

Diesel Smoke And Lung Cancer



Dr Kitty Little

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By

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FROM about 1930 it became apparent that there was an increase in the incidence of lung cancer that was out of proportion to the increase in cancer as a whole, and that the causative agent must be something comparatively new, probably something that had made its appearance during the 1930's. What was it?

To elucidate such problems there are well-established methods of scientific investigation: evidence is collected, hypotheses suggested, further facts sought, hypotheses modified or if they are not in accord with the evidence abandoned, perhaps new hypotheses put forward, and so on - and always, when a fact and a hypothesis contradict one another, it is the fact that must be retained.

There are plenty of facts available about the increase in lung cancer, and by about 1940 three main hypotheses were being considered: the action of urban smoke, cigarette and tobacco smoke, and diesel smoke. We need to consider which, if any, of these is in accord with the available facts.

The increase in lung cancer was primarily an urban phenomenon, and it was not observed in genuinely rural communities. Further, in cities on windy sites (e.g. Port Elizabeth or Cape Town) the same increase was not found as in other cities with a more stagnant atmosphere (e.g. Durban or Johannesburg). Such observations might be thought to implicate urban smoke. But urban smoke levels were high well before 1920 to 1930 (Parliament first discussed the problem in 1306 when the use of coal started), while when they were reduced after the Clean Air Act of 1956 lung cancer levels were not reduced. This eliminates the urban smoke hypothesis.

Similarly, cigarette and tobacco consumption among men had been high for about half a century before the increase in lung cancer became apparent. Women took to smoking later than men, and it was not till 1961 that the female cigarette consumption reached the male consumption for 1922. The increase in lung cancer in women has not paralleled this increase in smoking, but started at the same time as men, from about 1930 onwards. (1)

Again, in the rural communities in South Africa, where detailed medical and commercial evidence is available, the level of lung cancer is low. (2) In Rhodesia, where the level of cigarette and tobacco consumption was high, lung cancer was virtually non-existent until after diesel was introduced.

Such observations eliminate cigarette and tobacco smoke from consideration, but strongly point to diesel smoke as the culprit. In Great Britain the increase started a few years after the introduction of diesel engines. In South Africa, in city after city, lung cancer followed a few years after diesel engines were introduced!. There seemed to be a lag of about 7 or 8 years between the critical exposure and overt symptoms. Diesel was introduced in Great Britain a few years before South Africa or New Zealand. During the next 20 years British immigrants to South Africa' and New Zealand (3) showed a higher lung cancer incidence than the local population of British origin, whether they smoked or not.

Statistics such as these that have been quoted provide almost complete proof that diesel smoke has been the cause of the rise in incidence of lung cancer, but statistics on their own can never provide complete proof. One also needs confirmation from an investigation into the biological mechanisms involved. This includes seeking to identify the carcinogenic agent or agents responsible.

Urban smoke and cigarette and tobacco smoke contain a chemical, 3:4 benzpyrine, that is weakly carcinogenic. However, it oxidises very easily, and has never been shown to cause lung cancer - conditions in the lungs would favour rapid oxidation to harmless compounds. There is, however, evidence that diesel smoke contains at least four strongly carcinogenic

compounds. (4) It has also been shown, from field observations, that local concentrations in some traffic conditions can be very high. (5)

By the middle of the 1950s it was quite clear that the increase in lung cancer had been due to diesel smoke, and that cigarette and tobacco smoke had nothing to do with it. Yet on 27th June 1957 the anti-smoking campaign was launched, (6) with the Health Education Council being formed to help push its propaganda. (The Health Education Council, and its successor the Health Education Authority, have been primarily concerned with promoting bogus medical propaganda).

As a result of the scare campaign there has been a decrease in tobacco consumption since 1962. Since 1962 there has also been an increased and increasing output of diesel smoke on all major roads, while in 1970 and since there has been an increase in lung cancer deaths in areas affected by this increase. Thus, in the Abingdon and Faringdon district lung cancer deaths rose by 65% in 1970 as compared with previous years. (7)

Yet another source of evidence has been the statistics provided by the Registrar of Births and Deaths. The occupation with the highest incidence of lung cancer was that of garage attendant, while long distance lorry drivers also showed a high incidence. All other categories showed far lower incidences. When attention was drawn to this fact the only reaction was to introduce self-service at garages.

One of the main props of the anti-smoking campaign was a paper suggesting, as a result of a survey among British doctors, that those who gave up smoking were less likely to get lung cancer. (8) The figures given in that paper indicated that those who inhaled the smoke were less likely to get lung cancer than those who did not, but the authors decided that these figures were not statistically significant. The figures suggesting that giving up smoking decreased the likelihood of getting lung cancer were much closer, but the authors deemed those to be highly significant. There was no attempt made to check if any doctor with an early lung cancer had some other condition recorded as a cause of death. One such case would have been sufficient to invalidate the conclusion.

Since then statisticians have repeatedly attempted to implicate cigarette smoke by ignoring the involvement of diesel smoke. This invalidates all their results, since statistics always seem to give an answer, but it is only the correct answer when all the relevant variables are taken into account - and the effect of diesel smoke is undoubtedly relevant. It is interesting that lawyers issued instruction on how to confuse a court should an action for damages resulting from diesel smoke be initiated. (9)

The fact that many of the cases of lung cancer involve non-smokers became something that could no longer be ignored. Therefore, as diesel family cars came onto the roads, an attempt has been made to implicate "passive smoking". Evidence already quoted shows that this suggestion must be false. Not only does tobacco smoke not contain a carcinogenic agent that could cause lung cancer, but the high levels of smoking, in this country before diesel was introduced, and in South Africa and elsewhere in places where diesel had not been introduced, never resulted in lung cancer from "passive smoking". If the suggestion was valid they would have done.

According to advertisements produced by the anti-smoking lobby there are over 30,000 deaths from lung cancer a year. Yet there has been evidence for over 40 years that those deaths were not due to cigarette or tobacco smoke. Since the effect of the anti-smoking campaign has been to prevent the genuine cause from being publicly acknowledged, there is a very real sense in which we could say that the main reason for those 30,000 deaths a year from lung cancer is the anti-smoking campaign itself.

Dr Little's paper confirmed

After Dr Little wrote the above paper, it found confirmation in a study of 6338 non-smoking men, aged 27-95, who lived in California between 1967 and 1992. This study, published in January 1999, (10) found that PM10 exposure was strongly associated with lung cancer, raising the risk by 2.38 times. PM10 exposure was also associated with all natural causes of death in men and with an increased mortality from non-malignant respiratory disease in men and women. PM10s are particles of less than 10 µm in diameter exhausted from Diesel engines. David Abbey, leading

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author of the study noted that men who spent longer outside were at greater risk than men who spent most of their time indoors .

In addition, ozone exposure was implicated in increased risk of lung-cancer mortality in men, and sulphur dioxide (SO₂) exposure was independently associated with increased risk of lung-cancer mortality in both men and women. These too are found in vehicle exhaust emissions.

'Clean' Diesel is even worse!

Recently there has been a move to reduce the size of Diesel exhaust particles - the new 'clean' city Diesel. However, these may be even more harmful. As Dr Abbey points out, "recent studies on the short-term effects of atmospheric particles on respiratory and cardiovascular diseases have shown that PM_{2.5}s and even smaller particles are more important than PM₁₀s."

Smoking may reduce cancer risk - Stomach cancer

There is other evidence that smoking might actually protect against cancer. Nitrates and nitrites, commonly found in vegetables and cured meats turn to carcinogenic nitrosamines in the stomach. Smoking inhibits the uptake of circulating nitrate into the saliva, especially at higher levels of dietary nitrate intake. (11)

Breast cancer

One out of every 250 women has one of the inherited mutated genes, BRCA1 or BRCA2, whose normal function is not yet fully understood. And 80 per cent of women with one of the mutated genes will get breast cancer before the age of 70. This means that 3200 women per million will get breast cancer. Dr Paul Kleihues, M.D., Director of the International Agency for Research on Cancer, WHO reported a study which found that smoking cuts the risk of developing breast cancer by 50 per cent in these women. "The protection associated with smoking increased with the amount smoked. . . The risk reduction associated with up to four pack-years (one pack-year equals one pack per day for one year) of smoking

was 35 per cent, and for greater than four pack-years of smoking was 54 per cent." (12)

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Dr Kitty Little

The late Dr Kitty Little was a research scientist for nearly fifty years. For ten of those years, early in her career, she worked in the medical division of the Atomic Energy Research Establishment, Harwell doing research into the effects of radiation on the body. She also worked in orthopaedics at Oxford University Medical School, with US Forces, Washington as a pathologist, and the MRC laboratory working on DNA and the causes of dental caries. At Oxford she wrote a textbook on bone pathology and bone cancer. Kitty died in late 1999.

