. As a result of all these potential exposures, there are significant implications for the development of lung disease.

2. Domestic solid fuel exposure

In early human history, exposure would have been the result of burning wood or dung and the naturally occurring wild fires or volcanic activity. However, these forms of exposure have persisted to modern day. Domestic consumption of indoor fuels includes solids such as wood and coal; liquids such as kerosene, lamp oil and liquefied petroleum gas (LPG); and gases such as methane and natural gas. The majority of solid fuel users live in lower socio-economic conditions but developed countries also take part and it is estimated that around 3 billion people are potentially exposed worldwide.

A comprehensive update on the issue of indoor fuel exposure [1], highlights the association between regular indoor solid fuel smoke exposure and COPD (both emphysema and chronic bronchitis). It raises the issue as to whether tobacco smoke may potentiate any lung disease associated with fuel smoke. In a systematic review and meta-analysis [2], studies with sufficient statistical power to calculate the health risk of COPD from the use of solid fuel were included. They concluded that exposure to solid fuel smoke was consistently associated with COPD and chronic bronchitis and that domestic wood smoke rather than other fuels posed the greatest risk (see Fig. 1).

A domestically acquired pneumoconiosis, affecting rural African women [3], was initially thought to be due to silica particles inhaled while grinding maize between rocks. However, it was subsequently termed 'Hut Lung' when evidence based on histology, lung physiology and respirable dust concentrations (and the fact that three cases were in women who did not grind maize), suggested that domestic smoke may be more important than maize grinding in the causation of this pneumoconiosis. Most of the women in this report were symptomless but the authors make the point that some may progress to cor pulmonale and death and differentiating these cases from miliary tuberculosis must be the first priority. A case report [4] demonstrated an association between inhaled wood burning particulates and interstitial pneumonitis. Particles derived from bronchoalveolar lavage were shown to be carbonaceous with a surrounding inflammatory response and their source was a poorly functioning wood-burner. Such cases may have advanced disease [5], with diffuse anthracosis and associated interstitial inflammation. Domestically acquired particulate lung disease, as it is now referred to, is thought to account for a considerable burden of environmentally induced lung disease (see Figs. 2 and 3).

3. Outdoor environmental hydrocarbon exposure

Outdoor air pollution is an all-embracing term for gases and particles which may typically arise from sources such as industry, wild fires, automobile exhaust and domestic wood or coal burning. Solid, liquid or gaseous hydrocarbons are major contributors to the pollutant load.

Fig. 1. Domestic exposure to wood burning.
Image provided by Professor Nigel Bruce, Department of Public Health and Policy, Liverpool University.

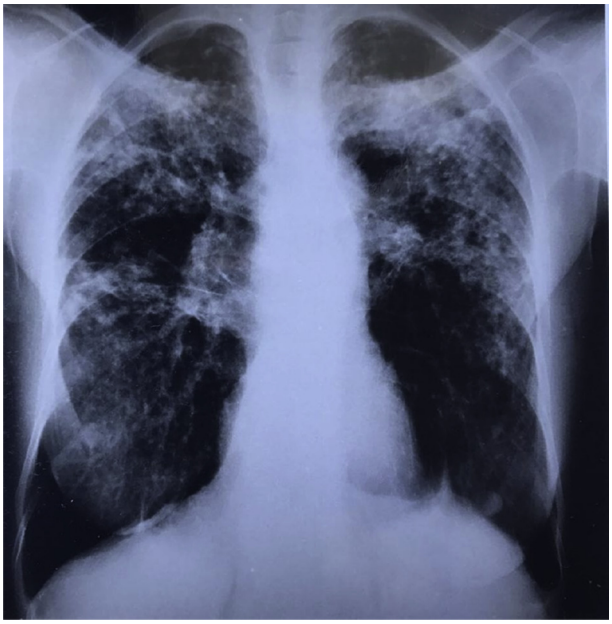

Fig. 2. Chest radiograph of a 54 year old woman, demonstrating features of progressive massive fibrosis due to domestically acquired pneumoconiosis.
Image provided by Dr J P Grobbelaar, Stellenbosch, SA.