Original Paper

Indoor and Built Environment

Indoor Built Environ 2008;0;0:1–34

Accepted: April 28, 2008

The California Coalinga Chrysotile Miners and Millers – Further Evidence for a Lack of Attributable Disease including a Refutation of Egilman and Roberts's [2004] Claims

E.B. Ilgren*

Suite No 503, 830 Montgomery Avenue, Bryn Mawr,[1] PA, USA

Key Words

Calidria · Coalinga · Chrysotile · Cohort · Egilman refutation

Abstract

The health risks to humans from exposure to ''pure'' short-fiber chrysotile, the most common form of asbestos, have been the subject of considerable debate. Identification of three cohorts of miners and millers exposed to amphibole-free short-fiber chrysotile has enabled this issue to be addressed. The findings detailed in this report strongly suggest that none of the exposed workers or others exposed para-occupationally or environmentally to the same form of asbestos, display any asbestos related disease.

In a letter to the editor of the International Journal of Occupational and Environmental Health in 2004 Egilman and Roberts claimed that 19 former miners and millers from one of these three cohorts developed asbestos-related disease due to exposure to Coalinga chrysotile. They also made other claims against the author's earlier work published in this journal (IBE). The data and findings in this report refute all of their claims. Egilman and Roberts also claimed that Union Carbide corporation concealed data from the NIEHS Coalinga chrysotile animal inhalation studies. This report shows that if there were any concealment of data it would have been by various government workers, not Union Carbide.

Introduction

In 1964, as a result of international concern about the carcinogenic effects of asbestos exposure, the International Union against Cancer (UICC) called, as a first priority, for studies of miners and other populations exposed to only one type of fiber [1]. Since that time, the health risks to humans from exposure to ''pure'' short-fiber chrysotile, the most common form of asbestos, have continued to be the subject of considerable debate. This has been due,

Los Angeles, London, New Delhi and Singapore
DOI: 10.1177/1420326X08094924
Accessible online at http://ibe.sagepub.com

Dr E.B. Ilgren,
Suite No 503, 830 Montgomery Avenue, Bryn Mawr, PA, USA 19010.
Tel. (610) 525 5960, Fax (610) 520 1156. E-Mail: dredilgren@aol.com
*The author is presently not affiliation with Bryn Mawr College.

in no small part, to the difficulty in identifying a type of amphibole-free short-fiber chrysotile to which cohorts of miners and other populations may have been exposed.

Coalinga chrysotile[1] is an amphibole-free form of short-fiber chrysotile [2,3]. Geological [2], mineralogical [2], hygiene [3–5] and experimental animal [6–10] evidence indicate Coalinga chrysotile lacks biological activity. This report provides human evidence that further supports Coalinga chrysotile's lack of biological activity including a detailed refutation of Egilman and Roberts' [11] claim that 19 former Union Carbide Coalinga miners and millers developed asbestos disease due to Coalinga chrysotile exposure.

The Coalinga Chrysotile Miners and Millers

Coalinga chrysotile was mined and milled by Union Carbide (UCC), Johns Manville (JM), and the Atlas Corporation. Work commenced at all three facilities in 1960 and the mining and milling operations ceased at JM in 1974, at Atlas in 1980 and at UCC in 2003[2]. The three work forces combined comprised approximately 1000 workers {UCC ($n = 460$); JM ($n = 275$); Atlas ($n = 390$)). Formal epidemiological cohort studies have never been conducted of these workforces. However, there has never been a confirmed case of attributable disease due to occupational, para-occupational, or "environmental" exposure to Coalinga chrysotile including those either residing near[3] and/or "recreating" [5,12] on the New Idria Serpentinite in which it forms [2]. The current health status of the Union Carbide workforce is described below and the 19 cases of alleged asbestos disease in that workforce are detailed in Appendix 1.

The Union Carbide Coalinga Chrysotile Miners and Millers

The Union Carbide Coalinga chrysotile mine was located at ca 4000 feet about 60 miles east-north-east of King City, California, the site of the mill. Mining was accomplished by scoop shoveling. Coalinga chrysotile lies loosely on the surface of the earth for many square miles and the terrain looks like fields of snow (see Figure 2(a) in [2]). To the naked eye, Coalinga chrysotile resembles "Lux laundry powder" in which the fibers are not macroscopically visible. This makes it totally distinct from most other types of chrysotile that are embedded as fibrous veins in dense host rock and have to be recovered with dynamite (detailed discussion in [2]).[4]

Annual health surveys of the UCC work force were conducted from 1970 to 2003. Dr Duane Hyde, Director of the Monterey Health Group, conducted a clinical survey of the UCC – King City Asbestos Corporation (KCAC) workforce in 1994 (see Hyde, [13–15] and Kumar [16]). Testimony by Dr Hyde in 2003 [17] (in: Szabo vs. Bindex et al. 5 Dec 03) revealed no evidence of asbestos disease in this workforce. In 1985, Dr Hilton Lewinsohn conducted a radiological survey of the UCC workforce as part of the sale of the UCC holdings to KCAC. The Lewinsohn report [18] also found no evidence of attributable asbestos related disease in the UCC workforce.

In 1980, McDonald and McDonald [19] performed a North American mesothelioma mortality analysis and failed to note an excess in the area around the New Idria Serpentinite. (The paper actually refers to "a chrysotile mining area in California." Mc Donald (pers commun., 1996) has said this was the New Idria area.) Coleman [20] and Ilgren [6] (and Ilgren, also, in an unpublished presentation to the International Mesothelioma Group, 1997, Philadelphia) have said disease due to Calidria asbestos had never been reported in this area. The California Cancer Registry (CCR) Public Use and Population Files (1988–1996) failed to identify a mesothelioma excess in the three counties in which the New Idria Serpentinite is found. This was confirmed in 2002 by Dr Lisa Scott (Scott pers. commun., 2002) of the CCR headquarters in Sacramento who found the mesothelioma incidence in Fresno, Monterey, and San Benito counties from 1988 to 2000 to be at "background" levels for men (1.1–1.8) and women (0.5) with no cases younger than 50 years of age. In 2002, Dr Paul Mills, the Director of Epidemiology at Region 2 of the CCR performed a descriptive epidemiologic analysis of mesothelioma in Fresno County (1988–2000), an area that encompasses some of the New Idria Serpentinite and serves as a significant catchment area for the region. A total of 83 residents of Fresno County California were diagnosed with mesothelioma. These cases generated an annual age adjusted incidence rate of 1.5/100,000 in males and 0.4/100,000 in females which are similar to national rates published by the "Surveillance Epidemiologist and End Results (SEER)" program of the National Cancer Institute [21]. Moreover, the distribution of the year of diagnosis of mesothelioma indicated that the actual age adjusted rates of mesothelioma had not changed appreciably (Table 1).

Plumlee et al. [22] from the US Geological Survey, Region 8 did not find an asbestosis excess in the region of the New Idria Serpentinite after examining national age-adjusted rates of asbestosis-related mortality by county for U.S. residents age 15 and over for 1970–1999. Plumlee et al. [22] also failed to find a mesothelioma excess in the same area on a combined epidemiological and geologic

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1–34] [Page No. 2] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

Table 1. Date of diagnosis of mesothelioma cases in Fresno Co., 1988–2000*

Year of diagnosis	No.	Percent (%)	Rate/100,00
≤1990	23	27.7	1.4
1991–1995	28	33.7	0.9
≥1996	32	38.6	1.0
Total	83	100	

*Some 80.7% of mesothelioma cases in Region 2 were histologically confirmed; 16.7% were diagnosed on the basis of positive exfoliative cytology; one case was based solely on clinical diagnosis and one only upon radiographic studies.

map (see Figure 11 in [22]) allegedly showing spatial correlations between ultramafic rock and mesothelioma rates by county in California.[5] Pan et al. [23] conducted a cancer registry based, case-control analysis, to assess a possible association between residential proximity to naturally occurring asbestos with the incidence of malignant mesothelioma in California. Such an association was not found in the area of the New Idria Serpentinite

Litigation against UCC prompted additional investigation into the health effects of Coalinga chrysotile. Nearly 20 years of intensive ''legal discovery'' have also failed to find evidence of attributable disease. In 1992, a third party action against UCC by the Conwed Corporation sought information about the health of the UCC miners and millers but after 11 years failed to identify any evidence of attributable disease [24]. In 1995, a large property damage case against UCC filed by the Chicago Schools system and a mass consolidated personal injury action filed in 2003 in West Virginia [25] also failed to reveal any evidence of attributable disease. Dr Peter Barrett conducted a detailed radiological review of the UCC miners and millers based on personal study of chest X-rays, medical records, and the Lewinsohn report [18] and correspondence with Hyde [13–15]. He also failed to find attributable disease [26]. In 2003, Kelly Moore Paint (KMP) [27] filed a large personal injury claim against UCC. To support their action, KMP retained Dr David Egilman to review the UCC asbestos document repository with a view towards, amongst other things, identifying attributable disease in the UCC miner and miller workforce.

In early 2004, Egilman and Roberts [11] published an eight page ''Letter to the Editor'' that allegedly identified 19 attributable cases of asbestos disease in the UCC workforce. Shortly after the ''Letter'' was published, Dr Egilman was deposed by lawyers representing UCC [28] during which he testified about various points made in the ''Letter.'' This report critically analyzes Egilman and Roberts' [11] ''Letter'' and Dr Egilman's testimony. The findings of this critical analysis demonstrate that not one of the 19 cases is a bona fide example of asbestos disease due to Coalinga chrysotile.

Materials and Methods

UCC – KCAC Medical records, death certificates, reports by Lewinsohn, Hyde and Barrett, a list of UCC baggers, long term (>20 years employment) UCC workers, and several letters written by OSHA to UCC workers were reviewed.[6] Additional information was obtained through discussions with UCC KCAC management, miners and millers; first hand inspection of the UCC mine and mill; and discussions with numerous other parties interested in and at times responsible for decision making regulatory efforts related to Coalinga chrysotile.[7]

JM Medical records and hygiene data were obtained with the assistance of the Marsh law firm in Denver and a local consultant Mr Dennis Christiansen. Former JM senior scientists and senior management,[8] medical personnel formerly overseeing the JM Atlas Coalinga operation,[9] as well as JM[10] and Atlas[11] miners and millers provided additional information. Atlas senior management, outside counsel, carriers, and successors also provided information about the miners and millers.

KCAC initiated a limited long term informal follow up of the UCC work force further to a request made by the author in 1999. Some of these data have been used in this report. Work history information for the 19 cases in Appendix 1 was provided by KCAC.

For the case control analysis, the records of the CCR Region 2[12] were assessed by Dr Paul Mills to identify cases of mesothelioma in Fresno County from 1988 through 2000.

For the Egilman and Roberts' [11] refutation, the individual ''comments'' cited by these authors for each case which allegedly represent attributable disease have been placed in italics underneath each worker number. This is followed by the information used to specifically refute each claim (see Appendix 1).

The symbols in the ''comments'' are those used by the International Labor Organization (ILO) as are typically found on ''ILO B reading'' radiology forms. The explanation of these symbols is shown in Appendix 2.

Egilman and Roberts [11] based their ''comments'' on the ''King City Asbestos Mill Surveillance Program'' Report and the ''Lewinsohn Report.'' Egilman's testimony [28] was based on these materials and others found in the

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 3] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

Union Carbide Document Repository received whilst serving as an Expert for Plaintiff in litigation against Union Carbide.

Overall Summary of the UCC – KCAC, JM and Atlas Coalinga Chrysotile Miners and Millers

The UCC – KCAC operations were situated on the northern part of the New Idria Serpentinite. UCC – KCAC employed 492 miners and millers. The records consisted of 460 folders, one per worker. Medical and employment data were only available for 375. Clinical records were available for 212, radiological records for 329, pulmonary function test data for 282, and smoking history information was available for 104. No information was available for 76 workers. No evidence of disease due to Calidria chrysotile was found in these records. It should be noted that the records included 37 men who worked 20 or more years at UCC – KCAC at least 9 of whom also worked in bagging; the dustiest part of the facility. Only one claim by a former UCC worker has been made in relation to Calidria exposure. The former employee who had worked in the KCAC mill thus alleged in Sept. 2000 that he suffered from pleural disease due to exposure to Calidria. However, in May 2002 he voluntarily dismissed the case against UCC.[13] Some believe the failure to observe disease in the UCC – KCAC workforce was due to the use of a wet processing method that kept fiber levels low. However, the UCC processing method was not exclusively wet since dry extruders and other methods were employed at different stages of the process.[14] In consequence fiber levels as high as 80 f/mL were measured in certain areas of the plant [5].

The JM and Atlas operations were located in the Southern part of the New Idria Serpentinite (also see Figure 6 in Ilgren [29]). JM employed 279 miners and millers. Clinical records were available for 161, radiology records for 141, and pulmonary function data for 21 workers. No information was available for 108 men. No evidence of disease due to Coalinga chrysotile exposure was found in these records. Atlas employed 239 workers, but no medical records were available for review.[15] Former JM miners, millers, senior management, and medical personnel formerly looking after the JM and Atlas workers corroborated the apparent lack of attributable asbestos disease. Particularly poignant testimony [30][16] was given by Mr John Davies, former mining manager of JM Coalinga who had also worked at Atlas. Mr Davies had, in fact, opened the JM facility in 1960 and closed it in 1974. Mr Davies told EPA IX about the health of the JM Atlas miners and millers at an EPA sponsored Community Meeting on May 30, 1990 in Sunnyvale, California. Davies lucidly described the extremely dusty conditions at both operations in discussion with an EPA Superfund toxicologist, Greg Hiatt. Davies said he was unable to name a single worker with asbestos disease 30 years after the operations started and 16 years after they closed Atlas senior management, outside counsel, carriers and successors did not know of any compensation claim awards made to Coalinga chrysotile miners and millers. The JM and Atlas operations used an exclusively dry processing method. Despite the high fiber levels with readings frequently in excess of 20 f/mL[17] there has been no evidence of attributable disease.

Finally, extensive discussions were held with various governmental agencies including though not limited to EPA IX, the Cal BLM, Cal EPA, and the ATSDR None knew of attributable disease in the Coalinga miners and millers.

Critical Summary of the UCC Cases cited by Egilman and Roberts [2004]

The results of the analysis of the 18 cases cited by Egliman and Roberts [11] are summarized in Appendix 1. One case (#549) was not analyzed since he only worked at UCC for one week and was then dismissed on the grounds of ill health.

Latency

Regarding asbestos related diseases, virtually all of the cases displayed insufficient latency. This was so for 13 out of 18 cases from the time the UCC Calidria operations began and for 16 out of 18 cases from the time they began working at UCC.

Asbestosis

None of the cases demonstrated asbestosis. Clinical, spirometric and/or radiological evidence refuted this claim. Only one case actually reached a profusion score of 1/1 diagnostic of asbestosis [31]. However, numerous films before and after that film was taken were normal. Since asbestosis does not ''regress,'' the diagnosis was not compatible with asbestosis. ''Regression'' was also seen in ten other cases.

Four cases were deceased. The Death Certificates of three did not list asbestos related disease as a significant contributor to the cause of death. Autopsy findings were available for one deceased worker. These failed to confirm asbestosis on histological study.

There was sufficient alternate causation in each case to explain the observed parenchymal findings.

These included various types of endemic chronic granulomatous disease (e.g., coccidiomycosis or "Valley Fever,"[18] histoplasmosis, and tuberculosis); tobacco abuse; different sources of industrial exposure (e.g., silica, fumes, and commercial amphibole asbestos from the large number of mines and oil wells in the Coalinga area[19] where the men may have worked before and/or after joining the UCC – KCAC workforce); and the complications of pneumonia and chronic heart failure.

Non Malignant Pleural abnormalities

Nine cases allegedly demonstrated pleural abnormalities due to Calidria asbestos exposure. None showed bilateral pleural plaques, the only pleural abnormality clearly linked with asbestos exposure [32], aside from the extremely rare examples of very severe bilateral pleural thickening [33]

Only one case (#383) had bilateral pleural abnormalities but these were adhesions, not plaques. The failure to find plaques was also confirmed by Professor Peter Barrett at Tufts and Professor George Jacobsen at UCLA. Seven cases demonstrated unilateral pleural abnormalities and alternate causes could explain them all.

Lung cancer

Five cases "suspicious" for lung cancer were claimed to be due to Calidria exposure. Four were not lung cancers since they survived more than ten years after diagnosis. The one proven case of lung cancer had no evidence of underlying asbestosis so it could not have been attributable [34]. This case was clearly due to tobacco abuse and possibly exposure to gases, diesel fumes and solvents.

Egilman and Roberts' (200) Claim of Asbestos Disease in the UCC Workforce

Egilman and Roberts [11] claimed 19 Union Carbide Coalinga chrysotile miners and millers had asbestos related disease. Critical analysis has shown not one of them was due to Calidria asbestos or asbestos of any kind. The specific areas of criticism include the following.

Egilman's Testimony

Egilman undermined his own proposal. Thus, "Q. Just as an overview question, is it your opinion, sitting here today, to a reasonable degree of medical certainty, that all of these individuals who you've listed in Table 1 (of the rerport) in fact had an asbestos-related disease? A. No." Egilman said that despite this admission, "the one proven autopsy case supports, by itself, that Calidria caused asbestos disease in miners and millers at the facility."

Latency

Nearly 90% of the cases can be excluded on the grounds of insufficient latency. Egilman himself supported the notion that latency could have been insufficient saying "there are three or four (cases) whose latent period is probably too short." Egilman testified that he could not verify latency because he was not able to get workers' names and identification due to limited access to medical records and other UCC documents. However, it would appear that Dr Egilman had access to the same materials as Dr Barrett and the author. Egilman almost certainly saw everything he needed to dismiss most of the cases on the basis of insufficient latency.

Asbestosis

Egilman said not "many legitimate pathologists would say that someone with 28 years of exposure at a mill, bilateral pulmonary fibrosis, bilateral pulmonary plaques with fibrosis on path with a chrysotile exposure only is anything but asbestosis." The "autopsy" case (#383) did not have asbestosis. The medical examiner did not subvert the case. He was not under some type of corporate "influence" nor was he incompetent.[20] The absence of asbestosis was independently confirmed by three senior pathologists, Dr Tom Colby (see above), Dr Allan Gibbs,[21] and Dr Victor Roggli.[22] The findings were attributed to, amongst other things, sepsis induced Adult Respiratory Distress Syndrome (ARDS) secondary to heart surgery and cardiac disease. Egilman admitted both conditions could provide alternate explanations for the observed fibrosis.

Egilman's claims concerning the criteria used to diagnose asbestosis are not legitimate and he is not competent to render such opinions. Egilman is not a qualified pathologist. He is not in a position to say asbestos bodies are not required to render a diagnosis of asbestosis. This is the widely accepted and practiced standard in the medical community. When Egilman said "The coroner was apparently under the impression that the diagnosis of asbestosis required the finding of asbestos bodies," the Coroner's impression was correct. Egilman suggested asbestos bodies were not needed in this case since he claimed a "secret" 1971 UCC Calidria animal study [35] failed to produce asbestos bodies. The secret 1971 "Mellon study" did not produce asbestosis. It produced severe nonspecific fibrosis. This was due to the massive doses used, the nonphysiological (intratracheal) method employed, contaminated starting materials and pulmonary infection. These and other criticisms i.e. inadequate controls, paucity of animals in each group, and the failure to histologically study all but a few animals, have been described by Ilgren

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 5] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

and Chatfield [6]. The proposal that human pathologists should alter accepted diagnostic criteria[23] on the basis of one animal study is as absurd as asking epidemiologists to confirm causation on the basis of a single case.

Lung Cancer

Egilman admitted he did not know what the normal expected number of lung cancer cases would be in a cohort the size of the Calidria workers. He also admitted he did nothing to determine if the possible lung cancer cases identified by Dr Lewinsohn were ever confirmed through follow up.

Benign Pleural Abnormalities

Egilman admitted the bilateral plaques allegedly found in the autopsy case were ''pleural thickenings.'' In fact these were just post-inflammatory ''adhesions.''

''Obstruction''

The obstructive findings could all be explained by alternate causes. The American Thoracic Society (ATS) [36] statement suggested asbestos could significantly contribute to obstructive findings. However, their statement is replete with contradictions on the point. Thus, under ''Evidence of Abnormal tests'' (Table 1: ''Criteria for diagnosis of non-malignant asbestos related disease''), the ATS [36] refers to obstructive patterns ''in context.'' It then says ''airway obstruction may be seen alone in nonsmokers who have asbestosis'' but ''isolated obstructive impairment is unusual.'' The ATS [36] admits that ''large airway function reflected in FEV1/FVC is generally well preserved'' and ''the role of asbestos as a cause of airway obstruction has been controversial.'' The ATS statement also says that while asbestos could be a specific cause of obstruction, other more viable explanations exist including nonspecific changes due to large dust burdens as noted with most inorganic dusts, the frequent association with exposure to other agents affecting airways, confounding by smoking and normal age related changes. It concedes that ''in general, the magnitude of the asbestos effect on the airway function is relatively small'' and that ''this effect by itself is unlikely to result in functional impairment or the usual symptoms and signs of COPD.'' The ATS statement even suggests, contrary to the expected progression of changes, the ''physiological findings associated with airflow obstruction e.g., the reduction in the FEV/FVC ratio, become less prominent as asbestosis progresses; this may reflect increased pulmonary recoil.'' Finally, the statement said that although there may be an interaction between smoking and asbestos exposure in the development of airway obstruction, this has only been demonstrated in animals, not in humans.

Mesothelioma

Egilman admitted he was unaware of a single case of mesothelioma amongst any former ''Union Carbide miners or millers.''

Overall Discussion of Findings: The Coalinga Chrysotile Miners and Millers

Nearly 1000 Coalinga chrysotile miner and millers worked at the UCC-KCAC, JM and Atlas operations. Conditions were often very dusty (Davies, pers. commun.) ([5]; also see ''Under a Cloud,'' 25 Sept 04, LA Times [37]). Many men worked in these dusty positions long enough for them to have developed an asbestos disease if the dust was pathogenic. Minimal exposure durations for mesothelioma have been set at ~2 weeks [38] and for Asbestosis ~ 1 month [39]. More than enough time has elapsed since these men were first exposed for asbestos related disease to develop.[24] However, to date, no asbestos related disease has been identified.

Conclusion

Calidria cannot produce asbestosis, lung cancer (for which asbestosis is a prerequisite [34]), or mesothelioma. Calidria chrysotile's purity [2], rapid rate of clearance [8,40,41] enhanced solubility [5], reduced respirability [4] and other unique features [3–5] explain its inability to produce disease. The human observations cited in this report fully support this view.

Potential Ascertainment Bias

The human observations noted herein, whilst incomplete, still add much further weight to the notion that Calidria chrysotile is innocuous. The failure to find one single proven mesothelioma attributable to Coalinga exposure in the former Coalinga chrysotile miners or millers is particularly telling despite the lack of formal follow up. And the failure to find attributable disease has not been due to poor ascertainment. The workers generally stay in the area (Kleber, pers commun.) and the ''local landscape'' has been extensively scoured for potentially compensable cases. The search has been greatest for mesotheliomas and the failure to identify one single attributable case, particularly in the last twenty five years, is especially noteworthy. During this time, the public has been and continues to be made aware of the association between mesothelioma and asbestos in newspaper and television adverts as well as at local public and

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 6] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

union meetings. Requests are continually made to report suspected mesothelioma cases to the Plaintiff bar. None has been found. One year after KCAC closed, the LA Times interviewed workers regarding fear of future disease. None of them named a single bona fide case of disease due to Calidria chrysotile exposure including mesothelioma [37]

The failure to identify a single attributable mesothelioma in the Coalinga chrysotile miner or miller workforce, their families, and/or in those seriously exposed "recreating" on [12] or residing near [42] the New Idria Serpentinite also cannot be due to inadequate regional surveillance. The CCR in Sacramento is one of the largest epidemiological units in the world. All cases of mesothelioma reported in the State are recorded in the CCR. However, the three counties covering the New Idria Serpentinite do not display a mesothelioma excess.[25]

Populations at Risk for Analysis

Some may argue the "population at risk" is simply too small for statistical analysis. That is not true. Nearly 1,125 men worked for at least several weeks at one of the three operations. All of the JM and Atlas workers (665/1165) ceased work 33 years ago. At least 75% of the UCC – KCAC workforce (350/460) were last exposed 20 or more years ago. The population at risk with sufficient latency (>20years) would be ca 1000 men. Thus, if the expected rate of mesothelioma is ca 2 per million per annum, at least four "background" cases should have occurred in the workforce unrelated to asbestos exposure. However, if Calidria chrysotile and amphibole asbestos were equally toxic for mesotheliomas, ca 9% or 90 mesotheliomas should have been found in the Coalinga chrysotile miners or millers, 86 being attributable to asbestos exposure (see for example [43]). However, none has ever been identified.

The actual population at risk from occupational exposure to Coalinga chrysotile from mining and milling on the New Idria Serpentinite could, in fact, be in the tens of thousands. This is evident from the fact that no less than 94 active mining sites were identified by EPA IX's superfund contractors (Figure 1(a) and 1(b)). These men worked the deposit not just for asbestos but also for chrome, mercury, graphite, and other minerals as well (to be discussed in a future report). In fact, the New Idria Deposit contained some of the largest mercury and chrome mines in the world (Figure 1) (also see [44]).[26] Their net area and theoretical degree of disturbance potential would far exceed the comparatively few asbestos mines.[27] Large amounts of crude oil are also extracted near the deposit by a number of companies and the extensive drilling causes the release of much asbestos (Divine, pers commun., Mar, 99). Workers are routinely screened for asbestos related disease but to date no mesothelioma excess has been found (Divine, pers commun Mar, 99; [45]).

The actual population at risk may be larger still if one includes families of those who ever mined or milled materials containing Coalinga chrysotile and those "recreating" on, residing near or agriculturally tilling the New Idria Serpentinite or nearby areas.

Potential for Diagnostic Confounding

The failure to identify a single attributable mesothelioma in the Coalinga chrysotile miner or miller workforce, their families, and/or in those seriously exposed "recreating" on or residing near the New Idria Serpentinite is almost certainly not due to misdiagnosis over the last 25 years. The regional hospitals and nearby tertiary referral centers have excellent diagnostic facilities.

Coalinga chrysotile can be used safely[28]

The net weight of evidence clearly indicates Coalinga chrysotile can be used safely. Its withdrawal from the market was unjustified. "Risks" were actually enhanced, not lowered (also see Maines [46]). Calidria was suited for commercial purposes where many other types of chrysotiles could not be used either at all or without extensive processing. The original UCC R & D team showed that Calidria could, amongst many other things, rapidly purify water sources (Skvarla pers commun., 1996) and possibly serve as a highly efficient fire retardant (Skvarla pers commun., 1996). Millions of cubic meters of Coalinga chrysotile still exist today [20]; a truly inexhaustible supply. It can be obtained at virtually no cost. However, scientifically unjustified regulatory actions have prevented the State from using this valuable asset and from possibly alleviating some of its most pressing problems: clean water and fire control.

Calidria chrysotile has also been shown to have the potential to produce unique materials such as nanomatrices [47]. It can be used as a dielectric host matrix, since Calidria's central pores, are totally free of matrix filler [2] and of a highly uniform diameter [4] and can be filled under pressure with molten metals including Hg, Sn, Bi, In, Pb, Se, and Te to form regular systems of ultrathin parallel filaments [47]. These thin metal and semiconductor filaments have thermal conductivity properties radically different from those of their constituent bulk materials.

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 7] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

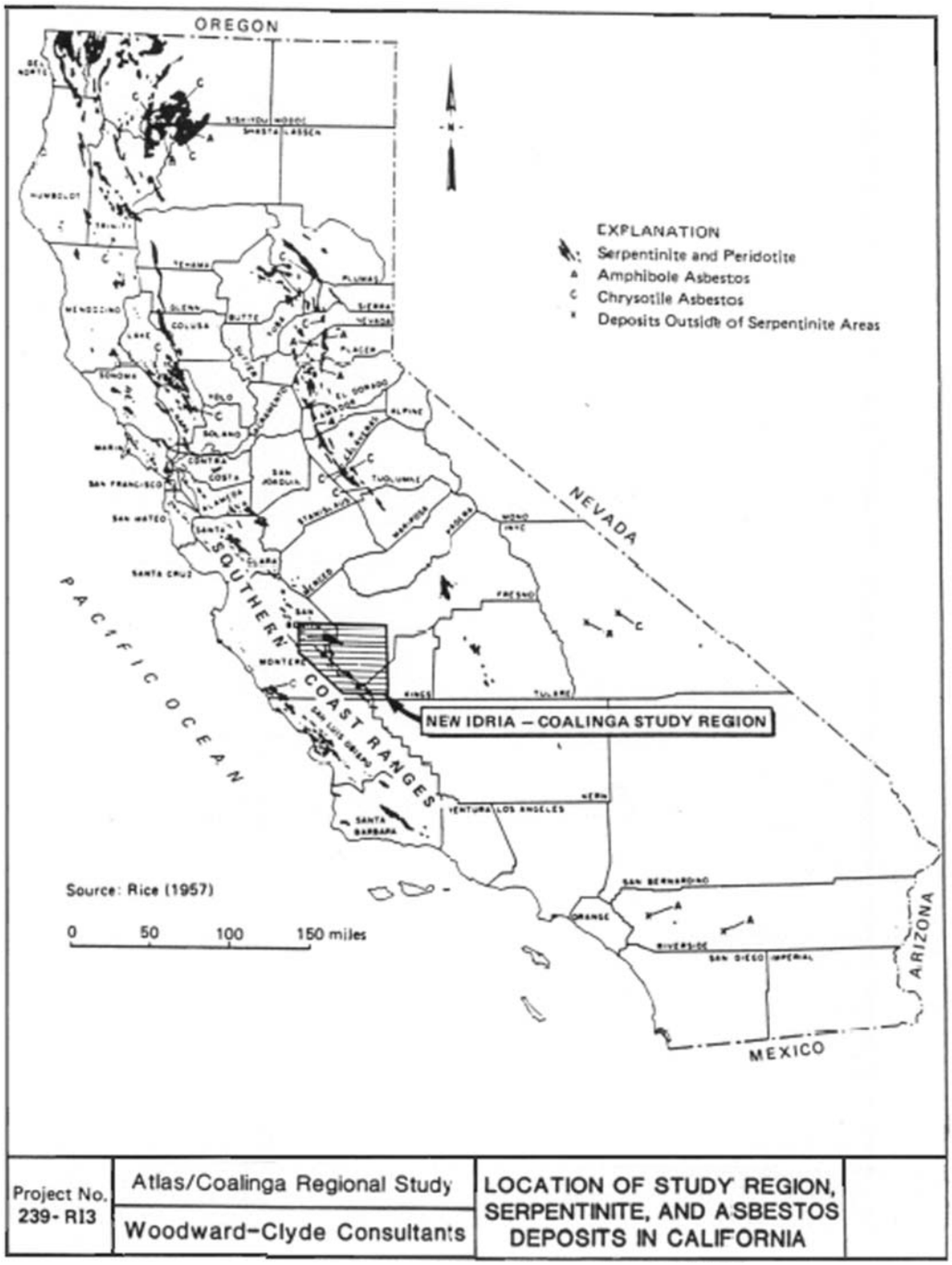

Fig. 1. (a) Serpentinite and asbestos deposits in California; (b) distribution of serpentinite and associated mines in study region. In total 94 active mining sites were identified by EPA IX's superfund contractors.

Refutation and Clarification of Claims made against Coalinga Chrysotile

Egilman and Roberts [11] in their report made various erroneous claims against the author about the ''Caldria'' papers he published in this Journal. The following section refutes and clarifies many of these claims.

Financial Support

Egilman and Roberts [11] claimed the author misrepresented his financial relationship with Union Carbide in earlier Calidria publications. The author never denied such a financial relationship. Dollar amounts were even stated in deposition in Conwed vs. UCC (1998) [48]. The author

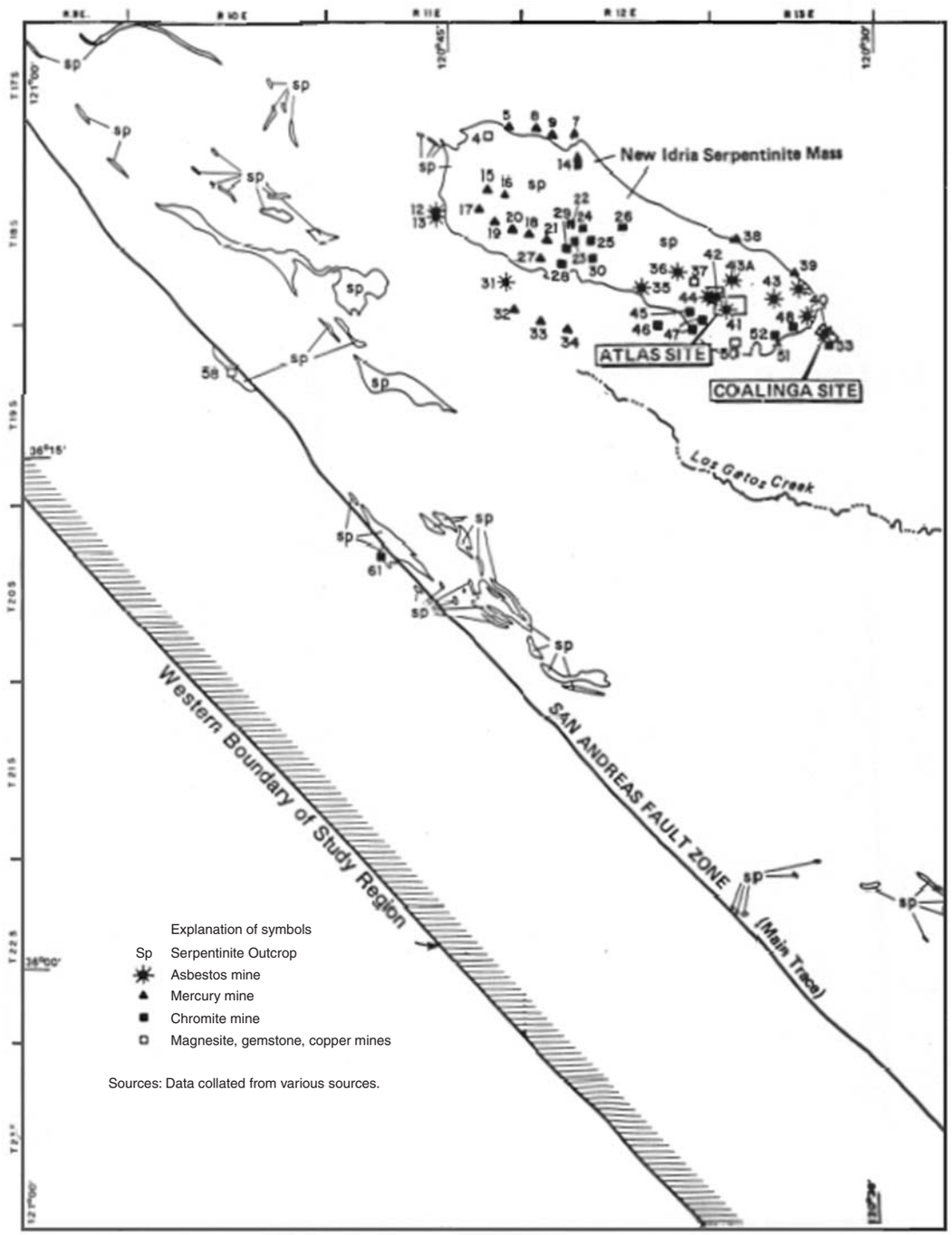

Fig. 1. Continued.

never received funds to specifically access, review or write up those studies. Researching and writing these papers entailed thousands of dollars in personal out of pocket expenses that the author was never obliged to spend on furthering Calidria research. He chose to do so since UCC refused to underwrite such research activities and there was no other way to get the work done.

By contrast, Egilman never told the readership of IJEOH, where he published the report, he ''was paid to read the documents'' used to produce his Letter.[29] Egilman and Roberts [11] merely said ''Many of the documents cited in this letter to the editor were produced in litigation where one of us (DE) was a consultant to Kelly Moore Paint in: Kelly Moore Paint Company vs. Dow Chemical

Corporation et al, No. 19785-BHO'' ([11] footnote on p. 99). Egilman was paid significant consulting fees for the work he did against UCC on behalf of the Conwed Corporation against Calidria chrysotile. Egilman also never divulged the $75,000 to $100,000 annual retainers he received from at least seven major corporations (General Electric, Georgia Pacific, Owens Illinois, Foster Wheeler, Dana, Robert Celo, Galbest) to conduct ''Consulting work'' on other asbestos related matters.[30] This clearly breached the mandate of IJOEH that required disclosures by authors of ''affiliations . . . with any organization with a direct financial interest in the subject matter or materials discussed.''

The author's 9/11 work

Egilman criticized the author regarding the ''World Trade Center'' paper he published in this Journal [49] six years ago saying: ''I think the people in New York would want to know that he was recommended by Gerson who was a previous Carbide lawyer to do the testing of the asbestos''. . .'' that's something that should have been on the public record.'' The people of New York City elected Alan J. Gerson to the post of First District Councilman of Manhattan by a wide margin. Gerson's constituents were very familiar with his background by the time he asked the author to help with the 9/11 matter. Gerson did not reach out to the author to provide an opinion favorable to Union Carbide. He asked for a totally independent opinion and he got one. In fact, Gerson asked the author to work with various scientists from Mt Sinai at the first post 9/11 planning session on 9.14.01 to determine the risks to residents from the Ground Zero exposures. Gerson asked the author, in concert with Mt Sinai, not to tell the residential committee he believed chrysotile was much less dangerous than amphibole asbestos. The author obviously did not follow that advice. Instead, he produced a totally independent report summarizing his findings of the 9/11 environmental study. The author did not discuss his findings or opinions with Gerson after 9/14. He provided Gerson and Congressman Nadler's Environmental Task Force with a copy of his Final Report after it had been submitted for publication in December 2001. The following year, Gerson and the New York City Council sent the author a ''Proclamation of Thanks'' for the work he did on the 9/11 matter.

Egilman's UCC Consulting

Egilman claimed he consulted for Union Carbide on matters related to Bhopal. However, Union Carbide and their attorneys have no record of any such consulting work.[31] Egilman claimed he worked with Mr Bob Brownson on that matter in 1986. Mr Brownson was not working with Union Carbide at that time.

IBE as a ''Nonexistent'' Journal

Egilman claimed the Calidria papers were published in a non-existent journal (he called it ''Safe Building Alliance Journal''). This is obviously not the name of the journal and IBE is certainly alive and well.

Calidria as a non-regulated form of asbestos

Egilman claimed Ilgren & Chatfield [7] said Calidria is innocuous because it ''is an unregulated short-fiber chrysotile and because none of the Calidria mine or mill workers ever developed asbestos related disease.'' Ilgren & Chatfield [7] never said Calidria was unregulated. Calidria was regulated the same way other forms of asbestos were.

The Rat as an Unsuitable Model for Assessing the Toxicity of Asbestos

Egilman suggested the Calidria animal studies were flawed by inferring the rat was an unsuitable model for the assessment of asbestos toxicity. Egilman thus claimed the rat was 260 times less sensitive to asbestos than human beings. He did not cite a single reference to support his claim. If anything, the opposite could be true as shown by an extensive body of experimental work. Indeed, the work done by Davis and his colleagues at the Institute of Occupational Medicine in Edinburgh actually suggest rats may be even more sensitive than humans to asbestos inhalation exposure (reviewed by Ilgren [50]) The validity of the model has been solidly reaffirmed by the National Toxicology Program (NTP)[32] (e.g., see NTP Annual Report, 1991, [51] pg 40) which states: ''At NIEHS, the 2 year exposure studies in rodents remain the most definitive methods for identifying chemicals with carcinogenic potential for humans.''[33]

Selective Presentation of Data

Egilman said the author selectively presented Pinkerton's data. However, Egilman failed to cite a single full text paper by Pinkerton or any of his colleagues to support his claim.

Authorship Issues

Egilman claimed Pinkerton told him in August 2003 he ''refused to co-author these (Calidria) papers'' since he ''did not agree that Calidria should be considered a nuisance dust.'' This is patently untrue. Pinkerton (pers commun., 1995) did not co-author the papers since

James Crapo advised him against doing so[34] and he decided to take that advice.[35]

Pinkerton (pers commun., 2008) told the author that he found it remarkable that despite the very high continuous exposures the animals incurred over the first 12 months of the study, there appeared to be near steady state clearance of the Calidria fiber whilst the Jeffrey and the UICC/B continued to increase in the various tissue compartments under study. Pinkerton said this accounted for the acute tissue damage seen with the Jeffrey fiber in the first 12 months and the progression of lesions seen with the UICC/B.

Peer Review

The letter by Egilman & Roberts [11] is not a peer reviewed publication. It was only reviewed by the Editor of the Journal. The Editor is a senior member of the Collegium Ramanzini and a staunch supporter of the global asbestos ban

Corporate Concealment of Calidria Animal Data by UCC

Egilman claimed UCC concealed the Calidria animal data (also see Egilman's NIOSH 2007 testimony re "secret" UCC studies [52]). He does not say how UCC did this. The data and underlying materials were in the NTP archives ever since the study was completed in 1984.[36] The archive is a limited access facility. The archivists keep detailed records of those who use the archive. The Calidria investigation was completed in 1983. There is no record of anyone ever reviewing the Calidria studies after the investigation was completed aside from the author.[37]

UCC personnel never visited the archive or had any role in the initiation, construction, execution, or analysis of the study. UCC learnt about the NTP inhalation Calidria studies from the author. Rather, US governmental scientists appear to have concealed the Calidria inhalation data and other parts of the study.

Finding the Calidria Inhalation Study

The author first sought the underlying Calidria inhalation data in 1992. He asked Dr Gene McConnell where these materials were. Dr McConnell said they should be in the NTP archives and that he would contact the archivists to see if the data could be found. Dr McConnell said the archivists told him the data could not be found and must have been discarded as being "too old" to keep.[38] Over the next three years, the author asked Dr McConnell several more times to request the archivists to search for the materials. Dr McConnell told the author on each occasion the data could not be found in the archives.

By September 1995, the author in collaboration with Prof. Kent Pinkerton and Dr Arthur Morgan completed the analysis of the morphometric data. The morphometric data clearly showed Calidria was far less injurious than long fiber chrysotile. By contrast, the summary tables[39] of the morphological findings received from Pinkerton in December 1991 and originally produced by McConnell suggested Calidria was as fibrogenic and tumorigenic as the two long fiber chrysotile preparations [29].

Given the obvious discrepancies between the morphometric and morphologic findings, the author called the NTP archives himself in November 1995 and asked them to recheck the repository. The archivists told the author they had never received a request to look for this material before and three days later found the requested materials.

A memo produced further to a FOIA request filed in July 2007[40] suggested the Calidria inhalation materials had not been studied before Jan 1985. In 1992 Crapo [53] testified the study materials had still not been reviewed and the workers responsible for doing so "quit."[41]

It is nearly impossible to understand how these materials could not have been studied when the "McConnell tables" [29] and Governmental reports say Calidria is pathogenic. Thus, the "Final Report" submitted by Crapo in 1984 [54] (and see [29]) to the NIEHS and the statements submitted by McConnell to the 1982[42] and 1983 NTP Annual Reports said Calidria was both fibrogenic and carcinogenic. Earlier papers by the same group [55–58] all claimed Calidria was injurious.

Reasons why the Calidria Inhalation Study was not Properly Handled

The author has tried to determine for nearly 17 years why these materials were not processed through the appropriate review channels. Some theories have already been discussed [29]. Several scientists originally associated with the NTP asbestos inhalation study have offered additional explanations. Thus:

"Pre GLP" – The NTP asbestos inhalation study was conducted between 1978 and 1983. Some scientists believed the NTP inhalation study was not "formally" processed since the NTP was going through a "transition" period from non-GLP to GLP oversight during this time period. This suggests studies done during this time period were either not done under GLP conditions or at least not done to rigorous NTP standards since the mechanisms for doing so had not yet been put into place. This is not true. At that time it was stated that compliance may be with "GLP regulations or NTP standards (as specified by each contract)" [51] (pg 140 "NIEHS" "Quality Assurance Program").

First, the initial phases of the studies[43] conducted by the contractors (Becton Dickinson, Mantech[44] and Northrup) appear to have been rigorously carried out.

Second, ''earlier'' NTP studies not originally subjected to detailed Quality Assurance review underwent detailed retrospective auditing.[45]

Third, Calidria was studied by ingestion in great detail at the same time (1977–1983) as it was on test by inhalation. Both studies used the same Calidria sample. Some of the same scientists oversaw both studies. Indeed, Dr McConnell, the ''Chemical Manager,'' appeared to be one of the most senior people involved with the Calidria ingestion study responsible for the ''evaluation of experiments, interpretation of results, and the reporting of findings.'' Moreover, many different groups oversaw the ingestion study to be sure there was detailed full disclosure and review of the findings. These included but were not limited to the following:

The ''Pathology Quality Assurance'' Group – Drs Busey and Proctor.

The Pathology Working Group – Drs Boorman, Gupta, Lomax, McConnell, Reznik, Stinson, and Ward.

The NTP/NIH Staff Peer Review Committee – Drs Douglas, Grieshaber, Hart, Haseman, Huff, McConnell, Moore, and Tennant

The NTP Board of Scientific Technical Reports Review Subcommittee – Dr Hitchcock, Harper, and Whittemore.

The Expert Subcommittee Panel – Drs Shore, Breslow, Highland, Mirer, Murphy, Nielsen, Schwetz, Swenberg, and Williams. The panel members came from academia (UNC, Stanford, NYU, U Wash, U Texas, U Conn), industry (Dow chemical, CIIT), the Unions (IUUAW), and various Foundations of uncertain affiliation (Environ. Defense Fund, Amer. Health Foundation).

The Calidria ingestion investigation underwent detailed stepwise review including but not limited to:

Assignment to an ''Executive Committee'' and a ''Staff Scientist'' (For information on the duties of the Staff Scientist in ''chemical management,'' see Report [51] (p. 139) (Contact Person – Dr W Eastin). In essence they were to ''ensure the chemical received a complete scientific evaluation.''

Preparation of the ''Experimental protocol for the investigation by the Staff Scientist.

Presentation of the Experimental Protocol to the Toxicology Design Review Committee'' and at times to an ''*ad hoc*'' Panels of Experts assigned to assist the ''NTP Technical Report Subcommittee'' (NTP TRS)

Open session discussion by the NTP TRS.

Production of Executive Summaries and underlying documentation forwarded to the NTP.

Board of Scientific Counselors for review.

Requests for and incorporation of pertinent public input into Final Executive Summaries.

Various governmental groups played a role in this process including many of those listed below:

NTP – Executive Committee, Steering Committees, Board of Scientific Counselors, Technical Reports Review Subcommittee, and Discipline Leaders.

NTP NIEHS – Director and the Membership, Quality Assurance Group, Study Evaluation and Reporting Section, QA Discipline leader, Pathology Working Group, Toxicology Pathology Group, Respiratory Toxicology Group

NIEHS Environmental Health Sciences Review Committee (EF–HRC), Division of Biometry and Risk Assessment, Computer Technology and Statistics and Biomathematics Branch.

NTP EPA FDA NIEHS GLP Regulatory Compliance Groups.

NTP Member Health Research and Federal and State Health Regulatory Agencies.

Key agency staff at ATSDR, OSHA, CPSC, EPA, NCI, NCTR, FDA and NIOSH.

NIEHS Division of Toxicology Research and Testing (DTRT) Subgroups (NIEHS DTRT) –

Management Committee, Experimental Toxicology Branch, General Toxicology Group, Chemical Dispo-sition Group, Chemical Carcinogenesis Branch, Laboratory Animal Management Section, Experi-mental Carcinogenesis and Mutagenesis Branch, Systems Toxicity Branch

Environmental Pathology Lab (EPL) quality assurance

The Calidria ingestion findings were appropriately processed in accordance with the NTP guidelines for ''Information generation and dissemination'' [51] (pp. 157–160).[46] This included but was not limited to the production of two highly detailed NTP Technical Report documents [59,60] and the equally detailed paper published in the scientific peer reviewed literature [61]. These reports and papers were cited by the international community in publications (e.g., [62]) concerning the safety of ingested asbestos. The Calidria ingestion findings were uniformly interpreted as being ''negative'' or without adverse health effects.

"Negative" Data

Crapo [63] testified that "they" chose not to publish (see endnote 43) the Calidria inhalation findings since "we did not publish "negative" findings in the 1970s." This argument falls flat when one considers the following:

The Calidria ingestion findings although contemporaneous were negative but still published in great detail.

Other Calidria studies [64] and short-fiber chrysotile [65] experiments contemporaneous with the NTP – NIEHS Calidria inhalation investigation were negative but these too were published in detail.

An entire IARC WHO Scientific Monograph was written on the value of negative data in the late 1980s [66].

Indeed Crapo [63] testified it took 20 years to discover "the most important finding (of the entire Calidria inhalation study) was actually the negative finding."

Technical Problems

Some (Eastin, pers commun., 2007) said the study may never have been published since "they may not have been convinced the study was run the way it should have been." This infers the problem had to do with the Contractors, not the Pathologists. Everything goes against that idea. The contractor's quarterly reports and attendant documentation are in good order and large portions of the study that used the long "pathogenic" chrysotile fiber preparations (acute [67–74], sub-acute [75] and chronic (e.g., morphology: [76–78]; e.g., morphometry: [79–81]) portions of the study were published in many different journals.

"Private" Study

Some (Boorman, pers commun., 1996, 2007; Busey, 2007 pers commun.) have said Dr McConnell, then the head of NTP pathology, made the Calidria inhalation investigation his own "private" study.[47] Since Dr Arnold Brody[48] and Dr James Crapo[49] also oversaw significant parts of the study and worked together on various segments of the investigation, they too were also probably part of this alleged "privatization" process. Various activities Brody conducted according to certain memoranda[50] support this.

Busey (pers commun., 2007)[51] argued McConnell was not obliged to turn the study over to the Government since he "was" the Government. Dr McConnell was not "the Government." He was a governmental employee. He did not fund the work. The study could no more be legitimately "privatized" than discarded for being too old (see above). Contractual agreements legally obliged these workers to provide the findings of the study to the Government in a timely, comprehensive, accurate, and detailed manner.[52] McConnell and his colleagues had no right to give the Government corrupted, incomplete, and highly misleading information [29] and to bury the primary study materials so they could not be independently and honestly reviewed, keeping the information from being disseminated through the scientific community.

The Calidria inhalation and ingestion studies were treated totally differently. The Calidria ingestion study underwent a highly rigorous open review process. The findings were widely published for the world to see. The Calidria inhalation study was "privatized," corrupted and then relegated to the bowels of the NTP archive. The Calidria inhalation and ingestion studies should both have undergone the same rigorous oversight. If that had happened, the numerous irregularities in the inhalation study [29] would have been detected and corrected.

According to Dr Gary Boorman,[53] the Calidria data would never have seen the light of day if the author had not found them. Given the difficulties attendant in locating the material, their discovery can only be likened to the discovery of the "Dead Sea Scrolls."

Mysteries and Motives

The role of fiber size in biological outcome was a central issue in this study and of great importance generally when the study was done. More than 20 years ago, Selikoff and his colleagues claimed short-fiber chrysotile was widely distributed from numerous natural (Coalinga chrysotile from the New Idria Serpentinite: [82]; Rockeville Maryland quarry: [83,84]) and commercial (Brakes: [85]; Spackling compound and Dry Wall: [86]) sources and was potentially very dangerous [87]. By 1980, the views of Selikoff and his colleagues were widely asserted by IARC [88], the EPA [89] and extensively cited by the NTP NIEHS as reflected in the "Introduction" to the Calidria feeding study [60].[54] The presence of three labor union scientists on the Calidria ingestion study Expert Review Panel was also suggestive of Selikoff's possible influence.[55]

Selikoff was integral to the establishment of the NIEHS (Wagner, pers commun., 1996). Not surprisingly, he appeared to exert great influence on the institute and its grant decision making policies (see "NIOSH Siberian study," report in preparation). In July 1981, the entire Carcinogenesis Bioassay Testing Program was transferred from the NCI to the NIEHS [60]. By 1981, the Calidria inhalation study, started in 1978, was nearly complete and ready for detailed final analysis. From 1981 onwards, the primary study materials should have undergone rigorous detailed review. Most mysteriously, this did not happen. Instead, the entire study was "privatized." In 1982 and 1983,

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 13] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

McConnell sent erroneous statements regarding the injurious nature of Calidria to the NTP which appeared in the Annual reports for those years [90,91]. In 1980 and 1982, Brody and Crapo also presented erroneous statements at meetings held by the ATS [56] and IARC [55] and in 1984 Crapo presented the "flawed" Final Report to the Government [54] (and see [29]). In ca. 1982, these workers also tried to publish full versions of these data in peer reviewed journals. The one manuscript describing the morphometric findings was rejected [92] (cited in the "Final Report" [54]). The other describing the morphological findings cannot be found [57] (cited as "reference 3" in McConnell et al. [77]).

Accountability problems were not limited to the chronic Calidria inhalation study. The long term (12/24 month) follow up data for the acutely exposed animals also "disappeared." Crapo said (Henderson pers commun., 1996) he had the 12 month follow-up tissues and data [29]. A sacrifice schedule in the Becton Dickinson "Final Asbestos Study" (ca. 1981) [93] indicated a 24 month follow up was also done but the archivists have not been able to find the underlying materials or data sets. The acute data have been the mainstay of Brody's testimony. Brody has not admitted to following up the study longer than 6 months. Since Crapo said these longer term follow up studies were negative, full disclosure of these results would seriously undermine Brody's testimony.

Crapo has refused to release the long term follow up materials (Henderson pers commun., 1996). He also refused to offer any opinions about Brody's animal studies[56] aside from saying Brody did important work [94], was a superb investigator [95] and was one of his closest friends [95]. His refusal to release the underlying materials or comment about Brody is not very surprising. Brody awarded Crapo the subcontract [96] to do the morphometric analyses of the inhalation materials and this award formed the foundation of most of the asbestos studies Crapo ever produced. Brody also continued to support Crapo's applications for other large more recent grants some amounting to millions of dollars: Crapo – ozone grant award notice, 1979 [97]; Brody to Crapo, September 16, 93;[57] NIH Grant 1992 [98]

If the NTP asbestos inhalation study had been fully disclosed and objectively analyzed in a timely way, the failure to find attributable disease would have negatively impacted Selikoff and Mt Sinai, among others as well as the Plaintiff bar and the testimony of their Experts very seriously. It would also have undermined many of unscientific notions that underpin the science the EPA and other regulatory agencies rely on.

In summary, scientists responsible for the Calidria inhalation study not only failed to turn over the full dataset to the Government as promised but their omission to report the findings has caused the Government and the scientific community at large to be misled for decades.

Conclusion

A review of the available health information for the Coalinga miners and millers and others potentially living with, residing near, recreating on, or farming the New Idria ore body has provided no evidence of attributable disease. Egilman and Robert's claims [11] to the contrary are without foundation and their claims against the author and the studies' motives and methods are equally unsupportable. Egilman and Roberts' claims of "corporate concealment of the animal data" are particularly misguided. Indeed, quite the opposite is true. The circumstances surrounding the long term inhalation study of Coalinga chrysotile done at the NIEHS strongly suggests the data from this study were intentionally concealed by those NTP scientists and their contractors originally charged with conducting the study in the first place.

If the animal data had been properly presented in a considered, scientific and a timely fashion to the Regulatory authorities, many hundreds of millions if not billions of dollars could and should have been saved. The EPA's JM Atlas Coalinga "Superfund" boondoggle should certainly never have been conducted and the myriad unfounded attempts to regulate short-fiber chrysotile in a manner similar to other forms of asbestos might have been diverted.

A further report is in preparation that extends this discussion further. It will describe in detail the flawed foundation of the EPA's Superfund "remediation" efforts at the New Idria serpentinite and their current efforts to close the Clear Creek Management Area (CCMA) due to their "extreme concerns" for those recreating on the site.

A final report in preparation for this series describes chrysotile exposed groups in Siberia, Arizona, China, and Maryland that further support the failure of chrysotile to produce attributable mesothelioma.

Notes

1. Coalinga chrysotile comes from the New Idria deposit, California and is also known as "Calidria" chrysotile.
2. The JM and Atlas mines and mills were situated on the Southern part of the New Idria Serpentinite (see fig. 6 in [29]) several miles north of the town of Coalinga The UCC

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 14] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

mine was located in the Northern part of the New Idria Serpentinite about 60 miles from the mill in King City (see fig 1a in [2] and fig. 6 in [29]). Some of the Atlas asbestos workers had also worked at Atlas Mercury Inc and Atlas Uranium at Moab. Utah. Some JM Coalinga chrysotile miners had also worked in JM Canada. In 1985 UCC sold its holdings to the King City Asbestos Company (KCAC)

3. In 1997, a personal injury suit against UCC [100] alleged that remote "environmental" exposures to Coalinga chrysotile were substantial contributors to Plaintiff's mesothelioma. Detailed analysis of the case demonstrated the cancer was due to significant alternate amphibole asbestos exposure elsewhere.
4. The reader may wonder if UCC and JM ever interacted in the mining, milling, and processing of Coalinga chrysotile. In fact, these two companies limited their exchange of information for proprietary reasons after the deposit was discovered but UCC apparently inspired JM to develop commercially their holdings in the southern part of the ore body. Dr William Dresher, one of UCC's original R & D team responsible for determining the exact nature of and uses for Coalinga chrysotile provided an interesting historical account of the discovery of this material and how UCC and JM viewed this unique mineral form. "At the time of discovery in 1957 UCC did not know what it had. The exploration crew was looking for lateritic nickel, not asbestos. The deposit was located from the air and it was a number of months before a team could visit the deposit on the ground. Lateritic nickel occurs with a brown cap over a white mass as it is a weathering product of serpentine. Thus, from the air, it appeared to be a nickel deposit. The first samples from the ground crew identified that it was not a nickel deposit but they did not know what it was. UCC hired a retired US Bureau of Mines nonmetallic mineral specialist, who advised them that the material was, in fact, 'mountain leather,' or paligorskite a clay mineral. It was not until we in research got our hands on a sample that we identified it as chrysotile. The material had a very pure chrysotile X-ray diffraction pattern. We then started our investigations into what it was that we had and why it was different than any other chrysotile. . . . Early on, a VP of UCC Chemicals Division played golf with a VP of Johns Manville. (JM was UCC's largest customer for polyvinyl chloride.) During the game he mentioned that UCC had located a significant deposit of asbestos in California. The JM man replied that they knew all about that deposit and it was totally worthless as it was equivalent to their 'shorts' – a waste product from their processing. He strongly advised that UCC drop the project. This was reported back to us in the lab and we were asked why were we pursuing the project? We reported that we knew that we had a unique material here, one that was significantly different than anything JM had, and we were confident that uses could be developed for it."
5. The map had been reproduced from a graphic prepared by UC Davis as originally developed by Pan et al. [24] based in part on geologic information compiled by Churchill and Hill [101].
6. Provided to the author further to some of the aforementioned litigation during which the author was retained as a Consultant and Testifying Expert to UCC (Conwed, Chicago Schools).
7. Agency for Toxic Substance Disease Registry (ATSDR) for EPA IX; California Cancer Registry (CCR); California Dept Health Services; California Air Resources Board (CARB); California Office of Health Hazard Assessment (OEHHA); California Mining Assoc.; California Bureau of Land Management (BLM); California Mining Safety Health Authority; California Occupational Safety & Health Authority (OSHA); California EPA, Office of the Deputy Director; California Dept of Water Resources; California Dept of Parks & Recreation; Coalinga EPA; Dept of the Interior, Pacific SW Region; EPA Region IX; PTI Environmental Services; American Motorcycle Assoc.; Southern Pacific RR; US National Institutes for Occupational Health/CDC; US Geological Survey; US Mining Safety Health Authority. Also see Ilgren [2].
8. Mr Bill Streib, JM Research coordinator, Geologist, and first JM Coalinga plant manager; Mr William Rietze, JM senior scientist; Dr Paul Kotin, former JM medical director; and Dr Fred Pundsack, former JM president).
9. Dr Bruce Baldwin, Coalinga Med Center Radio-logy; Dr Roland Icke, formerly Coalinga general physician; Dr Tom Nelson, formerly Fresno local pathologist; Mr Herb Watanabe, Coalinga pharmacist; also see Ilgren [2].
10. Mr John Davies, Mr Ivan Thompson, Ms Loretta Turner.
11. Mr Bob Simpson, Atlas miner and superintendent.
12. http://www.phifresno.org for a description of reports and publications.
13. also see footnote number 26 in Ilgren [2] and "the lawsuit filed against Carbide by an exworker", [28] (p. 13).
14. Dr William Dresher said "The real opening came in our efforts when the UCC sales people called upon duPont looking for uses. DuPont had previously developed a wet dispersion process for opening Quebec chrysotile to make a product useful to them in latex paint. The process was expensive and required a large amount of energy input. They indicated that they would buy our product if we could emulate the product from their process and provide it at a reasonable price. They gave us a copy of their patent. Having this in hand, Naumann and I developed a unique process for the Coalinga material and received a U.S. patent on behalf of UCC. This, like the duPont process, was a wet dispersion process in which chemicals (acetic acid) were added and the pH was adjusted. This led us into looking at the potential uses for colloidal chrysotile. In the end; however, we found that, by using the Coalinga material, we could produce an inexpensive product for fillers, etc. without going the chemical dispersion

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 15] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

route that was required for conventional asbestos to produce such a product. Thus, the chemical route that Naumann and I developed proved to be unnecessary commercially. Until the group was closed down due to a corporate reorganization, Naumann and I continued to search for uses for colloidal chrysotile.''

15. The Atlas records had been lost during the transfer of the company to its successors. Some JM records were also discarded by the hospital due to their 7 year retention policy and were also lost during a local earthquake and a flood.
16. pg 62: MR. DAVIES: ''In regard to all of this research that you gentlemen have done, how many of the men at work in the asbestos industry in the Coalinga area did you contact to see how much asbestos contamination they tolerated throughout the years that they worked in the asbestos industry before you come up with these figures? MR. HIATT: I am not aware that we contacted anyone. MR. DAVIES: That's just what I thought. MR. HIATT: The reason for that is that the association between asbestos exposure and cancer has been very clearly demonstrated in a number of other human exposure situations, including other mining and milling operations. The focus of the Superfund program is not on past disease incidences. The focus of our program is on preventing future – the occurrence of disease in the future and cleaning up environmental contamination situations so that they will be safe in the future, irrespective of what may have happened in the past. MR. DAVIES: I understand that. But there is a question in the back of my mind because I know an enormous amount of the boys that work and men that worked in these mines up here. And I happen to be one of them''.

    pg 72 MR. BAKER: ''Earlier the question was asked whether there was any attempt made to contact the peo-ple who worked up at the mines and he indicated that, in fact, we had made attempts and had contacted various people who had worked at the mines before. Most the information that was gathered from them did not pertain to whether they were having any personal health affects, it pertained to things they knew about the operation at the site and companies involved and things like that. So obviously, they never contacted you, Mr Davies, but there were some people who were working at the mine''.

    pg 79 MR. DAVIES: ''As I have said before, I have heard you talk about all of these percentages and all of this and all of that, and I can show you another gentleman that's right here in this town that worked with me except for about the time that we spent at Atlas, that all the time I spent with Coalinga Asbestos, except for about four months. In the first – I would say the first threeandahalf or four years that we were in that plant there was times that the tailings would stop up. We didn't stop the mill, we went right out in the tailings pile and cleared the shoot and went right on back – kept right on going. And sometimes, if he would have been as far as from here to that lady right over there, I couldn't have seen him because of the asbestos that we were in. And we have come out of there at times when we had to breathe through our mouth and our nose and be choked up. And I spent 13 years in the asbestos industry, and before that 20 years in submarines so you talk about them little things and I can show you a lot of other boys that's worked eight, nine, ten years in the plants. Of course, we cleaned them up as the years went along and it got a lot better, but since I was maintenance foreman, I was still – had to go into the tailings piles and had to get men in there with me and bag house and what have you and be out there on a windy day something when the dust was blowing and all of this and that. And I have – and then in the last five years I have had double pneumonia and acute valley fever and they can't find a one spot on my lungs even today. So when you breathed with them little things like the gentleman right here, he's worrying about him getting on a motorcycle, I probably breathed in more asbestos in one eighthour shift than he would if he road his motorcycle for 20 years. So I have got kind of an odd feeling and that's the reason I want to know who you talked to because I practically known everybody that worked in the asbestos industry here and several of the people that worked in Canada''.

    MR. HIATT: ''With respect to your personal experience, I would say you're lucky. MR. DAVIES: The only thing I am saying is that if I am lucky, I can name you about 500 other boys around California that must be awful lucky''.
17. Davies was also a maintenance foreman at Atlas for 8 months ca 1974. He said it was dirtier than JM. The ventilation was poorer and the dust collection equipment to bag house was bad. There was no requisition system in place to order new equipment when it was needed.
18. The Lemaire Airforce Base Medical Center tracks the incidence of Valley Fever in the area. This tends to follow locally seismic changes since New Idria is one of the most seismically active areas in the world.
19. Coalinga stands for ''Coalinga station A'' where the trains carrying various mineral ores were refueled. No less than 94 mines have been identified in this region [103]. Davies (pers commun.) has said possible commercial asbestos exposure came from asbestos from pipe and boiler wrapping in the oil fields run by Union Oil, Getty, Standard Oil, Texaco, Cambden, and Baker. Davies also noted other sources of potentially fibrogenic dust from local refractory works, chrome mines, JM (Asbestos) Canada, JM (diatomaceous earth) Lomtok, and UCC (uranium) Moab.
20. ''Is it your position that Dr Ozoa was lying on the path about what his – A. Absolutely not, no... I don't think he was lying on the other misdiagnosis he made out there in California that caused him to lose his license. Q. All right. A. It was a competence issue and/or influence issue, but... I don't think he was lying.'' Egilman p. 103.
21. Gibbs said ''the lung tissue showed organizing diffuse alveolar damage with hyaline membranes areas of necrosis, and bronchopneumonia. There was a fibropurulent exudates

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 16] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

affecting vessel walls. There was a florid fibropurulent exudate and granulation tissue affecting the pleura. I did not identify any asbestos bodies.... the changes were of relatively recent onset and consistent with severe lung injury occurring about one week prior to death. This fits well with the events described in the medical records. The fibrosis described in the coroner's report appears to be the young fibroblastic tissue that is seen in association with organizing diffuse alveolar damage. There was no evidence of asbestosis which is a chronic form of lung fibrosis''.

22. Roggli said ''the lung slides showed changes of organizing diffuse alveolar damage.. there is extensive alveolar duct fibrosis as well as organizing pleuritis. Other findings include patchy bronchopneumonia with dystrophic calcification. No asbestos bodies are identified. There is no histological evidence for asbestosis''.
23. The ATS 2004 guidelines were ''not limited to lung tissue'' for the diagnosis of asbestosis and suggest BAL could be used as well. However, the ATS said the use of of BAL remained to be established and even said BAL was unreliable since ''the AB count in BAL fluid appears to correlate with the presence or degree of fibrosis in some studies but not in others''. The ATS also said ''the presence of interstitial fibrosis in the absence of asbestos bodies is most likely not asbestosis although rare cases of pulmonary fibrosis with large numbers of uncoated asbestos fibers have been described''. The ATS thus concluded that ''the 2004 criteria are open to future testing modalities if and when they are validated''... ''These criteria and the guidelines that support them are compatible with the Helsinki criteria developed by an expert group in 1997 which represents substantial consensus worldwide. The guidelines supporting these criteria will undoubtedly change again in the future but the present guidelines should provide a reliable basis for clinical diagnosis for some years to come''. Indeed, the Helsinki expert group was an assembly of Plaintiff oriented scientists and physicians held in closed session to prevent independent review, oversight, or accountability (also see ''NIOSH Siberian Study'' in Ilgren (in preparation).
24. Attributable disease should have first become apparent in the early 1980's by which time there would have been sufficient latency. By this time, large numbers of asbestos related disease claims were being filed throughout California. These filings created not just a 'heightened' state of awareness of an association between asbestos and certain diseases particularly mesothelioma but also a keen incentive to identify such cases for the sake of compensation. Nonetheless, few Coalinga miners or millers have even filed against UCC, Atlas or JM and none, to my knowledge, has ever been awarded.
25. Schenker was asked why there did not appear to be a mesothelioma excess in the three counties (Fresno, Monterey, and San Benito) related to the New Idria Serpeninite [104]. He said county specific incidence rates are not very useful in looking at mesothelioma rates since these are ''fairly crude indicators... not for etiologic purposes... (and)... because (they) are so crude'' [103]. However, the entire University of California at Davis (UCD) effort to demonstrate an excess of mesothelioma in El Dorado County began on the premise that such data provided a reliable basis to make such claims.
26. Mercury mining in the Coastal ranges is the counterpart of gold mining in the Sierra Nevada. The histories of exploration for the two metals run parallel courses in the early mining development of the State partly because the possession and use of mercury was essential for the recovery of gold by amalgamation. Actually the mining of mercury began in the Coast ranges in 1845, prior to the discovery of gold in the Sierra Nevada, when ''Indian Paint Rock'' at New Almaden was identified as cinnabar, the chief ore of mercury. Subsequent successful developments encouraged prospecting both to the north and south and many more mercury deposits were discovered as were also deposits of other metallic minerals, principally chromite, manganese oxide and copper sulfide''.
27. ''A review of the 94 mines listed in the principal data source [102] provided an initial 60 mines believed to have potentially disturbed asbestosbearing materials. These mines had produced asbestos, mercury, chromite, or other commodities which are commonly associated with serpentinite''; ''Chromite mines and deposits are numerous within the New Idria/Coalinga area....Those chromite mines having the largest areas of disturbance (Big Ridge, Butler Estate, Tromby North, Sawmill Creek, and James/Corbett/Byles) are located in the southern third of the New Idria serpentinite. ''The largest mines of concern in the New Idria/Coalinga area (including the New Idria Group, Red Rock, Clear Creek, and Tirado/Shear/Monterey) are concentrated in the northern third of the New Idria serpentinite mass''.
28. Crapo testified [63] that ''looking backward'' it would clearly have been important 'to tell the world and publish the facts that... there was one type of safe asbestos' (p. 152).
29. ''I wouldn't tell you none of the money that I got from litigation didn't contribute to some of the work that went into this paper''.
30. The purpose of these retainers emerges in a three way discussion between Egilman, Counsel representing Union Carbide and Mr Al Parnell (long time President of the Defense Research Institute and lead Counsel for Georgia Pacific). Defense Counsel asked Egilman what he does for these payments to which he said ''charges for the right to be listed'' and for ''other Consulting''. The author asked Parnell (Parnell 22 April 04, pers. commun.) what Egilman meant by 'other Consulting' and Parnell said ''Egilman is paid not to testify''. This is clear from Egilman's testimony when he asked Mr Al Parnell if he had ever testified at trial against Georgia Pacific and Parnell said: ''not that I am aware of''. When Egilman was asked: ''is it your opinion that Georgia Pacific knew of the health risks of asbestos no later than the

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 17] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

mid-1960s?'' Egilman replied: ''. . .the answer to that question would be yes but because of the previous dialogue 'I don't recall'''. When Defense counsel asked what the consulting relationship with Georgia Pacific consisted of, Egilman said he would need to get their permission to discuss it as Al Parnell usually objects. When Defense counsel asked Egilman if he could estimate the number of hours he did pursuant to those Consulting Agreements - even asking whether it was more or less than ten hours - Egilman said he did not know.

31. Q. The last thing I just want to be clear. . . in the letter to the editor you indicate that you had served as a consultant to Union Carbide in matters related to health problems; is that correct? A. True. Q. All right. Can you just tell me the details of that? A. Sure. Q. What did – what did you do? A. Well, my recollection is I just had several discussions with Mr Brownson, who is the – either by – via mail or phone. And at least – well, let me – or the – or the lawyers, either he and/or the lawyers, that I sent them a bunch of my assignat literature, which was voluminous at the time, and I attended one all-day meeting at Kelly Drye – Q. And – A. – and answered some other questions at other times. Q. Okay. You attended one meeting; is that right? A. Yeah. Q. Okay. And when did that consulting relationship end? A. I don't think there was a formal end, that I recall. Q. Okay. Well, you – is it fair you haven't – to say you haven't consulted on any matters related to – for Union Carbide since – what was it? was it mid 198 – what – A. '86, I think it was. Q. Since that time, you haven't been working as a – A. They haven't called me up and asked me to do anything. That's right. Egilman, David S. M.D. [28] vol 1 - 2004/03/26 - p. 169.
32. ''Composed of four charter DHSS agencies: the National Cancer Institute; the National Institute of Environmental Health Sciences; the National Center for Toxicological Research, Food and Drug Administration; and the National Institutes of Occupational Safety and Health, Centers for Disease Control'' (see [60]).
33. ''the two year studies in rodents remain the most definitive methods by which chemicals or physical agents are identified as having carcinogenic potential other than epidemiological studies in man'' [. . . while the two year animal studies have been criticized for often not providing qualitative or mechanistic data on the process by which the chemicals exert their toxicity and carcinogenicity, animal studies remain at this time, the only mechanism for identifying human carcinogenic risk other than by epidemiological studies which are an unacceptable alternative''. [51], p. 69].
34. This is particularly ironical since Crapo provided UCC with an Affidavit in 2001 [105] supporting the relatively innocuous nature of Calidria.
35. When Crapo learnt the author had ''discovered'' the Calidria files (Pinkerton pers commun Dec 1995) he became extremely agitated and tried to shut down the four year collaboration the author had established with Pinkerton.
36. The study materials were in the NTP Archives from 1978 to 1985 and then from 1993 to the present time. The study materials were sent to the EPL that served as the NTP contractor facility from 1985 to 1993. A one page request form dated 3 July 85 re 'NTP asbestos' – 'Northrop laboratory' per ''Rats, male and female: Group Control, JM, I, S, B'' was also produced by the NIEHS further to the 2007 FOIA. This requested the ''Release'' of ''1 Inventory Booklet; 1 Discrepancy Report; 2 Cartons of Slides including 17 slide boxes re male rats, 15 slides boxes re female rats, and 1 special slide set in 1 box''. The release of those materials from the NTP archives was authorized by Dr G Boorman. [105–108] The requester also happened to be Dr Boorman. The Calidria study materials were returned to the NTP archives from the EPL (vi) on 11 March 93: ''Non Key employee, Lily Hong gave me these slides and paperwork on 3/11/93 to go back to the archives. There are seven boxes of slides, necropsy forms and handwritten notes in the box. There is a note on one of the slide boxes that has a date of 1980. Dr Eustis did not know what they were, so he told me to send them to you. He would like for you to go through the data over there and see if you can find out what they are and what was the purpose for them being here. They would have probably have come over here for either Dr Boorman, Lily Hong, or Dr McConnell. Dr Eustis said that if you could not find out Annette Shambley, a ''Non-Key Contract Employee'' of 12 March 93. ''Delivery Slip'' dated 12 Mar 93 from the Experimental Pathology Laboratories (EPL)[110] (''Contractor for the NTP Archives, Research Triangle Park, NC'') indicates the materials (''7 slide boxes, necropsy forms, and handwritten notes) were sent by Annette Shambley at EPL to Lily Hong, NTP.
37. Connelly, J (Supervisor, Pathology Support, NTP Archives) to Ilgren (16 Mar 04): ''As requested the NTP archives has reviewed the activity on the study Asbestos, Chrysotile IR/C611234A/F344 rats conducted by NIEHS, as far back as EPL had the contract to run the NTP Archives. Prior to your review (between 11/30/95 to 8/02/96) only program related reviews had been performed. . . . Subsequent to your visit . . . the only other request in regard the Asbestos Study was a 7/09/02 FOIA request for analysis by NIEHS of Asbestos or Asbestos containing material in paper products was received. No relevant data was found to be available in the NTP archives for this request.''
38. NTP studies of significant importance to human health are never discarded. ''**Management of the NTP Archives** – The NTP Archives is a unique national resource of over 6 million histological slides of spontaneous and induced lesions in mice and rats. The data are maintained in an access limited protected environment and are computerized for easy data access. . . . . Space has been saved by discarding studies more than 10 years old for which there is little interest. During the past 16 years, the NCI, now the NTP, has conducted over 400 two year studies in rats and mice. The slides, blocks and wet

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 18] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

tissues from these studies are archived for retrospective study and evaluation by the NTP and other interested scientists . . . (Contact Dr G Boorman)'' [51].

39. These summary tables were given to Prof. Pinkerton by Prof Crapo, his PhD supervisor, in 1991. Prof James Crapo originally received them from Mc Connell. Crapo suggested to Pinkerton the data in the summary tables should be published since these were 'important carcinogenicity' data [29].
40. ''I have instructed Mr Louis Cozart to forward the material from the inhalation asbestos study which was performed at the Northrup facility from the NTP Ware-house to the NTP archives. The material includes slides, blocks, wet tissues and animal data records. Please inventory the material, clean and prepare the slides for histopathological evaluation, and rebag and label the wet tissues. Please prepare animal data records as necessary.''. [109, 110].
41. Crapo [53] testified that ''The staff of the NIEHS who handled the necropsies at the end of the experiments . . . to evaluate carcinogenesis have never published their data.''. Crapo also said the ''tumor data'' belonged to 'staff of the NIEHS' (and that) ''we still have intentions to reevaluate that and see if there was a significant difference or an incidence with each of the tumors'' but ''I haven't done that and it wasn't my responsibility''. . . ''I'm only thinking of doing it now because the people who did it have quit and have never utilized their data''.

    However, it appears as if Crapo actually thought about doing this ten years earlier since in ca 1982, he and his colleagues [Pinkerton, Crapo, Brody, Pratt, and others] submitted a manuscript to Laboratory Investigation describing the morphological findings obtained with the Calidria and Canadian fibers, a paper that was cited as reference number three by McConnell et al. [77]. The paper appears to have been rejected.
42. NTP Annual Report [90] (p. 77, para 3) ''Inhalation exposure studies with chrysotile B (short and intermediate) and glass wool (JM100) have been completed. Increased incidences of pulmonary tumors were found in all chrysotile groups but not in the synthetic fiber groups'' (Contact person: Dr E Mc Connell, NIEHS); NTP Annual Plan [91] (p. 168, paras 4 & 5) ''Fibrogenic and carcinogenic potential of asbestos and man made mineral fibers'' – ''A joint study on the fibrogenic and carcinogenic potential of chrysotile asbestos and man made mineral fibers was conducted in conjunction with the Pneumocononiosis Research unit, Medical Research Council (MRC), Wales. . . . Fibers studied at both facilities were UICC Chrysotile B asbestos and microglass (JM100). In addition, short and long range chrysotile asbestos was studied at NIEHS while various types of rock wools were evaluated at the MRC. The results of this joint study were similar at both facilities. Fibrosis and neoplasia occurred with the asbestos fibers but not with the microglass fibers (Contact person: Dr E E McConnell)''.
43. these included, amongst other things, daily exposure measurements, daily animal assessments, interim and final sacrifices, the production of detailed necropsy reports, and the transfer of autopsy materials to the NTP pathologists and the NIEHS scientists as cited in Becton Dickinson Reports [111–114].
44. ''Respiratory Toxicology'' NIEHS – ''The Respi-ratory Toxicology Group designs and conducts studies of compounds administered by inhalation. The group develops and applies the technology necessary to accurately control and document the exposure environment. Studies involve the measurement of a variety of physiological parameters necessary to investigate the mechanisms and the time course of toxic effects. Exposures to particulates and gases are conducted by an on site contractor Mantech Environmental Technology Inc'' [51]. p. 116.
45. The NTP was established in 1978 and in July 1981 the Carcinogenesis Bioassay Testing Program of the NCI was transferred to the NIEHS [60]. After June 1981, ''the NTP adopted the policy that the experimental data and laboratory records from all NTP toxicology and carcinogenesis studies not yet printed and distributed would be audited.'' [60]. The audit reports were to be reviewed by NTP staff and kept on file and available for review at the NIEHS/NTP Quality Assurance Office [60]. e.g., see pp. 140 and 141, 1991 NTP Annual Report [51] particularly indicating the (''primary activities of the Quality Assurance (QA) Office within the Chemical Carcinogenesis Branch (CCB), DTRT'' especially the performance of ''retrospective data audits of all completed, long term studies and NTP Technical Reports . . . Contact person: Mr David Bridge)'' NTP Technical Report after final interpretation of the results. In addition, the extensive ''Quality Assurance Program'' of the NIEHS is described at length in the NTP Annual Reports; ''Study Evaluation and Reporting'' [51], p. 104: ''the Study Evaluation and Reporting Section (CCB, DTRT, NIEHS) is responsible for the accuracy and validity of the pathology data derived from the two year toxicology and carcinogenesis studies. This section coordinates the histopathology quality assessment activities, organizes, and conducts Pathology Working Group (PWG) reviews of the data from chronic studies and evaluates and interprets the data for preparation of the NTP Technical Reports.'' ''Pathology Support for the NTP – Quality Assurance'' – Pathology data are subjected to a thorough pathology quality assessment through a contract with Experimental Pathology Laboratories, Inc (EPL). The objectives of the contract are to validate the pathology data derived from all NTP studies by assessment of histotechnique quality, tissue accountability, tissue trimming, and the accuracy of the histopathologic diagnoses. (Contact person: Dr G Boorman)''. ''All NTP toxicology and carcinogenesis studies are subjected to a data audit before being presented for peer review'' [59]: p. 2.''

+ [A3B2 Ver: 8.07r/W] [21.6.2008–12:11pm] [1–34] [Page No. 19] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

46. "The effective deployment of scientific information about the properties and biological activities of toxic or potentially toxic chemical substances is a critical component of the basic goals of the NTP. Active dissemination of plans, experimental results, interpretations, and basic concepts and policies forms the bases for identifying and/or initating the resolution of many human health and environmental concerns relevant to chemical substances." . . . Scientific Coordination and Information Dissemination [51].
47. Dr Boorman said that Dr Mc Connell kept him at 'arms length' from the investigation which he found odd since it was such an important study.
48. Dr Brody was the NIEHS Project Officer of the Calidria inhalation study. He assisted in the management of the NTP long-term animal study (morphology segment) and the morphometric sub-study conducted by Duke (see below). "The majority of the NTP long term toxicology and carcinogenesis studies are managed by the NIEHS component" [51], p. 69. Dr Brody was therefore generally responsible for the entire investigation (Busey, 2007 pers commun). This comports with the general job description of an NTP Project Officer, i.e., "The Project officer provided the technical guidance and monitored the projects; served as the 'technical' officer to the Contracting officers who were generally not scientific (Eastin, pers commun., 2007)"; "Project Officers – are NTP scientists who are responsible for monitoring and evaluating the overall operations of the laboratories performing toxicology and carcinogenesis studies for the NTP in accordance with the standards and practices set forth in the NTP General Statement of Work. To establish that the studies are well conducted (scientifically, cost efficient and timely), these scientists initiate: 1. Annual program reviews by peer groups of scientists; 2. Quarterly site visits 3. Ad hoc visits by chemical management and experts in various scientific disciplines as needed. 4. Communication with the laboratory principal investigator and relevant NTP personnel and 5. Interaction with NTP contract specialists. These project officers/scientists are the Program's official representatives in all dealings with the laboratories performing the toxicology studies [51] p. 139. (Contact person: Dr W Eastin)]". It was also consistent with the description of a Project Officer as described in the NIEHS Contract Grants for the Calidria inhalation study signed by Dr Brody "Article VII – Project Officer: "The following Project Officer will represent the Government for the purpose of this Contract: The Project Officer is responsible for: 1. Monitoring the Contractor's technical progress, including the surveillance and assessment of performance and recommending to the Contracting Officer changes in requirements; 2. Interpreting the scope of work; 3. Performing technical evaluation as required; 4. Performing technical inspections and acceptances required by the contract; and 5. Assisting the Contractor in the resolution of technical problems encountered during the performance. . . . For guidance from the Project Officer to the Contracting Officer to be valid, it Must: 1. Be consistent with the description of work set forth in this contract; 2. Not constitute new assignment of work or change to the expressed terms, conditions, or specifications incorporated into this contract; 3. Not constitute a basis for an extension to the period of performance or contract delivery schedule; 4. Not constitute a basis for any increase in the contract fee and/or cost" and to the extent the Project Officer was responsible for overseeing the Contractor, he would also be responsible for ensuring the Contractor fulfilled the necessary "Reporting Requirements" under "Part IIA. 2. Technical Reports. C. Final Report" which states that "this report shall document and summarize the results of the entire contract work for the entire contract period of performance and shall be in sufficient detail to explain comprehensively the results achieved" to be delivered to the Project Officer "on or before the expiration date of the contract". [96,99]

B. Level of Effort. 2. "The Contractor agrees to use his best efforts to accomplish all the work described above. His obligation will be deemed complete if the work is performed in accordance with high standards of scientific and professional skill and the approximate level of effort has been substantially applied; except, however all other requirements must be met including delivery of reports and materials as may be required". C Report Requirements. 1. Manpower Report a. The Contractor shall complete and submit a Manpower Report, form NIH – 1749 . . . 2. Technical Reports. a. Final Report: "The Contractor shall submit a final report which documents and summarizes the results of the entire contract work for the entire contract period of performance. This report shall be in sufficient detail to explain comprehensively the results achieved. Specific requirements if any are set forth in Article I, Description of Work, above (re . . . morphologic and morphometric studies on the tissues from the lungs of 24 rats previously exposed to chrysotile fibers . . .) Final reports shall be submitted within 30 days after the expiration of the contract. Copies of the Comprehensive Report shall be delivered to the following: (6 copies) Dr Arnold R. Brody, Lab of Pulmonary function and toxicology, NIEHS' and (1 copy) to "Contracting Officer, Procurement Office, OAM, NIEHS (Dr Wm. R Johnston)"; "Article X – Key Personnel – Dr James D. Crapo, MD. Title: Assistant Professor of Medicine/Principal Investigator; Article XI – Project Officer – Primary Project Officer: Dr Arnold Brody (vs)" [118].

'Article I. Description of work – '. . . The Contractor shall . . . exert its best efforts to conduct particle translocation, morphologic and morphometric studies on animals exposed to asbestos and fiberglass for varying periods of time. More specifically, the Contractor shall perform the work set forth below: 1. Carry out morphologic and morphometric studies using electron microscopy on tissues of rats exposed to asbestos and fiberglass . . . tissues from

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:11pm] [1-34] [Page No. 20] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

about 100 animals distributed over about 15 exposure and control groups will be included in these studies''. 3. The Contractor agrees to use its best efforts to accomplish all work described in Article I. its obligation will be deemed complete if the work is performed in accordance with high standards of scientific and professional skill and the approximate level of effort has been substantially applied; except however all of the requirements must be met including delivery of reports and materials as may be required. D. Reporting Requirements c. Final Report. This report shall document and summarize the results of the entire contract work for the entire contract period of performance and shall be in sufficient detail to explain comprehensively the results achieved. (due) on or before the expiration date of the contract'' (4 copies to the Project Officer and 1 copy to the Contracting Officer). Article VI. Project Officer. A. Dr Arnold Brody.'' Duties of Project Officer identical to those described above in ''Request for Proposal. 4 Sept 79. RFP No. NIH – ES – 79 – 13'' [96]; Article VII. Key Personnel – Dr James D. Crapo, Principal Investigator''. Ford, E to NIEHS (undated one page letter) – ''Transmitted herewith is an executed copy of Contract Number N01-ES-9-0007 dated 1 May 1979. Dr Arnold Brody has been designated as the Contracting Officer's Technical Representative (COTR). The COTR is responsible for bringing to the attention of the Contracting Officer any deviation from the Contract requirements. However the COTR does not have the authority, implied or otherwise, to authorize any changes in the terms, provisions, and/or specifications of the contract. All changes in the contract shall be in writing and shall be made by the Contracting Officer only''.

49. Dr James Crapo, was the Principal Investigator overseeing the morphometric analyses of the Calidria inhalation study. He and his colleagues did this study as Duke Subcontractors. Crapo [63] testified he assumed much responsibility for the investigation. Dr Crapo admitted he possessed some of the missing study materials i.e., the 12 Month follow up of the acute exposure studies reported by Brody but would not publish them since they ''were only of use to lawyers'' (Henderson and May pers commun., 1996). The close relationship between Crapo and Brody is reflected by the fact that Brody awarded Crapo the subcontract to do the morphometric studies under an Request for Proposal (RFP); by study Memoranda; and by their co-authorship on papers that stated Calidria was injurious by inhalation (e.g., [55, 56])
50. Cc Brody as ''Project Manager'' re. permission to purchase equipment – Johnston to Anlyan 17 Feb 81[119]; Miller to Henschel 10 Feb 81[120]; Crapo to Henschel 10 July 80; [121]; Anlyan to NIEHS 13 April 83 [122]; Crapo to Brody 17 Jan 83 [123]; Crapo to Brody 7 Feb 84 Request for Contracting Officer's Authorization re N01-ES-0-0004 for travel expenses ($1,157.31) to go to meeting (Duke to NIH) [124].
51. Dr Wm. Busey of the Environmental Protection Lab, Inc. was a senior Pathology Quality Assurance officer involved with the oversight of the Calidria ingestion study.
52. According to Busey (pers commun., 2007), there was a legal obligation to report the findings of studies such as these done under Contract to the Government (Also see Ilgren [29]).
53. A member of the NTP Pathology Working Group (November 14, 1980) who worked on the Calidria inhalation study under Dr McConnell.
54. ''Excellent reviews of the carcinogenic and public health effects associated with asbestos are those of Selikoff [115], the EPA [89], Selikoff and Hammond [116] and the . . . Cooper et al. [12] confirmed the association between asbestos levels in San Francisco Bay area drinking water and cancer of the digestive tract'' NTP Tech Report 295.
55. Primary Union study reviewers Union Members as Primary Reviewers of the Calidria ingestion study: Hamsters – Dr Frank Mirer, PhD (Principal Reviewer) Intl. Union United Auto Workers, Detroit; Rats – Dr Louis Belickzky, MS MPH, Director, Dept. of Industrial Hygiene, United Rubber Workers Intl. Union, Akron, Ohio; Dr David Kotelchuck, PhD, Research Dept. United Electrical, Radio, and Machine Workers of America, New York.
56. p. 7 - A. {Crapo is expected to testify on} . . . the meaning of studies of asbestos exposed animals done by Dr Arnold Brody. PG 43 Q. What opinions are you prepared to express about Dr Brody's animal study data? I haven't prepared a specific opinion on that and am not prepared to answer the question that would be asked about it by the attorneys. Q. Has Mr Harvard talked to you about that at all? A. No. Q. What has he talked to you about? A. He hasn't talked to me. MR. RION: Bill, you are just sitting there like a potted plant? MR. HARVARD: Pretty much. MR. RION: I just figured if you went all of the way up there, you must have had some important mission. You are going to do that after the deposition, when I can't ask him about it; right? MR. HARVARD: Draw your own conclusions. MR. RION: I have that figured out. [115]
57. ''Dear Dr Crapo – I am pleased to act as an external advisor to your Program Project Grant on Acute Lung Injury. I understand that the work in your proposed experiments involves considerable expertise in experimental pathology and cell biology and I look forward to aiding this work in an advisory role. Arnold Brody, Prof. Director Lung Biology, Tulane University Medical Center''.

Acknowledgment

Union Carbide provided no financial support for this report. Over the last 4 years the author did no work for Union Carbide on asbestos related matters and only 3 h for a project not related to asbestos.

References

1 UICC: Working group on asbestos and cancer. Report and recommendations of the Working Group convened under the auspices of the geographical pathology committee of the international union against cancer: Arch Environ Health 1965;11(2):221–229.
2 Ilgren E: Coalinga fibre - a short, amphibole - free chrysotile. Part 6 lack of amphibole asbestos contamination: Indoor Built Environ 2004;13:325–341.
3 Ilgren E: The fiber length of coalinga chrysotile: Enhanced clearance due to its short nature in aqueous solution with a brief critique on ''short-fiber toxicity'': Indoor Built Environ 2008;17:20–24.
4 Ilgren E: The fiber width of coalinga chrysotile: reduced respirability due to its thick nature in an aerosol and its ''ultra-thin'' nature in aqueous solution (In Vivo): Indoor Built Environ 2008;17:20–41.
5 Ilgren E: Coalinga chrysotile: Dissolution, concentration, regulation and general relevance: Indoor Built Environ 2008;17:42–57.
6 Ilgren E, Chatfield E: Coalinga fibre: a short amphibole-free chrysotile. 1. evidence for lack of fibrogenic activity: Indoor Built Environ 1997;6:264–276.
7 Ilgren E, Chatfield E: Coalinga fibre-a short, amphibole-free chrysotile. Part 2: Evidence for leak of tumourigenic activity: Indoor Built Environ 1998;7:18–31.
8 Ilgren E, Chatfield E: Coalinga fibre: a short, amphibole-free chrysotile. Part 3: lack of biopersistance: Indoor Built Environ 1998;7:98–109.
9 Ilgren E: Coalinga fibre: A short, amphibole-free chrysotile. Part 4: further evidence for a lack of fibrogenic and tumorigenic activity: Indoor Built Environ 2002;11:171–177.
10 Ilgren E: Coalinga fibre - a short, amphibole - free chrysotile. Part 7 additional evidence to support lack of fibrogenic potential: Indoor Built Environ. 2004;13:375–382.
11 Egilman D, Roberts S: Controlled use of asbestos: Intl J Occup Environ Health 2004;10:99–103.
12 Cooper W, Murchio J, Popendorf W, Wenk H: Chrysotile asbestos in a california recreational area: Science 1979;206:685–688.
13 Hyde DS to Myers J: Correspondence re health status of KCAC mine and mill workers. 12 June 84.
14 Hyde DS to Myers J: Correspondence re health status of KCAC mine and mill workers. 19 Feb 88.
15 Hyde DS to Myers J: Correspondence re health status of KCAC mine and mill workers. 31 July 89.
16 Kumar S to Myers J: Correspondence re health status of KCAC mine and mill workers. 26 May 92.
17 Hyde, D. (2003) in Szabo vs. Bindex et al. 5 Dec 2003.
18 Lewinsohn report December 12, 1984. A Review of Chest Radiographs of Calidria Corporation Employees on 12/12/84.
19 McDonald, A., McDonald, J: Malignant mesothelioma in North America: Cancer 1980;46:1650–1656.
20 Coleman R: New Idria serpentinite: a land management dilemma: Environ Eng Geosci 1996;2:9–22.
21 Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Edwards BK (eds): SEER Cancer Statistics Review, 1973–1999, National Cancer Institute. Bethesda MD, http:/seer.cancer.gov/csr/1973_1999/ (2002).
22 Plumlee, G, Morman, S, Ziegler, T: The toxicological geochemistry of earth materials: an overview of processes and the interdisciplinary methods used to understand them: Reviews in Mineralogy & Geochemistry 2006;64:5–57.
23 Pan X, Day H, Wang W, Beckett L, Schenker M: Residential Proximity to naturally occurring asbestos and mesothelioma risk in California AJRCCM articles in press: Published on June 23, 2005 as doi:10.1164/rccm.20041:17310C
24 Ilgren, E: Trial testimony In: Conwed v Union Carbide Corp. Case No. 5–92–88. 11 Sept. 2002b. US District Court, St. Paul Minn.
25 West Virginia Mass Consolidation against Union Carbide Corporation.
26 Barrett, P: Testimony in: Conwed v Union Carbide, 2002.
27 Kelly Moore Paint Company v Union Carbide Corp., 23rd District Court, Brazoria County, TX. Trial Court Cause No. 19785-BH02. 2004
28 Egilman, D: Testimony in: Lustgarten v A W Chesterton et al. 26 Mar 2004.
29 Ilgren E: Coalinga chrysotile - the case of the missing ''asbestos study'': corporate connivance or plaintiff ploy? Indoor Built Environ 2004c;13:357–373.
30 EPA Sunnyvale Community Meeting: 1994. Supplement No. 3 of documents: AR670 AR672 (Administrative record indices) of the atlas mine area operable unit of the atlas asbestos company NPL site.
31 Murphy R, Becklake M, Brooks S, Gaensler E, Gee B, Goldman A, Kleinerman J, Lewinsohn H, Mitchell R, Utell M, Weill: The diagnosis of nonmalignant diseases related to asbestos: Amer Rev Resp Dis 1986;134:363–368.
32 Hoskins J: Eliminating the confusion. In: Pathogenesis, diagnosis and clinical relevance of pleural plaques. (Hoskins JA, Kummer HW, Solèr M (eds.)). Indoor Built Environ 1997;6:69–71.
33 Stephens M, Gibbs AR, Pooley FD, Wagner JC: Asbestos induced diffuse pleural fibrosis: pathology and mineralogy: Thorax 1987;42: 583–588.
34 Weiss W: Asbestosis: a marker for the increased risk of lung cancer among workers exposed to asbestos: Chest 1999;115:536–553.
35 Mellon Institute: Report No. 34–70 produced for the Union Carbide company 1971 (accessed from Union Carbide on request).
36 American Thoracic Society: Diagnosis and initial management of nonmalignant diseases related to asbestos: Am J Resp Crit Care Med 2004;170:691–715.
37 LA Times ''Under a dust cloud'' (25 Sept 04) home edition
38 Ilgren E, Browne K: Asbestos-related mesothelioma: evidence for a threshold in humans and animals: Reg Tox Pharm 1991;13:116–132.
39 Goff A, Gaensler E: Asbestosis following brief exposure in cigarette filter manufacture. Case report from the thoracic services of the Boston university medical school: Respiration 1972;29:83–93.
40 Bernstein DM, Chevalier J, Smith P: Comparison of Calidria chrysotile asbestos to pure tremolite: inhalation biopersistence and histopathology following short-term exposure: Inhalation Toxicol 2003;15:1387–1419.
41 Bernstein D, Chevalier J, Smith P. Comparison of Calidria chrysotile asbestos to pure tremolite: final results of the inhalation biopersistence and histopathology following short term exposure: Inhal Tox 2005;17:427–449.
42 Baxter D: Final report – ambient asbestos concentrations in California. Volume I. Submitted by Science Applications, Inc. to CARB. Contract number AO-103–32. 1 Dec 83.
43 Berry G, de Klerk N, Reid A, Ambrosini G, Fritschi L, Olsen N, Merler E, Musk A: Malignant pleural and peritoneal mesotheliomas in former miners and millers of crocidolite at Wittenoom, Western Australia. Occup. Environ. Med. 2004; 61: e14 (http://www.occenvmed.com/cgi/content/full/61/4/e14). Doi:10.1136/oem.2003.008128.
44 Davis F: Economic mineral deposits in the coast ranges. Geology of Northern California. Bulletin 190. Cal. Div Mines Geol. pp. 315–322. (1966)
45 Divine BJ, Hartman CM: Update of a study of crude oil production workers 1946–94 Occup. Environ. Med. 2000; 57(6): 411–417
46 Maines R: Asbestos & fire – technological trade-offs and the body at risk. 2005. ISBN 0-8135-3575-1. Rutgers University Press.
47 Kumzerov Y, Parfen'eva L, Smirnov I, Misiorek H., Mucha J., Jezowski A: Thermal conductivity of crystalline chrysotile asbestos. Physics of the Solid State 2003;45:57–60. (translated from Fizika Tverdogo Tela.)
48 Ilgren Deposition re Dollar amounts. Conwed v UCC 1998.
49 Ilgren E: Health risks from exposures to asbestos, inorganic metals, and various chemicals due to collapse of the world trade center: an environmental residential survey with a commentary related to ground zero workers: Indoor Built Environ 2002;10:361–383.
50 Ilgren E: Mesotheliomas of Animals. A comprehensive, tabular compendium of the world's literature, CRC Press, Boca Rotan 1993; 356.
51 NTP Sixth annual report on carcinogens. Research Triangle Park, NC: National Toxicology Program, 1991.
52 Egilman D: Testimony to NIOSH – public meeting – asbestos and other mineral fibers: a roadmap for scientific research NIOSH Docket No. NIOSH – 099. 4 May 2007.
53 Crapo J.: Testimony given In: Monongalia Co., W. Va. 17 March 1992
54 Crapo J: Final report – ''Morphometric studies on the lungs of rats exposed by inhalation to mineral fibers'' Crapo. N01-ES-0-0004 (9 April 84) (10 pages)
55 Crapo J, Barry B, Brody A, O'Neil J: Morphological, morphometric and X-ray microanalytical studies on lung tissue of rats

+ [A3B2 Ver: 8.07r/W] [21.6.2008–12:12pm] [1–34] [Page No. 22] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

exposed to chrysotile asbestos in inhalation chambers. in ''Biological effects of mineral fibers'' (Report on a WHO/IARC meeting), Copenhagen, 20–22 April 1982. Annex 1980;l:273–283.
56 Pinkerton K, Pratt C, Brody AR, Crapo JD: Lung injury patterns in rats as a result of exposure to different chrysotile asbestos fibers. Abstract - 1982 ATS Kansas Mtg pg 250 [Environmental and Occupational Health section] (1 page) (1982).
57 Pinkerton K, Pratt C, Crapo J Morphologic lung capacity following exposure to chrysotile asbestos fibers. Abstract - 1982 ATS Kansas Mtg pg 251 [Environmental and Occupational Health section] (1 page) (1982).
58 O'Neil J, Pinkerton K, Crapo J. Lung volume changes in rats exposed to chrysotile asbestos. Abstract - ? 1982 ATS Kansas Mtg pg 146 [Environmental and Occupational Health section] (1 page) (1982).
59 NTP Tech Report 295: Toxicology and carcinogenesis of chrysotile asbestos in F344/N Rats.
60 NTP Tech Report 246: Toxicology and carcinogenesis of chrysotile asbestos in Syrian Golden Hamsters. DHSS. 1982.
61 Mc Connell, E, Shefner, A, Rust, J, Moore, J: Chronic effects of dietary exposure to amosite and chrysotile asbestos in Syrian golden hamsters. Environ. Health Perspect. 53: 11–25, 1983.
62 Truhaut R, Chouroulinkov I: Effect of long-term ingestion of asbestos fibers in rats. In: Non occupational Exposure to mineral fibers. (Bignon J, Peto J, Saracci R, eds) IARC Sci. Pub. 90, Lyon (1989), pp 127–135.
63 Crapo J.: Testimony given In: Fernandez v Union Carbide et al. Civil Action No. 02–21136, 12 Sept 2003
64 Muhle H, Pott F, Bellman B, Takenaka S, Ziem U: Inhalation and injection experiments in rats to test the carconogenicity of MMMF: Ann Occup Hyg 1987;31:755–764.
65 Platek S, Groth D, Ulrich C, Stettler L, Finnell, M, Stoll M: Chronic inhalation of short asbestos fibres: Fund Appl Toxicol 1985;5:327–340.
66 Wald NJ, Doll R (eds.): Interpretation of negative epidemiological evidence for carcinogenicity, IARC Sci Pub 65, Lyon, 1985; 232.
67 Brody AR., Hill LH, Adkins B, O'Connor RW. Chrysotile asbestos inhalation in rats: deposition pattern and reaction of alveolar epithelium and pulmonary macrophages: Am Rev Respir Dis 1981;123:670–679.
68 Brody AR, Soler P, Basset F, Haschek, WM Witschi H: Epithelial-mesenchymal association of cells in human pulmonary fibrosis and in BHT-oxygen induced fibrosis in mice: Exp Lung Res 1981;2:207–220.
69 Brody A, Hill L, Adkins B, O'Connor R: Chrysotile asbestos inhalation in rats: Deposition pattern and reaction of alveolar epithelium and pulmonary macrophages: Am Rev Respir Dis 1981;123:670–682.
70 Brody AR, Hill LH: Interstitial accumulation of inhaled chrysotile asbestos fibers and consequent formation of microcalcifications: Am J Pathol 1982;109:107–114.
71 Brody AR, Hill LH: Deposition pattern and clearance pathways of inhaled chrysotile asbestos: Chest 1981;80:64–67.
72 Barry BE, Wong KC, Brody AR, Crapo JD: Reaction of rat lungs to inhaled chrysotile asbestos following acute and subchronic exposures: Exp Lung Res 1983;5:1–21.
73 Warheit DB, Chang LY, Hill LH, Hook GER, Crapo JD, Brody AR: Pulmonary macrophage accumulation and asbestos-induced lesions at sites of fiber deposition: Am Rev Respir Dis 1984;129:301–310.
74 Warheit DB, Hill LH, Brody AR: Surface morphology and correlated phagocytic capacity of pulmonary macrophages lavaged from the lungs of rats: Exp Lung Res 1984;6:1–82.
75 Chang L, Overby L, Brody A, Crapo J: Progressive lung cell reactions and extracellular matrix production after a brief exposure to asbestos: Am J Pathol 1988;131:156–170.
76 Wagner JC, Berry GB, Hill RJ, Munday DE, Skidmore JW: Animal experiments with MMM(V)F - effects of inhalation and intrapleural inoculation in rats; in: Biological Effects of Man-Made Mineral Fibres (Report on a WHO/IARC meeting), Copenhagen, 20–22 April 1982. (Annex 1984;42:210–235).
77 McConnell EE, Wagner JC, Skidmore JW, Moore JA: A comparative study of the fibrogenic and carcinogenic effects of UICC Canadian chrysotile B asbestos and glass microfibre (JM 100); in: Biological Effects of Man-Made Mineral Fibres (Report on a WHO/IARC meeting), Copenhagen, 20–22 April 1982. (Annex 1984;45:234–242).
78 McConnell E, Wagner J, Skidmore J, Moore J: Two inhalation studies: A comparison; in: Biological Effects of Man-Made Mineral Fibres (Report on a WHO/IARC meeting), Copenhagen, 20–22 April 1982. (Annex 1984;38:118–120).
79 Pinkerton K, Pratt C, Brody AR, Crapo JD: Fiber localization and its relationship to lung reaction in rats after chronic inhalation of chrysotile asbestos: Am J Pathol 1984;117:484–498.
80 Pinkerton K, Plopper CG, Mercer RR, Roggli VL, Patra AL, Brody AR, Crapo JD: Airway branching patterns influence asbestos fiber location and the extent of tissue injury in the pulmonary parenchyma: Lab Invest 1986;55:688–695.
81 Pinkerton K, Young S, Fram E, Crapo J: Alveolar type II cell responses to chronic inhalation of chrysotile asbestos in rats: Am J Respir Cell Mol Biol 1990;3:543–552.
82 Langer A., Mackler S, Pooley F: Electron microscopic fibers investigation of asbestos: Environ Health Perspect 1974;9:63–80.
83 Rohl A, Langer A, Selikoff I: Environmental asbestos pollution related to use of quarried serpentine rock: Science 1977;196:1319–1322.
84 Langer AM, Wolff MS, Rohl A, Selikoff I: Variation of properties of chrysotile asbestos subjected to milling: J Tox Environ Health 1978;4:173–188.
85 Langer AM, McCaughey WTE: Mesothelioma in a brake repair worker. Lancet 1982;ii: 1101 1103. (November 13); (8307):
86 Rohl A., Langer A, Selikoff I, Nicholson W: Exposure to asbestos in the use of consumer spackling, patching and taping compounds: Science 1975;189:551–553.
87 Langer A, Rohl A, Selikoff I: Asbestos on Maryland's roads: Lancet 1978;ii:1263–1264 (10 June).
88 IARC monographs on the evaluation of carcinogenic risk of chemicals to man, Asbestos, Vol. 14 IARC, Lyon, France, 1977; 106.
89 EPA. Ambient water quality criteria for asbestos. EPA 440/5–80–022. US EPA, Wash., DC, 1980.
90 NTP Annual Report 1982.
91 NTP Annual Report 1983.
92 Pinkerton, Pratt, Miller, and Crapo [∼1982] ''Lung reaction to chronic inhalation of three chrysotile in the F344 rat. I. Morphometry. (query - Submitted to (and probably rejected by) Environ Res as cited in Final Report above) (23 pages).
93 Becton Dickinson ''Final Report on Asbestos Study.'' Undated. 41 pages.
94 Crapo J.: Testimony given In: Civil Action No. 92-C-8888, Kanawha, Co., W. Va. 4 May 94.
95 Crapo J.: Testimony given In: Civil Action No. 95–8888, Kanawha, Co., W. Va. 5 Sept 95.
96 NIEHS Invitation to Request for Proposal. 4 Sept 79. RFP No. NIH – ES – 79 – 13 (Morphometric studies on the lungs of rats exposed by inhalation to mineral fibers) from Dr Elizabeth B. Ford, Procurement Officer, OAM, NIEHS.
97 ''Notice of Research Project: R807255 ''Morphometric studies of the effects of ozone on rodent lungs.'' 12 July 79.
98 NIH Grant PO1 HL31992–11A1 ''Acute Lung Injury – Mechanisms and therapy [1995–2000] $7,069,364.''
99 NIEHS Grant N01-ES-9-0007 (1 May 79 – 31 Jan 80) ''Negotiated Contract'' [30 April 79] – 14 pages - ''Morphologic and morphometric studies on rat lungs exposed to chrysotile asbestos'' TB#82.
100 Waggoner v Raybestos Manhattan et al. (1997) Sup. Court Calif. #005856
101 Churchill R, Hill R: A general location guide for ultramafic rocks in California: areas more likely to contain naturally occurring asbestos. CA Dept. Conserv., Div. Mines and Geol., DMG Open-File Report 2000–19. http://www.consrv.ca.gov/
102 Woodward Clyde Consultants (WCC) and Camp Dresser & Mc Kee: ''Draft Regional Study of Mining Disturbances of Asbestos - Bearing Material in the New Idria - Coalinga - Table Mountain study region - Performance of Remedial Response activities at Uncontrolled Hazardous Waste Sites'' under EPA Contract No. 68 - 01 - 6939 [28 Feb 89]
103 Schenker National Cancer Institute Project Grant: IR03CA81615-01 ''Mesothelioma in California''.
104 Schenker deposition testimony In: Sierra Terra v CARB et al. (2006) pp 127–128].
105 Crapo, Affidavit: Exposure to Calidria asbestos and other forms of asbestos and their roles in disease causation. 16 Apr 03.
106 NIEHS Memo: 3 Jan 85 Boorman, G, NTP to Eustis, S, EPL cc Dr Mc Connell;
107 NIEHS Memo: 3 July 85 re 'NTP asbestos' – 'Northrop laboratory' requesting the ''Release'' of study materials by Dr G Boorman.
108 NIEHS Memo: 11 March 93: from Non Key employee EPL to Lily Hong NIEHS acknowledging return of study materials to NIEHS.
109 NIEHS Memo: 3 Jan 85 Boorman, G to Cozart, L, NIEHS, NIEHS].

110 NIEHS Memo: 12 March 93 from Dr. Scot Eustis to Annette Shambley, a ''Non-Key Contract Employee'' as ''Delivery Slip'' from EPL Annette Shambley to Lily Hong, NTP.
111 Becton Dickinson Quarterly Report ''Effects of chronic exposure to airborne environmental agents.'' 1 April 1978 – 31 July 78. ''Submitted to NIEHS'' under Contract Number NIE – NIEHS – N01-ES-4-2164. Stone & Carter. 49 pages.
112 Becton Dickinson Quarterly Report ''Effects of chronic exposure to airborne environmental agents.'' 1 August 1978 – 31 Oct 78. ''Submitted to NIEHS'' under Contract Number NIE – NIEHS – N01-ES-4-2164. Stone & Carter. 48 pages.
113 Becton Dickinson Quarterly Report ''Effects of chronic exposure to airborne environmental agents.'' 1 Nov 1978 – 31 Jan 79. ''Submitted to NIEHS'' under Contract Number NIE – NIEHS – N01-ES-4-2164. Stone & Carter. 36 pages.
114 Becton Dickinson Technical Report No. 79 – Expermental protocol for fiber exposures. Feb., 1978 34 pages. Cavendar, Stone, and Steinhager.
115 Selikoff I, Irving: Health Protection in Construction: Problems and Prospects. Hazardous Materials Management J 1980; January/February: 8–13.
116 Selikoff IJ, Hammond EC: Asbestos and smoking: J Am Med Assoc 1979;242:458–459.
117 Brody A.- telephone dep - 4 May 94: Kanawha county, W Va [Civil action No. 92 - C - 8888]
118 NIEHS Negotiated contract N01-ES-9-0004 (June 20, 1980 to June 19, 1983) 27 June 80; ''Morphologic and morphometric studies on rat lungs exposed to chrysotile asbestos fibers,'' ($327,446).
119 NIEHS Memo: Johnston to Anlyan 17 Feb 81Cc Brody as ''Project Manager'' re permission to purchase equipment
120 NIEHS Memo: Miller to Henschel 10 Feb 81;
121 NIEHS Memo: Crapo to Henschel 10 July 80;
122 NIEHS Memo: Anlyan to NIEHS 13 April 83;
123 NIEHS Memo: Crapo to Brody 17 Jan 83;
124 Memo – Request for Contracting Officer's Authorization re N01-ES-0-0004 to Brody, Project Officer. From Crapo for Travel expenses $1,157.31. (1 page) (7 Feb 84)

Appendix 1

Individual Summaries of the 19 allegedly Attributable UCC Cases

Worker 12

''(*Right*) *upper lobe_ (lung)? Carcinoma'' (1982)*

Pleural abnormalities consistent with pneumoconiosis (1982)

''*od*'' *Indicated in comments (1982)*

''pi'' *indicated in comments (1982)*

Source Cited Lewinsohn Report

The patient was alive in November 2005 so the questionable carcinoma noted in a 1982 chest film could not have been the correct diagnosis. The hilar ''clips'' (found) ''in the vicinity of the right upper lobe'' (as indicated in the ''comments'' section as ''od'') must therefore have been mistaken for the ''carcinoma.'' This worker began working at UCC in 1963 therefore none of the findings noted in 1982 could have been due to Calidria exposure due to insufficient latency. There is no radiological evidence of asbestosis reported in any of the chest film reports. The radiological findings recorded in 1982 were due to cardiac disease, pulmonary thrombo-embolism, chronic pulmonary infection, smoking, and trauma. Indeed, parenchymal changes due to chronic infection predated the findings cited by Egilman by nearly 20 years. The pleural changes (including the diffuse left sided pleural thickening and the ''pleural thickening in the inferior fissure or mediastinum'' (pi) noted in the 6 April 82 film) are also due to one or more of the aforementioned factors.

Work history: DOH 7/16/63 as a Mill laborer; Shipping foreman (6/1/65); Shipping supervisor (2/1/68); Shift supervisor (1/1/70); and disability and retirement due to heart disease (2/3/78).

Worker 25

'''*Asbestosis*' *written on report*''

Source – ''King City Asbestos Mill Surveillance Report''

Asbestosis was only cited in an undated Union Carbide Periodic Medical Examination sheet that stated: ''Do you have regular significant exposure to . . . dust, specify type if possible.'' Immediately to the right of this comment is written the word ''Asbestos.'' In a separate column further to the right entitled ''Detail dates complications etc'' is written ''4. Asbestosis.'' There is nothing in this worker's records to suggest he had asbestosis. This is further supported by the fact that the second page of this same medical examination form said the worker was ''not required to wear respirator''. There was no radiological evidence of asbestos related disease. Ten chest X ray reports from 1974 to 1989 were read as normal. A chest X ray report of 4 April 91 reported ''some minimal apical pleural thickening bilaterally.'' This was probably due to heart disease and/or pulmonary infection. Its apical location is also not diagnostic of asbestos exposure. Dr Peter Barrett also independently reviewed five chest films from 1987 to 1992 and found no evidence of asbestosis or pleural plaques. There was no spirometric evidence of asbestosis. Thus, 11 PFT reports from 1975 to 1989 were reported as normal. A sixth reported minimal ''restrictive indications'' (VC 79%) but these were said to be due to smoking and obesity. A chest X ray taken at the same time as this PFT (14 May 86) was read as normal. There was also no clinical evidence of asbestosis. Thus, rales were not reported and he was said to be ''fit'' for work. He died after 1992. Death certificate could not be found.

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:12pm] [1-34] [Page No. 24] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

Work history: 2/8/73, mill laborer; bagger, palletizer, 3/18/73; Operator, outside, 12/28/75; Rock fiber operator, 4/26/81; Press dryer operator, 4/27/87; Retired, 7/12/92.

Worker 72

Parenchymal abnormalities consistent with pneumoconiosis (*1966*)

ILO section ''small irregular opacities'' filled in: 1/0 (1966)

''*pi*'' *indicated in comments (1966)*

''*cn*'' *indicated in comments (1966)*

''*cn*'' *indicated in comments (1982)*

Source Cited ''Lewinsohn Report''

Union Carbide Calidria operations did not commence until 1963 so the questionable findings noted in 1966 could not be due to exposure to Calidria asbestos due to insufficient latency. Since this worker did not start working at UCC until 1966, all of the aforementioned comments are also not attributable for the same reason. There is no radiological evidence of asbestosis. Asbestosis is diagnosed on the basis of lesions 1/1 or higher whilst those reported were only 1/0. The lesions of asbestosis also do not regress but the films from 1966 to 1994 failed to note parenchymal abnormalities. The ''other abnormalities'' ''cn'' (calcification in small pneumoconiotic opacities) and ''pi'' (pleural thickening in the inferior fissure or mediastinum) are not diagnostic of asbestos related disease but highly suggestive of chronic granulomatous disease. Some of the radiological findings may also have been due to smoking and obesity. There was no spirometric evidence of asbestos related disease as numerous spirometric tests were normal and clinically he was always said to be ''fit for work,'' having neither dyspnea nor rales. This worker was alive and well in November 2005.

Work history: Laborer, 8/22/66; shipping, 10/1/66; bagger, 2/1/67; lab technician, 4/1/67; warehouseman, 7/1/67; accountant, 6/1/84; terminated 9/30/02.

Worker 83

''Appearances on the left chest wall maybe due to previous trauma or pleurisy'' (1979)

Pleural abnormalities consistent with pneumoconiosis (1983)

Source Cited Lewinsohn report

This worker did not start working at UCC until 1980 so the changes noted in 1979 cannot be attributable. The changes reported in 1983 also cannot be attributable due to insufficient latency. There is no radiological evidence of asbestosis. The pleural changes are not attributable since they are unilateral whilst asbestos related pleural plaques are bilateral. The pleural changes noted in 1979 and 1983 are therefore nonspecific and probably due, as Egilman suggests, to ''trauma or pleurisy.'' Five chest X rays taken between 1980 and 1984 were reported as normal. Dr Peter Barrett also independently read a report in 1984 as normal. Since asbestos related changes do not disappear, this constitutes yet further evidence that the findings observed in 1979 and 1983 were not due to asbestos exposure. There is no spirometric evidence of asbestos related disease. Thus, 4 PFT's performed between 1980 and 1983 were normal whilst one in 1980 suggested mild obstruction which was probably due to smoking. There was no clinical evidence of asbestosis. Thus, there were no rales or shortness of breath as he was said to be in good health and fit for work. He died of myocardial infarction and diabetes type II in 1988. The death certificate did not mention asbestos related disease.

Work history: laborer, 11/25/80; bagger, 1/25/81; laborer, 3/1/82; bagger, 8/3/82; terminated 10/7/85; deceased, 2/2/88.

Worker 108

parenchymal abnormalities consistent with pneumoconiosis (1969)

ILO section ''small irregular opacities'' filled in: 0/1 (1969)

''co'' indicated in comments (1969)

parenchymal abnormalities consistent with pneumoconiosis (1983)

ILO section ''small irregular opacities'' filled in: 1/1 (1983)

''the smaller capacities (sic) are more obvious than they were in 1969'' (1983)

''pi'' indicated in comments (1983)

''ki'' indicated in comments (1983)

Sources cited Lewinsohn Report

This worker did not start working at UCC until 1976. Thus, the findings cited in 1969 could not be attributable. Those cited in 1983 would not be attributable due to insufficient latency. Similarly, the changes noted in 1969 are not attributable to Calidria exposure due to insufficient latency as the Union Carbide Calidria operation did not start until 1963. There was no radiological evidence of asbestos related disease. The diagnosis of asbestosis requires a 1/1 grade or higher so the 1969 ILO film read as 0/1 is not attributable. Nine films taken up to 19 years after 1969 were read as normal. Since the lesions of asbestosis do not regress, these findings were not due to asbestos exposure. The 1/1 changes seen in the 31 March 83 film do not represent asbestosis since four films were reported as normal after that date, a finding consistent with the fact that Dr Peter Barrett personally reviewed

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:12pm] [1-34] [Page No. 25] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

nine other films after 1983 and found these to be normal as well. Variability in the radiological findings was probably due to heart disease, a long history of smoking (vi) and progressive emphysema. There was no clinical evidence of asbestosis. Thus, rales were not reported; there was no record of shortness of breath; and he was said to be fit for work. There was no spirometric evidence of asbestosis. Thus, his spirometry was either normal or demonstrated slight obstruction. This worker died from cancer of the esophagus on 16 June 95 probably due to smoking, chronic gastro-esophageal reflux, and esophageal stricture. The death certificate did not mention asbestos related disease. The comments cited by Egilman namely ''pi'' (''pleural thickening in the interior fissure or mediastium), kl (''septal (Kerley) lines'') and ''co'' (an ''abnormality of cardiac size or shape'') are not asbestos related changes.

Work history: 1/22/76, bagger; 4/30/78, shipping II; 6/24/78, press, dryer operator II; 3/30/80, press dryer operator I; 2/15/90, retired; 6/15/95, deceased.

Worker 131

Parenchymal (1981) abnormalities consistent with pneumoconiosis

ILO section ''small irregular opacities'' filled in: 1/0 (1981)

Pleural (1981) abnormalities consistent with pneumoconiosis

''co'' indicated in comments (1981)

Parenchymal (1982) abnormalities consistent with pneumoconiosis

ILO section ''small irregular opacities'' filled in: 0/1 (1982)

''co'' indicated in comments (1982)

''Mild restrictive pulmonary impairment'' (1993)

Source cited Lewinsohn Report

Neither the 1981 nor the 1982 parenchymal abnormalities were due to Calidria exposure since he only started working at UCC in 1981. Moreover, neither the 1981 nor the 1982 chest films reach the 1/1 profusion score required for the diagnosis of asbestosis. Multiple X ray reports between 1981 and 1994 were reported as normal and this was confirmed by Dr Barrett's personal review of films from 1986 to 1992 for which he found no evidence of asbestosis or pleural plaques. Since the lesions of asbestosis do not disappear with time, these findings provide further evidence that the 1981 and 1982 parenchymal abnormalities do not represent asbestosis. Technical problems noted by Dr Lewinsohn with both X-rays (an ''I graining effect'' in 1981 and over penetration in 1982) may also have contributed to the observed abnormalities. There is no clinical evidence of asbestosis. Neither rales nor shortness of breath were noted and he was said to be fit for any type of work. The spirometric data do not confirm a diagnosis of asbestosis. Thus, five PFT's were normal. Three were mildly restrictive but this finding was ascribed to obesity. An undated PFT also said there had been ''improvement from the previous study of 12/17/92'' which is not in keeping with a diagnosis of asbestosis. The pleural abnormalities noted in the 1981 ILO reading could not have been bilateral pleural plaques due to his work at UCC, since pleural plaques cannot form within a year after first exposure. The ''co'' comments refer to ''abnormalities of cardiac size and shape'' which do not denote asbestos related disease. This worker was alive and well in November 2005.

Work history: 4/21/81, bagger; 6/21/86; 2/1/88, pkg operator; 9/10/98, terminated; 2/10,99, retired, laborer; 4/30/03, terminated.

Worker 178

''Shows X-ray evidence of an early pneumoconiosis, must wear respirator in dust.'' (1970)

Source Cited King City Asbestos Mill Surveillance Program

The ''comment'' cited above does not constitute evidence of asbestos related disease. There is insufficient latency for the findings in 1970 to be causally related to Calidria exposure since the Union Carbide Calidria operations did not begin until 1963. The same holds true in relation to his date of hire in 1960. There is no radiological asbestosis. No less than 17 chest film reports from 1966 to 1994 were read as normal. Dr Peter Barrett also personally read five chest films from 1984 to 1994 and found no evidence of asbestosis or pleural plaques. Smoking and possibly heart disease may have contributed to the radiological appearances noted in 1970. There was no spirometric evidence of asbestosis. Thus, 19 spirometric readings over the 20 year period from 1974 to 1994 were read as normal. There was no clinical evidence of asbestosis. Thus, rales were not heard, there was no shortness of breath, and he was fit for work. He was alive and well in November 2005.

Work history: 3/28/60, surveyor's helper; 5/4/64, engineering assistant; 2/1/71, mine foreman; 7/1/85, mine superintendent; 3/31/95, retired.

Worker 217

Parenchymal abnormalities consistent with pneumoconiosis (1966)t

ILO section ''small irregular opacities'' filled in: 0/1 (1968)t

*Minimal increase in the interstitial lung markings'' (1977)**

*''Mild to moderate obstructive disease'' (1983)**

Parenchymal abnormalities consistent with pneumoconiosis (1984)t

ILO section ''small irregular opacities'' filled in: 1/0 (1984)t

*''There is a moderate obstructive lung defect'' (1986)**

*''Mild obstructive lung disease'' (1986)**

*''There is a mild obstructive lung defect'' (198_)**

Sources Cited King City Asbestos Mill Surveillance Program*/Lewinsohn Report *t*3

This worker started working at UCC in 1966 so none of the changes noted in that year, in 1968, or in 1977 could be due to Calidria exposure due to insufficient latency. In addition, the 1966 ILO report said ''this film may be normal but in view of technique has been read as showing evidence of small opacities.'' The 1966 film was then personally reviewed by Dr Peter Barrett who said that it failed to show any evidence of asbestosis or pleural plaques. Moreover, 22 X-ray reports between 1972 and 1987 were read as normal. Dr Barrett also personally reviewed 13 X-rays from 1966 to 1987 and said these also failed to display asbestosis or pleural plaques. Moreover, since the changes due to asbestosis do not regress, none of the findings noted above in 1966, 1968 (This is probably a typo. since there is no 1968 film in the records.), 1977, or 1984 could be asbestos related. The UCC Calidria operations also started in 1963 so the findings noted in 1966, 1968, and 1977 could not be attributable to Calidria exposure again on the basis of insufficient latency. The X-ray scores cited above were 0/1 and 1/0. Since a profusion score of 1/1 is required for the diagnosis of asbestosis, neither film would be diagnostic of this condition. Moreover, the abnormalities initially appeared in the lower and middle zones but asbestos related interstitial markings typically begin in the lower lung fields. The variability seen in the radiological picture may have been due to technical problems (vs.) and possibly early obstructive emphysema due to asthma. He never smoked. The asthma developed shortly after birth probably on an allergic basis, was manifested as wheezing, and recorded as mild to moderate obstruction in 17 PFT reports from 1975 to 1987. This was explicitly said to be due to asthma in four reports. There was no clinical indication of asbestosis. Thus, rales were not found and he was said to be fit for any type of work. There was no spirometric evidence of restriction noted in 19 pulmonary function tests conducted between 1974 and 1987. He was alive and well in November 2005.

Work history: 17 Jan 66, Laborer; 31 Oct 66, Op II; 15 Apr 74, Mechanic; 20 July 75, Rock fiber Op/Outside Op; 1 Jan 76, RG Op; 8 Oct 79, Mech I

Worker 218

''pi'' indicated in comments (1958, 1961)

parenchymal abnormalities consistent with pneumoconiosis (1973)

''Appears to be developing bilateral basal fibrosis''

ILO section ''small irregular opacities'' filled in: 1/0

''pi'' indicated in comments

parenchymal abnormalities consistent with pneumoconiosis (1983)

''co'' ''od'' ''kl'' ''pi'' indicated in comments

ILO section ''small irregular opacities'' filled in: 1/0

parenchymal abnormalities consistent with pneumoconiosis (1984)

ILO section ''small irregular opacities'' filled in: 0/1

''moderate obstructive disease''

''co'' ''od'' ''kl'' ''pi'' indicated in comments

Source Cited Lewinsohn Report

The UCC Calidria operation did not start until 1963 and he did not begin work there until 1978. Therefore, the parenchymal changes noted in the 1973, 1983, and 1984 films could not have been due to Calidria exposure on the basis of insufficient latency since the changes were even noted 25 years before he began working at UCC (23 June 53). Moreover, asbestosis is diagnosed on the basis of ILO grades 1/1 or higher. As this criterion was not met in any of the films, the changes are not diagnostic of asbestosis. Similarly, the grades appeared to lessen with time whilst those of asbestosis do not decrease. Nine chest X-ray reports from 1953 to 1985 were read as normal. Dr Peter Barrett also personally reviewed a 1985 X-ray and felt this was normal. The variability in the radiological appearances was probably due to heart disease, post op changes following coronary bypass surgery, smoking, and possible chronic granulomatous disease. There was no clinical evidence of asbestosis. His lungs were clear without rales, He was in good health (Aug 84) and fit for work (1 Aug 84). There was no spirometric evidence of restriction. The last pulmonary function test was normal (16 July 85) whilst the preceding eight demonstrated mild to moderate obstruction clearly due to alternate causes cited above. None of the ''symbols'' cited above (i.e., pi (''pleural thickening in the inferior fissure or mediastinum''), co (an ''abnormality of cardiac size or shape''), od (''other significant abnormality''), and ki (septal (Kerley) lines)) denote asbestos related disease. The ''pi'' (pleural) changes noted above were not even recorded as ''pleural abnormalities'' in any of

the four (1958, 1961, 1973, 1983, 1984) films in which they were cited. Dr Peter Barrett also failed to find evidence of pleural plaques on a 9 July 85 film he personally reviewed. He was alive and well in November 2005.

Work history: 1/1/78, master mechanic; 1/1/82, mill main. Super.; 6/28/85, retired.

Worker 291

*''There is a moderate obstructive lung defect'' (1986)**

*''Moderate Restrictive pulmonary impairment'' (1993)**

Source Cited King City Asbestos Surveillance Program*

Worker 291 did not start working at King City until 1981. Therefore, the lung function changes cited above in 1986 and 1993 could not be due to exposure to Calidria asbestos due to insufficient latency. The same applies to a PFT said to display mild restriction in 1983. Five pulmonary function tests were said to be normal between 1981 and 1986. Three others reported mild to moderate obstruction. The findings on spirometry were thus due to smoking and rheumatoid arthritis. There was no radiological evidence of asbestosis. Nine films from 1981 to 1989 were reported as normal. Three others reported obstruction. Dr Peter Barrett personally reviewed eight chest X-rays and found no evidence of asbestosis or pleural plaques. There is no clinical evidence of asbestosis. Thus, there was neither dyspnea and nor rales. He was said to be fit and considered himself to be in good general health. He walked 3 miles a day, jogged daily for 40 years and got ''regular vigorous exercise.'' He died in 2002 of cancer of the esophagus.

Work history: 1/20/81, bagger; 11/3/86, packaging operator; 8/1/88, rock fiber operator; 1/28/94; died, 8/10/02.

Worker 311

Cancer listed as ''ca'' under ''other abnormalities'' (1984) t

Lewinsohn Report

Worker 311 never had cancer as he was alive and well in November 2005 after 11 years of follow up. The original suspicion that the lesion was a ''nipple shadow'' was therefore correct. This is confirmed by the disappearance of the suspect lesion in subsequent films from 1986 to 1994, all read as normal. There was no radiological evidence of asbestosis in 23 films from 1970 to 1994. Therefore, even if the lesion had been was cancerous, it would not be attributable to asbestos since asbestos related cancer requires an asbestosis prerequisite [34]. This worker was hired in 1969 so there was insufficient latency for the lesion to be attributable. There was no spirometric evidence of asbestosis as all 24 pulmonary function tests were normal. There was also no clinical evidence of asbestosis. Thus, there were no rales and he was always fit for work. Dyspnea was noted in 1983 and 1987 but was not mentioned in any other clinical record from 1979 to 1994. It was undoubtedly due to smoking, chronic bronchitis, chronic allergy, and possibly infectious sinusitis. This worker was alive and well in November 2005.

Work history: 10/5/69, laborer; 10/7/72, mechanic; 10/4/99, accident – disability.

Worker 358

''od'' indicated in comments (1966) t

''co'' indicated in comments (1966) t

*''May be the first indication of obstructive lung disease'' (1970)**

Parenchymal abnormalities consistent with pneumoconiosis (1972) t

0/1 profusion (1972) t

''pi'' indicated in comments (1972) t

''Contraction of Right lower lobe and pleural thickening in horizontal fissure'' (1972) t

*''Suggestive of asbestosis'' (1974)**

''co'' indicated in comments (1984) t

''pi'' indicated in comments (1984) t

''The appearances are consistent with pleural calcified plaques—? Asbestos-related'' (1984) t

No definite asbestos seen. (1984) t

Indefinite opacity on (right) lobe above diaphragm—rule out ca'' (1984) t

Cancer listed as ''ca'' under ''other abnormalities (1984) t

Pleural abnormalities consistent with pneumoconiosis (1984) t

Source cited King City Asbestos Mill Surveillance Program */Lewinsohn Report t

Worker 358 did not start working at the UCC Calidria facility until 1964. Therefore, none of the findings cited above between 1966 and 1974 could be attributable to Calidria asbestos exposure due to insufficient latency. There is no radiological evidence of asbetosis despite the one record that was said to be ''suggestive of asbestosis (1974)'' by a local radiologist at the Mees Hospital in King City This film was sent to Professor George Jacobsen, Chairman of Radiology at UCLA for independent review on two occasions. He was unable to confirm asbestos related disease on either (see letter below):

> 3 May 74: Letter: John Welsh, Corporate Medical Director UCC to George Jacobsen, Radiology, LA County hospital: . . . ''I am sending you six films on worker 358 (9/3/64; 3/8/66; 2/23/67; 9/29/70; 6/29/72; 12/20/73) . . . presently employed in an asbestos mill in King City . . . on a recent visit to this location I reviewed

the medical records of all employees and found this one to be questionable. The spirometry reported had been unavailable until recently . . . I am sending them to you so that you can give us your opinion concerning the diagnosis and if a diagnosis of asbestosis has not been made radiologically, what your recommendations would be.. The levels of chrysotile to which this man has been exposed were never excessive; and although the requirements have recently been lowered, we feel that we have maintained a safe workplace from the start; 8 May 74 Jacobsen to Welsh: 9/3/64 – pleural spaces are normal and the lungs are clear; 8 Mar 66: ''several pleural diaphragmatic adhesions are now present at the right base. No other changes'' 2/23/67; 12/20/73 – ''The pleural diaphragmatic adhesions are still present, though not as prominent as on the film of 3/8/66'' Impression: ''The pleural changes which appeared on the film of 3/8/66 are presumed to have been the result of an inflammatory process and not related to the question of asbestosis. In all other aspects the CXR's are within normal and there are no changes indicative of asbestosis or, for that matter, obstructive airways disease''; 7 June 74: Welsh to Jacobsen: ''I have just been informed that an X-ray taken recently on worker no. 358 has been diagnosed as asbestosis by the radiologist at the George Mee hospital in King City. He also stated that he had not yet received the X-rays from your office . . . therefore I have asked that the new CXR be sent to you for review''; 14 June 74: Jacobsen to Welsh: ''I have reviewed the CXR of worker 358 dated 6 May 74: ''There is a density in the right cardiophrenic angle which requires actual comparison with the previous chest films to determine whether it is of recent origin. I doubt that it is of clinical significance. Again, however, I see no changes to suggest asbestosis or any other form of pneumoconiosis''

Dr Peter Barrett also personally reviewed nine chest X-rays from 1967 to 1994 and found no evidence of asbestosis or pleural plaques. Twenty-one chest film reports from 1964 to 1987 failed to report asbestos related disease. Since the lesions of asbestosis do not regress, the parenchymal findings reported above are not due to asbestos exposure. Egilman himself noted that the radiologists failed to find asbestosis in the 1984 chest film. There was no clinical evidence of asbestosis. Thus, rales were not heard; there was no shortness of breath; he was believed to be in good general health; and was fit for work. There was no spirometric evidence of asbestosis. Thus, 12 pulmonary functions tests were normal whilst six showed early minimal obstruction probably due to ''emphysema, entrapment or asthma.'' These may have been due to exposure to acid and solvents. Spirometric results for tests done in 1974 and 1975 were sent to Dr Clark Cooper, M.D. for outside review (referred to as Case 4 on p. 5 In: 'Review of occupational health program at the King City Plant of Union Carbide Corporation (with emphasis on asbestosis), 9 Aug 76) and he found ''no evidence of functional abnormality.'' As the pleural abnormalities cited above pi (''pleural thickening in the inferior fissure or the mediastinum''); od (''Contraction of Right lower lobe and pleural thickening in horizontal fissure'') are unilateral and pleural plaques are bilateral, the findings thus cited are not due to asbestos exposure but more likely related to chronic granulomatous disease. Once again they are not attributable due to insufficient latency. The ''co'' symbols cited above in the 1966 and the 1984 reports represent ''abnormalities of cardiac size or shape.'' These are not diagnostic of asbestos related disease. Another note states: ''May be the first indication of obstructive lung disease'' (1970). Obstruction is not diagnostic of asbestos related disease. The lesion cited in the 1984 report could not have been a lung cancer since a chest X ray report indicates he was alive in 1994 and was probably alive and well in November 2005.

Work history: 9/14/64, laborer; 3/8/65, operator; 3/26/74, mechanic; 8/2/88, terminated.

Worker 360

Death certificate— ''lung cancer'' and ''chronic obstructive pulmonary disease'' (1994)

Source Cited King City Asbestos Mill Surveillance Report

This worker began working at UCC in 1971. He died of a smoking related lung cancer – he admitted to at least a 30 pack year history.

He was told to quit numerous times since his continued smoking produced numerous atypical sputum samples as well as obstructive changes on spirometry and radiography. COPD was listed as a contributing factor to his demise on his death certificate (29 Feb 94). His risk factors for a smoking related cancer were increased by obesity, possible excessive alcohol intake, and exposure to gas, diesel, and solvents. There was no radiological evidence of asbestosis. Thus, twelve chest film reports from 1971 to 1986 were normal; one showed ''effects from his smoking.'' Dr Peter Barrett personally reviewed six chest X-rays and said none of these demonstrated evidence of asbestosis or pleural plaques. There was no spirometric evidence of asbestosis. Thus, eight PFT's were normal or showed

minimal changes whilst the other 12 showed mild to moderate obstruction. There was no clinical evidence of asbestosis. Thus, there were no rales or shortness of breath and he was said to be fit for work. This worker died in 1994. The death certificate listed lung cancer as the primary cause of death and COPD as a significant contributing condition. There was no autopsy.

Work history: 8/4/71, laborer; 2/7/72, operator; 3/6/78, mechanic; 8/4/71, maintenance; 6/30/93, terminated disability.

Worker 362

ILO section ''small irregular opacities'' filled in: 0/1 (1972) t

parenchymal abnormalities consistent with pneumoconiosis (1972) t

*''evidence of obstructive Lung disease'' (1980)**

ILO section ''small irregular opacities'' filled in: 0/1 (1982) t

parenchymal abnormalities consistent with pneumoconiosis (1982) t

pleural abnormalities consistent with pneumoconiosis (1984) t

Sources Cited Lewinsohn report t/King City Asbestos Mill Surveillance Report *

This worker began working at UCC in 1973 so none of the ''comments'' cited above could be due to Calidria asbestos exposure due to insufficient latency. There is no radiological evidence of asbestosis. Thus, the parenchymal changes noted above in 1972 (0/1) and 1982 (0/1) do not reach a profusion score of 1/1. In addition, since numerous chest X-ray reports after 1972 and 1982 were read as normal, the changes noted in 1972 and 1982 are not diagnostic of asbestosis since such lesions do not regress. Also, the poor quality of the 1972 chest X-ray made the reader ask if the SIOs (or small irregular opacities) may have been artefactual. Dr Peter Barrett also reviewed a recent chest film for this worker and saw no evidence of asbestosis (or pleural plaques). The variability in the appearance of the radiological findings may have been due to smoking, welding fumes, obesity, and subpleural fat. There was no clinical evidence of asbestosis. Thus, there were no rales, and he was fit for work. There was no spirometric evidence of restrictive disease or asbestosis. Thus, seven PFTs between 1974 and 1986 were normal whilst two demonstrated obstructive disease. He was alive and well in 2005.

Work History: Laborer and bagger: 1973 to 1986

Worker 371

''Calcified opacities'' (1982) t

Parenchymal abnormalities consistent with pneumoconiosis (1982) t

''small irregular opacities'' filled in: 0/1 (1982) t

''od'' indicated in comments (1982) t

Sources Cited Lewinsohn report *t*

This worker did not start working at UCC until 1979. Therefore, none of comments cited above represents asbestos related disease due to insufficient latency. There is no radiological evidence of asbestosis. Thus, eight chest X-ray reports from 1979 to 1985 were read as normal. Since the lesions of asbestosis do not regress, the findings in the 1982 are not asbestosis. In addition, the 0/1 reading in 1982 film is also not diagnostic of asbestosis since it does not fulfill the ATS criterion for a 1/1 reading. The ''minimal'' changes noted in the 1982 chest film may have been due to technical factors, old granulomatous disease, and spondylitic arthritis. The ''calcified opacities'' noted above were also hilar in origin, not pleural. There is no spirometric evidence of asbestosis. Thus, eight PFTs from 1979 to 1982 were also normal. Two pulmonary function tests in 1985 and 1986 raised the possibility of a very mild (VC $\sim$71%) restrictive defect. However, the 1985 report said the ''mild decrease in FVC . . . could indicate some mild restrictive disease . . . related to spondylosis arthritis'' a known cause of restriction. There was no clinical evidence of asbestosis as rales were never heard and he was fit for work except in 1985 when he was restricted from lifting due to the arthritis in his back. This worker was alive and well in 2005.

Work history: 1/8/79, bagger; 4/12/81, palletizer operator; 6/21/81, outside operator; 3/1/82, palletizer operator; outside operator, 5/21/84; 6/5/86, laid off.

Worker 380

Parenchymal abnormalities consistent with pneumoconiosis (1963) t

ILO section ''small irregular opacities'' filled in: 1/0 (1963) t

''hi'' indicated in comments (1963) t/''cn'' indicated in comments (1963) t

*''The chest X-ray does show changes suggestive of chronic pulmonary fibrosis'' (1965)**

''od'' indicated in comments (1984) t

Sources Cited Lewinsohn Report t/King City Asbestos Mill Surveillance Report*

This worker began working at UCC in 1959. Therefore, the changes noted in 1963 and 1965 could not have been due to asbestos exposure from UCC due to insufficient latency. In fact, chronic fibrosis was first seen in 1954. Subsequent films taken in 1955, 1957, 1959, and 1961 were said to be essentially unchanged from 1954 showing

a generalized increase in pulmonary markings and hilar calcifications. Such changes were reported to be essentially unchanged from 1954 until the time he left work at UCC in 1986 in no less than 13 chest films, often being found in an otherwise ''normal'' chest. In addition, 19 chest film reports were read as normal between 1968 and 1986. The only ILO reading was done in 1963 (above). This recorded ''1/0 in all six zones'' as opposed to the change found in asbestosis that begin in the bases. Dr Peter Barrett also reviewed 20 chest films taken almost annually and found no evidence of asbestos related disease. Moreover, both Dr Lewinsohn and Dr Barrett also read the last chest film (25 Sept 86) as normal. Since asbestosis is a condition that does not regress, this indicates the earlier findings are not evidence of asbestosis. The radiological findings are very largely due to chronic granulomatous disease (as suggested by hilar, mediastinal lymph node, and/or parenchymal calcification described in seven chest film reports and specifically stated in two chest film reports). This condition was almost certainly contracted from his grandfather as a child since he was TB positive. Some of the radiological changes may also have been due to smoking and asthma. The radiological finding cited above in the 1984 chest film as ''od'' or ''other significant abnormality'' specifically referred to ''?nipple shadows right and left zones osteoarthritic lipping of thoracic spine.'' This has nothing to do with asbestos exposure. There was no spirometric evidence of asbestosis. Thus, 28 PFT's from 1972 to 1986 were read as normal. None displayed evidence of restriction. There was no clinical evidence of asbestosis. Thus, rales and dyspnea were not reported. He was always fit for work and was even encouraged to ''keep skiing'' and ''keep up the exercise program''. He died in 2004 at the age of 80 from pneumonia.

Work history: 4/48–11/57, UCC Nuclear Division, Cortez, Col. Bookkeeper; 11/57–7/59: Citizen's State Bank, Col – Teller; 7/1/63, UCC Coalinga asbestos plant accountant; 6/1/70, purchasing agent; 9/30/86, retired; 1/30/04, died.

Worker 383 {''The Autopsy Case''}

''od'' indicated in comments (1963) T

0/1 profusion noted (1981) T

parenchymal abnormalities consistent with pneumoconiosis (1981) t T

''Rule out (inflammatory changes) before considering asbestosis'' (1981) T;

''od'' indicated in comments (1981)T;

''Asbestosis'' listed on initial death certificate (1991);*

Sources Cited * King City Asbestos Mill Surveillance Program/T Lewinsohn Report.

Parenchymal abnormalities consistent with pneumoconiosis ILO box checked on X-ray report.

This worker was hired as a laborer in the mill in 1963 and then became a mill operator in 1965. The ''od'' comments could not have been due to Calidria exposure due to insufficient latency as the plant did not open until 1963. The ''od'' comments or ''other abnormalities'' did not refer to asbestos related changes but to ''congenital abnormalities of the 4th and the 5th rib on the right.'' The 1963 ILO reading associated with these ''od comments'' said the film was actually too ''hazy'' and unclear to score. However, a chest X-ray done on the same day was actually read as normal. There was no radiological evidence of asbestosis. Indeed, only the 1 April 81 ILO chest film displayed parenchymal abnormalities. These were, however, (atypically large) ''tu'' changes. These are not consistent with asbestosis where the abnormalities are small and round. Fifteen chest films taken between 1963 and 1987 were read as normal. Since the lesions of asbestosis do not regress, the parenchymal abnormalities found in the 1981 ILO film are not due to asbestos exposure. The parenchymal abnormalities did not reach a profusion score of 1/1. Dr Peter Barrett personally reviewed three chest X-rays (30 April 86; 19 November 87; 22 December 88) and concluded that none showed evidence of asbestosis or pleural plaques. Egilman also cited ''Rule out (inflammatory changes) before considering asbestosis'' (1981) – ''od indicated in comments (1981)'' as evidence of attributable disease. The entire commentary from which these two were taken reads: ''the basal changes may be due to the chronic inflammatory changes of congestive heart failure. Rule out before considering asbestosis.'' Indeed, most of the radiological changes are attributable to heart disease. This was diagnosed as ''ischemic cardiomyopathy secondary to arteriosclerosis'' (23 Dec 88) which produced three heart attacks (4/83, 6/84, and 11/90) and two cardiac bypass operations (1984, 1991). His cardiac illness thus caused him to take ''nonoccupational disability'' leave from 19 April to 6 August 1990. Whilst on vacation, he entered hospital on 12 Sept 90 and was again off work until 9 Nov 90 due to cardiac illness. He was diagnosed with another heart attack on 23 Nov 90 and hospitalized at Stanford University Medical Center. ''Per Dr Oyer, the decedent was being worked up for heart transplant however he was turned down due to his acute atherosclerotic cardiovascular disease and a option of multiple bypass was done. The decedent developed sepsis postoperatively and died some seven days later'' on 8 Jan 91. Some of the radiological findings could also be accounted for by smoking – related bronchitis,

+ [A3B2 Ver: 8.07r/W] [21.6.2008–12:12pm] [1–34] [Page No. 31] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

COPD and obesity. These were clearly significant contributors to his heart disease as were alcohol ingestion and a ''lack of regular exercise.'' Some of the radiological changes may also have been due to sinusitis and bronchitis due to an allergy to chemicals and dusts. There was no clinical evidence of asbestosis. Thus, there were no rales. Dyspnea was only recorded on one occasion (20 Nov 87) clearly due to severe heart disease, smoking and obesity. There was no spirometric evidence of asbestosis as all twelve of his PFT's performed between 1974 and 1987 were normal. There was no pathological evidence of asbestosis. This was independently confirmed by Dr Tom Colby at the Mayo Clinic who said ''the autopsy lung showed acute and organizing diffuse alveolar damage with some foci of acute inflammation consistent with sepsis. There is extensive fibroblastic reaction associated with diffuse alveolar damage. There is a moderate amount of dust filled macrophages consistent with the patient's smoking history. In my opinion, the fibrosis that is present here is all recent and occurred within the one week period following cardiac surgery and its complications following that. I do not see evidence of chronic scarring as one sees in asbestosis and I did not identify any asbestos bodies..... I think that the ''pulmonary fibrosis'' referred to in the coroner's report relates to the marked fibroplasia encountered in organizing diffuse alveolar damage, which is a nonspecific reaction pattern in this case probably triggered by sepsis and complications following cardiac surgery.''

''Asbestosis'' was listed on the original death certificate (1991) but was not confirmed at the time of autopsy. The final death certificate replaced asbestosis with ''diffuse fibrosis'' (not with ''nonspecific fibrosis'' as Dr Egilman states). Since asbestos bodies were not identified on routine or special stains, the revised Death Certificate (14 June 91) did not list asbestosis as a contributing cause of death. Egilman [11] however remarks: ''The coroner was apparently under the impression that the diagnosis of asbestosis required the finding of asbestos bodies.'' He argued this was incorrect on the grounds that ''UCC's secret animal studies'' never found asbestos bodies in animals injected with Calidria (see Discussion above). An autopsy was done on 9 Jan 91 and the report was given to, amongst others, Mr Myers the President of KCAC the successor to UCC. He in turn, gave a copy to one of the company physicians for review. The physician reviewed the report and ''concurred that asbestosis was not mentioned in the report nor was it a factor in the decedent's death.'' (31 May 91). The diagnoses listed on the autopsy report by Dr Angelo K Ozoa, MD, the Assistant Medical Examiner – Coroner were:

1. Arteriosclerotic cardiovascular disease: a. Calcific atherosclerosis of the aorta, coronary arteries, and other major aortic stenosis. b. Marked cardiomegaly (770 g) c. Focal ischemic myocardial fibrosis; d. Status post coronary artery bypass grafts, old and recent; e. Pericardial – pleural adhesions; 2. Acute bilateral bronchopneumonia secondary to #1. 3. Sepsis (by history). 4. Severe pulmonary fibrosis; 5. History of asbestos exposure, remote; 6. Embalming procedures.

Cause of death: Acute bronchopneumonia and sepsis. Due to: Coronary artery bypass grafts, Due to: Arteriosclerotic cardiovascular disease; Contributory: Pulmonary fibrosis.

The autopsy report indicated calcified plaques were not found on the pleura. There were bilateral fibrous adhesions present between the parietal and visceral pleura and these and the extensive fibrous scarring of the pericardium were due to surgery and inflammation. Thus, ''The myocardium showed many scattered foci of fibrous scarring.... There is evidence of previous and more recent surgical procedures;... of the three coronary artery graft orifices, two of are completely closed.....'' Both lungs were markedly heavy (right 1430 g; left 1230 g) due to acute bronchopneumonia, acute and chronic passive congestion. The revised Death Certificate issued on 14 June 91 by the County of Santa Clara listed the causes of death as:

21A – Acute Bronchopneumonia and sepsis; 21B – Coronary Artery Bypass Graft; 21C – Arteriosclerotic Cardiovascular Disease; 25 – Pulmonary fibrosis; 29 – Natural.

Egilman said ''the autopsy report listed pleural thickening, diffuse pulmonary fibrosis, and history of asbestos exposure (remote) as diagnoses.'' However, pleural thickening and diffuse pulmonary fibrosis are not listed amongst the diagnoses; only ''pleural adhesions'' and ''severe pulmonary fibrosis''

This worker died on 8 Jan 91. The events surrounding his death were described in an ''Investigation Report'' issued on 9 Jan 91 by the Medical Examiner and Coroner of Santa Clara County. Dr Oyer, an intern at Stanford University Medical Center, was said to have signed the death certificate and told the Medical Examiner that the decedent was being worked up as a heart transplant receiver. However, according to Dr Oyer, he was not a good candidate for this procedure due to acute ASCVD so they opted for a multiple bypass surgery. This was done at Stanford seven days prior to death. Dr Oyer apparently diagnosed ''asbestosis'' as a contributing cause of death (as listed on the death certificate) since the decedent's

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:12pm] [1-34] [Page No. 32] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

"chest x rays prior to the surgery had shadows that were consistent with fibrosis and the he did have some pulmonary problems" and because "he had questioned the decedent in detail prior to the bypass as to his past history and that included working at a business that crushed asbestos ore in to powder for sale to other companies that made roofing material." Dr Oyer "noted asbestosis on the death certificate even though no biopsy or other diagnostic work was ever made for asbestosis." The original death certificate was therefore signed by an Intern, not by his "treating thoracic surgeon" as Egilman claimed (also see [27] p. 77). Egilman insists Oyer was not an intern but "one of the top heart transplant doctors in the country" and also note Egilman's admission that even "heart transplant doctors" are "not experts in diagnosing asbestosis" [27] (p. 77). The body was sent to the Whitehurst Mortuary in King City and the mortuary attempted to file the death certificate in the County. The funeral services were set for the evening of 7 Jan 91. However, since the death certificate stated that other significant conditions included asbestosis, the County reported the case to the Coroner (as it was obliged to do under the law). Egilman said "The worker's family was not consulted" inferring some gross inconsideration or connivance on the part of the company had occurred. In truth, the family was not consulted about the autopsy because under the law one had to be done and there was no need to ask the family whether they wished one to be done. Dr Oyer said "he was not aware that it needed to be reported to the Coroner." The County Health Department then advised the mortuary that they were not to issue a death certificate since the body would have to be returned to their office for autopsy. Therefore, contrary to Egilman's assertion, the "UCC mine manager and president of KCAC...did not arrange to make the worker a coroner's case." The County did. The County also told the President of KCAC, John Myers, one of their workers had asbestosis listed on their death certificate. Mr Myers then spoke with the local doctors that had looked after the decedent. Egilman attempts to denigrate Mr Myers by saying "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." This is totally irrelevant to the case and also incorrect since he does not drives a hearse in his retirement. Egilman thus attempts to defame various parties by suggesting Myers, KCAC the Office of the Coroner, and the mortuary are part of a grand conspiracy to illegally alter the decedent's death certificate and hide the fact that the decedent has "asbestosis" (which he clearly did not). Egilman thus infers "criminal activity" without a shred of proof to back it. Indeed, Myers said: "We have never hidden anything from employees and will answer any questions.... I will be glad to talk to your employees if they have questions" (Communique from John Myers 8 Jan 91).

Work history: DOH (7/29/63) as a laborer; oiler (4/1/81); shipping foreman (4/1/81); lab technician (4/27/87); rock fiber operator (6/29/87); died 1/3/91.

Worker 385

Cancer listed as "ca" under "other abnormalities" (1984)

"A rounded opacity is present in the left hilium, It appears larger than on 4-30-84, but was probably

present on 1981 films, Although unilateral, rule out carcinoma" (1984)

Source - Lewinsohn report

The "rounded opacity" seen in 1984 could not have been a lung cancer since a follow-up film of the same date and a film taken three days earlier were normal and two subsequent films in 1987 and 1994 did not show any evidence of a mass. The chest film report of 30 Apr 84 film cited above described left lower lobe bronchopneumonia so the lesion seen four months later may have been a post obstructive infectious change related to smoking and allergy. Obesity may also have been a contributing factor. Even if the mass seen in 1984 was a lung cancer, it would not be attributable to asbestos exposure since there was no evidence of underlying asbestosis. Thus, no less than 10 films from 1976 to 1987 were read as normal. There was no clinical evidence of asbestosis. There was no shortness of breath or evidence of rales. He was said to be fit for any type of work and to be "relatively healthy." There was no evidence of asbestosis on spirometry as four PFT's performed between 1980 and 1987 were normal. Finally, since he started work in 1980, none of the findings described in 1984 could have been attributable due to insufficient latency.

Work history: 5/9/80, laborer; 6/3/80, bagger, palletizer, 8/3/80; 6/2/86, laborer; 2/4/91, press dryer operator; 2/28/94, bagger; 9/4/01, quit.

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)

King City Asbestos Mill Surveillance Program

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:12pm] [1-34] [Page No. 33] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword

This man only worked for one week at UCC (1 – 8 May 74) since he failed his physical due to the lung problems shown above. The chest X-ray (17 Apr 74) to which the above cited comment applies said: ''Calcific scar from old granulomatous disease and calcification of the left diaphragmatic pleura suggests asbestosis.'' Such pleural changes are not diagnostic of asbestosis since it is a parenchymal disease. There was also no clinical (''clinically normal'' 17 Apr 74) or spirometric (normal PFT 17 Apr 74) evidence of asbestosis at the time this film was taken.

Appendix 2

pi – pleural thickening in the interior fissure or mediastium
kl – Kerley lines
co – abnormality of cardiac size or shape
od – other significant abnormality
cn – calcification in small pneumoconiotic opacities
hi – Hilar changes
(atypically large) ''tu'' changes

+ [A3B2 Ver: 8.07r/W] [21.6.2008-12:12pm] [1-34] [Page No. 34] FIRST PROOFS {SAGE_FPP}Ibe/IBE 094924.3d (IBE) Paper: IBE 094924 Keyword