Editorials

Industrial carcinogens: the extent of the risk

Experts, especially those with proselytising inclinations, have a weakness for ex-cathedra pronouncements. The widely quoted statement that around 80% of human cancer can be attributed to environmental factors belongs to this genus (Higginson, 1969). An exaggeration in the first place, the statement has been taken out of context and used by some misguided environmentalists and other interested parties as an indication that exposure to chemicals and other toxic substances, such as asbestos, is the major cause of cancer in contemporary Western society. The arguments marshalled to support this view rely heavily on the differing incidence of various forms of cancer in different countries, plus a so-called dramatic increase in the death rate from cancer in the USA and Western Europe over the past two to three decades. The Occupational Safety and Health Administration (OSHA, 1977) and the Environmental Protection Agency (EPA, 1975) along with sundry other US Government agencies have endorsed this hypothesis with unseemly fervour, and are currently proposing draconian measures that will regulate all carcinogens and potential carcinogens in a standard fashion from now to eternity. OSHA (1977) has requested “that the proposed procedures, model standards, and classification criteria will be consistently applied and foreclosed from reconsideration in any future regularity activity.” Such regulations can hardly be put forward as representing a disinterested impartial assessment of the present situation and suggest that those who are proposing them have closed minds and do not wish to be confused by facts or new information. Were such regulations put into effect, not only would the US chemical and agricultural industries be profoundly affected, but as a result of receiving US Government blessing, the regulations would attain an unmerited respectability that would undoubtedly influence and put pressure on other Western countries to follow suit.

The hypothesis that environmental factors are the main cause of human cancer does not withstand critical examination. Firstly, although there are some 30 to 40 substances known to be carcinogens in man (Smith, 1978), and of these several, including asbestos, the halo-ethers, and various aniline derivatives are occupational hazards, there is no evidence that the increase in cancer in the USA or Britain is related to exposure to industrial chemicals. Indeed, the overall cancer rates in women have remained relatively unchanged for the past 15 years, and the only type of cancer that has increased greatly in women is lung cancer. Meanwhile carcinoma of the uterus and stomach have been slowly declining. In men the modest increase is due partly to a decrease in other causes of death, in particular those caused by infections such as tuberculosis, and partly to the massive increase in lung cancer deaths, 90% of which result from smoking. Secondly, the restricted and artificial use of the term environment to include only the chemical environment is misleading, and environmental exposure in its broader sense should include cigarette smoking, exposure to ionising radiation, sunlight, and family size; all of which play a far more important role in the production of cancer than industrial chemicals.

The proposed OSHA regulations define a carcinogen as “any substance that has been validly shown to produce tumours, either benign or malignant, in animals, or which decreases the latent period between exposure and development of tumours.” The production of tumours in any species suffices for the substance to be designated a carcinogen, yet the induction of cancer in experimental animals, with certain well-recognised exceptions, remains uncertain and unpredictable, and depends on the route of administration of the substance being tested, on its dose, on the length of exposure, and the species of animal, not to mention the diet, age, and sex of the animal used (McLean, 1973; Stevenson et al, 1978). The acceptance of the development of tumours in any one of a variety of species under vastly differing circumstances—tumours some of which might well be regarded as arising spontaneously—increases the number of recognised carcinogens from around 150 to several thousand. The inference that the results of animal carcinogenesis can be applied across the board to man is clearly unwarranted. Despite well-recognised species differences, OSHA subscribes to the view that positive studies in
animals should be given more weight than negative studies in man. Are we then expected to assume that cancer epidemiology in man is worthless unless it shows a positive association?

The testing of every new chemical in several species under a variety of different conditions is not feasible and would be inordinately expensive. Thus simpler methods for detecting carcinogens have been advocated (Bridges and Fry, 1978). These include the Ames test, which depends on the ability of the substance being tested to influence the mutation rate of a nutritionally deficient strain of Salmonella typhimurium. The other popular test is known as the mammalian cell transformation test and depends on the fact that cultured cells are known to reflect certain in-vivo malignant characteristics. In this regard a particular type of colony growth suggests that a substance may be a carcinogen. When these tests are used, nine times out of ten known carcinogens will give positive results, but an appreciable number of false-positives and false-negatives will occur. Those who advocate these and other similar so-called reproducible tests—and neither of the two tests mentioned above fits this criterion—tend to forget that no test has yet been or indeed can be devised that will indicate whether there is a threshold below which a suspected carcinogen is innocuous in man.

Until recently it was accepted that occupational exposure played a part in between 1 and 5% of all cancers (Doll, 1977). A recent report from several US Government agencies (Bridge et al, 1978) categorically asserted that previous estimates were erred, and that occupational carcinogens are likely to be wholly or partly responsible in around 30% of all cancers that will develop over the next few decades. They further suggest that asbestos exposure will be involved in 15% of all cancer that occurs over the same period; an inference that is based on the findings of one study (Selikoff and Hammond, 1978). This report has caused considerable consternation, and its contents have been and continue to be regarded as sacrosanct. The Secretary of Heath, Education, and Welfare has quoted sections of it in an address to the AFL-CIO National Conference on Occupational Safety and Health, an organisation that is inherently sympathetic to the notion that industry rather than the employee's habits is to blame. The report is notable mainly for the peremptory dismissal of the numerous studies that express divergent views. In putting together an official position paper such as this, impartiality and objectivity are essential, but in this case they are conspicuously lacking.

Unfortunately, environmentalists, be they employed by Federal or State Government or by various voluntary organisations, are invariably anxious to attract as much publicity as possible, since in doing so they ensure that popular acclaim keeps them in business, whether by putting pressure on politicians to maintain financial support or through voluntary contributions to the appropriate organisation. A more objective assessment of the problem would be far more likely to emerge from the National Academy of Sciences, an organisation that has no axe to grind.

The attitudes and assumptions that have evolved in regard to industrial carcinogens have split over to occupational lung cancer, and the publicity given by the news media to the hazards of asbestos exposure has created the erroneous impression in some members of the general public, especially in North America, that most lung cancer results from occupational exposure. Moreover, because many of the agents responsible for the development of occupationally related lung cancer have been identified, it is being too readily assumed that environmental contamination by such agents can be easily controlled, and will result in an appreciable decline in the number of deaths from lung cancer. In reality, subjects who develop lung cancer as a result of their occupation represent a small but important group, and their importance lies in the fact that it is possible to control to some extent exposure to the responsible carcinogens. Of the 90 000 or so deaths that will occur in the USA this year from lung cancer, occupational exposure will be a factor in 2000 to 3000, but even in these cigarette smoking will still be an important factor, asbestos, radon daughters, the halo-ethers, etc, playing the lesser role of the co-carcinogen.

It has also been assumed that because certain groups of industrial workers are known to be at greater risk from lung cancer, routine screening of such workers will detect the condition earlier, thereby permitting more effective treatment and a greater chance of cure. To achieve this end OSHA and the National Institute of Occupational Safety and Health (1973) have been advocating chest films and sputum cytology at specified intervals. The arguments used in favour of routine screening are specious but a more critical examination shows many flaws in them. So far as the management of lung cancer is concerned, there is little evidence to suggest that early diagnosis can be equated with improved prognosis. To endorse a programme of routine screening is premature. Before any screening test is used on a
population it is mandatory to determine the test’s specificity, sensitivity, and validity, and the cost. Consideration must be given to the percentage of false-positives and false-negatives, and what can be done to affect the prognosis when the condition has been diagnosed. Clearly, if one cannot alter the course of the disease then the whole exercise has been futile. The Mayo Clinic is presently conducting a large study to answer some of these questions (Fontana, 1977), and it would be appropriate to await the results of this and other similar studies.

Those who are concerned with introducing legislation to control carcinogenic substances need to tread warily. Preconceived ideas and doctrinaire attitudes can lead only to the promulgation of restrictive regulations that will not benefit the community. The benefits that occur from the use of a particular substance have to be weighed against its possible harmful effects. To think of asbestos in terms of carcinogenesis without considering the lives it has saved as a result of its fireproof properties is intellectual dishonesty. Similarly, for the US Food and Drug administration to ban medroxyprogesterone acetate (Depo-provera) as a long-acting contraceptive because of the doubtful demonstration that under certain circumstances it can be a carcinogen in animals, makes it difficult for developing countries with an entirely different set of problems to use the preparation. Yet in India the chance of developing tetanus after childbirth is infinitely greater than is the theoretical chance of cancer induced by the long-acting progesterone derivative. Legislatures and their scientific advisers need to think seriously before condoning or permitting the passage of legislation or regulations that will permit a particular substance to be banned on tenuous evidence, while at the same time protecting the sacrosanct right of the individual to do away with himself by his continued indulgence in cigarette smoke. One wonders how John Stuart Mill would react to a legislative body that bans cyclamates and yet tolerates, indeed in Canada and the USA, subsidises, those who grow and sell tobacco. Arbitrary legislative decisions based on insufficient evidence are likely to have serious economic, social, and biological consequences without doing anything to lessen the incidence of cancer.

W K C MORGAN

Sir Adam Beck Chest Diseases Unit,
University Hospital, London,
Ontario, Canada

References


Industrial carcinogens: the extent of the risk.

W K Morgan

Thorax 1979 34: 431-433
doi: 10.1136/thx.34.4.431

Updated information and services can be found at:
http://thorax.bmj.com/content/34/4/431.citation

Email alerting service

These include:

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/