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 "shortfiber" chrysotile (less than 5 pm) 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 um in length, but only 66% of the combined fibers and fiber clusters in the Coalinga chrysotile aerosol were less than 10 µm.*5 Edward Ilgren, a UCC litigation consultant, published selected portions of Pinkerton's thesis but misrepresented his data on fiber length. figren repeatedly referred to Calidria as a short fiber: "Fibres from . . . Coalinga, Calif., are almost all less than 5 µm in length. . . . " 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

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.** 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-

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.

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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 cwww.egilman.com/UCG_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. 18 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. Secret, unpublished, UCCfunded studies performed by the Mellon Institute support these conchisions. 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 aspestos 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. WIO

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." 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 ligren 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 exposed to much less asbestos than the other exposed animals.15 Despite the data, ligren 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.¹⁸⁻¹⁹ 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.⁵ Many studies have shown that grinding or manipulating the asbestos structure alters the toxicity and subsequent pathogenicity of asbestos.^{21,22}

Prinkerton refused to co-author these papers. He did not agree that Calidria should be considered a nulsance 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 OSHA23 In the 1960s, UCC implemented a program of periodic surveillance of asbestosexposed workers, including pulmonary function tests and chest xrays. 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 "hing 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. "37,28 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. 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 1983,52 L'CC'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." 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." 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-

fAll medical records referred to in this article may be found at www.egibnan.com/UCC_Corruption.

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	as asbestasis, not even knowing this was a Union Carbide worker, and comparison with films from 1969 shows definitely increased activity in the lungs." (1974)
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† Parenchymal abnormalities consistent with pneumoconlosis ILO box checked on x-ray report.

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† "pi" is the symbol used to indicate "pleural thickening in the interior fissure or mediastrum"

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**Co" is the symbol used to indicate on "abnormality of cardiac size or shape."

**This is the symbol used to indicate on "other significant abnormality."

**Min"is the symbol used to indicate a "calcification in small pneumoconlotic 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. 35 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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