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Editorials

EXPERIMENTAL LUNG CANCER

GREAT caution must be exercised in assigning a causative relation between inhaled materials and lung cancer in human beings. In considering the cause of lung cancer and in experimental studies of lung cancer, one naturally examines critically those materials which get into the lungs and which may have a local effect on lung tissue. Thus, various industrial dusts and fumes, tobacco smoke, and exhaust gases from heavy automotive traffic have been suggested as possible factors in lung cancer. These materials have been tested chiefly on the skin of mice. However, the mere demonstration of materials in the air which are known to be carcinogenic to mouse skin is not proof that they can cause human lung cancer. Experimental carcinogenesis has taught that different species react differently to the same material (i. e., the mouse skin is very receptive to methylcholanthrene but the rat and guinea pig skins are very insensitive) and different tissues of the same species have widely varying sensitivity to the same material (i. e., the rat skin is unresponsive to carcinogens but the rat subcutaneous tissues are so reactive that they respond with sarcoma production even to glucose).

Human epidemiological data give the only really sound way of associating human cancer with an environmental exposure. In the collection of such data, a sufficiently large sample must be obtained to permit statistical analysis and valid conclusions. Even so, causal relationships must be further validated by continuing epidemiological methods, supplemented by animal experimentation. It is neither valid to conclude that animal experimentation provides definitive answers to the human cancer problem nor valid to conclude that it offers nothing to an understanding of the human cancer problem. Intelligent use of data obtained with animals should be coupled with intelligent application of the information learned from human epidemiological data, in order to understand the further research effort needed and the practical control methods indicated.

Skin carcinogenesis in the mouse has been used, and effectively, as a guide to the control of potential occupational skin carcinogens in man. The whole field of experimental skin carcinogenesis arose out of the epidemiological studies of occupational skin cancers in humans, begun some 35 to 50 years ago, and yet today a complete understanding of the mechanism of human skin carcinogenesis has by no means been realized. For instance, the relative importance of carcinogens and co-carcinogens in skin carcinogenesis is still being intensively investigated.

The mechanism of lung carcinogenesis in the mouse has not yet been worked out, let alone this mechanism in the human. We are today in about the same stage of understanding lung carcinogenesis as we were in skin carcinogenesis in 1900, prior

to the artificial production of skin tumors in experimental animals. A study of occupational lung cancers suggests that a completely different set of mechanisms may be at work. Thus, chromium and nickel under some conditions, asbestos, and an as yet unidentified factor in isopropyl alcohol manufacture are today most suspect as human pulmonary carcinogens. The only experimental lung cancers produced by inhalation have been in response to beryllium and radioactive metals.

It therefore seems highly dangerous to draw conclusions about human lung cancer from experimental evidence based on the presence of mouse skin carcinogens. Rather would it be wiser to display temperance in the interpretation of present day data until we have developed a much greater fund of knowledge concerning the mechanisms of lung carcinogenesis.

ROBERT E. ECKARDT
PHILIP DRINKER

(Courtesy of the *Saturday Review* and of the artist, Robert Kraus.)

EXPERIMENTAL INFECTIVE PNEUMOCONIOSIS. E. J. KING, C. V. HARRISON, G. P. MOHANTY, and D. A. MITCHISON, Arhiv hig. rada 4:289-300, 1953.

Quartz and coal-mine dusts were introduced into the lungs of rats by inhalation in a high concentration for four months, and then groups of these dusted animals were infected with a virulent strain of human tubercle bacillus (37 Rv). Dusting was continued for a further period of 10 months in the case of the coal-mine-dust group. No variation in lung pathology was seen in the quartz group, although both groups produced classical silicotic nodules. In the case of coal-mine dust the infective group showed slightly more fibrosis, diffuse in nature, than either the noninfected or the tuberculous control group. These are preliminary experiments, and the results must be considered as both tentative and not very conclusive. Work is being continued.

FROM THE AUTHORS' SUMMARY.

BREATHING OF PNEUMOCONIOSIS-PRODUCING DUSTS. C. N. DAVIES, Arhiv hig. rada 4:301-306, 1953.

Pneumoconiosis-producing dusts must be insoluble or have a very slow solubility. Dusts which caused long-term cumulative effects act in three ways—by stimulating the growth of fibrous tissue (quartz), by producing mechanical injuries (asbestos, talc), and by accumulating large quantities of particles in the lymphatic system and throughout the lungs.

Another kind of risk from dust accumulating in the lungs arises from radioactive materials, particularly from solid products of radioactive decay of radon. An association between the normal content of radon in the atmosphere and lung carcinoma in the general population cannot be dismissed as impossible when varying susceptibility is taken into account.

FROM THE AUTHOR'S SUMMARY.

PNEUMOCONIOSES WITHOUT FIBROSIS. D. M. MARCHAN, Arhiv hig. rada 4:402-427, 1953.

The avowed object of this article is to bring some order into the vast tableau of those pneumoconioses which are not associated with pulmonary fibroses. All dusts are more or less injurious, depending upon their nature and amount; no dust is really inert if in excess. Size is always important, since particles more than 5 μ in diameter cannot get down into the pulmonary alveoli. The construction of the upper air passages decides why some persons are less prone to dust damage, as the particles are caught there instead of penetrating deeper down.

First, the fibrosis-causing dusts are dismissed, including those consisting of silica wholly or in part. The presence, if any fibrosis develops, of some percentage of silica must always be suspected. No dusts are harmful on account of their hardness or sharpness. Short allusions are made to a great number of dusts, some of which are said to be pathogenic. Pure coal and graphite are considered nonpathogenic. There are the metalloconioses which are caused by toxic lead, beryllium, and manganese as well as nontoxic iron and zinc. Then come the mycoconioses, including weavers' cough and cotton-strippers' asthma, the latter being known medically as byssinosis. Among the diseases surnamed allergoconioses are cannabiosis and bagassosis. [Adding the suffix "osis" does not seem to introduce any order, but readers will be grateful for the excellent list of references.]

E. L. COLLIS [BULL. HYG.].

CLINICAL EVALUATION OF DISABILITY IN SILICOSIS. J. FROST and J. GEORG, Acta med. scandinav. 147:349-357 (Dec. 30) 1953.

In cases of silicosis there has been established no correlation of the x-ray picture, the symptoms experienced, and the degree of disability. In order to reexamine the position, clinical examinations and measurements of pulmonary function were carried out on 50 persons with silicosis (from among whom patients with tuberculosis, cardiac diseases, or high blood pressure were excluded). X-ray films were taken, and fluoroscopic examinations were made of diaphragmatic movements; electrocardiograms were made. Vital, residual, and total capacities of the lungs, together with maximum breathing capacity, were measured, and a special working test on a bicycle-ergometer was made.

The cases were grouped into three stages of silicosis. When the observations were analyzed, a significant difference was noted between the total capacity for Stage 3 and for Stage 1; the same held good for vital capacity and maximum breathing capacity, but differences between