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**THE EFFECT OF DIRECT CIGARETTE SMOKE INHALATION  
ON THE  
RESPIRATORY TREE OF DOGS**

A tracheostomy was performed on 10 beagles, 9 males and 1 female, ages 9-30 months, and weighing 17-30 pounds. Ten additional dogs were used as controls. Two of these had tracheostomies kept open by hollow teflon tubes.

Every morning and afternoon of the experiment 10 dogs smoked cigarettes through a specially designed tube with auxiliary apparatus for handling. The number of cigarettes smoked per day was gradually increased to a maximum of 12 per day per dog. Five of the ten dogs died during the experiment. Four of these had thrombi with pulmonary infarction. The remaining 5 were killed after 421 days of smoking.

Immediately after death, the lungs were removed and filled with formalin instilled into the trachea. The tracheobronchial tree was then dissected out of the lungs and divided into portions by essentially the same procedure we have used with human material. We planned to divide each tracheobronchial tree into 133 portions (each from a specified location), embed each portion in paraffin, and then cut out one section from each block for microscopic examination.

Hyperplasia with distended goblet cells in the glands was not found in any of the sections from the 10 nonsmoking control dogs, but was observed in 96.7% of the sections from smoking dog 29 and in 98.9-100% of the sections from each of the other 9 smoking dogs. None of the glandular epithelium from the nonsmoking dogs showed any evidence of cells with atypical nuclei. The cells with atypical nuclei increased as the smoking habits of these dogs increased.

In 8 of the 10 nonsmoking dogs, no sections with areas of epithelium averaging more than 2 cell rows in thickness were found - that is, no more than 2 rows of basal cells, plus a surface row of ciliated columnar cells. The other 2 nonsmoking dogs showed minimal changes in this respect.

Every section from the 10 smoking dogs had epithelium consisting of 3 or 4 rows of basal cells plus a row of ciliated columnar cells, or 3 or more rows of cells but without ciliated columnar cells. Where ciliated columnar cells were lacking, the surface cells appeared typically squamous. Sections with areas having 6 or more rows of epithelial cells were not found in dog 29 that died on the 24th day and occurred more frequently in the 5 dogs killed after 421 days than in the dogs that died earlier.

Every section with epithelium was examined for the presence or absence of cells with atypical nuclei. There were no atypical nuclei in any of the sections from the 10 nonsmoking dogs and none in any of the sections from smoking dog 29. However, atypical nuclei were found in the epithelial cells of all the sections from the other 9 smoking dogs. Furthermore, the proportion of cells with atypical nuclei was considerably greater in sections from the 5 dogs killed after 421 days than in sections from dogs 30 and 35 that died on days 229 and 278, respectively. In the 7 smoking dogs that survived 410 days or longer, most sections had lesions in which 50-69% of the epithelial cells had atypical nuclei.

These findings parallel those in our study of human beings; namely, that as a result of the inhalation of smoke, there is an increase in the number of basal cells and as the smoking habit continues, the nuclei become atypical, increasing and extending toward the surface. The studies of these dogs show dyskeratosis present in a number of sections from the heavy-smoking dogs.

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