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UNITED STATES DEPARTMENT OF INTERIOR BOARD OF LAND APPEALS

SALINAS RAMBLERS MOTORCYCLE CLUB; AMERICAN MOTORCYCLIST ASSOCIATION DISTRICT 36; CALIFORNIA ASSOCIATION OF 4 WHEEL DRIVE CLUBS; CALIFORNIA OFF-ROAD VEHICLE ASSOCIATION; OFF-ROAD BUSINESS ASSOCIATION; and THE BLUERIBBON COALITION; Appellants, vs. BUREAU OF LAND MANAGEMENT; MIKE POOL, State Director, BLM California; Bob Beehler, Hollister Field Office Field Manager; Respondents.	IBLA NO. 2005-217 **DECLARATION OF DR. E.B. ILGREN**

1. My name is Dr. E.B. Ilgren, and I reside in Bryn Mawr, Pennsylvania. I have been licensed to practice medicine in the United States (New York, Pennsylvania) and the United Kingdom. I am Board Certified in Anatomic Pathology by the American Board of Pathology. I

was formerly a member of the Faculty of Biological Sciences and the Subfaculty of Biochemistry at the University of Oxford (1984 – 1992); the mineral fiber subcommittee of the International Congress of Occupational Health [ICOH]; and Congressman Nadler's (World Trade Center) Environmental Task Force [Ilgren, 2001] work for which I was cited by the City of New York. I was also invited by Senator John Kyle to comment on the Asbestos Medical Criteria in the 108th Congressional Record [Ilgren, 2003a]. I have published extensively in the areas of asbestos related disease and pathology. My professional study, expertise and consultation broadly include pathology, epidemiology and toxicology. My educational background, certifications and profession experience are detailed in the attached curriculum vitae (Exhibit "A.") and summarized in attachment B (Exhibit "B").

2. I have researched, written, and lectured about the effects of asbestos for more than 20 years with a particular focus on mesotheliomas [Attachment C as "Exhibit C"]. Moreover, over the last 15 years I have single-handedly published more on the nature of Coalinga chrysotile, the fiber found at the CCMA, than anyone else in the world. I have thus personally reviewed most of the clinical and pathological information related to the animal and human data concerning the biological effects of Coalinga chrysotile from any source. In this, I have personally reviewed virtually all of the available medical records of the miners and millers that worked with Coalinga chrysotile from 1962 to 2002 [Attachment D as "Exhibit D"]. In the course of undertaking this review, I have had extensive discussions with others concerned with non-occupational and occupational effects of Coalinga chrysotile and testified extensively

regarding same [Ilgren 2002b, 2003b]. Similarly, I have personally studied the pulmonary histology slides of nearly every animal ever exposed to Coalinga chrysotile by inhalation. Regarding such animal studies [Ilgren & Chatfield, 1997, 1998a,b; Ilgren, 2002a; Ilgren, 2004c,d], I personally 'discovered' and described in full the findings of the NTP – NIEHS animal inhalation investigation of Coalinga chrysotile, part of one of the largest animal inhalation studies ever conducted [Ilgren, 2004d] I also personally reviewed the data and histology of the only other animal inhalation study to use Coalinga chrysotile namely that carried out at the Fraunhofer institute in Germany [Muhle et al, 1987; Ilgren, 2002a]. These two, very large investigations were conducted more or less contemporaneously and totally independently, the one being in most likelihood, carried out unbeknownst to the other. I also personally reviewed another animal inhalation study of short fiber chrysotile done within the same time frame as the other two conducted at the NIOSH in the early 1980's by Platek et al [1985]. I also personally went to NIOSH to discuss their findings in 1992. I have also extensively studied and published papers on the geological formation [reviewed by Ilgren 2004a,b][1], mineralogical composition [reviewed by Ilgren 2004a], chemical purity [reviewed by Ilgren 2004a], fiber dimensions [Attachment E and F as "Exhibits E and F"; Chatfield, 2002, 2003 as Attachments (and "Exhibits") G and H], dust concentration [Ilgren 2002c, 2004c; also Ilgren 2002b, 2003b as Attachments I and J], and the solubility and biopersistence [Ilgren & Chatfield, 1998b] of Coalinga chrysotile.

[1] The EPA states that "CCMA's geology makes it an unsuitable location for OHV recreation". They fail to cite any supporting references. See

In 1994, I formally requested and received through a FOIA request to EPA IX all of the documents in their possession concerning the EPA JM Atlas Superfund Site and thus received information concerning the CCMA including the PTI [1992] document and others related to same. I had detailed discussions with the BLM personnel at Hollister and also presented a lecture in 1995 to Mr. Ed Hastey, former Director of the California BLM, and his staff on the biological effects of Coalinga chrysotile. In 1996, I also lectured to the Cal EPA OEHHA (Office of Health Hazard Assessment) {Drs. Mike Lipsett and Melanie Marty} and to the EPA IX Superfund Group in San Francisco at the request of the Superfund Manager, Dr. Richard Procunier, again on the biological effects of Coalinga chrysotile (other individuals with whom I have spoken concerning Coalinga Chrysotile can be found in the Acknowledgments of several of my publications on this topic [Ilgren, 2004a,c,d]) I also presented some of the findings of the biological effects of Coalinga chrysotile at two international conferences in 1997 in Kyoto and in 1998 in Philadelphia. Subsequently these findings were recognized as important contributions regarding the health effects of short fiber by various regulatory bodies and congresses in different parts of the world i.e. in 2002 by the European Directorate General Health and Consumer Protection, Scientific Committee on Toxicity, Ecotoxicity, and the Environment and the "International Workshop on The Health Effects of Chrysotile Asbestos" [cited in Ilgren,

2004d] and in 2003 by the EPA's ATSDR Expert Panel on the Health Effects of Asbestos and Synthetic Vitreous Fibers and the Influence of Fiber Length [cited in Ilgren 2004d].[2]

3. I have been retained by the BlueRibbon Coalition and other organizations to review the recent analysis by the BLM and Environmental Protection Agency regarding BLM's May 25, 2005, closure order and associated documents. I have specifically reviewed and analyzed the following documents: US Dept Interior BLM Environmental analysis – 11 May 05; US Dept Interior BLM Finding of No Significant Impact – 25 May 05; EPA Detailed Comments on the CCMA RMP Amendment DEIS – Nov. 2004; EPA IX Comments on BLM Draft Resource Plan – 1 Dec 04; EPA (Hanf) to BLM (Beehler) – after Mar. 05; Tech. Memo. Human Health Risk Analysis – Asbestos Air Sampling. CCMA 15 Sept 04; Coleman, 1996; Cooper et al 1979; and many other documents including but not limited to those cited in the first two sections of this "Declaration".

4. Based upon my analysis of all of these materials, my training, qualifications and experience in these matters generally and specifically (vide supra), I believe that the BLM lacks scientific support for the closure order based on the asserted risks to human health. Thus, Coalinga is neither an "occupational" [Ilgren 2004c; Crapo 2002 as Attachment (or "Exhibit") K] nor an "environmental" [Mc Donald & Mc Donald, 1980] health hazard. It is an innocuous, nuisance dust [Ilgren & Chatfield, 1997] prototypic of short fiber chrysotile from many other sources [Ilgren 2002a]. Short chrysotile fiber is ubiquitous [Ilgrren 2002a, 2004a,d; Schhrier,

[2] The EPA issued a letter [Marty, 1999] critical of some of the findings described by Ilgren & Chatfield [1997, 1998a,b]. I then responded to each

1989]: it is in the air we breathe, the food we eat, the water we drink. Short fiber chrysotile is therefore one of the most common types of fiber in the human lung typically found in concentrations ranging from 50,000 to 100,000 fibers per dried gram of lung. The New Idria serpentinite formation is a global source of airborne short fiber chrysotile [Klein, 1993]. Serpentine is California's State stone and Coalinga chrysotile is one of the most common forms of serpentine in the State of California. Short fiber chrysotile is therefore almost certainly in the lungs of virtually every resident in the State of California. Those that recreate on the CCMA are not at risk of asbestos related disease[3]. There is thus no rational justification for closure of the CCMA [4]. There is no evidence of attributable disease in animals [Ilgren & Chatfield, 1997;

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of these criticisms [Ilgren, EPA 2002c] and I am not aware of further response or rebuttal by the EPA to my remarks.

[3] The EPA's risk estimates have not been 'protective' overestimations but preposterous distortions. Thus, one out of every 100 agricultural workers in the Central Valley exposed to Coalinga chrysotile was allegedly at risk from lung cancer [discussed by Ilgren, 2004d]. Coleman [1996] said the calculations (for risk estimations) prepared by PTI for the BLM appear to be arbitrary".

The EPA risk estimates have been replete with 'uncertainty' [Tech. Memo. Human Health Risk Analysis 25 Sept 04]. Thus, "The quantitative unit risk estimate is limited by uncertainty in the exposure estimates which results from a lack of data on early exposure in occupational studies and the uncertainty of conversions between various analytical measurements for asbestos" ... "Uncertainty analysis: … these numbers do not predict actual health outcomes … (and are likely to be) overestimates" … "Specific uncertainties that should be considered when interpreting the results for this risk assessment include" … (the) representativeness of rider position, rider route, sampling season, "current practice of expressing TEM analysis results in terms of PCM equivalent values", and source of asbestos data. They also said that "The Unit Risk Factor should not be used if the air concentration exceeds 4E-02 fibers / ml" but have found higher concentrations than this at the CCMA yet again brining into question the validity of their risk estimations.

The ASTDR [1988] modeled the estimates of risk to Coalinga chrysotile. In its "most vigorous activity related experiments", it used a vehicle scenario in which a car was driven back and forth along a 100 foot section of dirt road in California containing about 1 to 4% asbestos. The maximal downwind sample concentrations were said to 0.9 structures per cc of air. On the basis of these estimations the ASTDR [1988] determined that the maximum air samples that were modeled for the Atlas Site piles would be up to three orders of magnitude higher than the road study. If this were the case, the Site readings could reach 900 structures per cc of air levels or those that could only be found on some other planet like Mars.

[4] The EPA and the BLM do not provide a single instance where Coalinga chrysotile has caused disease in humans nor have they cited a single publication to support their claims that it could make such disease. Moreover, the EPA continues to violate its mandate under CERCLA in its failure to rely on the Best Available Information. Environmental risk analyses must be site – specific. Indeed, the EPA has stated [Human Health Risk Analysis at the CCMA 25 Sept 04] that "The [IRIS] hierarchy recognizes the EPA should use the best science available on which to base

risk assessments. In general, if health assessment information is available to the IRIS for the contaminant under evaluation, risk assessors normally need not search further for additional sources of information"

The EPA made no attempt to provide any Coalinga specific information in the EPA [1990] Technical Background document. Not one single citation specifically referable to Coalinga chrysotile is given. The EPA is legally mandated under section 104 (I) (7) (A) of CERCLA (the Comprehensive Environmental Response Compensation, and Liability Act of 1980) to require its health assessment arm, the ASTDR, to provide them (the EPA) with the best and most appropriate data to formulate an assessment of the potential risks to human health. In the case of the New Idria formation, this pertains to exposure to Coalinga chrysotile. However, the EPA and its risk assessment arm do not appear to have made any attempt to review the "best and most appropriate data". The best risk assessment data are generally acknowledged to be epidemiological studies of workers exposed, under conditions of known intensity and duration, to the agent in question and next robust well controlled animal studies. The ASTDR [1988] document makes it clear that the EPA knew that information pertaining to such workers existed [cf: section 6-51]. My discussions with the director of the Region IX ASTDR unit, Dr. Bill Nelson, provided no insight into why the EPA had not even attempted to review these records. Thus, in 1990, there was more than sufficient information available to the EPA but they failed to cite a single article in their Superfund documentation.

Ilgren [2004d] states: "The EPA JM – Atlas Superfund Action -- The EPA JM – Atlas Superfund Action is a good example of wastage due to misguided regulatory policy toward asbestos. It stems in no small part from many of the issues discussed in the first part of this report. The EPA JM – Atlas Superfund Action is tantamount to an 'abate–the–earth' policy. The central driving force behind this Action has been the mistaken notion that Coalinga chrysotile is pathogenic and is equally toxic to all other fiber types of any size in any dose. The EPA Atlas JM Coalinga Superfund sites are located at the former JM and Atlas Coalinga mines at the southern end of the New Idria Serpentinite ore body located approximately one half-way between San Francisco and Los Angeles (fig. 6). The entire ore body occupies approximately 60 square miles and is estimated to extend almost 6 miles down virtually to the limit of the earth's crust (see [Crapo, 1994] for detailed discussion) with an estimated volume of 1 trillion cubic meters. It is therefore thought to be the single largest mineral deposit in the world being so large that it is felt to have been a continuing global source of airborne short fiber chrysotile since the Pliocene [Klein, 1993;, Crapo, 1994]. The New Idria Serpentinite is also contiguous with other serpentine formations along the Pacific rim making its true volume so vast as to be inestimable. By contrast, the amount of ore body disturbed through the former mining activities is so small as to be incalculable. This is highly relevant to the EPA Coalinga Superfund Action since it attempts to remediate the Coalinga situated land fill sites allegedly contaminated by asbestos through anthropomorphic activity. The remediation efforts are thus based on the premise that the earlier mining activities caused air and water borne fiber release significantly above that which would occur naturally from "undisturbed" areas through erosion and tectonic activity. Those residing near and / or recreating in the area are thus claimed to be at risk. However, the enormous size of the deposit (notwithstanding the innocuous nature of Coalinga fiber itself), makes these remediation activities tantamount to an "abate the earth policy" and are without any scientific merit whatsoever. Clearly, no amount of remediation (in a manner similar to the mining activities that took place earlier) could ever significantly alter the degree of exposures which occur at this site above and beyond those which occur naturally. To perform such remediation activities at the Coalinga site, the EPA was first required {cf: Section 104 (I) (7) (A) of the Comprehensive Environmental Response, Compensation, and Liability Act [CERCLA] of 1980} to request its health assessment arm, the ASTDR, to provide them with a "preliminary assessment of the potential risks to human health" posed by the site using the best and most appropriate data. The EPA [62] even acknowledged that their studies needed to be site specific. However, no site specific information was reviewed [EPA, 1990a,b]. Thus, the literature analysis done for the EPA [EPA 1990b] failed to cite a single reference about Coalinga chrysotile though such publications existed at the time (1990) e.g. Muhle et al. [1987]; Pott et al. [1987]; Langer et al. [1978]; Yeager et al. [1983]; Wylie [1979]; Pinkerton et al. [1983] amongst others. It also made absolutely no attempt whatsoever to determine the fate of the Coalinga miners and millers [Ilgren, 2002b] or do a case – control analysis of mesothelioma incidence in the relevant three county area. Review of Pinkerton et al. [1983] would have provided a good indication that Coalinga did not behave like other forms of asbestos in an aerosol and that this departure from normal behavior through clustering reduced its biological potential. The other publications [Mc Connell, 1984; Pott et al, 1987] indicated Coalinga chrysotile's markedly reduced tumorigenic potential which was clearly due to its very short length [Langer et al, 1978, Wylie, 1979]. Epidemiological information either from a cohort or a case control analysis would certainly have been very insightful.. The EPA Superfund document [1990b] said it was important to investigate the "the biological activity of clusters" and these may have accounted for ca 50% of the structures collected in the vicinity of the Atlas and Coalinga mines [EPA 1990b]. However, investigators appear to have arbitrarily excluded data related to such structures [EPA 1990b]. Pinkerton et al. [1983] demonstrated Coalinga clusters are thicker in air than fibril bundles of other forms of asbestos. Since the EPA [1990b] has recognized the biological importance of fiber width ("thin fibers were the most biologically active") consideration of the available literature could have led the EPA to conclude Coalinga chrysotile was far less toxic than other forms of asbestos."

Indeed, by 1987, the EPA should have been aware of the net cumulative data set demonstrating a failure of long term high dose inhalation exposure to Coalinga chrysotile to produce disease as evidenced by two totally independent, very large, nearly contemporaneous, governmental [EPA, NTP, NIEHS, Duke, Hannover University] - academic well controlled studies [Muhle et al, 1987; discussed by Ilgren 2004d] and another equally negative governmental [NIOSH] study using non Coalinga short fiber chrysotile [Platek et al, 1985]. Failure of the EPA – BLM to consider yet again a single study using Coalinga chrysotile is completely outrageous. Oversight of Cooper et al's [1979] report published more than 25 years ago is particularly amazing since it directly deals with recreational use of the CCMA. Careful study of this paper demonstrates glaring differences with the EPA – BLM data. Such differences relate to Coalinga chrysotile's lack of biological activity. Cooper et

al's data thus demonstrate the highly unusual nature of airborne Coalinga chrysotile 'fibers' to which the motorcyclists were exposed. TEM photos show non-respirable structures called 'chunks', highly 'clustered' collections of very short (97% <5u) chrysotile fibrils [Attachment E] found in concentrations as high as 61 'c'/ml! None of the EPA – BLM data mention such structures even though they were found at the CCMA and in other parts of the New Idria Serpentinite [Attachment E and figures contained therein]. Clearly, all of the BLM and EPA data sheets and representative portions of the sample grids warrant re-examination.

Cooper et al [1979] found high levels of "long" Coalinga fibers on personal samplers worn by motorcyclists recreating on the Coalinga serpentinite. In fact, these workers claimed these were some of the highest [max 5.9 fml > 5u] asbestos readings ever recorded from a naturally occurring environmental site. However, the actual counts included numerous "chunks" [see fig 1a in Cooper et al, 1979]. These were clearly clusters of the kind seen by ourselves and Pinkerton et al [1983, fig 4a] under experimental conditions and by earlier workers [Mc Daniel to UCC, 13 Sept 63; Mc Daniel to Riddle, 17 Nov 69 & 21 Aug 70] in occupational settings. Included "clusters" can significantly reduce the reliability of concentration measurements. This was seen in Cooper et al [1979] in the extreme variation in fiber [fml >5u] to total dust [mg/m3] ratios found between different motorcyclists, motorcycle runs, and sample positions; in the two to three fold variation in mass doses recorded for similar fiber levels; the tremendous range in mass concentrations [0-61]; and the marked discordance between PCM and EM - based fiber counts [see Tables 1 & 2 from Cooper et al (1979) reproduced below in modified form].

Table 1 Concentrations of airborne fibers longer than 5 µm detected by light microscopy and concentrations of total dust collected on personal samplers. [Cooper et al, 1979]

Motorcyclists		Run 1, 5.1 km (15 minutes)			Run 2, 1.2 km (5 minutes)		Run 3, 10.3 km (41 minutes)		(2)*
	Position on line	Fibers per milli-liter	Total dust (mg/m^3)		Fibers per milli-liter	Total dust (mg/m^3)		Fibers per milli-liter	Total dust (mg/m^3)
	1	0.9	0.97	1	0.6	0.0	1	0.3	l.0
	2	**5.6**	**31**	**2**	**3.0**	**12**	1.9	17	
	3	**2.3**	**19**	**3**	**3.0**	**37**	(3)	3.2	13
	4	4.3	21	4	4.9	20	(4)	2.9	11
	5	2.8	59	5	4.4	13	(5)	1.7	9
	6	**5.3**	**61**	6	3.1	22	(6)	2.9	22

*positions not fixed after rider 1.

Table 2. Comparison of light and electron microscopic counts of fibers collected on personal samplers worn by motorcyclist No. 4 and by the ranger. [Cooper et al, 1979]

	Light microscope Length > 5 µm (fibers per milliliter)	Electron microscope Length > 5 µm fibers per milliliter)	Length < 5 µm (fibers per milliliter)	Chunks (number per mill - liter)
Motorcyclist				
Run 1	**4.3**	**1.0**	**13**	**3.3**
Run 2	**4.9**	**23**	**188**	**61**
Run 3	2.9	6.5	42	24
Ranger	0.4	0.1	3.8	0.5

Berman also recognized that clusters were frequent at the Atlas and Coalinga Superfund sites stated that "data … indicated that 50% of the structures collected in the vicinity of the Atlas and Coalinga mines are matrices or other aggregates". Nonetheless, whilst admitting that the "biological activity of … clusters … has not been investigated directly" and that it would be important to do so (see section 4.1d, pg. 49 of the 1990 Technical Background Document).

1998a,b; Ilgren 2002a, 2004 a,c,d] or humans [Ilgren 2002b, 2003; 2004a; Crapo 2002; Barrett, 2002, 2003][5] exposed to high concentrations of Coalinga chrysotile either by inhalation or oral ingestion[6] and there has been more than sufficient latency for such disease to develop (>20 years)[7].

There are only three recognized forms of asbestos related disease: lung cancer, asbestosis, and mesothelioma[8]. Asbestos related lung cancer requires asbestosis [Weiss, 1999; Crapo 2002] and smoking to develop [Weiss, 1999]. Asbestosis in turn requires long term occupational

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The notion that the closure of the CCMA would "improve air quality" and improve human health risk [BLM Finding of no Significant Impact. – 25 May 05] is completely ridiculous and totally reminiscent of the EPA's Superfund Actions on that same area tantamount to an "Abate the earth policy" [Ilgen 2004d]. Coleman [1996] expresses the same view differently (also see Ilgren, 2004 a,b]: "the tectonic exposure of this serpentinite was followed by 15 million years of denudation and dispersal of asbestos which pre-dates asbestos mining and recreational activities" …"The anthropogenic disturbance of the New Idria serpentinite body by mining and recreation pales in comparison to the naturally occurring and continuing erosion of the asbestos bearing clastic debris throughout the Holocene".

[5] The only claim I am aware of regarding disease allegedly due to exposure to Coalinga chrysotile was made by a former California highway patrolman. He alleged that cruising for traffic violators on roads and freeways many miles from the CCMA between 1978 and 1983 tortiously exposed him to asbestos thus contributing to his alleged mesothelioma. [Waggoner Summary Judgment Motion In: Waggoner v Raybestos Manhattan et al]. Notwithstanding the fact that he had also worked with, amongst other things, transite pipe years before becoming a police officer, the claim also indicated that such highway related exposures could even occur when the windows of his car were rolled up!.

To the extent the EPA may wish to fall back on the 'policy' of single fiber – no threshold related asbestos induced disease, they should be reminded that such policy is clearly more political than scientific. Thus, Ilgren [2001] described how that EPA went from a pre 911 "zero tolerance" stand to a "post 911 only harmful if breathed in at high levels and over sustained periods of time" position. Moreover, review of the "LOEL, NOEL" tables in the EPA Toxicology Profile for Asbestos [2004; also see Table 3.1 in 2001] support their post 911 position.

[6] Exposure by Ingestion: Various animal and epidemiological studies done to assess the long term effects of drinking large quantities of asbestos, including Coalinga, have been negative. One of the largest experimental feeding and drinking studies exposed rats and hamsters to large quantities of Coalinga chrysotile and the rats and hamsters with no observable ill effects. This finding is consistent with the large numbers of long term animal asbestos feeding and drinking studies performed over the last twenty years that have also been negative. Thus, there is no reason to believe there is any need to 'adequately protect water quality' from activities taking place at the CCMA [EPA detailed comments. Nov. 2004].

[7] The EPA states that "Young children exposed to asbestos in the CCMA have a higher risk of developing mesothelioma during their lifetime … because the latency period … can be 30 to 40 years" [EPA Detailed Comments – Nov. 2004]. However, young children are not at greater risk since Coalinga chrysotile is not able to induce mesothelioma.

[8] The EPA said that "the observation of increased mortality and incidence of lung cancer, mesotheliomas, and GI cancer in occupationally exposed workers are consistent across investigators and study populations [US EPA 2004]" [Human Health Risk Analysis – Asbestos Air Sampling CCMA – 25 Sept 04]. This is not true. Some cohorts do not display a significant increase in lung cancer whilst other fail to demonstrate a marked increase in mesothelioma [HEI, 1991]. None of the studies show an across the board increase in GI cancers and these are not believed to be attributable to asbestos exposure.

exposures to long asbestos fiber to develop [Doll & Peto, 1985; Berman & Crump, 1999].[9] Such concentrations and exposure durations do not apply to those recreating on the CCMA. Therefore, neither asbestos - related lung cancer nor asbestosis merit further discussion [also see Ilgren 2004c]. Mesothelioma does not require asbestosis to develop. Nonetheless, mesotheliomas appear to require cumulative exposures of at least two weeks and where such exposures are 'brief' they are in turn intense [Ilgren & Browne, 1991; Crapo 2002][10]. Asbestos fibers capable

[9] The EPA further supports this view by stating that: "Furthermore, intense repeated exposures during periods as short as several months can lead to asbestosis [ATS 2004]" EPA Detailed Comments – Nov. 2004

[10] Non Occupational Studies: Non occupational or environmental exposures involve individuals residing or recreating on or near asbestos containing areas which, in the case of Coalinga, live on or adjacent to the New Idria serpentinite ore body. They do not include domestic exposures that can be quite high (e.g. 75 fml). There are only three environmental studies of the Coalinga residents, a California state mortality survey which failed to reveal a cancer excess; a mesothelioma survey by Mc Donald and Mc Donald [1980] that stated that there was no mesothelioma excess in individuals living near mining town in California that through discussions with the Mc Donalds turned out to be Coalinga; and a preliminary Case series analysis of mesothelioma in the Fresno and San Benito counties in 2003 by Dr. Paul Mills (unpublished) at the regional California Cancer Registry that also failed to find an excess of mesothelioma. Regarding, environmental mesothelioma, Ms. Felicia Marcus was the EPA Region IX Director under which the Coalinga chrysotile asbestos Superfund activities have been conducted. Sen. Samuel Farr wrote to Ms. Marcus [16 Aug 96] asking her to scientifically justify these Superfund activities. Ms. Marcus stated that the main scientific justification underlying the Superfund activities was based on the generalized observation that "indirect exposure to asbestos from nearby mining activities produced an increased number of cancer deaths" worldwide. Cancers arising under such circumstances are known as "environmental cancers". Ms. Marcus, however, failed to say in her response to Sen. Farr, that proven "environmental" cancers and, in this case, mesotheliomas are exceedingly uncommon; are largely limited to crocidolite exposures; are basically unheard of in residents environmentally exposed to chrysotile even when contaminated with tremolite; and are from high not low amphibole exposure. All of this is very relevant to the CCMA case since the EPA claims environmental exposures to chrysotile emanating from the CCMA can cause asbestos related disease. Given the importance of the topic of environmental mesothelioma to this case, particularly as it relates to fiber dose and type, more information will be provided below. Firstly, with regards fiber type, the following observation merits serious consideration: for almost 100 years, many families of the more than 11,000 Canadian (tremolite contaminated) chrysotile miners and millers lived at the "base" of tailing piles some over 100 feet high. Pictures of their homes are very telling as they are within ten feet of the tailings. Accounts of their neighborhoods are equally telling with chrysotile dust laying inches thick on car hoods, front lawns, and golf greens. Nonetheless, there has not been one proven attributable mesothelioma found amongst these Canadian mining residents. So under what types of conditions have proven environmental mesotheliomas been found? Some have been found in the environment of crocidolite mines and a few have been due to the natural environmental contamination of soils by amphibole. As for cases arising near crocidolite mines, these have been seen in the two areas of the world where most of the crocidolite is mined: South Africa and Western Australia. Since the South African workers are so migratory, we don't know exactly how many environmental mesotheliomas exist amongst them. However, 13 out of Wagner's original 33 cases of mesothelioma had no direct occupational exposure. If one disregards at least one of the 13 cases that was domestically exposed, the remaining 12 are probably "environmental". In all probability, all 12 grew up playing on the "asbestos mountains" that were a part of the semi-arid, almost desert - like conditions that characterize the area. Since the asbestos in South Africa comes to the surface of the earth it also covered the roads and tracks as well. Cumulative fiber levels downwind of the "tailing dumps" would be thousands of times greater than anything motorcyclists could be exposed to. What's more, these environmental crocidolite exposures continued for 24 hours a day, seven days a week, all year long. Regarding biopersistence, Coalinga chrysotile is totally cleared whilst, being highly durable, the crocidolite would have been almost completely retained. Since South Africa and Western Australia were "joined" many eons ago, it is not surprising that the physical conditions that prevail at each location are very similar. The mining area in Western Australia is also semi-arid and, being subject to strong winds, not infrequently very dusty. More importantly, the crocidolite tailings and dust were transported from the mine to the town and used "to keep the red (iron ore) dust down"; to protect the garden beds; to line the roads, race track, airport runway, tennis courts, and to provide children with "sand box" materials (as sand was lacking) to play in. This explains the fact that childrens' nappies were sometimes blue from eating the crocidolite fiber in their sand boxes. Not surprisingly the estimated fiber levels in the town were similar to those noted in South Africa (0.5 f/cc) though the real levels were probably higher. This is

of making mesothelioma must be long (>5u) [Crapo 2002; Berman and Crump, 1999; ATSDR 2002][11], thin (<0.5) [Berman & Crump, 1999] and hard i.e. composed of or contaminated by amphibole asbestos [Ilgren & Chatfield 1998a; Ilgren 2001, 2004 a; Crapo 2002][12]. Coalinga

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supported by the fact that fiber levels of 2 f/cc were recorded in 1978 in air samples collected from a car driven around the mining town more than 10 years after the mine had closed. In 1998, I personally visited this part of Western Australia to appreciate first hand what the conditions were like and what the fiber levels would have been. On the basis of my personal observations, discussions with residents, former miners, and local scientists, and my review of most of the available literature and data, I can state categorically that those recreating on the CCMA could never encounter environmental conditions resembling those found in Western Australia. It is also very informative that of the families of the 6500 crocidolite miners and millers that worked with crocidolite in Western Australia, there has only been one proven environmental mesothelioma as of 1998.

There are a few case reports of environmental mesotheliomas arising in individuals living on amphibole contaminated soils in Cyprus, Turkey, Greece, and New Caledonia. These mesotheliomas arose, however, in individuals that apply large amounts of long, thin tremolite fiber as whitewash, stucco, or plaster for their houses and / or from baby powder used on their children. The fiber types and exposure levels generated by these activities do not, in any way, resemble those encountered on the CCMA. Browne and Wagner [2001] refute the claim that 'neighborhood' exposures e.g. of the kind described by Newhouse et al [1964] can produce asbestos related disease which again supports the rarity of bone fide 'environmental' mesotheliomas.

Paraoccupational / Domestic Studies: There have been no confirmed or alleged cases of asbestos related disease due to domestic Coalinga chrysotile exposure. All discussions of 'tracking' in and out of the CCMA [EPA Detailed comments Nov 2004] re exposures per cars, campers, cows or other alleged vehicles of exposure do not apply to the CCMA given the lack of biological activity of Coalinga chrysotile.

[11] Fiber Length - Short versus Long Fibers and the Induction of Mesotheliomas: Inhalation and injection studies using specific short and long fiber preparations clearly demonstrate the role of fiber length in the induction of mesothelioma. Short (< 5u) fibers, or those less than the diameter of a macrophage are cleared and are thus unable to induce mesotheioma. Long (> 5u) fibers, however, are retained and therefore are able to induce mesothelioma. Macrophages are found throughout the lung but typically "reside" in the walls of the grape like sacs called alveoli where gaseous exchange takes place. When a fiber reaches the alveolus, the macrophage (which is a highly mobile cell) literally eats or ingests the fiber through a process called phagocytosis. In order for the macrophage to totally ingest the fiber, the fiber has to be less than the macrophage's width. Once ingested, the short fibers are carried by the macrophages into the lymphatic vessels where they in turn go into the blood stream and finally are excreted through the urine and the feces. Such short fibers are said to have been "cleared" by the macrophage. A common response following the ingestion of fiber (or any foreign material for that matter), is for the macrophage to release the acid enzymes contained within its cell body onto the fiber. Since chrysotile tends to be relatively soluble, the enzymes may dissolve much of the fiber and with time much of the chrysotile is said to undergo "dissolution". Finally, fibers that land on the bronchial mucosa may be swept up the bronchus by the ciliary action of the bronchial epithelium ("the so called broncho-ciliary escalator") and coughed up.

The fact that fibers less than 5u long cannot produce disease is supported by EPA sponsored research [Berman et al, 1995; Berman and Crump, 1999] and by EPA sponsored International Panels brought together to discuss this very topic [ATSDR 2002, also cited by Ilgren, 2004d]. It is also consistent with many comments made in the EPA [1990] Asbestos Technical document. Dr. Berman also repeatedly indicated the need to consider the features that were known to determine fiber related disease particularly fiber length. Berman continually states that the "longest" fibers are the "most biologically active" [see citations on pp. 41, 48, 49:4.1c; 50,60, & 72 of "background document"] whilst he states subsequently in an EPA supported scientific study [Berman et al, 1995] that fibers less than 5u long are biologically inactive. Although Dr. Berman does not relate any data cited by the EPA on the length and width of Coalinga fibers measured in air and water anywhere in the Background Technical Document, the ASTDR [1988] provides some indication that it is short. "The EPA (1988) considers AP42 emission factors for TSP matter to be a reasonable approach for risk assessments of activity related exposures providing that the asbestos fibers are relatively short. The fact that the TEM fiber counts are much greater than PLM counts at the Atlas and the JM Coalinga mill site is an indication of short lengths".

[12] Fiber Type - Failure of Amphibole - free, chrysotile to Induce mesothelioma - The "Amphibole Hypothesis of Mesothelioma Induction": The "amphibole hypothesis of mesothelioma induction" states that amphibole - free chrysotile cannot induce mesotheliomas. In our [Ilgren and Chatfield, 1998b] summary of the animal and human evidence that supports the "amphibole hypothesis", a notion strongly endorsed by other experts [e.g. Mossman and Gee, 1997; Mc Donald and Mc Donald, 1996], we point out that most of the mesotheliomas claimed to be due to

chrysotile is "short" (in vivo) [Crapo, 2002; Attachment E][13], "thick" (in air) [Crapo 2002; Attachment E] and "soft" (or amphibole free) [Ilgren 2004a][14]**.** In fact, virtually all long Coalinga chrysotile fibers are non-respirable [Crapo, 2002; see Berman & Crump, 1999 for discussion of respirability and fiber diameter]. Therefore, Coalinga chrysotile cannot induce mesothelioma [also see Crapo 2002]. Certainly, if it could, such tumors would pandemic. However, mesothelioma is one of the rarest tumors in the world.

In summary, no amount of Coalinga chrysotile will induce mesothelioma. It is not a hard enough to biopersist [Ilgren & Chatfield, 1998b; Crapo 2002][15] being amphibole free [Ilgren 2004a,b]; the majority of long (>5u) Coalinga chrysotile 'fibers' in an aerosol are too wide to be respirable [Berman & Crump, 1999; Crapo 2002; Attachment E] and thus fail to even enter the lung; and the few long Coalinga chrysotile thin enough to enter the lung either fall apart into

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chrysotile were actually caused by a small, unrecognized natural level of tremolite contamination and / or exposure to other kinds of amphibole {vs} that may have been used commercially. For the purpose of understanding this case, it is important to understand that Coalinga chrysotile does not contain tremolite. Indeed, the type of asbestos fibers which naturally occur in the CCMA (as well as throughout the New Idria region) is pure chrysotile and pure chrysotile is not a type of asbestos known to cause mesothelioma. In a broad analysis of the available data, we [Ilgren and Chatfield, 1998b] concluded that there probably has never been an attributable, clinically and pathologically proven case of mesothelioma in any chrysotile mining or cement, friction or textile product manufacturing industry amongst the many tens of thousands of workers where chrysotile alone has been used. Amongst the detailed listing of virtually all of the asbestos cohorts (such as that found in the Health Effects Institute [HEI, 1991] report), two specialty industries are worth pointing out. This is so since chrysotile and crocidolite were used "in parallel" . Comparison of groups thus exposed provide some of the most powerful evidence that chrysotile can not induce mesothelioma. The first of these groups are the WWII gas mask makers. These were entirely women and those who constructed masks for the military used crocidolite and suffered a very high mesothelioma incidence. By contrast, those who produced masks for the civilian population used chrysotile and these women failed to develop any attributable mesotheliomas. In the second group, namely the cigarette filter factory workers, those who used chrysotile in factory "A" displayed no disease whilst those that employed crocidolite in "B" demonstrated the highest mesothelioma ever recorded.

The EPA also appears to acknowledge the role of fiber type in asbestos related disease in other regions e.g. EPA Libby Superfund action The findings of the Health Effects Institute [1991] specifically produced to guide EPA policy, further contradict the EPA's views of fiber type.

[13] By contrast, the EPA stated: "The DEIS describes chrysotile asbestos found at the CCMA as short fiber asbestos, which is not supported by the data. The 1992 risk assessment relied on PCM measurements which only detect long fibers. EPA's current exposure evaluation is examining both long and short fibers and has found significant levels of long fibers [PCME]" [EPA Detailed Comments – Nov. 2004]

[14] Various US Regulatory Agencies also believe Coalinga is exclusively chrysotile [Ilgren 2004a].

their constituent very short thin fibrils that can be rapidly cleared by the macrophage from the body or transiently remain as long fibrils that can be readily dissolved through acidic macrophage digestion. Thus, Coalinga chrysotile's length, width, purity, and solubility characteristics account for its inability to produce disease.

Current regulatory methods do not adequately represent Coalinga chrysotile's true biological potential[16]. Indeed, limitations in the methods used to measure the size and number of Coalinga-type chrysotile fibers have caused the risks attributed to this form of asbestos to be greatly over-estimated [see "EPA Coalinga Superfund Action" in Ilgren, 2004a,d]. Standard regulatory counting methods rely upon phase contrast microscopy (PCM). These cannot differentiate between true long chrysotile fibers and long Coalinga-type pseudofibers [Chatfield, 2002, 2003] and are therefore not suitable for risk assessment. True long chrysotile fibers are composed of tightly bound fibrils, oriented in parallel, that run the length of the fibers[17]**.** By contrast, long Coalinga type pseudofibers consist of assemblies of numerous, overlapping, weakly bound short fibrils, in which each fibril extends over only a small part of the length of the pseudofiber. Large numbers of "long" Coalinga chrysotile pseudofibers seen under experimental

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[15] The EPA states that "Even brief exposure to asbestos levels found in the CCMA may cause fibers to remain in the lungs for an extended period of time. Therefore the exposure does not end when the visitor leaves the CCMA". [EPA Detailed Comments – Nov. 2004]. This is not true since Coalinga chrysotile does not display biopersistence.

[16] Regarding "Complex structures (asbestos aggregates)" Berman and Crump [1999] stated "The need to include components of complex structures in the exposure index for asbestos is further supported by inferences that such structures may degrade in vivo to their component fibers and bundles … their consideration may also have been ignored during most of the published inhalation studies due to the wide use o SEM to characterize asbestos exposure and the limited ability to distinguish the internal details of asbestos structures with such instrumentation". This emphasizes the need to use indirect examination to assess the biological potential of Coalinga type fibers. "PCM doesn't tell the whole story". A "pseudofiber" would be counted as an OSHA fiber [Chatfield, 2002].

[17] Each fibril is a single crystal produced by unidirectional crystalline growth [NRC, 1984].

[Muhle et al, 1987, e.g. 131 fml], occupational [e.g. UCC dust sample, 1972, 43 fml], and "environmental" [Cooper et al, 1979, 5 - 6 fml] conditions do not portend an increased risk. PCM counts that include large numbers of long pseudofibers are thus very misleading [Hodgson, 1986]. The fiber counts[18] taken by the EPA and the BLM at the CCMA do not represent the actual risks posed to those recreating in the area. In fact, they do not portend any risk at all. [19]

In accordance with 28 U.S.C. § 1746, I declare under penalty of perjury that the foregoing is true and correct.

DATED this 27th day of July, 2005, at Bryn Mawr, Pennsylvania.

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Dr. E.B. Ilgren, MD, D.Phil.

[18] The EPA is also relying on mass dose to assess risk at the CCMA [EPA detailed comments – Nov 2004]. There is no scientific basis for reliably measuring the risk of asbestos related disease using mass dose e.g. see discussion in Ilgren 2004c.

[19] Some of the EPA – BLM actions seem to be totally arbitrary e.g. where some of the CCMA is closed whilst others are not. Thus the EPA said that all routes except R001 through R019 would be closed during 'dry' conditions based on air monitoring if the OSHA PEL is reached in two consecutive weeks'. EPA Detailed Comments – Nov. 2004. The EPA also said that 'the DEIS should discuss how the BLM would ensure compliance with appropriate standards'. EPA Detailed Comments – Nov. 2004. However, the EPA is aware of the fact that there are no appropriate standards for making such determinations particularly since the currently used measuring methods don't adequately represent the biological properties of Coalinga chrysotile [also see Ilgren 2001 re standards].