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*Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are being received that the omission of some is inevitable. Letters should be signed personally by all their authors.*

## Relationship between lung cancer and chronic bronchitis

SIR,—In their report on 233 men with chronic bronchitis, Drs M Caplin and Freda Festenstein (20 September, p 678) found that 60 died after surviving on average for two years and 10 months. Of 76 men with severe airways obstruction 41% died, none from lung cancer. In the group without severe obstruction only 18% died, but over one-third of these did so from lung cancer. They seem to imply that severe airways obstruction protects people from developing lung cancer.

None had detectable cancer when they entered the survey. Severe airways obstruction has a profound effect on survival. Nearly half died within three years, so they had little chance of getting cancer. The death rate in the mild group was much lower and they lived longer. People in this group would be unlikely to die unless they developed some complication like lung cancer. This disparity between the two groups will increase with time. Isn't this the simple answer?

In their statistical analysis Drs Caplin and Festenstein compare 11 lung cancer deaths out of 29 in the mild group with no cancer deaths out of 31 in the severe group. Surely the comparison should be between 11/157 and 0/76, also taking into account the very different duration of observation and survival in the two groups? They find it surprising that men presenting with lung cancer and incidentally found to have chronic bronchitis had less severe airways obstruction than those referred because of bronchitis. In the former group bronchitis was diagnosed on answers to direct questions and the disease was often

mild. I fail to understand their surprise, without doubting that smoking makes a contribution to both cancer and airways obstruction. The all-or-none law surely does not apply here.

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D DAVIES

SIR,—I read with great interest the excellent article by Drs Maxwell Caplin and Freda Festenstein (20 September, p 678) regarding the absence of an association between lung cancer and airways obstruction. Their findings confirm impressions which I have long held but have never attempted to verify that many heavy smokers who develop bronchial carcinoma have little or no airways obstruction whereas comparatively few patients with severe chronic obstructive bronchitis develop bronchial carcinoma.

Many studies have shown a broad correlation between the quantity of tobacco (of any kind) smoked and the prevalence of symptoms of mucus hypersecretion. Rimington<sup>1</sup> found that smokers with simple chronic bronchitis appeared to run a higher risk of lung cancer, an association confirmed by the findings of Drs Caplin and Festenstein. This can readily be explained if one accepts that the risk of developing lung cancer and the liability to mucus hypersecretion both depend largely on the quantity of cigarettes smoked, the former being due to the carcinogenic properties of cigarette smoke and the latter to its irritant effects. However, the fact

that it is by no means uncommon for patients with lung cancer to deny that they have ever had habitual expectoration suggests that individuals differ in the susceptibility of their bronchi to the irritant effects of cigarette smoke. Might I suggest that "host factors" could also provide an explanation of the dissociation between lung cancer and chronic obstructive bronchitis found by Drs Caplin and Festenstein?

No epidemiological study has shown as clear a quantitative relationship between smoking consumption and airways obstruction as that between smoking consumption and lung cancer. For instance, in a study of male post office workers in the United States and in London<sup>2</sup> the differences in FEV<sub>1</sub> between non-smokers and light, moderate, and heavy smokers were much smaller than those between the American and London postal workers within each smoking category. Read and Selby,<sup>3</sup> who found no association between impairment of peak expiratory flow and the amount smoked in the subjects whom they studied, invoked genetic factors in an attempt to explain their findings.

It seems clear that other factors besides smoking quantity are concerned in the pathogenesis of chronic obstructive bronchitis. Rather than postulate that the presence of severe airways obstruction is in some manner inimical to the development of lung cancer, is it not simpler to explain the dissociation between chronic obstructive bronchitis and lung cancer on the basis of host differences in the development of the former which are completely independent of susceptibility to the carcinogenic effects of cigarette smoke?

The apparent dissociation between asthma and lung cancer mentioned by Drs Caplin and Festenstein may depend upon an atopic constitution rather than upon asthma per se. The findings of several studies, all of somewhat questionable validity because of their retrospective nature, have suggested that all