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Review Article

Apportionment in Asbestos-Related Disease for Purposes of Compensation

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Abstract: Workers' compensation systems attempt to evaluate claims for occupational disease on an individual basis using the best guidelines available to them. This may be difficult when there is more than one risk factor associated with the outcome, such as asbestos and cigarette smoking, and the occupational exposures is not clearly responsible for the disease. Apportionment is an approach that involves an assessment of the relative contribution of work-related exposures to the risk of the disease or to the final impairment that arises for the disease. This article discusses the concept of apportionment and applies it to asbestos-associated disease. Lung cancer is not subject to a simple tradeoff between asbestos exposure and smoking because of the powerful biological interaction between the two exposures. Among nonsmokers, lung cancer is sufficiently rare that an association with asbestos can be assumed if exposure has occurred. Available data suggest that asbestos exposure almost invariably contributes to risk among smokers to the extent that a relationship to work can be presumed. Thus, comparisons of magnitude of risk between smokers and nonsmokers are irrelevant for this purpose. Indicators of sufficient exposure to cause lung cancer are useful for purposes of establishing eligibility and screening claims. These may include a chest film classified by the ILO system as 1/0 or greater (although 0/1 does not rule out an association) or a history of exposure roughly equal to or greater than 40 fibres/cm^3·y. (In Germany, 25 fibres/cm^3·y is used.) The mere presence of pleural plaques is not sufficient. Mesothelioma is almost always associated with asbestos exposure and the association should be considered presumed until proven otherwise in the individual case. These are situations in which only risk of a disease is apportioned because the impairment would be the same given the disease whatever the cause. Asbestosis, if the diagnosis is correct, is by definition an occupational disease unless there is some source of massive environmental exposure; it is always presumed to be work-related unless proven otherwise. Chronic obstructive airways disease (COAD) accompanies asbestosis but may also occur in the context of minimal parenchymal fibrosis and may contribute to accelerated loss of pulmonary function. In some patients, particularly those with smoking-induced emphysema, this may contribute significantly to functional impairment. An exposure history of 10 fibre·years is suggested as the minimum associated with a demonstrable effect on impairment, given available data. Equity issues associated with apportionment include the different criteria that must be applied to different disorders for apportionment to work, the management of future risk (eg. risk of lung cancer for those who have asbestosis), and the narrow range in which apportionment is really useful in asbestos-associated disorders. Apportionment,

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attractive as it may be as an approach to the adjudication of asbestos-related disease, is difficult to apply in practice. Even so, these models may serve as a general guide to the assessment of asbestos-related disease outcomes for purposes of compensation.

Key words: Asbestos, Workers' compensation, Apportionment, Epidemiology, Lung cancer, Mesothelioma, Chronic obstructive pulmonary disease, Asbestosis, Pleural plaques, Equity, Exposure assessment, Occupational history

Introduction

Asbestos may cause a variety of health outcomes. Some of these are characteristic but not specific, some are highly specific but uncommon, and some are nonspecific and difficult to attribute[1–3]. Much is known about these conditions, but this knowledge is derived mostly from population studies.

Workers' compensation systems provide insurance for the medical costs of treatment and diagnosis and replace lost income associated with disability resulting from the functional impairment caused by occupational disease. Workers' compensation deals exclusively with disorders arising from occupation or significantly aggravated or contributed to by workplace exposure. Discriminating between occupational and non-occupational causes of disease is fundamental to proper adjudication. It is also necessary in fairness to the interests of employers who fund the system and cannot be held responsible for disorders arising from personal lifestyle, behaviour, or causes unrelated to the workplace. Bringing evaluation down to the individual case is often an ambiguous and uncertain undertaking. However, individual evaluation is essential to the fair adjudication of such cases under workers' compensation. *Apportionment*, which is the estimate of the contribution of a particular cause to the outcome in an individual case, may be a part of this individualized approach[4, 5].

The number of cases attributed to a particular cause in a population is called the *attributable risk* by epidemiologists. The fraction of cases attributed to the cause is called the *attributable fraction*. Attribution, using either measure, is an important public health indicator and may inform the interpretation of workers' compensation claims. However, assignment of attributable risk is an epidemological concept and does not apply to the individual case. Apportionment must be understood always to apply to the individual. For the individual, the attributable fraction is a best estimate only. The fundamental issues of apportionment have been discussed in detail elsewhere[4, 5]. This article will explore the apportionment of cause in asbestos-related diseases where other putative risk factors, such as cigarette smoking, may be present.

Apportionment in Principle

In almost all Canadian jurisdictions, workers' compensation boards are required to accept claims in their totality if a substantial component of the disease is work related. However, defining what constitutes a substantial, significant, or minimal component is often difficult. A possible alternative approach is apportionment, which some boards have already used on a relatively informal basis to allocate responsibility for claims.

Workers' compensation boards in all jurisdictions are faced with an expanding challenge in the management of claims related to occupational disease. Questions of causation, the presence of multiple risk factors, and modifications of the characteristic presentation of occupational diseases greatly complicate adjudication.

Asbestos-related diseases are particularly problematical in this regard and illustrate these problems well. Among these fundamental issues is the relative contribution of different causes, such as cigarette smoking or asbestos exposure, to the risk of a disease such as lung cancer or to overall impairment from on outcome, such as chronic obstructive airways disease. It is generally easier to distinguish occupational from nonoccupational disease when characteristic outcomes are specific to the exposure, as occurs with pneumoconioses such as asbestosis or when the association is so great that a presumption is reasonable, as in mesothelioma. However, when the outcomes are not specific, and especially when they may also be caused by other common environmental exposures such as cigarette smoking, defining causation can be problematic.

Causation may be reduced, in most cases, to a proposition of "but for", a term commonly used in law. If "but for" exposure to the hazard, the condition would probably not have occurred, the hazard can be considered to be the cause. Another way of saying this is that the cause was necessary, even if it was not sufficient. Applied to asbestos-related

disease, assessing that the possible causes include asbestos exposure at a level that may have substantially contributed to disease is the first step. The second would be to assess the relative contribution of asbestos compared to other causes, the step called apportionment.

Apportionment by cause

The process of adjudicating workers' compensation claims involves a differentiation between occupational and nonoccupational causes of disease and injury. Though in practice this can be exceedingly difficult, and in some cases impossible, the requirement to consider causation is fundamental to the philosophy of workers' compensation. That is because workers' compensation systems are mandated to resolve individual claims on the best evidence, not to generalize to groups or classes.

Faced with a large number of difficult occupational disease cases, workers' compensation agencies have considered apportionment by cause. Apportionment by cause is the estimation in an individual case of the relative contribution to an outcome, such as a multi-factorial disease, of several risk factors or potential causal exposures that are present in the case and that are known to be associated with the outcome. Apportionment by cause is a way of apportioning responsibility and contribution to the final outcome. In workers' compensation, it principally applies to apportioning causation between occupational and non-occupational risk factors.

There are other ways to apportion. Apportionment of impairment and disability, for example, is common in multiple injury cases. In the tort system, the equivalent concept is apportionment of harm (meaning responsibility for causing harm) but because workers' compensation is a no-fault insurance system the assignment of blame or responsibility is not so useful.

Apportionment by cause must be performed on the individual case. Individuals may vary in their characteristics from the population as a whole. Often, apportionment cannot be determined with certainty and epidemiological data may then be used to derive an estimate of the relative contribution of a risk factor in an individual claim. However, this must be understood to be a derived estimate, not to be confused with attribution, which uses the population attributable fraction, or the apportionment of impairment or its social derivative, disability, which can be done by specific measurement in the individual case.

The benefits of fair and accurate apportionment are obvious: adjudication may be simpler, adjudication may be fairer to employers and some injured workers and financial resources would be conserved for workers with greater impairment. Workers might be encouraged to take responsibility for their own health, fiscal exposure would be more fairly shared among health care funding agencies and the relative contribution to disability benefits for permanent impairment could be divided among payers, such as provincial health care plans, Social Security or Canada Pension, and workers compensation. Although apportionment is an attractive option for adjudication in compensation, it has many drawbacks and uncertainties. These are explored in detail elsewhere[4, 5].

For apportionment to work in practice, two related concepts must be introduced: presumption and substantial contribution.

Presumption

A presumption exists when a worker with a compatible exposure history develops a particular disease and the condition is assumed to be related to the exposure. The principle of presumption requires that the disorder be sufficiently common among workers with that exposure that in any given case it is more likely than not that the disorder is work-related. The logic of presumption requires that a risk attributed to exposure in an exposed population must equal or exceed double that of people without exposure, because a relative risk of two corresponds to even odds which corresponds to the legal requirement of "more likely than not", all other things being equal. A rebuttable presumption is one that can be challenged on the particulars of the case, for example when the claimant or plaintiff had not accumulated sufficient exposure to expect a substantial contribution.

Substantial contribution is, simply, the requirement that a claimant have been exposed to a sufficient quantity, concentration or duration of exposure of the hazard, in this case asbestos, to cause at least a minimal injury that could contribute to the outcome. This is not quite the same as a threshold because a threshold may be defined in various ways. As a practical matter, the purpose of the requirement for a substantial contribution is to reduce the number of claims without real merit and to increase the likelihood that those claims remaining are associated with work-related exposures and are therefore apportionable.

One may propose the following essential criteria for a definition of substantial contribution:

- The contribution to the outcome (regardless of the subsequent impairment) should be demonstrable in some way or inferred from population data; a history of nominal exposure or the presence of a marker that does not correlate with risk is not enough.

- The contribution should be on the same order of and significant relative to natural individual variation and the loss of function in progression of disease.
- For example, if the normal adult change in FEV_1 is –30 ± 7 ml/year and –60 ± 10 ml/y is associated with chronic obstructive pulmonary disease by age 60, an additional incremental loss of 10 ml/y due to an occupational exposure would clearly be significant (representing one-third of the contribution leading to pathology) but 5 ml/y would not so clearly be significant, because it falls within the range of measurement error and normal variation. In practice, the "noise" in measurement and lack of baseline measurements may make this difficult to apply.
- In cases where impairment results from loss of function due to the disease outcome, the proportion of impairment contributed by the cause in question should be enough to change the prognosis or clinical course; in other words, enough to make a difference in a borderline case.
- Whatever the contribution to the outcome, it should plausibly relate to the permanent impairment; in other words, if the presence of a pleural plaque does not predict airflow obstruction, demonstration of a pleural plaque cannot be used to suggest a substantial contribution of asbestos to causing airflow obstruction, notwithstanding their association with a restrictive component of reduced ventilatory capacity[6].

One approach to defining substantial contribution is to identify a level of exposure commonly associated with definite functional changes that may be of significance in the progression of disease. In the real world of workers' compensation, detailed exposure information over the lifetime of the worker is simply not available. More robust approximations are needed. In practice, this may mean resorting to general or approximate categories.

When there is a possibility of error, workers' compensation policy is almost always to give the benefit of the doubt to the worker. Usually this is written into the legislation creating the workers' compensation system. Estimates of substantial contribution should therefore be set at a level that will include all or almost all claimants who are likely to be affected by their exposure. The tradeoff is to be less efficient to exclude as many as possible of claimants who are not likely to have been affected, erring on the side of inclusion.

Asbestos-Related Disease

Occupational disease claims, including asbestos-related cases, tend to be complicated and less certain than occupational injuries. Asbestos-related claims may be more amenable to adjudication than occupational asthma but remain open to interpretation and subject to assumptions that are difficult to prove. In a detailed study of the handling of claims by Washington state in the period 1982–1986[7] for a high-risk population in which occupational disease had been diagnosed at a university-affiliated clinic, only half of claims in the state system were accepted and there were suggestions of bias in the adjudication against nonwhite claimants and by adjudication system. Criteria for acceptance were inconsistent among systems and within the state system; there was no or unexpectedly low correlation between claim acceptance and chest film (ILO category), presence of restrictive changes, smoking status, or concurrent obstructive lung disease. Other, older studies have shown similar findings (cited in 7).

More recent studies suggest that in British Columbia and possibly Australia only about 10% of asbestos-related lung cancer cases have been recognized and compensated appropriately[8]. A high mortality from potentially asbestos-related disease, including asbestosis, has been reported among workers potentially eligible for compensation in Ontario. These workers also often did not file claims[9]. The problem appears to be not one of acceptance but of the claims not having been filed in the first place.

Chrysotile

In this discussion, no distinction will be made between chrysotile and amphibole forms of asbestos, except as noted. Although there are apparent differences with respect to potency for different outcomes, some risk is present for all forms and these differences play little role in apportionment[2, 10].

Chrysotile has been the leading form of asbestos used for industrial insulation in the Americas and the UK and the experience reflected in epidemiological studies of end-users, such as insulators, reflects predominantly chrysotile exposure. Insulation is the source of exposure of greatest concern in Japan, as elsewhere. Most of the asbestos on which the earlier insulators studies were conducted were also associated with chrysotile exposure, mostly from Quebec. Some of the highest risk estimates reported in the asbestos industry (e.g. the South Carolina textile plant) were in fact associated with chrysotile exposure (without obvious contamination by tremolite)[11, 12]. The conclusion is inescapable: chrysotile is itself a cancer hazard[10].

The data on chrysotile-associated risk among Quebec asbestos miners is irrelevant. It is true that many of the studies used to calculate risk estimates for exposure to chrysotile reflect the exposure of miners and mining

communities. However, miners consistently show less risk than would be predicted based on the experience of end-users, such as insulators. This is so consistent that it is now generally accepted that the experience of miners is a poor guide to the assessment of risk, probably because fibre size and degradation to fibrils is less advanced in mining and refining and further advanced in manufacturing and application of insulation. Although chrysotile may be less potent than other forms of asbestos for most outcomes, it is still hazardous and responsible for the observed health effects[11, 13].

Chrysotile and amphiboles

Chrysotile has been contaminated with amphibole forms of asbestos, especially with tremolite, in the past. Some investigators believe that the small residual amphibole content of chrysotile asbestos is responsible for the cancer risk associated with chrysotile-exposed workers. Even if this were true, the outcome would still be work-related and therefore compensable. The end users described above, especially insulation workers, generally used products in which amphibole contamination was not likely to be a major factor. The entire issue is therefore irrelevant for purposes of compensation management[13].

Bronchogenic Carcinoma

Lung cancer is the most difficult problem in apportionment problem among asbestos-related diseases[14]. There are many causes of lung cancer, many of them occupational, and one major lifestyle cause, cigarette smoking. Apportioning between occupational and nonoccupational causes of lung cancer in a worker exposed to asbestos, therefore, is almost always an issue of ruling out the significance of other occupational exposures and then estimating the most likely contribution of asbestos against that of cigarette smoking.

Smoking and asbestos exposure

Complicating matters is the fact that there is a positive interaction between asbestos exposure and smoking in conferring risk of lung cancer. In the classic studies conducted on insulation workers and other groups in the 1970's, it was observed that asbestos exposure alone conferred a risk of lung cancer approximately 5 times the baseline risk of a nonsmoking person not exposed to asbestos. Cigarette smoking alone conferred a risk approximately 10 to 15 times that of the baseline. However, the combination of work-related asbestos exposure and cigarette smoking was associated with a risk of 50 to 100 times the baseline, far greater than if both risks were simply added, and roughly what one might expect if they were multiplied, and provides a classic example of multiplicative (synergistic) interaction.

This interaction reflects an underlying biological mechanism. This mechanism clearly acts to amplify the effects of the exposure to asbestos to greatly enhance the risk following combined exposure and does so in a non-linear fashion. This means that it is not possible to trade off the effects of asbestos and smoking as if their contributions were additive, or linear. Because the risks of lung cancer are nonlinear, simple regressions or calculations of relative risk associated with a given level of asbestos exposure and a given smoking history cannot resolve the problem. A much more complicated interactive regression, or curvilinear function, would be required to estimate the contribution of each factor. In practice, an attempt to apply such a complicated formula based on statistical patterns in a large population, with large variance, would appear arbitrary in the case of an individual and would be open to challenge based on the characteristics of the individual claimant.

One problem in dealing with this interaction is that past studies of lung cancer among smoking asbestos-exposed workers were based on much higher asbestos exposure levels than occur today, and were documented in populations with a generally higher prevalence and intensity of smoking than occurs today. (They also did not break down this observed interaction by age group, which would be helpful in thinking about apportionment.) The old rules of thumb may no longer apply in an era when asbestos exposure is far less, with concomitant reduction in cigarette smoking. As the magnitude of each exposure is reduced, it is likely that the interaction becomes less as well, because it too is likely to be exposure-dependent. Thus, one must conclude that although the apportionment by cause of a lung cancer to asbestos or cigarette smoking is not a simple linear tradeoff, it is probably no longer a tradeoff between steeply exponential curves either. Paradoxically, this reduces the influence of cigarette smoking as the dominant factor in the equation and makes it easier to conceptualize a tradeoff between the two factors.

At first, it might seem that because cigarette smoking accounts for most of the risk for developing lung cancer, the odds that a cancer was caused by cigarette smoking in a person who smoked but was not exposed to asbestos was 10 to 1. Applied as an estimate of apportionment in someone who only smokes, this results in 90% apportionment by cause. This leads to a clearly justified presumption that in all cases of comparable smoking history a lung cancer would have been caused by the cigarette smoking. Correspondingly,

the odds that a cancer was caused by asbestos in a person who was exposed but did not smoke would be 5 to 1, clearly justifying the presumption in a nonsmoker. If the tradeoff were linear, it might be tempting to compare the tenfold risk against the fivefold risk and to conclude that cigarette smoking was twice as important a factor, for odds of 2 to 1.

However, this is not logical in the context of workers' compensation. It does not take into account the interaction or modification of risk between cigarette smoking and asbestos. Because employers, or government regulations, did not or could not ban smoking among their employees, both on and off the job, as a condition of employment, they must "take the worker as they come". The preferred analysis would be to observe that risk is excessive *among smokers*. This is the only relevant comparison if one "takes the worker as he (she) comes" and applies the "thin skull" rule, that unusual susceptibility in the injured party does not absolve the tortfeasor of liability (In workers' compensation, of course, the employer is not held liable. The principle merely shifts the burden of liability to the system to accept the claim.).

The rules of rebuttable presumption remain useful in this application. The evidence suggests that in the majority of cases, the risk of lung cancer in an asbestos-exposed smoker is more than double that of a smoker not exposed to asbestos. If so, then *among smokers* it is more likely than not that "but for" the asbestos exposure the exposed worker would not have developed the cancer. This applies the usual legal test for causation. The odds that a cancer was associated with asbestos exposure in a cigarette smoker compared to a nonexposed cigarette smoker would then be around 5 or 10 to 1. This is more than enough to justify a presumption that in any smoker exposed to asbestos, the cancer in question was due to the asbestos exposure.

The fact of smoking increases risk for the worker but it also increases the potential effect of asbestos exposure. "But for" the asbestos the probability of the individual smoker developing the lung cancer would have been much less. Not even a positive interaction between asbestos exposure and cigarette smoking is required to justify a presumption on this basis, as long as the combined risk is at least double that of cigarette smoking alone.

Given this analysis, it is clear that in either smokers or nonsmokers, the occurrence of bronchogenic carcinoma in a worker exposed to asbestos at a substantial level should be apportioned 100% to the asbestos exposure. The issue of apportionment in lung cancer should therefore become a rebuttable presumption.

"Substantial contribution" in lung cancer

An index of exposure is required to separate claims for lung cancer that may have an association with asbestos exposure from those that probably do not. This derivation applies only to risk of lung cancer and is consistent with levels used for purposes of settlement in a class-action suit in the United States.

We have previously applied[15] a quantitative risk assessment of exposure to airborne asbestos in an office building, based on a simple mathematical model developed by Hughes and Weill[16]. This model is consistent with that used for asbestos-related claims adjudication by the Central Claims Facility (CCF) in the U.S. We now have adapted this model with a slightly different derivation and have adjusted assumptions to conform to the group of asbestos workers showing the highest risk for lung cancer (asbestos textile workers). These are very conservative assumptions, meaning that no asbestos worker who develops lung cancer as a result of asbestos exposure is likely to be omitted but that some who develop lung cancer unrelated to asbestos exposure will be accepted.

The derivation is as follows:

O = observed cases, E = expected number of cases,

SMR = standardized mortality ratio ($O/E \times 100$, equivalent to relative risk expressed as a percentage),

B = slope of the linear extrapolation of the incidence curve related risk of lung cancer to cumulative asbestos exposure expressed in fibres per cubic centimeter per year (this is adapted from Hughes and Weill[16] and equals 'b/100' in this equation. We used b/100 because it was more logical and to separate out 'd';

d = total cumulative dose (in terms of fibres/$cm^3 \times$ years, the terms *presumably* convertible to fibre-years if ventilatory volume and clearance could be accounted for).

The derivation of a reasonable "threshold" exposure for substantial risk is governed by the equation of Hughes and Weill[16]:

$$\text{Excess deaths} = O - E = EBd$$

For purposes of legal criteria, we are interested in the risk level at which it is "more likely than not", giving benefit of doubt to claimant, that a lung cancer is associated with asbestos exposure. This risk level compounds to even odds, a relative risk of 2.0, a relative attributable risk of 1.0, and an SMR = 200.

Therefore: $0 - E = 2E - E = E = EBd$

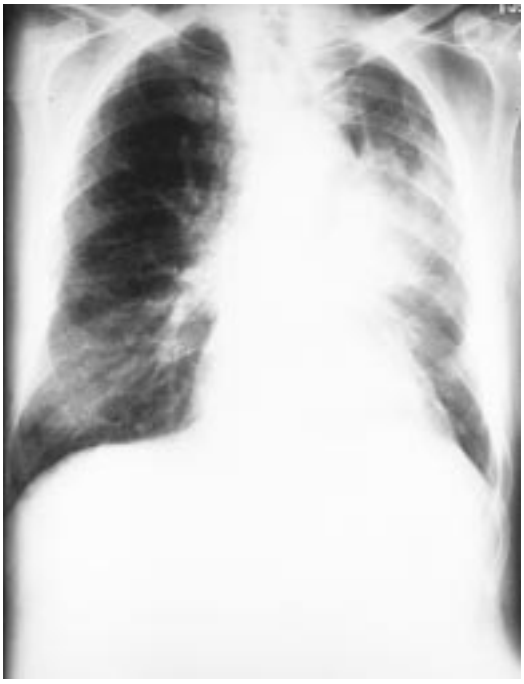

Fig. 1. Bronchogenic carcinoma in an asbestos cement pipe worker, against a background of asbestosis.

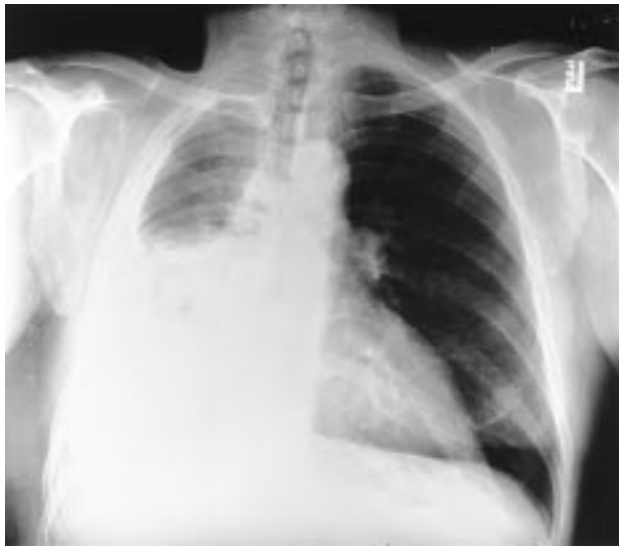

Fig. 2. Mesothelioma in another asbestos cement pipe worker, with no radiographic signs of asbestosis.

The value of 'B' is taken from Fig. 1 of Hughes and Weill[16], B = 0.025 (in inverse units of f/cm^3·y) and from the *highest* risk group (textile workers):

$$d = 40 \text{ f/cm}^3\cdot\text{y}$$

This means that any combination of fibre exposure and duration of employment that yields this rate for 'd' will correspond to a legal definition of "more likely than not" + benefit of doubt.

Translated into terms of duration of employment, this means:

- 8 years at 5 f/cm^3·yr, consistent with CCF high risk group
- 10 years at 4 f/cm^3·yr, consistent with CCF intermediate group
- 15 years at 2.7 f/cm^3·yr, consistent with CCF low risk group.

If an individual shows a mixed employment history, moving among occupations in different risk categories, one may apply a very simple weighting system as follows:

- high risk occupations: count 1.25 years of eligibility for every year of employment
- intermediate risk occupations: count 1.00 years of eligibility for every year of employment
- low risk occupations: count 0.67 years of eligibility for every year of employment

Although occupational histories may be only approximate in reflecting level of exposure, recent studies suggest on acceptable correlation for this type of classificiation[17].

This set of criteria is actually relatively conservative compared to other jurisdictions. The German "Berufgenoßenschaften" (workers' compensation panels) have recently adopted a threshold of 25 f/cm^3·y for accepting claims in that country (Information supplied by the International Labour Organisation.). This is a widely accepted "threshold" estimate (not a true toxicological threshold) originally proposed by the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario[18].

Clinical markers of substantial contribution

Although this article is primarily concerned with apportionment, the issue of causation in asbestos-associated lung cancer requires further attention. Fundamentally, this is a problem of identifying markers of effect that suggest that the claimant was exposed at a level that makes a substantial contribution to risk. As a practical matter, the markers of greatest interest have been radiological, the early identification of fibrosis and the role of pleural plaques.

For many years there has been a dispute over whether asbestos-associated lung cancer can occur in the absence of interstitial fibrosis and early asbestosis. This has resulted in a great deal of confusion[19, 20]. However Churg and Green[18] have argued persuasively that fibrosis is a necessary concomitant of asbestos-related bronchogenic cancer risk.

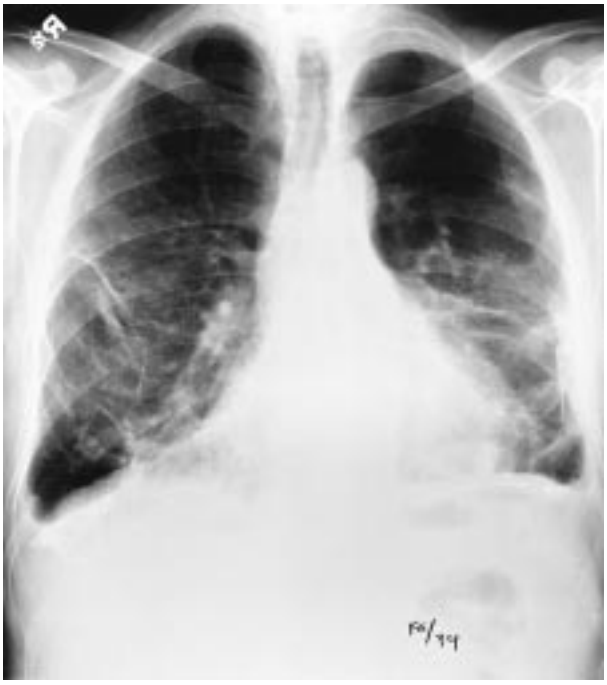

Fig. 3. Chest film showing classical features of asbestosis: irregular opacities, fibrotic bands, interlobar fibrosis, blunted costo-phrenic angles, diaphragmatic tenting and plaques, pleural plaques, shaggy heart border, mediastinal displacement and parenchymal nodule.

The clinical and medicolegal issue is how much fibrosis is required for risk to be demonstrated and can this level of fibrosis be detected by routine clinical tests[21].

Recently, a major paper by Weill[22], following up on earlier findings by Hughes and Weill[23], suggested that among asbestos cement workers who had 20 or more years of experience, only those with category 1/0 disease or greater involving small irregular opacities on their chest film (by the ILO classification of the pneumoconioses) were at risk of lung cancer. This article was widely interpreted as suggesting that some degree of early asbestosis was necessary to conclude that the degree of asbestos exposure was sufficient to be associated with an excess risk of lung cancer.

However, this is a flawed interpretation. Category 1/0 is not clear evidence of disease and is just over the boundary from a nominally normal film. There is no "bright line" boundary between 0/1 and 1/0, only an interpretation of profusion that differs in degree. Lung content of asbestos fibres shows a continuous trend from low levels at 0/0 progressing through 0/1 and 1/0 to 1/1, not a clear threshold. Since there is no threshold for asbestos exposure and risk of lung cancer, one would not expect an arbitrary threshold for risk associated with category 1/0 profusion. Finally, Weill did not explain how workers who had gone that long exposed to asbestos without developing 1/0 profusion may have differed from those who did; it may be possible to develop up to a 0/1 film on the basis of cigarette smoking alone and cigarette smoking accelerates the appearance of opacities among asbestos-exposed workers[24, 25]. For all these reasons, this study is not definitive in suggesting that changes compatible with interstitial fibrosis are necessary to accept a lung cancer as asbestos-related, although it has been so interpreted (Weill's major point in the paper was actually that the mechanism of lung cancer is associated with the alveolitis that occurs as the first pathological event in asbestosis.).

An equally careful study by Wilkinson *et al.*[26] demonstrated that asbestos-exposed workers with category 0/1 or 0/0 (normal) films had an increased risk of lung cancer compared to workers who had no history of asbestos exposure, regardless of film category. The risk was less than that of asbestos-exposed workers with 1/0 changes, with odds ratios of 1.56 and 2.03, respectively. In their data, the association was clearly present, it was statistically significant, and it was dose-dependent, with the chest film category presumably crudely indicating dose.

One reasonable interpretation of Wilkinson *et al.*[26] is that it supports the idea that a chest film of 1/0 or greater is needed for the presumption of lung cancer as asbestos-related but that chest films at 0/1 do not exclude asbestos as a cause. Chest films classified as 0/0 suggest that an association between lung cancer and asbestos exposure is less likely but cannot rule out such an association.

Histological studies tend to confirm this interpretation; in a significant proportion of cases of lung cancer in asbestos-exposed workers, parenchymal fibrosis is not visible on the chest film[27]. Histological or microscopic interstitial fibrosis also may not be a necessary concomitant of asbestos-related lung cancer. Individual studies have suggested that asbestos-related bronchogenic carcinoma is "almost always" associated with histological asbestosis but have also demonstrated a relationship between degree of fibrosis and risk that is compatible with an excess risk at lower levels of fibrosis, below 1/0[19, 28]. Egilman and Reinert[16] reviewed the available evidence for an association between fibrosis at the tissue level and lung cancer (as they did for a clinical or radiographic correlation) and concluded that although several different studies used rather different approaches and methods, they were consistent in suggesting that there was only a statistical association reflecting the history of asbestos exposure. They concluded that although workers exposed to asbestos were more likely to have fibrosis at the

time of resection or death from lung cancer, many asbestos-exposed workers with lung cancer did not have microscopic fibrosis, occasionally despite greatly elevated fibre burdens. They suggest that the alveolitis that results in fibrosis and that probably predisposes to lung cancer is not invariable and that epithelial metaplasia and proliferative fibrosis do not necessarily occur together or stepwise in progression, although both may be caused by asbestos fibres.

Egilman and Reinert[19] do not address the issue of whether the cases in which this association does not occur at necropsy might just represent "background" lung cancers not associated with asbestos. However, they cite individual studies that suggest that this is not the case. On a group basis these cancers were more frequent and more likely to be distributed in the lung in areas likely to be affected by asbestos (for example, in the lower lobes) compared to persons who were not exposed to asbestos[6]. If histologically demonstrable asbestosis is not associated with lung cancer, then advanced methods for detecting early asbestosis[29] such as HRCT[30] would not be useful either in ruling out an association with asbestos either but are valid markers of past asbestos exposure.

Pleural plaques are also not satisfactory predictors of asbestos-related lung cancer. Weiss[31] has critically reviewed this literature and has pointed out the methodological limitations in all extant studies. However, for the purposes of apportionment a more useful question is whether workers who develop lung cancer are more likely to have pleural plaques than asbestos-exposed workers who did not develop cancer. Unpublished data from Hughes cited by Weiss[31] describes an odds ratio of 1, suggesting that the presence of pleural plaques cannot be used as a marker to associate lung cancer causally with asbestos exposure. Subsequent studies[32] and a more recent review[33] have not changed this conclusion.

It is often difficult to demonstrate asbestos fibres in cases of lung cancer, even with a clear history of exposure to asbestos[27]. For this and other reasons related to under-recognition, British Columbia investigators[8] have concluded that asbestos-related lung cancer is substantially underrecognized in both Canada and Australia and that as many as 90% of cases may be missed.

The most reasonable conclusion with respect to apportionment among cases of lung cancer in asbestos-exposed workers appears to be to treat the association as a rebuttable presumption. If there is a confirmed history of exposure to asbestos, neither pleural plaques nor parenchymal fibrosis is required to demonstrate sufficient exposure. If the British Columbia investigators are correct, fewer cases will be misclassified by a presumption than by rigorously enforcing the requirement for objective evidence of an asbestos-related effect. Obviously, that policy would require acceptance of many more claims, raising the question of setting limits.

Mesothelioma

The most dread outcome of asbestos exposure is mesothelioma, a cancer with a poor prognosis and an almost invariable association with asbestos exposure. Mesothelioma in the presence of a history of asbestos exposure must be presumed to have been caused by asbestos. Chrysotile asbestos is generally considered less likely to induce mesothelioma than amphibole forms[34]. In practice, even a history of exposure to chrysotile alone does not rule out an association because of contamination or concomitant use of amphiboles. Cigarette smoking does not increase the risk of mesothelioma and there is no evidence that it modifies the clinical course or progression of the cancer.

Thus, any impairment associated with the cancer, including pain, chest wall mechanical problems, respiratory insufficiency, and disabling symptoms, are apportioned entirely to asbestos. Given the poor prognosis for recovery, the subjective symptoms that will accompany progressive impairment, and the conversion of these realities into reduced capacity to work and to disability, it is only reasonable to apportion both cause and impairment to the asbestos as soon as the symptoms or signs of mesothelioma become manifest. Both the original impairment and the prognosis for permanent impairment are soon determined by the tumour, and the cause of the mesothelioma can be presumed in almost all cases to be the asbestos exposure.

Asbestosis

Asbestosis is the characteristic pneumoconiosis associated with inhalation of asbestos fibres. The term should never be used generically to refer to asbestos-related disorders, as this leads to unnecessary confusion[36].

Like all pneumoconioses, asbestosis as a process consists of the direct effect of the dust, and also of the effect on the lung of the reaction to its presence. In asbestosis the pulmonary response is exuberant fibrosis, occurring in parenchyma (alveolar region) of the lung, initially adjacent to the airways in response to an alveolitis, or inflammation of the airspaces. Early asbestosis resembles the disease known as usual interstitial pneumonia (UIP), a synonym for fibrosing alveolitis and idiopathic pulmonary fibrosis. Indeed, there is a hereditary form of UIP that may conceivably

place some workers at risk for fibrotic lung diseases such as asbestosis, but this has not been adequately studied.

Characteristic of both early asbestosis and UIP is the presence of an inflammatory reaction that can be measured by bronchoalveolar lavage (BAL), in which cells and secretions from the deep lung are obtained by bronchoscopy. With advancing disease the fibrosis becomes more extensive, and is more likely to be associated with other asbestos-related changes in the thorax. The diagnosis of asbestosis is usually made on the chest film, but computerized tomography (CT) and high-resolution computerized tomography (HRCT) are increasingly used to establish the diagnosis[36]. Both are more sensitive than conventional chest radiography in identifying interstitial fibrosis[1].

The final common pathway for both asbestosis and UIP, and for a variety of other pneumoconioses, is a coarse pattern of parenchymal fibrosis called honeycombing. Asbestosis is characterized by the presence of asbestos fibres and asbestos bodies, which distinguishes the condition from UIP and other fibrogenic pneumoconioses. Asbestos bodies are much easier to see, but are much less common than asbestos fibres. New cases of asbestosis in recent years have usually not been so severe as in the past, when honeycombing and fibrous bands were common in advanced asbestosis cases. Fibres from tissue recovered at autopsy or biopsy were sometimes difficult to visualize because of the mass of scarred tissue, but total fibre counts from ashed tissue were very high in such cases[20].

The fibrosis associated with asbestosis rarely occurs in complete isolation. More commonly it is associated with a variety of asbestos-related changes in the thorax that are more or less characteristic of asbestosis as a disease and are seen only rarely in other conditions. These include:

- pleural fibrosis with diffuse and circumscribed plaques, especially on the diaphragm;
- progressive loss of definition of other structures in the thorax, especially the heart; bullae (large thin-walled holes in the lung);
- asbestos-associated cancers (often difficult to see by chest film in the fibrotic lung); and
- distortion of organs in the mediastinum.

These secondary changes are now uncommon because exposure levels are substantially lower, and are unlikely to produce such extreme manifestations of disease.

The process of fibrosis in asbestosis is relatively localized to the interstitium (the structural connective tissue in the lung that lies between alveoli) and over time becomes thicker and more diffuse. Initially the fibrosis begins as isolated patches that coalesce into rough or spiky-shaped masses that appear as irregular opacities on a chest film. These opacities are most frequent, and therefore most dense, on the chest film in the lower lung fields. Over time, they tend to coalesce into larger masses or opacities and may sometimes present as nodules, in which case cancer must be ruled out, or as bands of fibrosis. Ultimately, the scarring may become gross and interfere with the mechanical function of the lung.

In asbestosis the airways are also affected but not as much as the parenchyma. Pulmonary function studies may show a mild obstruction to airflow, particularly early in the course of the disease[36, 37]. In more advanced or rapidly progressing cases of asbestosis, this obstructive component is usually soon overwhelmed by a progressive restrictive disease, at least in part due to air trapping[38] that limits the capacity of the lungs and that ultimately may cause respiratory insufficiency. In less advanced or progressive disease, there is an accelerated loss of ventilatory capacity, sometimes appearing before radiographically evident asbestosis. In such cases, however, the progression of the chronic airflow obstruction is greater with greater profusion of irregular opacities on the chest film[39]. The apportionment of chronic obstructive airways disease as an outcome of asbestos exposure is discussed in a later section. Combined restrictive and obstructive deficits in asbestos-exposed workers seems to be associated with greater functional impairment[40].

Because it is difficult to appreciate obstructive disease against a background of severe restrictive disease, the airways component of asbestosis has not received much attention until recently. Pleural fibrosis is particularly associated with these restrictive changes and probably represents the contribution of mechanical changes in the chest wall, but this is a relatively minor effect[41–43]. Pulmonary function studies also show a reduced diffusing capacity, both because of delayed diffusion across the thickened interstitium and mismatching of blood and air in the alveolar region due to the disruption of the fibrosis. This mismatching is also a reason for the progressive desaturation of oxygen in the blood that eventually results in hypoxemia and clinical respiratory insufficiency in severe cases. Mild cases of asbestosis may not necessarily show this interference with gas exchange and blood gases may be normal in such cases.

Unlike other outcomes associated with asbestos, there is no evidence that cigarette smoking plays any role in contributing to the onset of asbestosis, or that the effects of asbestos exposure and cigarette smoking are positively interactive in causing enhanced asbestosis[44]. There is some evidence that once established, asbestosis may be enhanced by cigarette smoking with an increased frequency of opacities

detectable by HRCT for the same degree of asbestos exposure[45]. Since the frequency of opacities does not correlate closely with changes in pulmonary function and therefore impairment, it is not clear that this finding can be used as the basis for an apportionment formula.

Possible susceptibility states may contribute to risk of asbestosis, for example glutathione-S-transferase deficiency[46]. This is a common condition, affecting some 50% of Caucasian males, that might well be considered within the range of normal but that appears to predispose to asbestosis and may modify the outcome. However this observation is not helpful in apportionment. It is an inborn condition of the worker and so common that it may be considered a variant of normal.

The implications of these data simplify apportionment in most cases. Because asbestosis is a disease only caused by exposure to asbestos, and because other risk factors play only a minor role in modifying the outcome associated with the fibrosis (as opposed to complications such as cancer), there is no basis for apportionment by cause. If the diagnosis is asbestosis and causation can be established, the apportionment by cause is 100% attributable to asbestos and all respiratory impairment resulting from the fibrotic component of the disease is asbestos-related. Examiners often acknowledge the presence of asbestosis, but apportion the resulting respiratory impairment between asbestos and cigarette smoking, particularly when there is mixed obstructive/restrictive impairment. It is difficult to do this by cause for the obstructive component and the progression of mixed impairment makes separation of the restrictive and obstructive components uncertain. Given the caveat in workers' compensation that any substantial contribution by a workplace exposure is sufficient to consider the outcome to be work-related, the presence of any documentable asbestosis-related impairment, for example mild restrictive impairment, should be sufficient to apportion all impairment to the asbestos exposure.

The general rule that in the presence of asbestosis all respiratory impairment should be apportioned to asbestos. The exception may be a very mild case of asbestosis with minimal or no functional impairment associated with marked obstructive changes in a heavy smoker, a characteristic smoking-related respiratory impairment. In such a case, the restrictive component of the disease would be considered asbestos-related and the obstructive component, taken as FEV_1/FVC(%) rather than FEV_1 compared to predicted, would be more likely to reflect the influence of cigarette smoking. The treatment in such a case would then parallel that given below for chronic obstructive airways disease. However, even in this case there is evidence that the asbestos-related airways changes modify the effects of cigarette smoke, at least in experimental studies[46, 47]. The relative contribution by cigarette smoking may be overestimated by this approach in such cases.

Chronic Obstructive Airways Disease

It has been known for many years that exposure to asbestos is associated with obstruction to airflow as well as restrictive changes[50–52]. Functional changes are also correlated with respiratory symptoms such as cough, wheeze, and shortness of breath[53]. However, chronic obstructive airways disease (COAD) has not been emphasized as an asbestos-related outcome and has not been accepted by compensation agencies as a presumption or scheduled occupational disease. There are several reasons for this reluctance to recognize asbestos-related chronic obstructive airways disease. The most influential has probably been that the effect of cigarette smoking is not easily separated from asbestos exposure and has confounded the association, influencing agencies and adjudicators to attribute all of the cause to the smoking[50]. Another factor is that the predominant effect in advanced asbestosis is restrictive disease and the obstructive changes associated with lesser degrees of asbestosis have been largely overlooked[37, 39]. Yet another factor is that mandated surveillance for asbestos-exposed workers, such as the OSHA asbestos standard in the United States and the Alberta Fibrosis Program in Canada, have emphasized the early identification of restrictive changes and changes in the FEV_1, which will reflect changes in the FVC, rather than an interpretation that emphasizes airflow taking changes in vital capacity into account.

Adults lose a fraction of their lung capacity and airflow velocity, as measured by routine spirometry, due to aging; this loss is predictable, and for FEV_1 averages 30 ml/y. In theory, any person who lived long enough would develop obstructive disease, once the natural loss progressed far enough. Pulmonary injury may accelerate this loss and in cigarette smokers this rate of loss may easily double or triple, so that during their lifetime they dip well below the normal range and develop incapacity, the condition known as chronic obstructive pulmonary disease (COPD). (COPD and the less common term COAD are usually synonymous. Here, COAD is the more general term, and is used to avoid confusion with the complex illness associated with cigarette smoking that most clinicians have in mind when they refer to COPD.)

It is now well established that asbestos-exposed workers

show accelerated loss of airflow and are at risk for obstructive airways disease[54–59]. Those with signs of early parenchymal fibrosis appear to be at higher risk for more rapid decline[60]. Asbestos-exposed workers who develop persistent respiratory symptoms are at risk for even more rapid loss of pulmonary function[61]. There is also experimental evidence for a positive interaction (synergy) in airflow obstruction between asbestos exposure and cigarette smoking because of changes in compliance in the wall of small airways[47].

Studies of nonsmoking asbestos-exposed workers confirm that asbestos exposure alone can accelerate loss of pulmonary function[36, 54, 55, 62–64]. The two studies that permit inference of the rate of loss of FEV_1[55, 64] suggest that the accelerated rate of decline, over the usual 30 ml/y, is on the order of 30 to 60 ml/y or a doubling or tripling of the normal rate. The decline in FEV_1 was greater with higher exposure levels. This is in the same range as the effect of cigarette smoking.

The pathology and physiology of this effect is reasonably clear. The alveolitis induced by asbestos begins at the respiratory bronchiole, which is anatomically adjacent to the terminal and other small bronchioles. As well, there may be direct inflammation of the bronchiolar wall in response to deposited asbestos fibres[47, 65]. The adjacent alveolitis changes the compliance of the wall of the small airways (which is membranous, unprotected by cartilage) and, together with loss of the elastic recoil of the surrounding lung parenchyma, causes a progressively larger fraction of the population of small airways in the lung to close earlier on expiration, trapping air and introducing resistance to airflow. Asbestos therefore causes a small airways disease that appears first as reduced flow rates in the mid-expiratory part of the spirogram, which reflects airflow in the small-diameter but high-cross section peripheral airways, where there should normally be very little resistance to flow. This may occur with or without early signs of asbestosis[36]. Šaric and Peric[66] have proposed that this process follows an initial phase of several years in which small airways airflow actually increases due to stabilization of the bronchiolar wall by fibrosis.

Cigarette smoking induces a focal bronchiolitis and minimal adjacent alveolitis in much the same way. Over time, a loss of elastic recoil, early collapse of the bronchiole, and small airways disease ensues. An important component of this process, also presumably critical in asbestos-related bronchiolitis, is the release of inflammatory mediators and protease enzymes that degrade structural protein, which result in local tissue destruction. This chronically progresses to overt emphysema. To date, there is no evidence for interaction between cigarette smoking and asbestos as a cause of small airways disease or loss of FEV_1[54, 55, 63, 64, 67]. One may therefore assume that the two exposures contribute more or less independently to risk.

Given this apparently relatively independent contribution to risk, apportionment by cause can be applied as a tradeoff between the contribution of asbestos exposure and the contribution of cigarette smoking to the degree of impairment, since COAD is manifested by and defined by increased resistance to airflow. A reasonable method is therefore needed for apportioning the relative contribution of cigarette smoking and asbestos in an asbestos-exposed worker who is impaired, with a reduced FEV_1. This might be done in three ways:

1. Assessing the rate of loss of pulmonary function characteristic of the worker, smoking or nonsmoking, prior to exposure to asbestos, extrapolating the rate of loss, and determining the difference between the predicted rate of loss and that observed, which is assumed to be due to asbestos exposure. The relative contribution of each to the last relevant set of pulmonary function studies would be the apportionment attributed to each cause.

   This approach is most rigorous but depends on having at least two FEV_1 determinations prior to beginning work involving exposure to asbestos. This is not realistic in most cases. Variability in spirometric measurements is enough to obscure or exaggerate such changes when the tests are performed in different laboratories. Workers who have had routine spirometry are also likely to have had the test as surveillance for dust exposure in an earlier job or because they had a lung disease; in either case the predictive value of the baseline rate of change of FEV_1 is reduced but it would be even more important to obtain individualized results. It may be challenged if the worker then quits smoking, although rates of decline in FEV_1 only recover after some time. Removal from exposure to asbestos would not normally present a problem in interpretation because the accelerated decline in FEV_1 continues for at least 10 years[55].
2. When a baseline FEV_1 is available, assume that the rate of loss of pulmonary function due to aging is the average of 30 ml/y, extrapolate the expected rate of loss to current pulmonary function, and determine the difference between the predicted rate of loss and that observed. This difference is assumed to be due to asbestos exposure. The relative contribution of each to the last relevant set of pulmonary function studies would be the apportionment attributed to each cause.

   This method can be used in cases where pre-exposure pulmonary function levels are not known, which is the

majority of cases. This is not individual-specific but it is based on group norms for rate of change of FEV_1. Spirometric variability remains a problem.

3. Assess current pulmonary function, and compare with predicted values, then apply a crude rule of thumb to the difference: 50% apportionment to asbestos and 50% to cigarette smoking, of the respiratory impairment. This method has the advantage of simplicity but cannot take into account degrees of exposure or smoking history. It is probably an overestimate (thereby "giving the benefit of doubt to the worker", appropriate to workers' compensation) since it is unlikely that asbestos exposure would be responsible for as much as 50% of isolated obstructive impairment.

Applying the criteria for substantial contribution, one may derive a reasonable test for substantial contribution in asbestos exposure, as demonstrated in the next section. As a practical matter, individual awards at such low levels of impairment in the absence of a test would be small but there could be many of them. A small error on the side of inclusiveness is not very expensive but the total absence of a test would place a huge demand on the system.

Substantial contribution in chronic obstructive airway disease

In chronic obstructive airways disease, the outcome is the physiological impairment. Apportionment of cause therefore apportions impairment, and vice versa. If more than half of the impairment is due to an occupational cause, then the disorder is presumptively occupational and qualifies as an occupational disease. If less than half, then the contribution may be significant but it is by definition not the major determinant of disease. If the impairment is not sufficient to push an otherwise fit person into a level of impairment recognized by workers' compensation, it would be inconsistent to call it a substantial contribution for purposes of compensation. Therefore an exposure that causes a lesion so trivial that it cannot be discerned in the contribution to total impairment cannot be considered a substantial contribution. As a practical matter, therefore, one is concerned about contributions to the apportionment of predominantly nonoccupational disease from, say, 5% to 50%.

Ohlson *et al.*[37] presented data that relate lung function as a percentage predicted from regression equations by exposure category for asbestos workers. These data are cross-sectional in a stable, aging workforce without evidence of asbestos-related disease or evidence of significant out-migration. Although a longitudinal study would be preferable, these data do reflect the realities of clinical presentation, as they would be enrolled as workers' compensation claims. Notwithstanding that the regression never dipped below the range of normal, their data provides a relationship between very mild impairment and exposure. These data are particularly useful in defining the relationship between exposure and response for changes so subtle that they could not be appreciated by any other means. The table is adapted in Table 1.

Table 1. Lung function as a percentage predicted from regression equations by exposure category for asbestos workers (Data from 37)

Fibre·years:	0 – 14 (n=41)	15 – 22 (n=42)	23 + (n=41)
FVC	96.1	95.4	94.6
FEV_1	92.8	91.8	90.5

There are two ways of reading the regression. It may be read as a prediction for the entire population and therefore a best estimate for the individual, or as an average for the population with variability among individual subjects, so that a small subset of subjects might have a markedly greater loss than the average. The authors comment that "the group exposed to dust with comparatively low asbestos fibre concentration had a minor impairment of lung function…", both smokers and nonsmokers, and variance was low in this population. They do not identify a subset with disproportionately poor pulmonary function, although such a subset would be of greatest concern.

The Ohlson data[37] show a linear relationship with a very slight slope and are clearly reflective of a mild effect in a population with generally preserved pulmonary function. It is therefore a useful data set for the purpose of defining substantial contribution. A longitudinal study would be even more useful.

The standard convention in pulmonary function testing is to consider both FVC and FEV_1 as abnormal only when they fall below 80% of predicted. Functional impairment for most people, other than athletes, is generally not demonstrable until at least this much function has been lost. This convention is reflected in the *AMA Guides to the Evaluation of Permanent Impairment*, which does not recognize impairment as existing until this threshold is reached. Category 1, involving either FVC or $FEV_1 > 80\%$ predicted, is associated with 0% impairment of the total person. FVC is less obviously linked to symptomatic impairment than FEV_1 and seems to be less impaired in asbestos-related disease than FEV_1, at least in the earliest stages. Therefore FEV_1 should be used as the most sensitive

indicator of effect. If one assumes that 20% of FEV_1 must be lost before impairment is obvious, what fraction of that 20% must result from a given cause before it can be considered "substantial"?

For a disorder to result in a loss of FEV_1 sufficient to push a normal person who smoked across the line into clinical impairment, perhaps half of this residual may be required; this is a clinical impression not easily validated by data. Thus, a level of exposure sufficient to result in loss of 5% of function is a reasonable threshold for what is substantial. This is also reasonable considering that it exceeds the measurement error of careful spirometry by the ATS criteria.

Referring to Table 1[37], a loss of only 5% of FEV_1 would correspond to approximately 10 fibre·years of asbestos exposure. This number can now be compared with other derivations as an estimate of a reasonable exposure level constituting substantial contribution.

If the effect of an exposure to asbestos, for example, was only to produce a pleural plaque, that might qualify as a tissue injury in pathological terms, but not as a cause of an outcome leading to impairment. The tissue injury did not interfere with function. In some compensation systems, the worker is still entitled to compensation for an asbestos-related condition, i.e. medical costs for annual surveillance, but not for permanent impairment. However, if one may demonstrate that the same exposure to asbestos resulted in a decrement in pulmonary function that falls outside the range of normal variability and could mean the difference between impairment and freedom from impairment in a worker developing chronic obstructive airways disease, that would constitute a substantial contribution. Unfortunately, there is no relationship demonstrable between the loading of fibres required to produce a plaque and that required to contribute to airflow obstruction, so plaques cannot be used as a marker of substantial contribution and the absence of plaques cannot be used to rule out a substantial contribution[6].

Conclusion

Asbestos-related diseases are attractive models for the application of apportionment. In practice, apportionment is less useful as a rigid approach or formula for managing claims than as a conceptual framework for thinking about the problem. The models presented here may serve as a general guide to the assessment of asbestos-related disease outcomes for purposes of compensation.

Asbestos-related outcomes vary greatly in their suitability for apportionment. For mesothelioma and asbestosis, apportionment is not a very meaningful process. For airflow obstruction, it is a complex and technical but theoretically valid approach. For lung cancer, it is complicated and there are no markers or approaches that support apportionment in the individual case. This means that different asbestos-exposed workers with different outcomes are being judged differently by the system of adjudication. In some cases, e.g. patients with asbestosis who have a predictably high cancer risk, the sequence of these outcomes are almost matters of chance and the injured worker may as easily presented with lung cancer first as asbestosis.

Unlike apportionment of impairment, where there are consensus standards such as the *AMA Guides to the Evaluation of Permanent Impairment*[68] apportionment by cause has achieved no consensus, defies the imposition of rigid standards, and is not convertible (as is percentage impairment of the total person) from one disease category to another. Within this class of injured workers, is it reasonable to apportion in some cases and not others simply because apportionment is possible in those cases?

This raises the issue of equity. On the one hand, it is standard operating procedure for the workers' compensation to evaluate hand injuries, occupational lung disease, noise-induced hearing loss, and brain injury by different criteria. The "apportioned" causation may be reflected in the apportioned impairment (in these cases always for aggravational injury) so that eventually these very different cases are evaluated on a comparable scale. However asbestos-related diseases reflect different outcomes of a common exposure in a situation where the effect is not aggravational but simultaneously causal. Is it reasonable to treat these related disorders so differently?

This is a fundamental issue in workers' compensation policy and falls outside the scope of this report. It is raised, however, to suggest that apportionment may not be equitable if its application is constrained in some cases more than others[4, 5].

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