



strengthen her will to live. On our very first consultation he said 'Out there is somebody who has been cured of cancer of the ovaries. If somebody could be cured why not you also?' What a boost it was for her spirit.

I know of many cancer patients who died within the time specified by oncologists. Their predictions became reality for their patients.

Patients go to an oncologist for treatment in the hope of beating the disease so that they can live longer. Rather than keeping the hope of a patient alive, oncologists seem to be over-zealous to kill them off with their predictions of death.

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Straight thinking about the aetiology of endemic cancer of the oesophagus

To the Editor: Despite decades of research, no carcinogen has yet been identified as the cause of endemic squamous cancer of the oesophagus (SCO), yet there is still a view current that endemic SCO in South Africa may be caused by a single potent carcinogen. Logic does not support this view.

Smoking has been identified as having a very strong association with SCO in endemic areas.^{1,2} Tobacco has a proven association with SCO in non-endemic situations also. It is therefore extremely unlikely that its association with endemic disease is spurious. Tobacco is a proven carcinogen. It is therefore logical to assume that its action in promotion of SCO is as a carcinogen.

Conclusion: tobacco is a significant carcinogen for endemic SCO.

Tobacco may act as a carcinogen for the majority of those who have SCO, but it cannot be involved for the significant

minority of approximately 30% who do not smoke, 19% of whom have never smoked.^{1,2} After excluding all patients who smoke, the remaining number who have SCO in endemic areas and who do not smoke would still provide a grossly elevated prevalence of the disease.

Conclusion: in addition to tobacco, there is another carcinogen or other carcinogens at work.

Although the white population of South Africa smokes more than the black population it does not have the same incidence of SCO. Two possibilities exist. The white population may be protected from the effects of tobacco. This idea is unlikely enough to be eliminated — the incidence for whites in South Africa is as for similar population groups in Europe and North America. The alternative explanation is that the black population is predisposed to the effects of tobacco or is exposed to co-carcinogens that work with tobacco and other carcinogen(s).

Conclusion: the affected population is predisposed to the action of tobacco as a carcinogen, or is exposed to co-carcinogen(s) that can work with tobacco.

The presence of a single powerful carcinogen acting on its own as the cause of endemic SCO is not a credible possibility.

The molecular and genetic background to the disease, the focus of much research at the moment, may well reflect only the end stage of the disease and may not furnish tools for prevention.

Effort should therefore be directed to pursuing the two remaining possibilities — predisposition and co-carcinogenesis.

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