# Letters

## RE: Controlled Use of Asbestos

> A lie can travel halfway around the world while the truth is still putting on its shoes.
>
> — Mark Twain

*To the Editor:*—Castleman's recent article, "Controlled Use of Asbestos" critiques industry's misrepresentation of the hazards associated with using asbestos.[1] We agree with Castleman when he states that, "'Controlled use' of asbestos is the asbestos industry's way of referring to business as usual with a false face." "Controlled use" is but the latest trend in a series of corporate misrepresentations relating to asbestos risks. Union Carbide Corporation (UCC) and its consultants have asserted that asbestos mined from the Coalinga deposit in California and sold by UCC as "Calidria®" is innocuous.[2] They base these assertions on two arguments: 1) Calidria is unregulated "short-fiber" chrysotile (less than 5 µm) and thus nontoxic, and 2) none of the Calidria mine or mill workers ever developed asbestos-related disease. UCC successfully marketed Calidria® as short-fiber asbestos, while suppressing evidence that more than 50% of Calidria fibers were actually longer than 5 µm. UCC concealed the results of animal studies, and to give the impression that Calidria was safe, they employed consultants to manipulate Calidria inhalation data from previous studies. As a result of legal actions instigated by injured workers, U.S. courts have forced UCC to divulge some of these secret studies and documents that reveal the perfidious nature of these assertions.*† We report this evidence here.

### *The "Short-fiber" Myth*

Union Carbide has given customers the impression that Calidria is innocuous because of its short fiber length. However, Kent Pinkerton's 1982 PhD thesis found that 48% of aerosolized Calidria fibers were longer than 5 µm, compared with 25% of Canadian (Jeffrey) and UICC B chrysotile.[3] Calidria also had a greater percentage of fibers more than 10 µm long: "At least 92% of the combined fibers and fiber clusters in the Jeffrey and UICC B aerosols were less than 10 µm in length, but only 66% of the combined fibers and fiber clusters in the Coalinga chrysotile aerosol were less than 10 µm."[3] Edward Ilgren, a UCC litigation consultant, published selected portions of Pinkerton's thesis but misrepresented his data on fiber length. Ilgren repeatedly referred to Calidria as a short fiber: "Fibres from . . . Coalinga, Calif., are almost all less than 5 µm in length. . . ."[4] In a 1998 deposition, however, Ilgren admitted that he had no data to support his conclusion that Coalinga asbestos was a short-fiber asbestos.[5]

Union Carbide undertook secret studies that revealed that Calidria fibers were thin, fine, and longer than 5 µm. They realized that Calidria fibers were on average thinner than those of Canadian chrysotile and they therefore were often missed when analyzed by light microscopy (which has a lower limit of detection of 0.25 µm). Three of these studies are highlighted below.

In 1975, UCC secretly compared the lengths of Canadian and Calidria fibers released during the use of two, otherwise identical, ceiling texture paints. The measurements taken during the dry mixing and spraying of the textures revealed similar exposures to long fibers: "There is clearly no difference between these types of asbestos for airborne concentrations of asbestos fibers longer than 5 µm."[6]

In 1977, some UCC customer and OSHA sampling of Calidria exposures determined that fiber levels were 20–40 times higher than previous UCC counts. In response to these reports, UCC reevaluated its sampling method and found that 20–40 fibers/cc had been missed. UCC noted that: "It has also been our experience in working with ultra-fine fibers that they are very easy to miss completely unless the operator specifically looks for such material."[7] UCC did not pass this information on to customers or government agencies. In 1983, when NIOSH made an undercount error at the UCC mine, UCC again confirmed that fine Coalinga fibers escaped detection: "It would appear that the NIOSH counting lab missed a significant amount of asbestos fiber in some of the samples. . . . Coalinga has a much smaller mean diameter and fiber length distribu-

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*Many of the documents cited in this letter to the editor were produced in litigation where one of us (DE) is a consultant for *Kelly Moore: Kelly Moore Paint Company vs. Dow Chemical Corporation et al.*, No. 19785-BHO.

†All Union Carbide Documents referred to in this article may be found at <www.egilman.com/UCC_Corruption>.

tion. Because of this and the fact that most counting laboratories are not familiar with Coalinga asbestos, their fiber counts are often artificially low."[8] UCC withheld this information from NIOSH.

*Exposure to "Short-fiber" Asbestos Does Cause Disease: Hidden and Deceptive Studies*

Ronald Dodson et al. reviewed the pathogenicity of various fiber lengths and concluded that all fiber lengths cause asbestos-related diseases.[9] Secret, unpublished, UCC-funded studies performed by the Mellon Institute support these conclusions. The Mellon Institute conducted at least two animal studies on Coalinga chrysotile, the first in 1966 and the second in 1971. In the 1966 study, Mellon researchers injected Coalinga into the peritoneal cavity in guinea pigs, rats, and rabbits. To mimic the human exposure route, Mellon researchers intratracheally injected a second cohort of rats. The asbestos tested included a Johns Manville Canadian long-fiber chrysotile and two short-fiber types, including a Coalinga sample (CMS-100). The Mellon researchers concluded that fiber length was unrelated to pathogenicity and that Coalinga fiber was potentially more harmful than the Canadian chrysotile: "The results . . . indicate that the asbestos products studied produced fibrotic lesions of the visceral organs . . . regardless of fiber length. Of the 3 products, CMS-100 (Coalinga fiber) produces the most severe reaction."[10]

After reviewing the results of the 1966 Mellon report, Dr. Dernehl, UCC's associate medical director, remarked that: "The only conclusion we can draw from this crude test is that it is possible that our Coalinga product may be more hazardous to use than long fiber asbestos in that it may induce the disease, asbestosis, at an earlier time after exposure."[11] Dernehl also concluded that the 1966 test called into question the adequacy of the threshold limit value to protect employees: "It is probable that the 5 million particles per cubic foot will not be acceptable for the prevention of mesothelioma."[11]

The 1971 Mellon study tracheally insufflated rats with a new UCC product, a silica coated asbestos pellet (RG-244). The rats were examined at 30-, 60-, 90-, and 180-day intervals, and the results provided UCC with clear evidence of the hazards associated with exposure to this double-hazard product: "In general, because of the overwhelming preponderance of effect in the asbestos dosed lungs versus in the controls, we have sufficient evidence of damage to warn us to do our best to prevent inhalation of concentrations of asbestos in excess of the Threshold Limit Value proposed for 1970. . . ."[12]

Unfortunately, UCC did not publish either of the Mellon studies. In 1982, after passage of the Toxic Substances Control Act (TSCA), which required companies to report toxicologic findings to the EPA, UCC lawyers and management made a decision to hide these findings from the EPA: ". . . the Mellon Institute studies are presently defined as confidential. Since they are more than 10 years old, however, they do not appear to be reportable."[13]

As mentioned above, between 1997 and 1998, UCC consultants Ilgren and Chatfeld published selected portions of Pinkerton's 1982 PhD thesis (and subsequent abstracts and articles), which compared the relative fibrogenicities of *ground* Calidria and Canadian and UICC reference chrysotile.‡ The consultants claimed that they performed a "careful review" of Pinkerton's data, and published their conclusion avowing that Coalinga was not fibrogenic: "Exposed animals displayed no fibrosis following exposure to Coalinga chrysotile, but showed fibrogenic responses with both Canadian fibres."[14] However, Pinkerton et al., the actual researchers, arrived at the opposite conclusion: "Interstitial fibrosis was seen histologically in all exposed animals [including the Coalinga group] at one year and increased in severity during the year in air [without exposure]."[15] This fibrosis developed even though the Coalinga-exposed rats were exposed to much less asbestos than the other exposed animals.[15] Despite the data, Ilgren touted Coalinga chrysotile as "innocuous" and declared that it should be treated as a "nuisance dust."[14]

Ilgren also claimed that none of the chrysotile fibers tested induced mesotheliomas in any of the rats.[4] This "finding" conflicts with other literature, which reveals a direct relationship between exposure to Coalinga asbestos and the development of mesothelioma.[16–19] Ilgren's conclusion also conflicts with almost all other animal studies that have examined this question.[20]

Although Pinkerton did not show that Coalinga asbestos was "innocuous," his experiment did indicate that it was less toxic than comparison chrysotiles. This apparent conflict with Mellon study findings is easily explained. Asbestosis is a dose–response disease, and Pinkerton's Coalinga-exposed rats were exposed to 66% less fiber by weight and five times fewer respirable fibers than rats exposed to the comparison chrysotiles.[3]

Additionally, the asbestos types were prepared differently prior to testing. The Coalinga asbestos was water processed and ground three times, while the Canadian fiber was passed through a hurricane pulverizer.[3] Unlike the Canadian fiber, which was a commercial sample, the Coalinga sample came from the cyclone overflow at the UCC mill.[5] The UICC B was untreated.[3] Many studies have shown that grinding or manipulating the asbestos structure alters the toxicity and subsequent pathogenicity of asbestos.[21,22]

‡Pinkerton refused to co-author these papers. He did not agree that Calidria should be considered a nuisance dust. Personal communication, August 2003.

Pinkerton confirms this fact, but does not provide any evidence that commercial Calidria is any less toxic than any other asbestos form.

Finally, Ilgren stated that Coalinga asbestos was "amphibole-free," but in his 1998 deposition, he admitted he had no data to suggest this assumption.[5]

*Asbestos disease in UCC Calidria mine and mill workers.* UCC has denied the presence of asbestos disease in any mine or mill workers in sworn testimony and has failed to report any cases to OSHA.[23] In the 1960s, UCC implemented a program of periodic surveillance of asbestos-exposed workers, including pulmonary function tests and chest x-rays. Additionally, Dr. Hilton Lewinsohn, their medical director, performed an additional review of the x-rays in 1984. Our review of these medical records and reports reveals that many workers had findings compatible with asbestosis on chest x-rays, four of whom were diagnosed as having asbestosis (see Table 1).[§] Four other employees exhibited possible asbestos-related lung cancers or other chest malignancies, one of whom had "lung cancer" listed on his death certificate.[24]

### *Additional Medical Information*

One worker died in 1991 after working at the King City mill for 28 years. On his original death certificate, his treating thoracic surgeon stated that asbestosis was a contributing cause of death.[25] After conferring with UCC corporate lawyers (despite the fact that UCC had sold the mine to a group of investors six years earlier), UCC's mine manager and president of KCAC, the successor corporation, obtained his death certificate, arranged to make the worker a "coroner's case" and had an autopsy performed on the worker.[26] The worker's family was not consulted. The coroner subsequently changed the death certificate, replacing "asbestosis" with "non-specific pulmonary fibrosis."[27,28] The mine president was a member of the city council at the time (he is now the mayor and has been for 12 years), was a friend of the owner of the funeral parlor (in retirement he currently drives the parlor hearse), and serves on the board of the only hospital in town.

The autopsy report listed pleural thickening, diffuse pulmonary fibrosis, and history of asbestos exposure (remote) as diagnoses.[30] The text of the report noted the absence of asbestos bodies. The coroner was apparently under the impression that the diagnosis of asbestosis required the finding of asbestos bodies. UCC's secret animal studies indicated, however, that Calidria caused asbestosis but did not induce the formation of asbestos bodies.[12]

### *The UCC Lewinsohn Report*

In 1984, in anticipation of the sale of the King City mill, Union Carbide's medical director, Dr. Lewinsohn, who previously had served as the medical director for the asbestos companies Turner & Newall and Raybestos Manhattan, reviewed all the employees' radiography reports. In his report, Dr. Lewinsohn, stated that ". . . 1/1 is regarded as definite radiographic evidence of the presence of changes consistent with pneumoconiosis."[31] Dr. Lewinsohn found that several asbestos millers had x-ray evidence of pneumoconiosis, including evidence of progression. One worker's x-ray progressed from 0/1 to a 1/1 profusion between 1969 and 1983.[32] UCC's medical director noted that this progression could have been related to asbestos exposure, but suggested the possibility that an unidentified asbestos exposure, other than the exposure at the asbestos mill where the worker was employed, might have caused this worker's disease: "Further investigation of this case is suggested to determine whether the changes noted are related to asbestos exposure at King City or elsewhere."[32] UCC's records fail to identify any other asbestos exposure for this individual. Even if the worker had other additional exposure, it is indisputable that his 14 year exposure to Calidria at the King City mine contributed to his asbestos-related disease.

Despite the fact that Dr. Lewinsohn told UCC that informing the employees of their condition was a legal requirement under the OSHA act, we have been unable to find any evidence that UCC, its medical director Dr. Lewinsohn, or any of its consulting physicians ever informed any of the workers that they might have had an asbestos-related illness. This medical information proves that Calidria causes asbestos-related disease.

### *UCC Successful Marketing: Customers Believed Calidria Was Safe*

Not only did United Carbide tell employees that Calidria was safe enough to eat, they also expended considerable resources to persuade customers that Calidria was "innocuous."[33] After receiving UCC promotional material and/or attending UCC lectures, several UCC customers believed Calidria was safe. After attending a UCC presentation, one Dow employee wrote, "Apparently there are four crystalline structures common to asbestos. Of the four, three are proven bad actors whereas the fourth (which they claim is the Calidria type) is much more innocuous . . . Union Carbide claims they have sufficient toxicological data that places Calidria in the nuisance dust category. . . ."[34] Other customers believed that Calidria was not carcinogenic: "The asbestos we use is a unique type available from just one mine in California. It is produced and processed by the Union Carbide Corporation. The have run exten-

[§]All medical records referred to in this article may be found at <www.egilman.com/UCC_Corruption>.

**TABLE 1. Disease in King City Mine and Mill**

| | |
|---|---|
| Worker 25 | "Asbestosis" written on report* |
| Worker 108 | ILO section "small irregular opacities" filled in: 0/1 (1969), 1/1 (1983);† parenchymal abnormalities consistent with pneumoconiosis‡,† (1969, 1983); "pi" indicated in comments (1983) p,†; "kl" indicated in comments (1983)k,†; "the smaller capacities (sic) are more obvious than they were in 1969" (1983) †; "co" indicated in comments (1969)co,† |
| Worker 178 | "Shows x-ray evidence of an early pneumoconiosis, must wear respirator in dust." (1970) * |
| Worker 217 | "Minimal increase in the interstitial lung markings" (1977) * "Mild obstructive lung disease" (1986)* "There is a mild obstructive lung defect" (198_)* "There is a moderate obstructive lung defect" (1986)* "Mild to moderate obstructive disease" (1983)* parenchymal abnormalities consistent with pneumoconiosis (1966, 1984)‡,† ILO section "small irregular opacities" filled in: 0/1 (1968), 1/0 (1984)† |
| Worker 291 | "Moderate Restrictive pulmonary impairment" (1993)* "There is a moderate obstructive lung defect" (1986)* |
| Worker 358 | "Suggestive of asbestosis" (1974)* "May be the first indication of obstructive lung disease" (1970)* "The appearances are consistent with pleural calcified plaques—? Asbestos-related. No definite asbestos seen. Indefinite opacity on (right) lobe above diaphragm—rule out ca." (1984)†; cancer listed as "ca" under "other abnormalities (1984)c,†, "pi" indicated in comments (1972, 1984)pi,†; "co" indicated in comments (1966, 1984)co,†; 0/1 profusion (1972)†; "Contraction of Right lower lobe +pleural thickening in horizontal fissure" (1972)†; "od" indicated in comments (1966)†; Pleural abnormalities consistent with pneumoconiosis (1984)§,†; parenchymal abnormalities consistent with pneumoconiosis (1972)‡,† |
| Worker 311 | Cancer listed as "ca" under "other abnormalities" (1984)c,† |
| Worker 360 | Death certificate—"lung cancer" and "chronic obstructive pulmonary disease"(1994)* |
| Worker 380 | "The chest x-ray does show changes suggestive of chronic pulmonary fibrosis" (1965)*; parenchymal abnormalities consistent with pneumoconiosis (1963)‡,† ILO section "small irregular opacities" filled in:1/0 (1963)†; "od" indicated in comments (1984)od,†; 'hi' indicated in comments (1963)hi,†; "cn" indicated in comments (1963)cn,† |
| Worker 383 | "Asbestosis" listed on initial death certificate (1991)*; "Rule out (inflammatory changes) before considering asbestosis" (1981)†; An 0/1 profusion noted (1981)†; "od" indicated in comments (1963, 1981)†; parenchymal abnormalities consistent with pneumoconiosis (1981)‡,† |
| Worker 385 | Cancer listed as "ca" under "other abnormalities" (1984)c,†; "A rounded opacity is present in the left hilium. It appears larger than on 4-30-83, but was probably present on 1981 films. Although unilateral, rule out carcinoma" (1984)† |
| Worker 549 | "This man should not be exposed to further inhalation of asbestos. Dr. Hughes, Radiologist read the file as asbestosis, not even knowing this was a Union Carbide worker, and comparison with films from . . . 1969 shows definitely increased activity in the lungs." (1974) * |
| Worker 72 | Parenchymal abnormalities consistent with pneumoconiosis (1966)‡,†; ILO section "small irregular opacities" filled in: 1/0 (1966)†; "cn" indicated in comments (1966, 1982)cn,†; "pi" indicated in comments (1966)pi,† |
| Worker 131 | Parenchymal (1981, 1982) and pleural(1981) abnormalities consistent with pneumoconiosis‡,§,†; ILO section "small irregular opacities" filled in: 1/0 (1981), 0/1 (1982)† "Mild restrictive pulmonary impairment" (1993)*; "co" indicated in comments (1981, 1982)co,† |
| Worker 362 | ILO section "small irregular opacities" filled in: 0/1 (1972, 1982)†; parenchymal (1972, 1982) and pleural (1984) abnormalities consistent with pneumoconiosis‡,§,†; "evidence of obstructive Lung disease" (1980)* |
| Worker 218 | "Appears to be developing bi-lateral basal fibrosis" (1973) † parenchymal abnormalities consistent with pneumoconiosis (1973, 1983, 1984)‡,†; "moderate obstructive disease" (1984)*; ILO section "small irregular opacities" filled in: 1/0 (1973, 1983) 0/1 (1984)†; "pi" indicated in comments (1958, 1961, 1973, 1983, 1984)pi,†; "co" indicated in comments (1983, 1984)co,†; "od" indicated in comments (1983, 1984)od,†; "kl" indicated in comments (1983, 1984)kl,† |
| Worker 371 | "Calcified opacities" (1982)†; parenchymal abnormalities consistent with pneumoconiosis (1982)‡,†; "small irregular opacities" filled in: 0/1 (1982)†; "od" indicated in comments (1982)od,† |
| Worker 83 | Pleural abnormalities consistent with pneumoconiosis (1983)§,†; "appearances on the left chest wall maybe due to previous trama or pleurisy" (1979)† |
| Worker 12 | "(right) upper lobe_ (lung)? Carcinoma" (1982)†; Pleural abnormalities consistent with pneumoconiosis (1982)§,†; "od" indicated in comments (1982)od,†; "pi" indicated in comments (1982)pi,† |

* King City Asbestos Mill Surveillance Program.
† Lewinsohn Report.
‡ Parenchymal abnormalities consistent with pneumoconiosis ILO box checked on x-ray report.
§ Pleural abnormalities consistent with pneumoconiosis ILO box checked on x-ray report.
c "ca" is the symbol used to indicate "cancer of lung or pleura."
p "pi" is the symbol used to indicate "pleural thickening in the interior fissure or mediastium"
k "kl" is the symbol used to indicate "septal (kerley) lines."
co"co" is the symbol used to indicate an "abnormality of cardiac size or shape."
od"od" is the symbol used to indicate an "other significant abnormality."
hi"hi"is the symbol used to indicate an "enlargement of hilar or mediastinal lymph nodes."
cn"cn" is the symbol used to indicate a "calcification in small pneumoconiotic opacities."

sive medical tests on this asbestos (Calidria RG-144) and we have their assurance it is non-carcinogenic."[35]

The asbestos industry continues to take advantage of the short-fiber myth to promote the use of chrysotile around the world. For example, the Asbestos Institute, the lobbying arm of the Canadian Asbestos industry, has recently touted a soon-to-be published study that concludes that chrysotile is safe to use.[36] This study relies on Ilgen's shortfiber misrepresentations.

The evidence presented in this paper illustrates only one example of how corporations have used science to achieve profit growth and escape liability at the expense of dead and injured workers.

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# Occupational Histories of Cancer Patients in a Canadian Treatment Center and the Generated Hypothesis Regarding Breast Cancer and Farming

*To the Editor*:—We commend Mr. Brophy and his colleagues for their recent article examining the relationship between farming and breast cancer.[1] The article focuses attention on occupationally related cancer, an area of research that is relatively understudied.

Although the proportion of cancer attributable to occupational exposure is much smaller than those attributable to other known risk factors for cancer (e.g., smoking and diet), knowledge regarding the relationships between exposures to hazardous agents at the workplace and their roles in the carcinogenic process is invaluable to many workers in Ontario. Unfortunately, the scientific evidence available to date, particularly in relation to farming and cancer, remains inconclusive.

A recent study conducted by Brophy and colleagues reported an increased risk of breast cancer in women associated with a previous history of farming for more than a year before age 56 compared with women with no previous farming history (OR = 9.05, 95% CI, 1.06–77.43). This estimate is based on a convenience sample of cancer patients from the Windsor Regional Cancer Centre. While the article suggests a number of interesting hypotheses, dependence on the convenience sample, small sample size, and the inadequate control of potential confounders limits the interpretability of study conclusions.

Duell and colleagues[2] conducted a large population-based study of farming and breast cancer in North Carolina, comparing 862 cases with 790 population controls. Unlike the Brophy study, the Duell study did not observe an increased risk for breast cancer associated with farming in general. The risk estimates obtained from the Duell study had been adjusted for potential confounders such as age at diagnosis, age at menarche, race, education, smoking, family history of breast cancer, and body mass index, among others. Recently, an agriculture job-exposure matrix was developed for British Columbia farmers.[3] This approach can provide more accurate historical estimates of specific exposures, thereby reducing misclassification and subsequent bias in estimates of cancer risks. It is likely that the heterogeneity of study results in the literature can be attributed in part to variations in sampling methods, measurements, and adjustments of potential confounders.

With the limited current body of evidence, it is difficult to determine whether farming activities are associated with breast cancer. With appropriate study design and analyses, these hypotheses could potentially translate into scientific knowledge invaluable to all stakeholders.

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*In reply*:—Do and colleagues at the Occupational Cancer Research and of the Surveillance Project of Cancer Care Ontario raise several issues regarding our study of breast cancer and farming,[1] including some of the acknowledged limitations of the study and our interpretation of the study by Duell et al.[2]

Because the incidence of female breast cancer has been rising steadily by about 1% annually over the last 30 years throughout the industrialized world, including Canada, this disease should be regarded as an unsolved public health crisis. The crisis is made more profound not only because the majority of breast cancer cases cannot be explained by known risk factors,[3,4] but also because relatively few epidemiologic studies have scrutinized the potential occupational breast cancer risks in general and for farming in particular,[5] and few new hypotheses have been proposed and incorporated into studies. For example, animal bioassays have identified over 200 chemical substances that trigger breast carcinogenesis[6–8] but although such agents exist in high concentrations in many workplace environments, their influences on breast cancer incidences among exposed workers remain largely unstudied.

Traditional toxicology and epidemiology are being challenged by

a new synthesis leading to new hypotheses, particularly regarding possible associations between prenatal exposures to agricultural chemicals and predisposition to cancer.[9] Increasing evidence is emerging from epidemiologic investigations and from laboratory research of an association between breast cancer and xenoestrogens; i.e., endocrine disruptors.[10] Some organochlorine pesticides, which "mimic" estrogens, are hypothesized to contribute to the proliferation of damaged cells, thus promoting the neoplastic process.[11] The herbicide atrazine is one of the most widely used agricultural pesticides. In animal studies, exposure to atrazine in utero delays mammary gland development and "may also confer an extended window of sensitivity to potential carcinogens after sexual maturity."[10]

The Windsor Regional Cancer Centre was the first local cancer treatment center in Ontario to participate in a program to track the occupational histories of its patients. Our original intention was not to study breast cancer and occupation, but rather to collect data on the occupational histories of all cancer patients. The breast cancer cases made up the largest proportion of the participating cohort, providing an adequate sample size with which to conduct data analysis. As this study was designed to capture limited data from a wide range of cancer patients, data regarding a few of the risk factors specifically identified with breast cancer were unavailable. We therefore suggested, based on our findings, that a more focused study was necessary, and funding was secured for a subsequent case–control study that did control for such risk factors.

The Duell et al. study[2] presented a complex picture of the associations between farming and breast cancer risk in women with and without pesticide exposure that supports our conclusions. The odds ratio was below that expected for farming women when analyzed without controlling for pesticide exposure or the use of protective equipment. However, women who reported being present in the fields during or shortly after pesticide application had an 80% increased risk of developing breast cancer (OR 1.8, 95% CI 1.1–2.8). Among those who reported using pesticides without protective clothing, there was a twofold excess breast cancer risk (OR = 2.0, 95% CI 1.0–4.3), while women with protective clothing had a lower-than-expected risk (OR = 0.8, 95% CI 0.4–1.8). Duell et al. conclude that while farming may not present an elevated risk per se, farming women exposed to pesticides may have an elevated breast cancer risk (p. 329). This reaffirms the weakness in using occupation as a surrogate for exposure, since misclassification of exposure occurs when subjects with less exposure are aggregated with the more highly exposed. Such non-differential misclassification decreases the probability of detecting associations[12,13] and tends to underestimate the actual risks.

In addition, Band and colleagues have recently reported the findings of a possible elevated breast cancer risk among farmers through a comprehensive population-based case–control study utilizing the British Columbia Cancer Registry.[14] The authors sought to investigate occupations while controlling for known or suspected hormonal risk factors. Women were stratified by pre- and post-menopausal status as well as by both combined. Among the combined pre- and post-menopausal group, there was a threefold elevated breast cancer risk among women ever employed in fruit and other vegetable farming (OR = 3.11, 90% CI 1.24–7.81); there was a sevenfold elevated breast cancer risk among women ever employed in other vegetable farming (OR = 7.33, 90% CI 1.16–46.2). The authors conclude:

> Several significant associations observed only in the combined group of pre- and post-menopausal women (in part owing to larger numbers) are of substantial interest, particularly those in crop farming and in the fruit and vegetable, publishing and printing, and motor vehicle repair industries. Farmers are exposed to pesticides, compounds suspected of being associated with an increased breast cancer risk, whereas the other occupations and industries entail exposure to various solvents and to carcinogenic substances such as aromatic amines in the printing and polycyclic aromatic hydrocarbons in the motor vehicle repair industries (p. 309).

While Do and colleagues acknowledge that understanding the etiology of occupationally related cancer is "invaluable" to Ontario workers, they somewhat arbitrarily assign the attributable risk of occupational exposures to a much less prominent place than such personal "lifestyle" causes as smoking and diet. Confidence in this hypothesized hierarchy of cancer causality may be misplaced, since no Canadian Cancer Agency, including Cancer Care Ontario, has begun to systematically collect or analyze the occupational histories of cancer patients. Given the parsimony of epidemiologic studies regarding occupational cancer risk factors, I believe that, as Doll and Peto[15] suggested more than 20 years ago, it is "impossible to make precise estimates of the proportion of the cancers today that are attributable to hazards at work." It might be more useful to acknowledge that our current state of knowledge does not allow us to confidently link cancer incidence to any one specific factor; it is more likely, given the current evidence, that it is related to a variety or combination of factors, including environmental, occupational, genetic, lifestyle, and socioeconomic[16] factors that should all be included in future epidemiologic studies.

This lack of scientific consensus about the causes of breast cancer has profound implications for this crisis in public health and for formulating comprehensive preventive strategies that could include protection from occupational and environmental exposures to carcinogenic and hormonally disrupting agents. Researchers, cancer agencies, and public health institutions with the goal of cancer prevention are urged to use their influence to actively support research efforts designed to explore links between environmental exposures and breast cancer risk.

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# Is Sleep a Priority for Yourself or Your Research?

To the Editor:—*Just how effective is a daytime nap or siesta in improving work performance? Should a nap be required for the night shift worker? How does chronotype affect work productivity? Can sleeping longer on weekends eliminate weekly sleep debt?*

If you cannot answer these few simple questions, you can relax—we actually know very little about sleep and how it impacts health and safety. If you ever worked a night shift, or missed a night of sleep for any reason, you would be well aware of how it can affect your judgment, your daytime functioning, and your quality of life. The public's health and safety are also at risk from those who drive trucks and automobiles while drowsy and deprived of adequate sleep. To a certain extent, medication errors and work-related injuries can also be blamed on sleep debt.

The 2003 Sleep Research Plan, recently approved by the National Center on Sleep Disorders Research, contains priorities for sleep research that would be of interest to researchers in occupational health and safety. This Center, located within the Heart, Lung and Blood Institute (NHLBI) at the National Institutes of Health (NIH) in the United States, released its original research plan in 1996. A task force of interdisciplinary sleep experts recently updated research priorities for the next five years, and the plan is available at the following NIH Web site: <http://www.nhlbisupport.com/sleep/research/research-a.htm>.

The 2003 Sleep Research Plan specifies ten priority areas, all of equal importance, for research during the next five years. One priority is the need for more training in sleep medicine, but other priorities are detailed for both basic and clinical research. Occupational health researchers will be interested in reading more about the priority for understanding the impact of sleep deprivation on work performance and safety (see pages 55-58 of the document). Sleep debt, defined as sleeping less than six hours a day, has become a major health concern in the United States[1] and in other industrialized nations.[2] Concerns include cumulative sleep debt and its detrimental effects on cognitive functioning over time,[3] accidents

and errors in the workplace, and alterations in metabolic and endocrine function.[4]

The best prescription for adequate amounts of sleep at different stages of life remains unknown, effects of chronic sleep debt are poorly understood, and the timing and effectiveness of naps or siestas during the day require further study. If you are not accounting for all the variance in your outcome of interest, perhaps it is time to consider adding sleep quality and quantity measures to your occupational research endeavors. A visit to the NIH Web site will help you learn more about the need for research in this area.

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# Bhopal Priorities

*To the Editor*:—One of the arguments that Carlsten[1] makes for giving priority to the prevention of Bhopal-type accidents over further research on its victims is that too much time has passed and that confounding factors and recall bias may limit the validity of any research studies.

While this is certainly true, we do not believe that it is an insurmountable barrier. Very large numbers of people in Bhopal (probably over 200,000) continue to experience persistent symptoms; this alone should warrant further population-based research. Furthermore, and crucially, there has been no research focusing on those who were children at the time of the disaster. Studies conducted in 1994 by the International Medical Commission on Bhopal addressed a number of the epidemiologic issues raised by Carlsten. For example, using a cross-sectional method, we found that by stratifying the current adult population by distance of residence from the Union Carbide plant to estimate exposure to the gas cloud, we could also control satisfactorily for confounding by various environmental factors such as air pollution and for socioeconomic factors.[2] We were also able to develop individual exposure estimates associated with subjective and objective measures of ill health.[3] These methods were rapid, cheap, and effective.

A large cohort of exposed and unexposed persons is still being examined periodically by the local government. Limitations of this effort include the relatively few health end points being studied, and that the results are not being widely disseminated. However, we believe that this effort can be expanded given the resources and commitment of personnel. In a recent meeting, Dr. N. K. Ganguly, Director General of the Indian Council of Medical Research, indicated that enough funds are available for research projects in Bhopal.

While prevention of industrial accidents is certainly paramount, we believe that valid research can and should continue to be done for the Bhopal gas victims.

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