life; and that the general theory extends to numerous non-neoplastic age-dependent diseases, for which it was first developed.

A good test is provided when my "Procrustean model" forces me to postulate, for example, that three distinctive types of malignancy are included under the rubric "malignant neoplasms of the large intestine and rectum". When the clinical evidence bears out the postulate (as it does in this and many other instances) the "Procrustean model" is usefully corroborated. Peto seems not to appreciate that the rigidity of a "simplistic Procrustean model" is a positive virtue in that it allows its predictions to be the more readily falsified. The type of "minor modification" (in other words, fudge factor) that Peto advocated in his review does not belong to rigorous science, and should be regarded as the last resort of a desperate theoretician. More interesting corroboration of my theory is provided when the progression of a neoplasm from one distinctive phase to the next is seen to correspond to a simple increase in $n$ with constant $r$ and $k$.

Finally, we should rejoice over the speed and promise of Peto's progress. An increase in lung cancer rates that was "largely due to cigarette smoking" in his review has been transformed in his letter to an equipartitioning between diagnosis and smoking. If this rate of improvement is maintained, Peto should rapidly approach the truth.

P. R. J. BURCH

REFERENCES

Burch, P. R. (1976) The Biology of Cancer: A New Approach. Lancaster: Medical & Technical Publishing Press Ltd.

Registrar General (1958) Statistical Review of England and Wales for the Year 1956. Part III Commentary. H.M.S.O.

SIR,—Professor Burch makes three points:

(1) That the anomaly in the sex ratio arises only if it is assumed that misdiagnosis rates have been similar in the sexes. This is the opposite of the truth. This assumption, coupled with the conventional model of lung-cancer incidence in smokers, predicts the observed ratio in each quinquennium and age-group with remarkable accuracy. The study he cites (Registrar General, 1958) confirms the age dependence of misdiagnosis that I hypothesized, and shows no evidence of any sex difference.

(2) That synchronous proportional changes in both sexes prove that cigarette smoking cannot have contributed substantially to the observed increase. Such changes will necessarily occur, whatever the pattern of the underlying cause, when substantial improvements in diagnosis occur. The widespread introduction of radiography after the First World War, and antibiotics to clear supervening infection during the Second, probably account for the increases in both sexes during these periods (Fig. 10.14).

(3) That my statement that the increase was largely due to cigarette smoking was misleading. This apparently trivial semantic point conceals an important scientific misunderstanding. If smoking had not increased there would have been a tenfold increase due to improved diagnosis, whereas recorded rates have increased a hundredfold due to the (multiplicative) tenfold increase caused by smoking. Roughly 90% of the increase is thus due to smoking. (By the same token, of course, 90% could be attributed to improved diagnosis.) As I mentioned in my last letter, Professor Burch assumed that these two factors of 10 should be added rather than multiplied.

Professor Burch still offers no quantitative explanation of the age-specific secular changes and sex ratio, nor any coherent critique of my detailed analyses, while attacking me as a "desperate theoretician" whose "remarks were so lacking in perception it would be a kindness to refrain from criticizing them". His persistent charity is becoming rather tedious.

J. PETO