A/C Pipe Producers Association

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ISSUE #1 VOLUME #1 This first in a series of "advisories" is designed to keep water utilities and public health officials current on information regarding usage of asbestos-cement pipe. Each Advisory issue will factually summarize data contained in scientific papers, government studies, media reports, and technical and educational source material.

GOVERNMENT ANIMAL FEEDING STUDIES SHOW NO CARCINOGENIC EFFECT OF INGESTED ASBESTOS

Preliminary results of government-sponsored studies on the effect of ingested asbestos on laboratory animals show no carcinogenic or cocarcinogenic effect.

The studies are being conducted by the National Institute of Environmental Health Sciences (NIEHS) under the direction of the Public Health Service's National Toxicology Program. The Environmental Protection Agency (EPA) has contributed a portion of the funding.

The aim of the NIEHS/EPA studies is to assess the biological (carcinogenic) effects of asbestos fibers which are ingested, or taken into the body through the digestive tract, as in drinking water.

The studies call for asbestos to be fed continuously in the diet over the entire lifespan of the animal, which is defined as the age at which the animal begins eating solid food until its death.

A total of 1,850 male and female hamsters were fed asbestos, and an equal number of hamsters were fed a control diet with no asbestos over their lifetime of 18-23 months. Two types of asbestos fibers (chrysotile and amosite) commonly used in the manufacture of asbestos-cement products were administered.

The dose of asbestos (1% of total diet by weight) fed to the hamsters was millions of times greater than levels which occur in drinking water consumed by the public in a lifetime, in areas where asbestos occurs naturally in the drinking water.

NIEHS reports, "There was no indication of major differences in the mortality rate between the hamsters receiving the asbestos diet or the control diet." Preliminary analysis of the hamster data indicates that no carcinogenic or cocarcinogenic effect was observed.

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The analysis included thorough gross pathological evaluation at time of autopsy, as well as microscopic examination of some thirty tissues from test animals.

In addition, preliminary results of rat studies in which a total of 5,158 rats were fed asbestos and control diets were also reported. The lifetime exposure phase of the study concluded, ". . . longevity was not affected by exposure to the various types of fibers."

These findings are consistent with a number of other studies of animals fed asbestos in food or drinking water. In 1980, the Health Research Institute at Fairleigh-Dickinson University reported no malignant tumors related to treatment in hamsters maintained on drinking water containing 13 billion asbestos fibers per liter.

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ing is massive, and it has the added advantage that clotting agents may be injected.[6] If bleeding continues skilled judgment is needed, and joint consultation is essential; a balance has to be struck between early (and perhaps unnecessary) surgical intervention and prolonged and repeated resuscitation and transfusion, which may well weaken the patient's resistance.

Since most patients do not require intensive treatment they may be successfully managed satisfactorily in an ordinary ward. In spite of the evidence of Avery Jones,[7] who showed that early feeding reduced mortality, the dangerous myth persists that patients who bleed from the gut should be given "nil by mouth." There is also widespread faith in alkalis, despite the clinical observation that in a patient who has bled previous dyspeptic pain (and therefore presumably acid) disappears. The immediate and long-term effects of bleeding on gastric acidity bear re-examination. A similar uncritical attitude has grown up to the use of cimetidine: several trials[8] have shown that it is valueless in the treatment, as opposed to the prevention, of bleeding, and its "routine" use intravenously should be condemned on the grounds of both illogicality and expense. For most patients admission to hospital should mean a few days of observation rather than treatment, and if bleeding does not recur they can be safely discharged home.

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Smoking, coal, asbestos, and the lungs

Over the past 20 years the number of British coalminers with pneumoconiosis has fallen substantially. In part the fall is due to the fact that fewer men work in the pits, but it is also a response to effective measures taken to reduce dust levels underground—on the evidence of research showing the association between exposure to respirable dust and the risks of developing pneumoconiosis.[1] Some 500 men a year are still diagnosed as having the disease by the pneumoconiosis medical boards, though the average ages at which men show signs of the different stages of the disease have been increasing steadily, largely reflecting the higher dust levels of earlier years.[2] Asbestosis, the other important pneumoconiosis in Britain, continues to be diagnosed by the pneumoconiosis boards in about 200 people a year; no decline in incidence has yet been seen, but current dust-control policies in the industry are expected to produce such an effect in the near future.

In theory occupational diseases are wholly preventable, but in practice so long as society requires an industry's products some men will fall ill and even die as a result. A cost in terms of illnesses and accidents is exacted for the benefits provided by most productive industry. Society and its elected representatives need to be informed of the risks in order that work can be made as safe as possible, consistent with the need to continue or increase production[3]; but the ultimate responsibility in a democratic society for occupational morbidity and mortality rests with every individual.

Two problems that exemplify the complexity of these issues are bronchitis in the coal industry and lung cancer in the asbestos industry. Chronic bronchitis is one of the most common disabling diseases in Britain and, though mortality from it has been falling, it still ranks high as a cause of death. Its relation to cigarette smoking is well known,[4] but it is also related strikingly to the Registrar General's social groupings (which are based on occupation), to dust exposure in industry, and to area of residence,[5] implying some influence of general atmospheric pollution (at least in the past), overcrowding, and other social factors. Cigarette smoking, with its effects on both smokers and those who surround them, is also strongly related to social class.[6] In clinical practice patients disabled by chronic bronchitis who have never been smokers are extremely rare. Talk of occupational bronchitis, as though occupation were the sole cause of the potentially disabling or fatal disease in an individual, is misleading. Claims that exposure to dust does not contribute to the disease are, however, equally false, since there is good evidence in the coal industry of relations between measured dust exposure and symptoms, impairment of lung function, and mortality from chronic bronchitis.[7,8] To disentangle the relative effects of dust and cigarettes in causing the disease is difficult epidemiologically[9] and impossible in the individual exposed to both; nevertheless, both have played their part—at least in the past. Whether current levels of dust exposure in the coal industry will be sufficient to cause important clinical effects on the airways remains to be seen, but the evidence suggests that this is unlikely.

Similar arguments apply to the relation between exposure to asbestos, cigarettes, and lung cancer. Here the two causal factors seem to act in a multiplicative manner. Exposure to asbestos probably has a linear relation with the risk of developing lung cancer.[10,11] This risk is very high indeed in the heavily exposed smoker, but the risks are also substantial in a heavily exposed non-smoker. Two forms of action would, therefore, reduce the number of workers at risk of lung cancer: reduction of asbestos levels in the industry and reduction of smoking. Cutting the proportion of smokers would have more effect in workers exposed to asbestos than in the general population.

Dust control has been much improved in both industries in recent years, and further improvements will be made in the near future voluntarily and in response to public pressure or legislation. As well as reducing the risks of the specific pneumoconioses in the workers, this will also reduce the risk of chronic bronchitis in miners and lung cancer in asbestos workers. Yet these latter diseases will continue to be a problem in industry as well as in the general population so long as people continue to smoke. An important reduction in their incidence will occur only when the same public and governmental pressure is exerted on smoking as on dust control. Banning tobacco advertising would be a sensible next step.

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Noise at work

Enormous numbers of people are exposed to potentially damaging levels of noise at work. In Britain, in manufacturing industry alone about 600 000 work in noise levels above an average of 90 decibels*—noisy enough to make shouting necessary for talking to someone standing at arm's length. Over 2½ million more work in levels over 80 decibels.

The Health and Safety Commission has now[1] proposed legislation based on the 1972 voluntary code of practice[2] and the specific regulations that apply to a few industries. The central provisions are that exposure to noise must be reduced to the lowest level that is "reasonably practicable"—whether by reduction of the actual noise or, failing that, by ear protection—and that no one must be exposed to more than 90 decibels. Is this level too high?

The risks of damage after given exposures are known quite precisely, largely as a result of the work of Burns and Robinson.[3] After a lifetime's exposure to 100 decibels, the document states, 32% of people will have a hearing threshold level of 50 decibels or more (averaged over 1, 2, and 3 kHz), and with 90 and 80 decibels the proportions will be 11% and 3%. A hearing threshold of 50 decibels is the point at which the DHSS starts to pay disability benefits; but this is a considerable level of disability. A hearing level of 30 decibels or more represents impairment of the understanding of conversation even in low background noise, and is now recognised as such by the Industrial Injuries Advisory Council.[4] A careful look at figure 1 in the document shows that on this basis at least 40% of workers exposed to 90 decibels will have some handicap. Not all of this will be due to noise, but the steepness of the curve from 90 down to 80 decibels, at which some 26% are affected, points to the size of the problem. Moreover, some of those in noisy jobs, now more than in the past, have appreciable amounts of noise in their leisure hours. An estimated 10-12% of the large population of disco attenders have noisy jobs[5]; and, though these kinds of pursuits are unlikely to extend over a working lifetime, such combined exposure should not be forgotten.

The Health and Safety Commission says that it selected levels over 90 decibels for mandatory action "to ensure that the greatest effort is directed to areas of greatest need." But this would leave unprotected the far greater proportion of workers exposed to noise of 80-90 decibels, as figure 2 in the document shows: the proportions suffering damage are smaller but the absolute numbers could be large. A further point, not dealt with by the commission, is that the damaging effects of noise are not necessarily confined to hearing. Other health effects are less certain[6]; but higher blood pressures and incidences of hypertension, for example, have been reported in some though not all industrial studies, and recordings of industrial noise have produced increases in diastolic blood pressure and total peripheral resistance lasting longer than the noise. Work performance, accident rates, and behaviour may also be adversely affected, though confounding factors are hard to exclude in real-life studies; and Broadbent cites a study by G Jansen suggesting that steelworkers working in noise had more domestic disputes than others.[8]

Reducing noise and even providing personal protection, however, cost money—how much is not clear, but the notes in the background paper accompanying the consultative document suggest large sums.[9] With limited resources more spent on reducing noise could mean less for other health and safety measures. The commission maintains that few if any workers will be exposed for long to exactly 90 decibels since in practice the design target will need to be two or three decibels lower than the limit. It also emphasises the general requirement to reduce noise as far as is reasonably practicable, and adds that it "will keep the question under review, and will consider whether to propose some lower value if this seems correct in the light of future developments." The present proposal, then, could indeed be seen as "a sensible first step in legislation."

Nevertheless, a general obligation to keep noise levels as low as possible and below the statutory limit would not influence the poorer or less conscientious firms as powerfully as would a lower statutory limit; and once the figure of 90 decibels is enshrined in legislation we cannot realistically expect any early change. The document speaks of the crippling costs to industries if the regulations were very stringent and the likelihood that some would have to close. The social implications of different limits, in terms of direct and indirect costs and benefits, need to be spelt out quite fully in the coming months, so that society can make an informed choice. We need too to hear more of the "strongly held and differing views" about adoption of 90 decibels as the main action level, to which the commission refers so laconically. Its final decision to propose 90 decibels must reflect Britain's present economic circumstances and a substantially lower limit may not be feasible. But would not 85 decibels, which would nearly treble the number of workers protected by legislation, be preferable? At the very least, this could be written into the regulations as a "warning level." At all events, we hope that many of those in the health professions will send their views to the Health and Safety Commission before the end of April 1982.†

*"Decibels" refers throughout to dB (A) over an eight-hour period.

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EDITORIALS

Asbestos and public health

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Asbestos workers are known to have an increased risk of pulmonary fibrosis, pulmonary carcinoma and diffuse primary interstitial mesothelial tumours of the pleura and peritoneum. Suspicions about the role of inhaled asbestos in these conditions date back to 1906, 1935 and 1960 respectively, and these suspicions were confirmed in the 1930s, 1960s and 1970s. However, the health effects of asbestos are highly complex and only because of recent intensive epidemiologic investigation has synthesis become possible. Most of the epidemiologic evidence is in the first three references;[1-3] clinical implications are discussed by Becklake,[4] although her epidemiology is somewhat outdated. It is now known that the probability of adverse effects from inhaled asbestos depends on the total number of fibres inhaled (a function of both duration of exposure and concentration) and the type of asbestos.

Most asbestos used in the Western World is "chrysotile", which is mined mainly in Quebec. The rest is "amphibole" — essentially crocidolite and amosite — which is now mined only in South Africa. Although the mechanisms of retention and elimination of asbestos are not fully understood, on average and where exposures are similar much less chrysotile than amphibole is found in the lungs post mortem.[5] Thus, amphibole might appear to be the more potentially hazardous agent; indeed, this has been confirmed in many epidemiologic studies. The findings conform to a "fibre gradient", with crocidolite the most hazardous and chrysotile the least.

In humans one cannot measure "dose" — that is, how much dust is retained in the target organ. At best, through personal samplers one can measure fibre concentrations in the air. However, in most studies even workplace asbestos levels have not been measured. Perhaps the best epidemiologic investigation was that of a birth cohort of all 11 379 persons born between 1891 and 1920 who worked in chrysotile production in Quebec;[2] this study was crucial for the Health and Safety Commission's advisory committee on asbestos in the United Kingdom.[6] The wide range of accumulated dust exposure — that is, the summation, job by job, of dust concentrations multiplied by the number of years in the job — allowed a study of the *shape* of exposure–response relationships; that for lung cancer, in particular, was effectively linear.[2,7,8] This finding and the results from other studies have provided strong evidence against a threshold or "safe" exposure, despite the hope expressed in an earlier review[9] that was cited, but not endorsed, by Gloag.[10]

There have been so few studies in which asbestos exposure has been assessed in more refined terms than duration of exposure that it is impossible to estimate exposure–response relationships for each fibre type and each response. However, we can evaluate, using certain reasonable assumptions, the fibre gradient for certain responses. Thus, the fibre gradient is almost certainly steeper for mesothelioma than for other responses.[11,12] With lung cancer the gradient remains quite definite; in chrysotile production the risk is only mildly increased;[2,13] in chrysotile processing the risk may be higher, although the few small studies are difficult to interpret; in pure crocidolite exposure the risk is always much higher;[14-16] and working with amphibole-rich mixtures carries some of the highest risks.[17,18]

The association of asbestos exposure with gastrointestinal cancer is uncertain; some other etiologic factor may also be involved.[11] Evidence concerning laryngeal cancer is even more equivocal,[1,2,19] but this cancer is so rare that even if asbestos exposure enhanced the risk the effect on total mortality would undoubtedly be small. Diagnosis of asbestosis is far from standard, if only because no clinical signs are specific and some history of asbestos exposure is required. However, there is some support for the usual "fibre gradient", although rather shallow.[1]

Among asbestos workers cigarette smoking increases the risks of lung and laryngeal cancer but not mesothelioma or gastrointestinal cancer and perhaps not asbestosis. In Quebec the relative risk of lung cancer appeared to depend on smoking habits, the slope of the asbestos exposure–response line being steeper for nonsmokers than for smokers;[2] however, in other studies the slopes seemed to differ only slightly.[20,21] Either way it is clear that exposure to chrysotile in mining and milling at the current control limits is equivalent in carcinogenic potency to smoking about three or four cigarettes a week.

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In all asbestos-related diseases there is a long interval between the first exposure and the onset of symptoms. Today's cases are attributable to working conditions 30 to 40 years ago. Over the next decade or so more cases will undoubtedly appear, perhaps decades after cessation of exposure. However, since conditions have generally been improving since about 1950 we may have already seen the worst of the pathologic effects of asbestos, despite some questionable forecasts widely circulated in the United States.[22] Cases arising from the current levels of exposure will not be seen until well into the 21st century.

Some of "today's cases" are discussed by Finkelstein and coworkers in this issue of the Journal (pages 259 to 262). They explain that most of their 172 former workers certified as having asbestosis had first been exposed "when hygiene conditions were considerably worse than they are today"; their general findings support earlier results.[23,24] However, specific interpretation is not easy because the 20 cases in which compensation was awarded between 1942 and 1969 may well have differed from the others for the following reasons: change in the criteria of certification during the three decades; a wide range of ages at the time of award; different types of exposure, particularly to amphibole; and accepted interactions of these factors. All these factors make Finkelstein and coworkers' discussion especially difficult to generalize from, particularly as, for most of the subjects, the period of observation following the award was rather short. Furthermore, their Fig. 1 is just another way of presenting the data for "all causes" that appear in their Table III; while there is undoubtedly a "horror story" from the past it is one told twice over and is not two such stories. Nevertheless, one can only agree with the sentiment in their final remark: that hygiene must be maintained so that the future risk of asbestos-related disease is eliminated.

No control limit can guarantee absolute safety; limits for industrial exposure are usually derived from findings in workers with low exposures (by the standards of the 1950s). At a factory in Rochdale, Lancashire, England (where chrysotile was used mainly, although crocidolite was probably used substantially, even into the 1960s) no relation was found, among workers who had entered the industry after 1951, between lung cancer and asbestos exposure up to the equivalent of eight respirable fibres per millilitre of air (well above most countries' control limits) for 50 years.[25] In Quebec no excess exposure was detected in those who had worked all their lives with asbestos at concentrations below about 20 fibres per millilitre.[7] Crocidolite should be subject to much more stringent regulations, including special care in any essential processing or handling. Because amosite is an amphibole and evidence against it is accumulating, it seems sensible for it to be subject to similarly stringent regulations.

Asbestos exposure has occurred in nonoccupational settings,[1] but it is unlikely that the concentrations can have been high enough for long enough[26] to constitute a serious hazard (except in instances of household contact with asbestos workers). Nonoccupational exposure may occur around demolition sites or when certain building materials are damaged; if the materials contain amphibole strict precautionary measures must be taken during repair or replacement of building materials. On the other hand, the hazards during such procedures must not be allowed to be worse than those of leaving the asbestos in place. No ill effects have yet been demonstrated from the presence of asbestos in drinking water, food or beverages, or in the general atmosphere. Indeed, the air pollution around Thetford Mines, PQ was quite severe for many decades, but there was no evidence that excess asbestos-related mortality or morbidity in the local population could be attributed to anything other than occupational exposure.

Finally, some adequate substitutes for asbestos do not resemble asbestos; however, of the ones that do, the better they mimic the properties of asbestos the more similar the effects on health are likely to be. Indeed, in experiments in animals glass fibres with the same dimensions as crocidolite produced biologic effects at least as serious as those of the amphibole.[27] At least 30 years' exposure of a large group of workers would be required to test the effects of any substitute; even then its safety could not be demonstrated.

Decisions on control limits, on whether to repair or replace asbestos after it has been damaged and on substitutes for asbestos, must be based not on emotions stirred by the results of past exposure to clearly excessive doses of asbestos but on the best available scientific evidence.

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College of Family Physicians of Canada's position on family medicine certification

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I am hesitant to comment on the views expressed in the editorial by Conn and colleagues, of the Canadian Federation of Medical Students (CFMS), on family medicine certification,[1] given the risk of further aggravating the serious level of emotionalism that has already developed over the decision by the College of Family Physicians of Canada (CFPC) to enforce its long-standing policy of limiting practice eligibility to sit its certification examination in family medicine to family physicians who graduated prior to 1981. As one might have anticipated, the undergraduate student body as represented by the CFMS is particularly sensitive to this issue.

I would be less than responsible, however, if I permitted a number of the statements made in Conn and colleagues' editorial to go unchallenged. Since the CFPC and myself, as the college's chief executive officer, appear to be the authors' main focus for attention, I want to share with the readers of the Journal several observations, in the hope that a more balanced perspective may be provided as to what the policy of the CFPC on practice eligibility is all about.

The following claims represent the major thrust, as I interpret it, of their editorial.

Claim 1. The College of General Practice of Canada, now the College of Family Physicians of Canada, was established by the Canadian Medical Association (CMA) in 1954, and was provided with a mandate as set out in the original objectives of the college.

Response: True. The CFPC, like the Royal College of Physicians and Surgeons of Canada, the Canadian Cancer Society, the Canadian Council on Hospital Accreditation and a number of other national associations, was established by the CMA. The CFPC is proud of this heritage and continues its close association with the CMA as an affiliate member. Like other national associations established by the CMA the CFPC is an independent, autonomous body, incorporated by an act of Parliament, that is free to establish its policies within the framework of its act of incorporation.

Claim 2. Over a period of 25 years the CFPC has digressed from its original objectives, which, in the words used by Conn and colleagues, represents a "clear violation of the second proviso set out by the CMA in 1954". They provide two examples: (a) the promotion of graduate (residency) training in family medicine and (b) involvement in economics and politics. They point out in particular that the CFPC has somehow performed a disservice to Canadian medicine and to the Canadian public by creating a two-tier system — certificated and noncertificated family physicians.

Response: True and false. The CFPC takes pride in the knowledge that its policy has changed significantly since its inception 25 years ago, in response to the changing needs of family physicians and the Canadian public for the services provided by family doctors. The decision to establish residency training programs in family medicine was made by practising family physicians who, on the basis of their own experience, acknowledged that a period of undergraduate training and an internship were inadequate preparation for family practice.

The CFPC is concerned and involved with large-E economics and large-P politics within the framework of doing those things necessary to maintain an attractive practice climate for its members. This interest has stopped short of involvement in the nitty gritty of how and how much a family physician should be paid and

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Editorial

Asbestos and public health

The highly complex health effects of asbestos have been the subject of many symposia and publications over the last two decades. The Health and Safety Commission's Advisory Committee on Asbestos produced its final report[1] late in 1979; in a second volume (of commissioned papers), there is an excellent review by Acheson and Gardner of the Ill-Effects of Asbestos on Health.[2] More recently, McDonald's team has published the latest report of mortality in a very large cohort of Quebec miners and millers,[3] and the proceedings of a symposium held at the (WHO) International Agency for Research on Cancer, in Lyon, in September 1979, have now appeared.[4] At the September 1980 British Occupational Hygiene Society's Symposium on Inhaled Particles, all but one of the asbestos papers fitted and helped to fill out the pattern. The findings from the last were completely out of line with anything that has gone before; so much so that reasonable scientists must await careful evaluation before allowing this paper[5] to influence judgement based on scores of well-authenticated reports. With this single exception, matters have become sufficiently clear that reasonable synthesis seems possible. This editorial draws freely on many sources but, to keep within reasonable limits, it concentrates largely on mortality in relation to asbestos exposure, with especial reference to death from lung cancer. Morbidity is given less weight because of the greater difficulties of diagnosis and attribution, particularly bearing in mind the interactions with smoking. In view of the recent editorial,[6] mesotheliomas are reviewed only cursorily.

The bulk of the Western World's asbestos is "chrysotile," a magnesium silicate mined mainly in Quebec; the remainder consists of the so-called "amphiboles," ferrous and ferric silicates—almost entirely crocidolite and amosite—both now produced only in South Africa. Russia produces almost as much chrysotile as the rest of the world, mainly for "home" consumption, but no amphibole.

It is now thought that the dimensions of retained fibres in the lung are more important in carcinogenesis than their chemical composition, while the processes of inhalation, elimination, and retention also seem to depend on physical characteristics. It is possible that chrysotile may not penetrate deeply and that those fibres which are initially retained become susceptible to the system of natural elimination including dissolution. Amphiboles, on the other hand, may penetrate quite deeply, and their relative indestructibility seems to lead to retention more or less indefinitely. Whatever the mechanisms, it seems that, on average and where exposures are similar, considerably less chrysotile than amphibole is found in the lung post mortem.[7 8] If these beliefs are well-founded, it is clear that, compared with chrysotile, the amphiboles have substantially greater potential for causing ill effects.

Also important is that, with all asbestos-related disease, there is a delay, usually of several decades, between first exposure to respirable fibres and the onset of symptoms. Thus, today's cases are not attributable to present conditions, but to those of 40 or more years ago. Meanwhile new cases can arise several decades after cessation of exposure. It is also clear that it could take up to half a century before there would be any serious possibility of discovering whether control had been satisfactory and perhaps a like period before the appearance of a new hazard.

In no study in man is it possible to measure the dose —that is, the amount of dust reaching or better, retained in, the target organ. At best, measurements can be made of the fibre concentrations in the air close to that inspired by individual workers. Personal samplers are now in use, but in the past only assessments in the general area of the work place were possible, and even these have been available in very few studies. With such measurements, linked to work histories, it is possible to assess roughly the exposure experienced by each worker in certain periods, and this may be a reasonably satisfactory index of dose. Without such measurements, the only available surrogate is duration of exposure; this has been used in most studies, but is obviously less than satisfactory and, indeed, of dubious validity.

If only for the difficulties just mentioned, there can be no ideal epidemiological study. The best have been in occupational settings and that of Quebec chrysotile production workers[3] has many advantages over most. A large birth cohort of 10 939 males and 440 females who worked at least a month in the industry, some as early as 1904, was followed to the end of 1975; only 2% of those known to be alive in 1936 were untraced

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and there had been nearly 4500 deaths. Estimates of respirable dust concentrations, job by job, were obtained to cover the relevant periods of exposure, and smoking histories were obtained for almost all men alive in 1950. The data have been analysed by different methods,[9] and the results have been consistent.[10] The inclusion of those with very short employment led to a very wide range of accumulated dust exposure—the summation, job by job, of dust concentration multiplied by years in the job. This overcame some of the problems of selection and survival that arise when a study group is defined in terms of long employment, or employment at a particular time, as has been usual in other studies.

The advantage of having such a wide range of exposures is that it allows study of the *shape* of the exposure-response relationship; in Quebec for lung cancer it was effectively linear.[3 9 11] Results from other studies have tended to support such a relationship[12] or one that is possibly "sub-linear"—that is, steeper for short periods of exposure and more shallow for longer periods.[13] Either way, there seems no evidence for a threshold or "safe" dose. The implications of this are important.

It is difficult to make *quantitative* comparisons between the health effects of the different types of asbestos fibre. For many purposes, mixtures of amphibole and chrysotile have been found satisfactory from a commercial point of view, and there are very few large groups of workers (other than miners and millers) who have been exposed to a single fibre type. Even were single-fibre studies possible, differences in selection and management policies, problems over reference populations, and the almost certain lack of information on dust concentrations, all militate against reliability of comparison. In mixed-fibre studies, where groups of workers have been distinguished by exposure to a single fibre, the above factors should be standardised. However, the classification by fibre type may have been imperfect (or there may have been contamination), and numbers have tended to be rather small.

The findings from most studies to date appear to support the hypothesis of a "fibre gradient", such that crocidolite has much the most severe health effects, and chrysotile the least, with amosite somewhere in between. This is compatible with the beliefs expressed in the third paragraph of this editorial, and the gradient seems to exist with all health hazards. For mesothelioma, the gradient is undoubted, and is almost certainly steeper for this outcome than for any other.[14 15]

As to lung cancer, the gradient still exists although it is probably not quite so steep. Chrysotile production has yielded a comparatively mild excess.[3 16] Only a handful of small studies in chrysotile processing have been reported, and their findings are all difficult to interpret; they are not incompatible with risks higher than in mining and milling, but much less than with the amphiboles (except in reference 5). The risk of lung cancer in pure crocidolite exposure was much higher in Nottingham,[17] Eastern Canada,[18] and Western Australia,[19] and some of the highest risks were in amphibole-rich mixtures.[20 21]

Gastrointestinal cancers appear to be asbestos-associated only in certain circumstances, so that some other aetiological factor may also be involved.[14] Cancer of the larynx was clearly unrelated to asbestos exposure in Quebec[3] and in London,[22] but has been associated with asbestos, probably amphibole, in a few other studies.[2] Fortunately, the tumour is rare and even if there is an enhanced risk from asbestos exposure, the absolute effect on total mortality is undoubtedly small. Diagnosis of asbestosis in life is so difficult (because even the radiological signs are not specific and some history of asbestos exposure is required) that reliable comparisons between fibre type can only be made where the diagnoses have been made by the same team. Diagnosis of asbestosis at death is inevitably related to awareness and to compensation procedures. However, what evidence there is suggests the same gradient, if perhaps even less steep, by fibre type.[2]

Cigarette smoking is an important factor in lung cancer and cancer of the larynx, but does not seem to affect the risk of mesothelioma or of gastrointestinal cancer, and perhaps not of asbestosis. In the latest Quebec data on lung cancer,[3] the slope of the asbestos exposure-response line appeared to depend on smoking habits, being steeper for non-smokers than for definite smokers; other data seem to fit the multiplicative model better.[23 24] However, from the Quebec data it is clear, whatever the model, that today's control limits for occupational exposure to chrysotile are equivalent in carcinogenic potency to very light smoking—that is, only three or four cigarettes smoked each *week*.

Bearing in mind the lag period, it is important to consider findings in men with low exposures to chrysotile. In analysis of the Quebec data on lung cancer up to the end of 1973, it was reported[9] that no excess was detectable (at anything approaching a conventional level of statistical significance) where exposure was less than a certain amount, based on the integration over time of a concentration of respirable asbestos dust. This is equivalent to saying that (on the linear hypothesis and with a *conservative* fibre/dust conversion ratio) in a 50-year working lifetime, in concentrations below about 20 respirable fibres per millilitre of air (ie 20 f/ml), no excess could have been detected with any confidence. The proposed control limit[1] for occupational exposure to asbestos is of a

concentration of 1 f/ml, or one-twentieth that indicated in parentheses above. At Rochdale (where the fibre was mainly chrysotile but where there was probably significant use of crocidolite even into the 1960s), no relationship was found—in those who had entered the industry after 1951—between lung cancer and exposure up to the equivalent of 8 f/ml for 50 years.[25]

Other low exposures have occurred in non-occupational settings[2]; it is difficult to see how concentrations can have been severe enough for long enough for them to have been a serious hazard in the past, except where there was domestic contact with asbestos workers.

Over the next decade or longer, more cases of asbestos-related disease will undoubtedly appear—but they will be the result of working conditions 30–40 years before their appearance. However, conditions have generally been improving for at least two decades, and it is possible that we are already over the worst of the pathological effects, despite some questionable forecasts informally, but widely, circulated in the USA.[26] Should any cases arise from today's levels of exposure they will not be seen until well into the twenty-first century.

The latest UK government regulations limiting occupational exposure to chrysotile to 1 f/ml from 1981 do not, of course, guarantee absolute safety. Even if it were assumed that there had been no crocidolite at the Rochdale factory, and putting a very gloomy interpretation on the findings there, 50 years' continuous exposure to the upper limit allowed by the regulations would lead to 1·25% excess mortality from lung cancer, or about 0·125% overall excess.[2] Crocidolite is, of course, subject to much more stringent regulations—no more imports and especial care in any necessary processing or handling. As amosite is an amphibole, and as evidence against it is accumulating, it would seem sensible to treat it on the same lines as crocidolite. One reason is that chrystotile can, perhaps with some ingenuity, often be used instead, although there will remain specific problems such as the production of large-diameter pressure pipes.

Non-occupational exposures to asbestos may continue because of demolition or damage to certain building materials, particularly those used for insulation where the materials often contained amphiboles. There is no doubt that any exposure to respirable asbestos is to be avoided wherever possible, and strict precautionary measures during repair or replacement are clearly indicated. Nevertheless, great care must be exercised that the hazards during such operations are no worse than those arising "naturally"—that is, from leaving the asbestos in situ. It is also important to note that the *peak* figure identified in a survey[27] of buildings containing asbestos materials in the UK was only 0·08 f/ml (one-twelfth of the proposed control limit for occupational exposure); however, it is agreed that more information is required "about asbestos levels in new and old buildings in relation to type and usage of asbestos-containing materials, particularly insulation materials."[2] No ill effects have yet been demonstrated of asbestos in drinking water, in food and beverages or in the general atmosphere. Indeed, the pollution of the air around Thetford Mines, by far the dustier of the two mining areas in Quebec, was for many decades quite severe, but there was no evidence that excess asbestos-related mortality or morbidity in the general population could not be attributed to occupational exposure.

A final point must be made about substitutes for asbestos. Some adequate substitutes exist which do not resemble asbestos; where they do, it should be borne in mind that the better a substitute mimics the physical and chemical properties of asbestos the closer the ill effects on health are likely to be. Indeed, there is animal evidence[28] that glass fibre of the same dimensions as crocidolite has biological effects at least as serious as those of the amphibole. It must also be emphasised that at least 30 years' exposure of a large group of workers would be required to "test" any substitute, and even then it would not be possible to demonstrate that it was "safe."

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