

continuation of the exposure to soot. While some of the lungs showed at histological study the changes characteristic of a chronic organizing pneumonia, none revealed any evidence of a neoplastic condition.

Atypical epithelial growth was observed by Ibuka in the lungs of rabbits fifteen to sixty-eight days following the implantation of blocks composed of a mixture of soot and paraffin. Similarly inconclusive results were obtained by Schmidtman, who exposed rabbits to the inhalation of the soot contained in the exhaust gases of a Diesel engine for periods up to 385 hours. The rabbits, which died 185 to 480 days after the cessation of this treatment, showed at the histological examination of the lungs a nodular type of anthracosis with scar formation and distortion of the bronchi. The bronchial epithelium had grown in some places into the alveolar lumina. These changes were most advanced in those animals which survived for the longest time. The epithelial proliferations, however, were in no instance of a neoplastic type. Schmidtman concluded that these growth manifestations were the result of a stimulus exerted by traces of the fuel oil mixed with the soot.

The exposure of mice to the inhalation of exhaust gases produced by a gasoline motor, which differ, however, fundamentally in chemical respects from those emitted by a Diesel engine, proved to be entirely negative regarding a causative influence upon the development of pulmonary neoplasms. Twenty-six mice exposed by Smith over a period of five months to the inhalation of the exhaust fumes delivered by a Ford engine remained free, with one exception, from pulmonary neoplasia. This incidence of pulmonary tumor formation is within the normal range and, therefore, etiologically insignificant. There was no significant increase in the number of lung tumors over those found among the controls in mice exposed for seven hours daily during five days of the week for the greater part of their lives by Campbell to the inhalation of exhaust gases produced by an internal combustion engine. The concentration used was comparable to that existing in traffic blocks and garages. These results were confirmed by observations made by Schmidtman. Experimental investigations of the carcinogenic potentiality of the sooty matter deposited in the exhaust pipes of gasoline motors are still outstanding.

A definite decision as to the role soot and sooty material may play in the production of pulmonary malignancy is not possible in view of the partly controversial and inadequate experimental evidence available. It may be mentioned that the clinical evidence, supporting a causative relationship between the inhalation of soot and the production of pulmonary carcinoma, is more suggestive than that provided by experimentation. This discrepancy possibly may be attributed to the fact that the duration of exposure to soot used in the experiments was perhaps too short to elicit a neoplastic response in the lungs of mice and rabbits. It seems to be advisable to alter the fundamental experimental approach to this problem by using animals which have a much longer normal life span than mice so as to be able to lengthen considerably the time of exposure. There does not exist any definite and fixed