DOCTORS for the ENVIRONMENT

AIR POLLUTION

PART 2

Before we look at the specific components of air pollution it may be worthwhile looking at the particle sizes of different air pollutants: tobacco smoke < 1µm, pollens 10-30µm, mould spores 2-50µm, Actinomycete spores 1-2µm and foundry dusts 50mm. Tobacco smoke will thus reach the alveoli with ease.

It is important to know how the tracheobronchial tree is cleared of foreign material. Three mechanisms are involved: mucociliary-, cough-, and alveolar clearance.

Mucociliary clearance involves mucus, its cellular contents, antiproteinasis, lysozyme and other antimicrobial substances. Ciliated cells line the airways from the anterior nasopharynx to the terminal bronchi. The cilia beat in a cephalad direction and has been compared to an escalator with progressively increasing speeds from the terminal bronchiole to the trachea. Healthy airways can clear particulate matter by means of this mechanism in six hours and the entire escalator can be cleared within 24 hours.

Cough clearance is particularly effective in the central airways. This mechanism is very important in diseases like chronic bronchitis where it may account for more than 50% of clearance. It is also of great importance in compensating for any defect in mucociliary transport. In healthy subjects with no sputum production, cough contributes little to tracheo bronchial clearance.

Less than 20% of extremely small particles between 0,1 and 0,5µm are retained by the alveoli. The alveoli are either cleared by transport or absorption. Absorption is accomplished by penetration into epithelical cells or by phagocytosis. Particles are also transported to the ciliated regions from where they are cleared.

Substances like sulphur dioxide, ozone, sulfuric acid, particulate matter from wood smoke and nitrogen dioxide all produce effects when investigated in laboratory or field studies.

In the United States, SO_2, total particulate matter, carbon monoxide, photochemical oxidants (O_3), nitrogen dioxide and lead concentrations are regulated by set standards.

Even with strict regulation it is estimated that over 100 million US individuals live in areas which do not meet the ozone standard. It is calculated that in a Manhattan street canyon the diesel concentration may reach 30µm/m^3 if 20% of the vehicles were diesel powered. That is way above the maximum standard.

Specific substances implicated in human respiratory disease

Ozone (O^3):
There are several sources of ozone in ambient air. One part of the ozone comes from stratospheric O_3. The rest comes from photochemical reactions between organic vapours, nitrogen oxide and radiation from sunlight. Some organic vapours such as olefinic hydrocarbons and formaldehyde can be controlled but others such as methane from agriculture and animals as well as isoprene and terpenes omitted from trees are less controllable. Nitrogen oxide has increased due to increased use of fossil fuels and may be the reason for the increased levels of ozone.

Ozone is a water insoluble gas. Up to 40% inspired ozone is taken up in the nasal passages during tidal breathing. It causes an increase in neutrophils, eosinophils, mono-nuclear cells and histamine in nasal lavage fluid. This may necessitate pharmacologic intervention.

Ozone also causes changes in the lower respiratory tract. Ten to 20% of individuals are more sensitive to ozone than the general population. FEV_1 values continue to decrease with exposure of up to six hours. There seems to be controversy as to whether asthmatics are more sensitive to ozone than non-asthmatics. The most common induced symptom of ozone inhalation is pain on deep inspiration. This may be blocked by lidocaine inhalation with resultant improvement in FVC. It was shown in southern Ontario that admissions for asthmatic attacks correlated with both ozone and suspended sulphates. The investigators Bates and Sizto coined the term "acid summer haze" as being responsible for this phenomenon. Apparently there is no rapid broncho constriction after ozone inhalation but rather a more gradual decline in lung function during exposure. This decline is not rapidly reversible but may last several hours after exposure.

As far as pharmacological intervention is concerned, atropine blocks changes in airway resistance but had no effect on FVC. Indomethacin partially prevented O_3 induced lung function on changes in normal subjects but did not prevent increases in airway reactivity. Albuterol was unable to block pulmonary function changes induced by O_3.

At present, ozone levels in ambient air is of great concern. Taking the sources of ozone into account it will be difficult and expensive to lower ozone concentrations to more acceptable levels.

In part three of this series we will look at a few more pollutants.

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