AHERA
Worker

U.S. EPA and Cal-OSHA Accredited

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by The Asbestos Institute

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POTENTIAL HEALTH EFFECTS ASSOCIATED WITH ASBESTOS EXPOSURE

The adverse health effects associated with asbestos exposure have been extensively studied for many years. Results of these studies and epidemiologic investigations have demonstrated that inhalation of asbestos fibers may lead to increased risk of developing one or more diseases. Exactly why some people develop these diseases and others do not remains a mystery. In this discussion, each of the major diseases associated with asbestos will be examined, along with the risk and how that risk can be minimized.

It is important to recognize that the majority of people in the data collected who have died as a result of asbestos exposure were industrial asbestos workers. These workers were frequently exposed to high concentrations of asbestos fibers each working day with little or no protection. The asbestos abatement worker of today follows specific work practices and wears appropriate protection, including respirators, to minimize the risk of exposure.

THE RESPIRATORY SYSTEM

Since the primary route of exposure is inhalation, it is necessary to gain a brief understanding of the respiratory system. Air which is breathed into the body passes through the mouth and nose into the windpipe or trachea. The trachea splits into two smaller airways called the bronchi. Each bronchus divides into smaller and smaller tubes which terminate into air sacs called alveoli. It is in these air sacs that oxygen is absorbed into small blood vessels and waste gases, such as carbon dioxide, pass out of the blood. (See Figure 3-1, following page.)

The lung itself is divided into two halves and sits in the pleural cavity. This cavity and the outside of the lung itself have a membrane lining (called pleural or mesothelial tissue) which looks somewhat like saran wrap. These linings are in contact with each other and are very moist. Just like two panes of glass with a drop of water between them, these linings slide easily across each other, but are difficult to pull apart. Accordingly, as the chest cavity expands, the lungs expand and air rushes in. If these lining were to become damaged, inhalation could not occur properly.

The body has several mechanisms by which it filters the air it breathes. First, very large particles are removed in the nose and mouth. Many smaller particles impact on the mucous coated walls of the airways and are caught. These airways have a hair like lining (ciliated cells) which constantly beat upward. Accordingly, particles caught in the mucous are swept up into the back of the mouth. From here it is swallowed or expelled. Unfortunately, cigarette smoking temporarily paralyzes these ciliated cells inhibiting the body's natural defense against unwanted dust. During the night, in the absence of smoke, the hair-like cells start working again and carry large amounts of mucous back into the mouth. This causes the so called "smoker's hack" in the morning. After

the first cigarette or two, the cleansing mechanism is paralyzed again and the coughing stops. It should now be evident why cigarette smokers who are exposed to asbestos appear to be at greater risk. Other reasons will also be discussed later in this section.

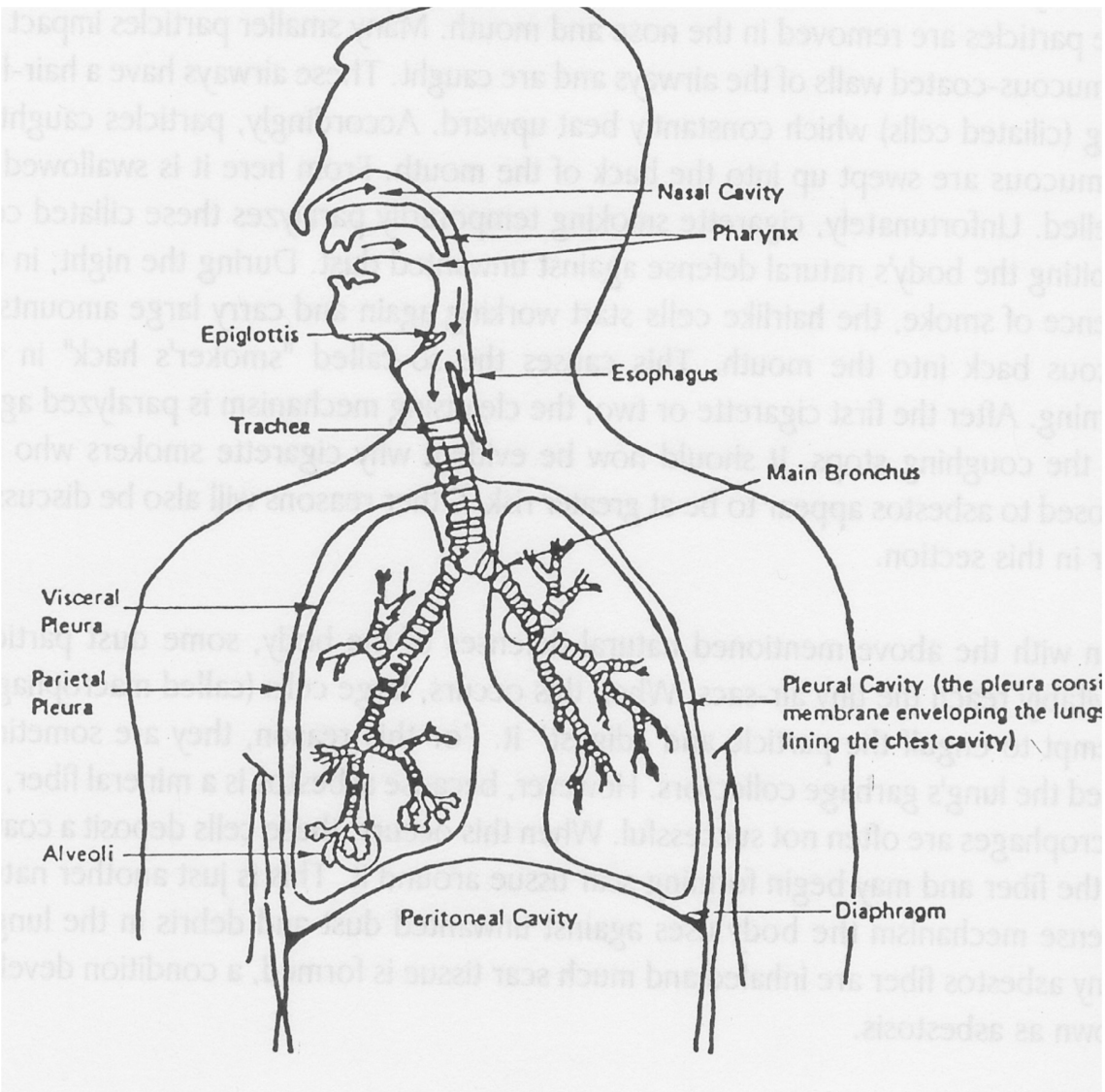

Figure 3-1: Routes of inhalation and ingestion of asbestiform fibers are shown by small arrows. Mesothelial cells line the outside of the lungs and the pleural and peritoneal cavities. Interaction of asbestos with these cells can result in either pleural or peritoneal mesothelioma. Adapted from Wagner, 1980*

* Figure from Asbestiform Fibers. Nonoccupational Health Risks, National Research Council, National Academy Press, Washington DC (1984), pg 101.

Even with the above mentioned natural defenses of the body, some dust particles inevitably reach the tiny air sacs. When this occurs, large cells (called macrophages) attempt to engulf the particle and digest it. For this reason, they are sometimes called the lung's garbage collectors. However, because asbestos is a mineral fiber, the macrophages are not successful. When this occurs, these cells deposit a coating on the fiber and may begin forming scar tissue around it. This is just another natural defense mechanism the body uses against unwanted dust and debris in the lung. If many asbestos fibers are inhaled and much scar tissue is formed, a condition develops known as asbestosis.

ASBESTOSIS

Asbestosis is a disease characterized by fibrotic scarring of the lung. This is a restrictive lung disease which reduces the capacity of the lung. The common symptom is shortness of breath. Asbestosis is prevalent among workers who have been exposed to large doses of asbestos fibers over a long period of time. Accordingly, there is a clear dose response relationship between asbestos exposure and developing this disease. This means the greater the asbestos exposure, the more likely asbestosis will develop. All forms of asbestos have demonstrated the ability to cause asbestosis. Like all diseases associated with asbestos exposure, it may take years for the disease to show up. The typical latency period for asbestosis is 15 30 years.

Even after exposure to asbestos has ceased, scar tissue will continue to form around existing scar tissue and fibers in the lung. Limiting exposure will reduce the amount of new scar tissue since additional fibers entering the lung will be reduced. The current Occupational Safety and Health Administration (OSHA) Asbestos Standards (29 CFR 1910.1001 and 29 CFR 1926.1101) were promulgated to greatly reduce asbestosis among asbestos workers by reducing their daily dose of asbestos.

LUNG CANCER

There are many causes of lung cancer, of which asbestos is only one. While employees exposed to industrial concentration of asbestos in years past have an increased risk of getting lung cancer (at least 5 times), their risk is not as great as the cigarette smoker (10 times). However, together, a cigarette smoker who also works with asbestos is more than 90 times more likely to contract lung cancer than the normal non-smoking population. Like asbestosis, there exists a long lag time between initial exposure and the occurrence of lung cancer, typically 20-30 years. There appears to be a dose-response relationship between asbestos exposure and lung cancer, although no “safe level” has yet been determined. It should be noted, however, that several research papers published in the mid 1980s suggest there may exist an exposure level for certain forms of asbestos, below which, the occurrence of lung cancer related to asbestos exposure will not