DISPARITIES BETWEEN ASBESTOSIS AND SILICOSIS CLAIMS GENERATED BY LITIGATION SCREENINGS AND CLINICAL STUDIES

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INTRODUCTION

In 2005, U.S. District Court Judge Janis Jack, presiding over a multi-district litigation involving 10,000 claims of injury from exposure to silica dust that were generated by litigation screenings, issued a 263-page opinion rejecting the validity of thousands of medical reports generated by those screenings. Before issuing her opinion, Judge Jack

1 In a litigation screening, potential litigants are solicited directly or indirectly by lawyers by use of mass mailings, newspaper and circular advertisements, television and radio announcements, and “800” telephone numbers. Those responding to the advertisements come to a strip mall, motel room, union hall or lawyer’s office where medical tests, including medical exams in some cases, are administered by a doctor or medical technician for the purpose of generating results to be used to support claims of injury and qualify the potential litigant for compensation. Litigation screenings were first used to generate nonmalignant asbestos claims in the mid-to-late 1980’s; those screenings usually involved use of mobile X-ray vans which were brought to the site of the screening. For a more detailed description of asbestos screenings, see Lester Brickman, On The Theory Class’s Theories of Asbestos Litigation: The Disconnect Between Scholarship and Reality, 31 PEPP. L. REV. 33, 62 (2004) [hereinafter Brickman, Asbestos Litigation]. Litigation screenings, such as those that have been used to generate hundreds of thousands of nonmalignant asbestos-related claims, are fundamentally different than medical screenings. Litigation screenings have no intended health benefit and are undertaken for the sole purpose of generating claims for compensation. For a listing of the criteria of a medically sound screening program for asbestos-related diseases, see The Ass’n of Occupational & Envtl. Clinics, Guidance Document: Asbestos Screenings (Spring 2000), available at http://www.aoec.org/principles.htm.

2 In re Silica Prods. Liab. Litig. (MDL 1553), 398 F. Supp. 2d 563 (S.D. Tex. 2005). About 10,000 of approximately 20,000 claims based on injury from exposure to crystalline silica (e.g.,
ordered that a Daubert hearing be held to assess the reliability of

sand dust or quartz) that had been filed mostly in state courts in Mississippi and Texas were removed to federal court and then transferred by the Judicial Panel on Multi-district Litigation to the U.S. District Court in Corpus Christi, Texas for consolidated pretrial proceedings under the federal MDL (multidistrict litigation statutes), 28 U.S.C. § 1407(a) (2000). In order for a case to be transferred, the civil actions pending in different judicial districts must have one or more questions of fact in common. Id. Additionally, the transfer must be convenient for the parties and the witnesses and must promote justice and efficiency. Id. The MDL process is used to manage mass torts. See James M. Wood, The Judicial Coordination of Drug and Device Litigation, 54 FOOD & DRUG L.J. 325, 337 (1999); Desmond T. Barry, Jr., A Practical Guide to the Ins and Outs of Multidistrict Litigation, 64 DEF. COUNS. J. 58, 66 (1997) (“[T]he procedures are intended only as a guide to promote the fair and efficient resolution of complex litigation.”); id. at 59 (noting the purpose of MDL is to “eliminate duplication in discovery, avoid conflicting rulings and schedules, reduce litigation cost, and save time and effort on the part of the parties, the attorneys, the witness and the courts”). Transfers are for pretrial management only. Gregory Hansel, Extreme Litigation: An Interview With Judge Wm. Terrell Hodges, 19 ME. B.J. 16, 18 (2004).

3 Daubert v. Merrel Dow Pharm., 509 U.S. 579 (1993). In Daubert, the Court established a two-part test for determining the admissibility of scientific evidence under FED R. CIV. P. 702. The Court found that (1) the Rule’s requirement of “scientific knowledge” establishes a standard of evidentiary reliability, including “trustworthiness” and “scientific validity;” and (2) that the Rule requires that the scientific testimony “assist the trier of fact ... [to make] a valid scientific connection of pertinent inquiry as a precondition to admissibility.” Id. at 590 n.9, 591-92. The Court also laid out a flexible, non-exhaustive, four-factor test to determine the reliability of scientific expert testimony, examining (1) whether the scientific technique or theory can be or has been tested; (2) whether the theory or technique had been subject to peer review and published; (3) whether the technique or theory has an established rate of error or is governed by a set of established standards; and (4) whether the theory or technique has achieved a status of general acceptance in the relevant scientific community. Id. at 593-95; see also Robert J. Berlin, Epidemiology as More Than Statistics: A Revised Text for Products Liability, 42 TORT TRIAL & INS. PRAC. L.J. 81, 82-83 (2000); Margaret A. Berger, The Supreme Court’s Trilogy on the Admissibility of Expert Testimony, in FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 9, 12-13 (2d ed. 2000) (providing a summary of the four factors). The first factor, testability, asks whether the hypothesis can be and has been challenged by conducting appropriate scientific testing. Daubert, 509 U.S. at 593. The Court described this as a “key question.” Id. The second factor, peer review, examines whether the theory or technique has been examined by the relevant scientific community. Id. at 593-94.

[Submission to the scrutiny of the scientific community is a component of “good science,” in part because it increases the likelihood that substantive flaws in methodology will be detected. The fact of publication (or lack thereof) in a peer reviewed journal thus will be a relevant, though not dispositive, consideration in assessing the scientific validity of a particular technique or methodology on which an opinion is premised. Id. (citations omitted). The third factor, existence of standards and rate of error, aids in assessing whether a scientific technique is likely to yield accurate results. Id. at 594. The final factor, general acceptance in the scientific community, harkens back to Frye but becomes only one non-determinative factor in the reliability analysis. Id. The Court emphasized that the multi-factor reliability standard is a flexible one. Id. “The inquiry envisioned by Rule 702 is, we emphasize, a flexible one. Its overarching subject is the scientific validity—and thus the evidentiary relevance and reliability—of the principles that underlie a proposed submission.” Id. at 594-95. The Court directed judges to employ these factors in a “gatekeeping role” and to exclude evidence which lacks reliability and fit. Id. at 597. “We recognize that, in practice, a gatekeeping role for the judge, no matter how flexible, inevitably on occasion will prevent the
thousands of medical reports generated by a handful of doctors. In addition to this unprecedented use of a Daubert hearing in a mass tort proceeding, Judge Jack compelled the production of a large volume of

jury from learning of authentic insights and innovations.” Id. Rule 702 “assigns to the trial judge the task of ensuring that an expert’s testimony both rests on a reliable foundation and is relevant to the task at hand.” Id.

4 What is unprecedented is Judge Jack’s use of a Daubert hearing to determine the reliability of the litigation doctors’ diagnoses of silicosis and, therefore, the admissibility of their testimony. To comprehend the significance of Judge Jack’s decision, it is necessary to have some understanding of how proof of causation is introduced in a products liability or toxic tort trial. In dealing with such litigation, courts differentiate between general causation and specific causation: General causation is established by demonstrating, often through a review of scientific and medical literature, that exposure to a substance can cause a particular disease (e.g., that smoking can cause lung cancer). Specific, or individual, causation, however, is established by demonstrating that a given exposure is the cause of an individual’s disease (e.g., that a specific plaintiff’s lung cancer was caused by his smoking). Mary Sue Henifin, Howard M. Kipen, & Susan R. Poulter, Reference Guide on Medical Testimony, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, supra note 3, at 439, 444 (footnote omitted).

There have been frequent occasions when federal judges have excluded plaintiffs’ medical (and scientific) experts in Daubert hearings on the grounds of unreliability or irrelevance of their general causation testimony. See, e.g., Ruggiero v. Warner-Lambert Co., 424 F.3d 249 (2d Cir. 2005) (excluding expert medical testimony that the diabetes drug, Rezulin, was capable of causing or exacerbating cirrhosis of the liver); Gen. Elec. Co. v. Joiner, 522 U.S. 136 (1997) (affirming exclusion of physicians’ opinions that PCB exposure can cause small-cell lung cancer); see also David Klingsberg & Bert L. Slonin, Physicians’ Differential Diagnoses as Causation Proof: Recent Case Law Holds the Line in Requiring Daubert Reliability, 33 PRODUCTS SAFETY & LIABILITY REP. 1129 (2005) (discussing courts’ rejection of differential diagnoses as not satisfying the Daubert reliability requirement with regard to general causation). In 1996, in an MDL proceeding, U.S. District Court Judge Robert E. Jones appointed independent advisors for the court on scientific issues and on the basis of their reports, held that testimony of plaintiffs’ experts that certain alleged diseases were caused by silicone breast implants was not based on accepted scientific evidence and would therefore be excluded. Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387 (D. Or. 1996).

General causation is not an issue in most silica and asbestos litigation because it is indisputable that long-term exposure to crystalline silica dust can cause silicosis and that exposure to asbestos dust can cause asbestosis (as well as mesothelioma and lung cancer).

The operative issue in silica litigation is whether there is specific causation, that is, whether a plaintiff has silicosis and if so, whether any exposure to silica dusts emanating from use of a plaintiff’s products or activity was a substantial factor in causing the silicosis.

Generally, in personal injury cases, physicians often testify on one or more of the ultimate issues in the case such as specific causation. Henifin, Howard, & Poulter, supra, at 445. Depending on the applicable substantive rule on the burden of proof, the physician may testify that a plaintiff’s disease is “more likely than not” due to exposure to plaintiff’s product or that such causation exists “to a reasonable degree of medical certainty.” Id.

A licensed physician who is a B Reader qualified by the National Institute for Occupational Safety and Health (NIOSH) to read chest X-rays and grade them on the International Labour Organization (ILO) scale or who is a pulmonologist, is qualified to testify as an expert on both causation and whether the plaintiff has a silica or asbestos-related disease. In testifying that the plaintiff has silicosis, a plaintiff’s medical expert properly bases the diagnosis on (1) an X-ray reading or pathology; (2) a history of occupational or other exposure to crystalline silica dust; (3) a sufficient latency period from time of first exposure; and (4) a differential diagnosis in which
medical evidence, under threat of contempt, that the screening companies and doctors would not have otherwise produced. In her opinion, Judge Jack documented in great detail the existence of a fraudulent scheme to create bogus medical evidence that led her to conclude that “it is apparent that truth and justice had very little to do with these diagnoses. . . . [Indeed] it is clear that lawyers, doctors and screening companies were all willing participants” in a scheme to “manufacture. . . [diagnoses] for money.”

Judge Jack’s findings largely corroborated my own conclusions, published a year earlier, with regard to the validity of X-ray readings, pulmonary function assessments and diagnoses of asbestosis produced in the course of litigation screenings. In that article, I described how an illegitimate “entrepreneurial” model had been devised by lawyers, doctors and screening companies to screen hundreds of thousands of potential litigants for the sole purpose of generating claims of nonmalignant injury from asbestos exposure.

More recently, U.S. District Court Judge James T. Giles, who succeeded the late Judge Charles Weiner in presiding over the asbestos MDL, reached the following conclusion after extensive hearings:

the physician has ruled out other possible causes of the opacities shown on the x-ray, based upon a physical examination and pulmonary function tests. Thus, if there is a positive diagnosis, the physician testifies (a) that the plaintiff has silicosis and (b) that the occupational (or other) exposure alleged was sufficient to cause the disease. That testimony may be countered by a defendant’s medical expert, resulting in a typical “battle of the experts.”

What was unprecedented in the silica MDL was Judge Jack’s use of a Daubert proceeding first to engage in discovery of the screening companies that had generated the approximately 10,000 claims in the MDL and of the doctors who had provided most of the diagnoses, and then on the basis of the results of that discovery, to conclude that the doctors’ diagnoses were unreliable and therefore inadmissible. Though not unprecedented, Judge Jack’s determination to have the screening company principals and diagnosing doctors deposed in her presence and to take an active role in the questioning was another key factor in unraveling the fraudulent scheme to manufacture diagnoses for money. Indeed, had she not personally presided over the Daubert hearing (and used her knowledge as a nurse and her legal and medical research skills to obtain scientific information about silicosis and its incidence), it is doubtful that the fraudulent scheme would have been so clearly illuminated.

5 MDL 1533, 398 F. Supp. 2d at 635. The court remanded virtually all of the cases back to state courts on the grounds that they had been improperly removed to federal courts. Id. at 567. Nevertheless, the court addressed “all of the diagnoses by all of the challenged doctors,” despite not having the jurisdiction to issue a ruling on the admissibility of the testimony regarding the diagnoses, id. at 637 (emphasis in original), because Judge Jack felt constrained to issue what was, in effect, an advisory opinion to state courts. Since the Mississippi Supreme Court had adopted the federal Daubert standards, thus leading to the same standards under which Judge Jack reviewed the issues, she wanted to document the results “in hopes that the state courts that ultimately must shepherd these cases to their conclusion will not have to re-hear Daubert-type challenges to these doctors and their diagnoses.” Id.

6 See Brickman, Asbestos Litigation, supra note 1.

7 In re Asbestos Prods. Liab. Litiga. (No. VI) (MDL 875), MDL Docket No. 875, 2002 U.S.
discovery, and motion practice:

Current litigation efforts in this court and in the silica litigation have revealed that many mass screenings lack reliability and accountability and have been conducted in a manner which failed to adhere to certain necessary medical standards and regulations. . . . This court will therefore entertain motions and conduct such hearings as may be necessary to resolve questions of evidentiary sufficiency in non-malignant cases supported only by the results of mass screenings which allegedly fail to comport with acceptable screening standards.  

A. The “Entrepreneurial” Model of Litigation Screenings

The core of the “entrepreneurial” model of nonmalignant asbestos litigation that I described is an unprecedented-in-scale litigant recruitment effort: the litigation screening. 9 Entrepreneurial screening companies have been hired by lawyers to seek out persons with occupational exposure to dusts such as those containing crystalline silica or asbestos. Mobile X-ray vans are brought to local union halls, motels, or strip mall parking lots, where X-rays are taken on an assembly-line rate of one every five to ten minutes. In addition to the X-rays, most screening companies also administer pulmonary function tests (PFTs) to determine lung impairment for the sole purpose of generating evidence for litigation purposes.10

The sole object of these screenings is to generate medical reports to be used to support claims of asbestosis, a scarring of the lung tissue caused by exposure to asbestos.11 In the 1988-2006 period, well over...
90% of the approximately 585,000 nonmalignant claims for compensation filed with the Manville Trust were generated by these litigation screenings.

To read the hundreds of thousands of chest X-rays and pulmonary function tests generated by the litigation screenings and to produce the massive numbers of medical reports needed to advance the scheme, plaintiffs’ lawyers and the screening companies have hired a small number of doctors who share one common characteristic: their apparent willingness to enter into business transactions with lawyers and screening companies for the sale of tens of thousands of X-ray readings and diagnoses in exchange for the payment of millions of dollars. These X-ray readers, usually radiologists and pulmonologists, have been certified by the National Institute for Occupational Safety and Health as “asbestosis.” W. Raymond Parkes, Occupational Lung Disorder 285, 411 (3d ed. 1994). Fibroses caused by exposure to different dusts encountered in occupational settings, as well as by numerous other causes, may manifest differently on an X-ray. See infra notes 162-170. While the determination of the cause of a fibrosis may have a medical purpose, the principal reason for determining that the cause is asbestos exposure is a function of the compensation system. Whereas a diagnosis of another cause of fibrosis may yield no compensable claim, a diagnosis of asbestosis may enable the subject to be eligible for substantial compensation.

In its mildest form, asbestosis may cause no breathing impairment and is detectable only by chest X-ray or high resolution CAT scan. In more severe cases, significant fibrosis can decrease the elasticity of the lungs, and “interfere with the lung’s ability to oxygenate the blood.” AM. BAR ASSOC., COMM’N ON ASBESTOS LITIG., ABA REPORT TO THE HOUSE OF DELEGATES, RECOMMENDATION & RESOLUTION 7 (2003) [hereinafter ABA REPORT] (“Asbestotic lungs are characterized by reduced capacity, i.e., they can process only a reduced volume of air compared to normal lungs. Workers who suffer from significant asbestosis generally have shortness of breath on exertion.”). In its most severe form, asbestosis is progressive and debilitating and can lead to death.

The Manville Personal Injury Settlement Trust (Manville Trust) is the entity created as a consequence of the bankruptcy of the Johns-Manville Corp. in 1982 to which all claims against Johns-Manville relating to asbestos exposure were channeled. Johns-Manville mined most of the asbestos used in the United States and was by far the leading manufacturer of asbestos-containing materials. Prior to its bankruptcy filing, the company was the one most frequently sued for causing asbestos related injury. See Brickman, Asbestos Litigation, supra note 1, at 54. The company’s filing for bankruptcy led plaintiffs’ lawyers to develop the “entrepreneurial” model described in this Article. See Lester Brickman, The Asbestos Litigation Crisis: Is There A Need For An Administrative Alternative?, 13 CARDozo L. REV. 1819, 1825 (1992) [hereinafter Brickman, Administrative Alternative?].

See S. COMM. ON THE JUDICIARY, THE FAIRNESS IN ASBESTOS INJURY RESOLUTION ACT OF 2003, S. REP. NO. 108-118, at Attachment A (2003) . The Senate Judiciary Commission cites to a letter from Steven Kazan to the Honorable Jack B. Weinstein, which states that David Austern reported at a conference that “90% of the [Manville] Trust’s last 200,000 claims have come from attorney-sponsored x-ray screening programs, [and] that 91% of all claims allege only non-malignant asbestos ‘disease.’” Id. Since about 10% of the claims were for malignancies, then the reference to 90% of claims generated by screenings is the equivalent of virtually 100% of the nonmalignant claims. See also STEPHEN CARROLL ET AL., ASBESTOS LITIGATION 75 (RAND Institute for Civil Justice 2005); Lester Brickman, Ethical Issues In Asbestos Litigation, 33 HOFSTRA L. REV 833, 834 (2005) [hereinafter Brickman, Ethical Issues].
Health (NIOSH) as B Readers, which is an indication of special competence in reading chest X-rays and classifying them on the International Labour Organization (ILO) scale. A small number of B Readers, perhaps 4-6% of all certified B Readers, are most frequently selected by plaintiffs’ lawyers to read most of the hundreds of thousands of X-ray films generated by screenings. These B Readers grade most of

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14 NIOSH, part of the Centers for Disease Control and Prevention (CDC), awards B Reader approvals to individuals who meet a specified level of proficiency in classifying chest X-rays according to the ILO scale. See infra note 15. These B Readers are usually, but not always, licensed physicians and must be re-certified at 4-year intervals. ABA REPORT, supra note 11, at 14.

15 The degree of fibrosis appearing on a chest X-ray is graded according to a classification system developed by the International Labour Organization (ILO) INT’L LABOR ORG., GUIDELINES FOR THE USE OF ILO INTERNATIONAL CLASSIFICATION OF RADIOGRAPHS OF PNEUMOCONIOSIS (rev. ed. 1980) [hereinafter ILO GUIDELINES]; see also DIV. OF RESPIRATORY DISEASE STUDIES, NAT’L INST. FOR OCCUPATIONAL SAFETY & HEALTH & CENTERS FOR DISEASE CONTROL AND PREVENTION, THE CLASSIFICATION OF RADIOGRAPHS OF PNEUMOCONIOSES, in STUDY SYLLABUS FOR CLASSIFICATION OF RADIOGRAPHS OF PNEUMOCONIOSES (2002) (acting as a study guide for the application of the ILO radiographic classification system; prepared under contract by the Task Force on Pneumoconioses of the American College of Radiology). The system uses a scale that was developed to systematically record the radiographic abnormalities in the chest provoked by the inhalation of dusts. ILO GUIDELINES, supra, at 1, 2. According to the ILO:

The object of the Classification is to codify the radiographic abnormalities of pneumoconiosis in a simple reproducible manner. The Classification does not define pathological entities, nor take into account working capacity. The Classification does not imply legal definitions of pneumoconiosis for compensation purposes, nor set nor imply a level at which compensation is payable.

The Classification is based on a set of standard radiographs, a written text and a set of notes. In some parts of the scheme the standard radiographs take precedence over the text for the definitions; the text makes it clear when this is so.

Id.

On the ILO scale, chest X-rays are classified according to the number of abnormalities (termed “opacities”) in a given area of the chest film. A zero corresponds to no abnormalities, one to slight, two to moderate, and three to severe. “Since this process is to some degree inherently subjective, readers give two classifications, the category that they think most likely and next most likely. The result is a 12 point scale, with results ranging from 0/0 (normal [X-ray] appearance) to 3/3 (severe abnormalities).” In re Joint E. & S. Dists. Asbestos Litig., 237 F. Supp. 2d 297, 308. (E.D.N.Y. & S.D.N.Y. 2002). The vast majority of screening X-rays (for which asbestosis is claimed) are read as 1/0, which means the X-ray on first impression is at the lowest level of abnormality (1), but may be normal (0). See infra note 206. A reading of 1/1 is stronger than a 1/0 and means that the reader found clear evidence of irregularities. ABA REPORT, supra note 11, at 13. For purposes of identifying and locating opacities, the ILO form divides the lungs into six zones, upper, middle, and lower, left and right. For a diagnosis of asbestosis, the opacities should be found bilaterally in the lower zones. Nonetheless, a B Reader may assign a 1/0 grade even if he finds irregular opacities in only one of the six zones.

these X-rays as 1/0 on the ILO scale and describe their findings of radiographic evidence of fibrosis as “consistent with asbestosis.” Along with a small number of other doctors, they diagnose the vast majority of litigants thus found to have lung profusions of 1/0 or greater as having mild asbestosis17 (or silicosis—if that is the purpose of the screening, or both asbestos and silicosis18). These B Readers and other doctors, numbering approximately twenty-five, have accounted for a dramatically disproportionate percentage of the total number of X-ray readings and medical reports that have been submitted as evidence in support of nonmalignant asbestos personal injury claims.19 Indeed, the reliance on a small number of B Readers and diagnosing doctors is a defining characteristic of the “entrepreneurial” model.20

Based on the evidence I examined, I concluded that the majority of the hundreds of thousands of medical reports generated by the litigation screenings were not the product of good faith medical practice; rather they were produced in the course of business transactions involving the sale of X-ray readings and diagnoses for tens of millions of dollars in fees. I opined that the vast majority of those diagnosed with asbestosis

17 A diagnosis of asbestosis, when done in a clinical rather than a litigation setting, is based on a chest X-ray, physical exam, including a medical and occupational history, and a measurement of lung function. Am. Thoracic Soc’y, Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos, 170 AM. J. RESPIRATORY CRITICAL CARE MED. 691, 695-97 (2004) (publishing the official statement of the American Thoracic Society as adopted by its Board of Directors on Dec. 12, 2003); see also infra note 141.

18 See infra note 219.

19 A study of a stratified sample of claims submitted to Owens Corning before its bankruptcy filing indicated that just five B Readers (Drs. Raymond Harron, Jay Segarra, Richard Keubler, Philip H. Lucas and James W. Ballard) had read over eighty percent of the X-rays, with Dr. Harron alone accounting for forty-six percent of the X-ray readings. Report of Dr. Gary K. Friedman Owens Corning Impaired Nonmalignant Claim Submissions 1994-1999 (approx.) at 11, 18, 21 (c. 2000) (unpublished report, on file with the Cardozo Law Review). The Manville Trust reported that of 199,533 claims it processed in the period January 1, 2002 to June 30, 2004, just twenty B Readers accounted for sixty-two percent of the total B Readings. See David T. Austern, Claims Resolution Management Co., 2004 Asbestos Claim Filing Trends 8 (Sept. 2004) (Unpublished Power Point Presentation, on file with the Cardozo Law Review). The Trust further reported that as of December 31, 2005 of the many hundreds of B readers in its files, the top twenty-five who authored B reads in support of claims submitted to the Trust accounted for sixty-six percent (89,092) of the 135,235 B reads in its records. CRMC Response to Amended Notice of Deposition Upon Written Questions at Exh B, In re Asbestos Prods. Liab. Litig. (No. VI), Civ. Action No. MDL 875 (E.D. Pa. Mar. 2, 2006) [hereinafter CRMC Response]. Of the thousands of doctors who submitted diagnoses, the top twenty-five who were identified in the Trust’s records as the primary diagnosing doctor accounted for forty-six percent (255,928) of the total of 552,045 claims that permitted such identification. Id. at exh. C.

20 In the silica MDL, Judge Jack noted that “the over 9,000 Plaintiffs who submitted Fact Sheets were diagnosed with silicosis by only 12 doctors . . . affiliated with a handful of law firms and mobile x-ray screening companies.” In re Silica Prods. Liab. Litig. (MDL 1553), 398 F. Supp. 2d 563, 580 (S.D. Tex. 2005).
would not have been found to have an asbestos-related disease if they were examined in a clinical setting by doctors without a financial stake in the litigation. 21

Subsequent to Judge Jack’s opinion, I published another article in which I concluded that Judge Jack’s findings with regard to silica claims applied in full measure to nonmalignant asbestos litigation. 22

Judge Jack’s opinion has been widely covered in the news media 23 and is still reverberating around the mass tort world. Much less heralded is the fact that Judge Jack ordered that the X-rays and medical records, generated by the unprecedented discovery that she had permitted, be placed in a repository where it could be accessed by the MDL 1553 parties. 24 These records, which include the N&M screening company’s files on asbestos screenings, are now being systematically examined.

In addition to the litigation screening files that have been made available by Judge Jack, additional such files are being accumulated in the course of ongoing discovery in the asbestos MDL—a federal proceeding that may include as many as 100,000 plaintiffs that has been underway for over fifteen years. 25 While this proceeding has been largely inactive for the nonmalignant claims for at least the past ten years, Judge Jack’s decision in MDL 1553 has motivated defendants to seek similar discovery as that permitted by Judge Jack. These attempts are being stoutly resisted by plaintiffs’ counsel. 26 While presiding Judge James T. Giles has been cautious in permitting discovery, 27 he

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21 See Brickman, Asbestos Litigation, supra note 1, at 42-43.
27 In response to motions to compel production of documents and countermotions to quash
has come to recognize that the medical reports generated by asbestos litigation screenings “lack reliability and accountability.” Some of the documentary evidence that has been ordered to be produced has already proved valuable in affording additional insight into litigation screening practices. Like the records produced in MDL 1553, the records being produced in MDL 875 are also being systematically examined.

In this Article, I present some of the findings of these ongoing examinations as well as other data which has recently become available that addresses the issue of whether a substantial proportion of the hundreds of thousands of medical reports generated by asbestos litigation screenings have also been “manufactured for money.” In particular, I focus on data indicating the percentage of X-rays read as indicating radiographic evidence of pulmonary fibrosis which is “consistent with asbestosis” (the “positives” rate or the “percent positive”) by the comparative handful of doctors who account for a majority of the hundreds of thousands of medical reports produced by litigation screenings and, as well, the percentage of these “positives” who are then diagnosed with asbestosis “within a reasonable degree of medical certainty.” To understand the significance of this data properly, I summarize the results of a review of clinical studies of the prevalence of radiographic evidence of fibrosis and diagnoses of asbestosis among workers occupationally exposed to asbestos and compare that to the prevalence rates of the doctors involved in the asbestos litigation screenings (litigation doctors).

I also summarize the results of seven clinical studies or their equivalent in which X-rays generated by litigation screenings and read as indicating radiographic evidence of fibrosis, which were “consistent with asbestosis,” were re-read by independent medical experts who found very high error rates.

Another comparison presented is that between the ratio of findings of pleural plaques to pulmonary fibrosis found in clinical studies to the ratio in litigation screenings. This ratio abruptly changed from one that was moderately consistent with clinical studies to one that was widely inconsistent when a global settlement (later invalidated by the U.S. Supreme Court) significantly devalued pleural plaque claims.

Another facet of litigation screenings that I examine in this Article is the administration of pulmonary function tests to determine the degree of lung impairment and qualify the litigant for increased subpoenas, Judge Giles stated: “This Court . . . is not an investigating Grand Jury . . . . I do not presume that there is fraud in mass tort litigation.” Transcript of Motions Hearing, MDL 875, 771 F. Supp. 415 (E.D. Pa. Jan. 31, 2007) (MDL 875).

28 See Administrative Order No. 12, supra note 8, at ¶ 7.
compensation. Here too, I summarize the findings in medical literature which are significantly inconsistent with the outcomes of the pulmonary function tests administered in litigation screenings.

I also compare the pandemic proportions of nonmalignant asbestos-related disease claims which were filed in the 1990-2004 period in the tort system and with asbestos bankruptcy trusts with the paucity of hospitalizations primarily for asbestosis in that period.

I then review some of Judge Jack’s findings in the silica MDL and how they bear on the reliability of X-ray readings and diagnoses of asbestosis and silicosis generated by litigation screenings. Evidence introduced in the silica MDL indicates that 60-70% of the 10,000 silicosis claimants had previously filed claims for asbestosis. Medical literature, however, indicates that having both diseases is a clinical rarity. One of the lead plaintiff’s counsel in the silica MDL attempted to exonerate his firm’s actions in filing dual disease claims by arguing that the previous diagnoses of asbestosis were “wrong” and that his firm did not file asbestosis claims. A careful recitation of the record, however, reveals that the firm had formed an affiliate firm which did file asbestos claims with the fees shared with the parent firm. Indeed at screenings sponsored by the firm, a litigation doctor made a diagnosis of silicosis and forwarded that to the firm and, at the same time and for the same litigant, made a diagnosis of asbestosis and forwarded it to the affiliate firm.

Finally, I examine the possibility that the litigation doctors have predetermined “signature” percentages of positive X-ray readings and diagnoses. Bearing on this is the detailed record I present of the concerted refusal of the litigation doctors to provide records of all of their X-ray readings and diagnoses in response to subpoenas and court orders—records that may enable calculation of their percent positives that could be “smoking gun” evidence of fraud.

The conclusion I draw from the data and evidence presented is that Judge Jack’s findings with regard to the medical reports in the silica MDL apply with at least equal force to nonmalignant asbestos litigation: the medical reports are mostly “manufactured for money.”

I. THE PREVALENCE OF FINDINGS OF FIBROSIS “CONSISTENT WITH ASBESTOSIS” AND OF DIAGNOSES OF ASBESTOSIS IN LITIGATION SCREENINGS

Asbestos litigation screenings are an enormously profitable commercial enterprise. The purpose of these screenings is to identify
potential litigants and generate medical reports to support claims for compensation. No health benefits are intended.\textsuperscript{29} In the 1990-2000 period, each screened litigant cost attorneys approximately $500-$1000 and represented a net potential value to the attorney paying for the screening of $30,000-$50,000 in the form of fees and expenses.\textsuperscript{30} To assure that a high percentage of those screened become litigants, lawyers select B Readers who have demonstrated a great propensity to find that a high percentage of the X-rays they are asked to review for asbestos litigation purposes are graded as 1/0 or higher on the ILO scale.\textsuperscript{31} ILO guidelines require that the B Reader read all X-rays blind to “any information about the individuals other than the radiographs themselves.”\textsuperscript{32} This includes information about an individual’s occupational and exposure history. Leaving nothing to chance, however, plaintiffs’ lawyers routinely instruct B Readers that the purpose of reading the X-ray is to determine whether the individual has a claim for asbestosis or silicosis. As concluded by Judge Jack:

"In the setting of mass screening and/or mass B-reading for litigation, the B-reader is acutely aware of the precise disease he is supposed to be finding on the X-rays. In these cases, the doctors repeatedly testified that they were told to look for silicosis, and the doctors did as they were told."\textsuperscript{33}

B Readers’ responsiveness to these directions from the lawyers that hired them is indeed impressive. As noted by Judge Jack:

"After December 31, 2000 (when N&M changed its focus from asbestos to silica litigation), Dr. Harron [working for N&M] found [lung] opacities (consistent with silicosis) in 99.6% of the 6,350 B-reads he performed for MDL Plaintiffs. But prior to December 31, 2000 (when N&M focused on asbestos litigation), Dr. Harron performed B-reads on 1,807 of the same MDL Plaintiffs for asbestos litigation and he found . . . opacities (consistent with asbestosis but not silicosis) 99.11% of the time."\textsuperscript{34}

The “entrepreneurial” business plan for generating claims by use of screenings has been highly effective. My research leads me to conclude

\textsuperscript{29} See Brickman, \textit{Asbestos Litigation}, supra note 1, at 64-65.
\textsuperscript{31} For an explanation of how a new B Reader was tested to see whether he measured up to the standard for selection, see Brickman, \textit{Asbestos Litigation}, supra note 1, at 86 n.174.
\textsuperscript{33} \textit{Id.} at 627. It was “the lawyers [who] determined first what disease [the litigation doctors] would search for and then what criteria would be used for diagnosing that disease.” \textit{Id.} at 634-35.
\textsuperscript{34} \textit{Id.} at 607-08 (footnote omitted).
that the comparative handful of B Readers employed by screening companies and plaintiffs’ lawyers are mostly reading 50-90% of the X-rays generated by screenings as indicating radiographic changes graded as 1/0 or higher on the ILO scale which are “consistent with asbestosis.” \(^\text{35}\)

A number of these B Readers have testified that their

\(^{35}\) See Brickman, Asbestos Litigation, supra note 1, at 84-89 nn. 159-64 (concluding on the basis of the evidence that I had then examined that 60-80% of the X-rays were being graded as 1/0 or higher); see also Joseph N. Githin et al., Comparison of “B” Readers’ Interpretations of Chest Radiographs for Asbestos Related Changes, 11 ACAD. RADIOLOGY 843, 844 (2004). (“A small number of B Readers has [sic] made reputations with attorneys by consistently interpreting chest radiographs of asbestos claimants as positive [i.e., 1/0 on the ILO scale] in 90-100% of cases.”).

The silica MDL generated a treasure trove of data about the activities of N&M, a screening company which did both asbestosis and silicosis screenings and which was responsible for a majority of claims included in the silica MDL. But for Judge Jack’s rulings that N&M and other screening companies and doctors submit to examination in her presence and provide extensive records under threat of contempt, this inculpatory data would never have seen the light of day. N&M was incorporated in Mississippi in 1996 by Heath Mason and Molly Netherland. Transcript of Daubert Hearings at 266-67, MDL 1553, 398 F. Supp. 2d at 563 (testimony of Heath Mason). N&M has screened over 47,000 individuals. Certain Defendants’ First Amended Supplemental Brief in Response to Plaintiff’s Challenge to the Constitutionality of Florida’s Asbestos and Silica Compensation Fairness Act at 9, Perry v. Am. Optical Corp., No. 99-0869-AI (Fla., Palm Beach County Ct., 2006) (citing to N&M records produced in MDL 1553, Sales by Item Summary). These screenings were held in Alabama, Arkansas, California, Florida, Hawaii, Illinois, Kentucky, Louisiana, Missouri, Mississippi, Ohio, Pennsylvania, Texas, Wisconsin, West Virginia, and the Virgin Islands. \(^{1}\) at 8. N&M’s financial records indicate gross receipts totaling over $25 million between July 1996 and April 2005. \(^{1}\) at 9 (citing to an N&M record, Income by Customer Summary). N&M did work for numerous law firms including: Reyes & O’Shea; Provost Umphrey; the Ferraro Law Firm; O’Quinn, Laminack & Pirtle; the Foster Law Firm; and Campbell, Cherry, Harrison, Davis & Dove. \(^{1}\) at 18. A review of specific screening records provided by N&M in the silica MDL indicates that at those specific screenings, N&M’s doctors found that between 80% and 95% of the individuals had “positive” ILO profusions of 1/0 or greater. Transcript of Daubert Hearings at 302, MDL 1553, 398 F. Supp. 2d 563 (Feb. 17, 2005) (testimony of Heath Mason).

One of the principal N&M doctors accounting for this positive rate is Dr. Ray Harron, who is the most prolific, by far, of the litigation B Readers. See CMRC Response, supra note 19, at Exh. F. Dr. Harron’s positive rate for X-ray readings for the West Virginia law firm of Perice, Raimond & Coulter since 2000 was approximately 97.5%. See Amended Complaint ¶36, CSX Transportation, Inc. v. Gilkison et al., Civil Action No. 05:05-cv-202 (N.D. W.Va. July 5, 2007) (citing to E-mail dated January 27, 2006 from Robert Potter, former defense counsel for the Perice firm, to J. David Bollen.) Dr. Harron’s diagnosis rate is set forth in infra note 37. Dr. James Ballard appears to have read 99 of 100 X-rays sent to him by the law firm of Nix, Patterson & Roach as positive for asbestosis. See Dr. Steven E. Haber, Diagnostic Practices in a Litigation Context’s Screening Companies and The Doctors they Employed, June 11, 2007, at 25, In re W.R. Grace & Co., No. 01-1139 (JFK) (Bankr. D. Del. 2006). “For another set of X-rays for the same law firm, Dr. Ballard invoiced only for positive reads and had a 97% positive rate involving 1000 films.” Id.; see also infra note 124 and accompanying text.

Another source of information about the results of litigation screenings are the records being produced in the course of the recent proceedings and discovery in MDL 875. A review of documents and materials produced by Respiratory Testing Services, Inc. (RTS) in MDL 875 indicates that RTS screened at least 40,507 individuals over the course of 669 days in 35 different states. See Certain Defendants’ Combined Motion and Brief to Exclude Diagnostic Materials
In litigation screenings of 700-750 active and retired tire workers done in the late 1980’s, 439 (58.5%-62.7%) were found to have radiographic evidence of exposure to asbestos and on that basis, had filed claims for compensation. See R.B. Reger et al., Cases of Alleged Asbestos-Related Disease: A Radiologic Re-Evaluation, 32 J. OCCUPATIONAL MED. 1088 (1990) [hereinafter Reger et al., 1990].

Other empirical and documentary data that I assembled indicates that PTS, another screening company, generated a 70% positive rate on initial X-ray screenings. See Brickman, Asbestos Litigation, supra note 1, at n.164. Most of PTS’ B readings were done by Dr. Richard Kuebler and to a lesser extent, Dr. Philip Lucas, his partner. Dr. Larry Mitchell testified that for the period 1990-1995, he had a positive rate of around 60%. Id. Other screening companies also generated positive rates in the 70-80% range. Id. Dr. Barry Levy testified in the silica MDL that he reviewed 860 reports in a 72 hour period and concluded that all 860 plaintiffs had silicosis. Transcript of Daubert Hearings at 67, MDL 1553, 398 F. Supp. 2d 563 (Feb. 16, 2005) (testimony of Dr. Barry Levy).

Dr. Jay T. Segarra is one of the most prolific B readers and diagnosing doctors in asbestos litigation. According to the Manville Trust, Dr. Segarra provided 38,447 positive reports in support of claims submitted to the Trust as of December 31, 2005. See CRMC Response, supra note 19, at Ques. 14(a) and 14(c). Only two doctors authored more positive reports than did Dr. Segarra. Id. For these services, Dr. Segarra has been paid “about $10 million.” Wade Goodwyn, Silicosis Ruling Could Revamp Legal Landscape, on All Things Considered (Nat’l Public Radio Broadcast, March 6, 2006), 2006 WLNR 22951933, available at http://www.npr.org/templates/story/story.php?storyId=5244935. On one occasion, Dr. Segarra testified that he found 20-35% of the X-rays he reviewed positive for asbestosis. Deposition of Dr. Jay T. Segarra at 40, Moorehouse v. N. Am. Refractories Co., No. CI-2002-00253(2), (D. Miss. Oct. 14, 2002). In depositions taken in 2002, 2003, 2004 and 2006, Dr. Segarra variously testified that in screenings, his percentage of positive X-ray readings ranged from 10-20% and 10-40%. Deposition of Dr. Jay T. Segarra, at 232-37, In re: W.R. Grace & Co., No. 01-1139 (JFK) (Bankr. D. Del. Nov. 20, 2006). On the basis of my own analysis of available data, I previously concluded that Dr. Segarra’s positive rate for X-ray readers was at least 40%. Brickman, Asbestos Litigation, supra note 1, at n. 164. In response to a recent subpoena for his records, Dr. Segarra produced a redacted version of the response he had submitted to an August 2, 2005 request from the House Committee on Energy and Commerce for all records relating to his diagnoses of silicosis and to his work for screening companies. See Deposition of Dr. Jay T. Segarra at Exh. No. 28, Ragsdale v. Able Supply Co., No. 2005 -76615 (Tex. Dist. Ct. June 29, 2006) Dr. Segarra’s response indicates that he did not turn over the requested records to the House Committee. Instead, he provided only his own statistical analysis of what he alleges his records contain—not the actual underlying records, and only for the period January 2003 through June 2005, and only for X-ray impressions and diagnoses. Dr. Segarra began providing litigation support for asbestos claims in 1992 for Pulmonary Function Laboratory. Deposition of Dr. Jay T. Segarra, Abernathy v. ACandS, Inc, No. A-290, 967-C. (Tex. Dist. Ct. Aug. 1, 1995). Moreover,
in responding to the subpoena in Ragsdale, Dr. Segarra deleted information that he had provided to the House Committee indicating which lawyers he had done screenings for in the two and a half year period. By limiting his response to January 2003 through June 2005, Dr. Segarra omitted, inter alia, providing data as to whatever role he may have played in the phantom silicosis epidemic that commenced in 2002, see infra notes 216-224, and his oversight of RTS screenings as its de facto medical director from 1995-2000.

According to the deposition, id., Dr. Segarra states that he reviewed 13,329 X-rays for a variety of purposes in the two and a half year period of which 266 were unreadable. Of the 13,063 films which were readable, Dr. Segarra states that he read 46.6% as positive (6,092) and 53.36% negative (6,471). Asbestosis was the most common impression (33%). Id. Of the 4,276 relevant medical reviews he rendered in this period which could be tabulated, he issued positive diagnoses in 82.8% (3,540) and negative findings for disease in 17.2% (736). Id. During this period, Dr. Segarra provided X-ray impressions and/or diagnoses for approximately 93 different attorneys, including Baron & Budd, Brent Coon & Associates, Ness Motley, Hissey Kientz, and Heard, Robins and Cloud. Id. Dr. Segarra identified three screening companies for which he did these readings and reports: N&M, RTS, and Holland & Bieber. Id. According to the limited information provided, Dr. Segarra invoiced a total of at least $889,220 for X-ray readings and diagnoses in 2004 and $380,735 for the first six months of 2005. Id. Annualized, this amounts to $847,000 per year. Additional data recently compiled indicates that Dr. Segarra made positive X-ray findings in 42% of 11,378 X-rays read for RTS, see Certain Defendants’ Combined Motion and Brief to Exclude Expert Testimony by Dr. Jay T. Segarra and to Dismiss the Claims of Plaintiffs Relying on Same at 23, In re Asbestos Prods. Liab. Litig. (No. IV), MDL Docket No. MDL 875 (E.D. Pa. Sept. 7, 2007), and made positive findings in 50% of 18,463 X-rays read for Workers’ Disease Detection Service. Id.

Dr. Segarra’s asserted positive rates of around 47% for thousands of X-ray readings and 83% for thousands of diagnoses appear to be attained irrespective of the work histories of those screened, their degree of exposure to occupational dust-containing products that allegedly caused the opacities or disease or any other factors that would appear relevant to the incidence of disease. By failing to turn over the actual records which were requested by the House Committee and subpoenaed in Ragsdale, Dr. Segarra has precluded analyses of both the reliability of his calculation of 46.6% positive and of whether he has a “signature” percentage of positive readings. While these percentages are consistent with the limited empirical data I previously examined, see Asbestos Litigation, supra note 1, at 92 n.164, the high degree of self-interest that may have motivated Dr. Segarra to understake his “positives” rate should introduce a note of caution with regard to the reliability of Dr. Segarra’s responses. Additional reasons for caution in accepting the veracity of Dr. Segarra’s testimony include: (1) the fact that he effectively acknowledges that his previous testimony on multiple occasions of a positive rate of 10-40% and averaging 10-20% for X-ray readings is not accurate; (2) that his response omitted results for the ten or more years prior to 2003 when he was participating in numerous screenings, beginning in the early 1990s, when he worked initially for the Pulmonary Function Laboratories, a screening company, see Deposition of Jay Segarra, In re W.R. Grace & Co., No. 01-1139 (JFK) (Bankr. D. Del. Nov. 20, 2006), including the screenings that generated the phantom silicosis epidemic in 2002-2004; his role as de facto medical director of RTS in 1995-2000; (3) that he has steadfastly refused to supply the underlying records on which he based his calculations for January 2003 through June 2005, see infra note 253; (4) the fact that in order for Dr. Segarra to even justify his acknowledged 47% positive rate, he would have had to have made over 40,000 negative findings which would have meant that during his 13 year career as a litigation doctor, he would have had to have examined at least 17 screened individuals every single day of the year, including weekends and holidays—a process that Dr. Segarra claims requires between 60 and 90 minutes per individual, In re Silica Prods. Liab. Litig. (MDL 1553), 398 F. Supp. 2d 594, 623 (S.D. Tex. 2005) (footnote omitted); and (5) the fact that Dr. Segarra has retreaded hundreds of the X-rays he graded as 1/0 and “consistent with asbestosis” by re-reading these same X-rays a few years later as indicating silicosis. See Certain Defendants’ Combined Motion and Brief to Exclude Expert
percentages of positive X-ray readings are in the 10-30% range or below, but the available evidence casts considerable doubt on the credibility of these assertions. In addition, it would appear that these same B Readers and other doctors are diagnosing 80% or more of those whose X-rays have been read as indicating radiographic changes graded 1/0 or higher with asbestosis “within a reasonable degree of medical certainty.” Based upon the data I have assembled, I conclude that

36 The comparative handful of B Readers most frequently used by plaintiffs’ lawyers generally claim that their percentages of positive X-ray readings are at most 30%. Dr. Alvin J. Schonfeld is one of the most prolific B Readers and diagnosing doctors in asbestos and silica screenings. According to the Manville Trust, which has kept records only since 2002, Dr. Schonfeld has authored 41,573 reports submitted to the Trust, is considered the “primary diagnosing doctor” on 31,211 reports, and his diagnoses make him the second most prolific “primary diagnosing doctor” in the Trust’s history. CRMC Response, supra note 19, at ques. 14(a) and 14(c). Dr. Schonfeld has testified that he never sees a “huge percentage of abnormal films” and that seventy five to ninety percent of the films he reads are normal and do not show signs of asbestos-related illnesses.” Deposition Testimony of Alvin J. Schonfeld at 19-21, Blackburn v. Ill. Cent. R.R., Civ. Action No. 04-L-25 (Cir. Ct. Ill. July 10, 2006). Dr. Schonfeld’s credibility is subject to question, however, in light of his steadfast refusal to produce his records, which would enable calculation of his percentage of positive X-ray readings and his policy of destroying his records in screenings. See infra notes 254, 259.

37 Dr. Ray Harron is the most prolific of the B Readers and diagnosing doctors; he has accounted for over 80,000 medical reports filed with the Manville Trust in support of asbestos claims generated by litigation screenings. See CRMC Response, supra note 19, at exh. F. Dr. Harron has testified that if he finds radiographic evidence of bilateral interstitial fibrosis and is provided a statement that the screened litigant had exposure to asbestos in the workplace (usually provided by the plaintiffs’ lawyer or in the screening intake process), then he finds that the litigant has asbestosis within a reasonable degree of medical certainty. See, e.g., Deposition of Ray Anthony Harron, M.D. at 60-62, Jurecek v. Quigley Co., No. 03-CV-0594 (Tex. Dist. Ct. Sept. 28, 2004); Deposition of Ray Harron, M.D. at 240-41, Owens Corning v. Glenn E. Pitts, No. 96-29601-MI-0001-688 (Ind. Sup. Ct. Nov. 25, 2002). None of these B Readers have provided access to their records which would allow calculation of their percentages of positive X-ray readings.
there is a significant likelihood that each of these B Readers and diagnosing doctors, as well as the screening companies that hire them, have predetermined “signature” percentages of positive X-ray readings and diagnoses that fall within the 50-90% range. Indeed the “product” that these doctors appear to be selling to lawyers and screening companies are high fixed percentages of “positive” X-ray readings and diagnoses of silicosis and asbestosis.

A. “Shopping Around” of X-Rays and Diagnoses

A screening-generated X-ray read as negative for fibrosis represents tens of thousands of dollars of lost revenue. Between 1990

into an Agreed Order whereby Dr. Harron agreed to cease practicing medicine in the period before his medical license expires and not to seek or grant renewal of that license. See Investigated Physician Surrenders Medical License, HOUSTON CHRON., Apr. 22, 2007, at B3. For additional commentary on Dr. Harron, see infra notes 216, 226.

As indicated, RTS screened at least 40,507 individuals. See supra note 35. Analysis of the diagnoses provided to the first 32,119 persons in this group reveals that 17,877 (56%) were provided with a diagnosis of asbestosis. See Motion to Exclude RTS claims, supra note 35, at 8. Assuming that RTS’s rate of positive X-ray readings was 55.5%, as indicated by an analysis of RTS records, see supra note 35, then 17,826 of the 32,119 had their X-rays read as 1/0 or higher. Since 17,877 of the 32,119 were diagnosed with asbestosis, it is clear that RTS’s rate of positive X-ray readings is higher than 56%. Assuming it was 60%, then RTS’s rate of diagnoses of asbestosis of those with positive X-ray readings is 92.8%. Dr. Todd Coulter saw approximately 600 litigants and diagnosed approximately one half with silicosis, during an eleven day silicosis screening. MDL 1553, 398 F. Supp. 2d at 616. Assuming that he read 60-70% of the X-rays as positive for fibrosis, then his diagnosis rate would have been 80%.

Dr. Gregory Nayden, working for American Medical Testing, a screening company, diagnosed 100% of the 14,000 persons he examined as having asbestosis based on positive B reads by other physicians such as Drs. Lucas and Ballard. Deposition of Dr. Gregory A. Nayden at 164-65, Bentley v. Crane Co., Civ. No. 92-7655 (Miss. Cir. Ct. Mar. 28, 2002).

Empirical data on the percent of those with X-rays graded as 1/0 or higher who are then diagnosed as having asbestosis is nonetheless sparse and is limited to the evidence cited above, though additional evidence is being produced in proceedings underway in MDL 875. My estimate of a diagnosis rate of 80% or higher is based, in part, on reading scores of transcripts of depositions of litigation doctors and screening company principals as well as the discovery done in the silica MDL. For a selection of data from the transcripts, see Brickman, Asbestos Litigation, supra note 1, at n.164. Based on these depositions and materials, I would expect that Dr. Segarra’s claimed diagnosis percentage, 80%, is at the low end of the range. Moreover, my estimate is conservative in that it does not fully reflect the full impact of Dr. Harron’s and Dr. Nayden’s 100% diagnosis rate.

Another factor that supports my estimate is the economic context. A litigant who was screened as 1/0 or higher could have generated $60,000-$100,000 in settlement payments in the 1990-2000 period and a lesser sum thereafter, of which the lawyer would take about one half for fees and expenses. See Korosec, Enough To Make You Sick, supra note 30; see also Brickman, Ethical Issues, supra note 13, at 841-42. Having incurred the expense of screening the litigant, approximately $1,000-$1,500, there is a substantial economic incentive to monetize the claim by obtaining a diagnosis of asbestosis.
and 2000, an unimpaired nonmalignant asbestos claim was worth $60,000-$100,000, of which the lawyer would claim half for fees and expenses. To avert the substantial revenue loss that a negative X-ray reading would yield, lawyers or screening companies often send the X-ray to another one of the cadre of litigation B Readers to re-read, without disclosing that the X-ray had previously been read as negative. This “shopping around” can include as many as four to six re-readings by other litigation B Readers until a positive reading is obtained. Accordingly, a 50-90% positive rate on initial X-ray readings may, in fact, be a 70-90% positive rate or higher when taking into account the subsequent re-readings. To be conservative, however, I will continue to use the 50%-90% estimate.

This same “shopping around” process is followed for diagnoses. If one doctor concludes that the evidence is “insufficient to diagnose pneumoconiosis,” the plaintiffs’ lawyers often send the medical record for re-evaluation; once the “correct” diagnosis is obtained, the lawyer submits the diagnosis of “bilateral asbestosis” without any mention of the initial doctor’s report that he did not find disease. Accordingly, a pre-shopped diagnosis rate of 80% could well become a post-shopped 90% plus diagnosis rate. Again, however, in this Article, I will continue to use the 80% diagnosis rate estimate.

II. CLINICAL STUDIES OF THE PREVALENCE OF FIBROSIS

One method of evaluating the reliability of the X-ray readings and diagnoses of the litigation doctors is to compare those results with

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38 See Korosec, Enough To Make You Sick, supra note 30, at 3. For discussion of fees and expenses charged in asbestos litigation, see Brickman, Ethical Issues, supra note 13, at 840-43. After a wave of bankruptcies that began in 2000, the value declined.

39 See Status Report on Non-Party Discovery, Brief of Debtors and Debtors in Possession, In re W.R. Grace & Co., No. 01-01139 (JKF) (Bankr. D. Del. Dec. 1, 2006) for examples of specific X-rays and diagnoses that were shopped around by plaintiffs’ lawyers. See also, David Egilman, MD, MPH, Letter to the Editor, Asbestos Screenings, 42 AM. J. INDUS. MED. 163 (2002) (“I was amazed to discover, that in some of the screenings, the worker’s X-ray had been ‘shopped around’ to as many as six radiologists until a slightly positive reading was reported by the last one of them.”).

40 Egilman, supra note 39; see also MDL 1553, 398 F. Supp. 2d at 601 (“Sometimes, law firms . . . would ask N&M to have another doctor do re-reads of the x-rays which had been read as positive for silicosis. And if the subsequent B-reader . . . did not make a positive silicosis finding, then N&M would send the x-ray to a third B-reader for yet another read . . . . [I]t was even possible that if the third reader also did not make a positive silicosis finding, then the x-ray would be sent to a fourth reader.”) (footnotes and references to transcripts omitted).

41 Egilman, supra note 39.
clinical studies of workers who were occupationally exposed to asbestos. There have been over eighty studies of both exposed and unexposed populations to determine the prevalence of radiographic evidence of fibrosis.\textsuperscript{42} It is not possible to state with certainty that the subjects in the clinical studies were workers with similar exposures to those that were screened for litigation purposes. Nonetheless, the number of clinical studies and the number of workers included in the studies, as well as the range of occupational groups included, appear to cover a sufficiently broad sample as to constitute a valid basis for comparison. Moreover, the analyses that I have undertaken in this Article, and others referenced herein, as well as the findings of U.S. District Court Judge Janis Jack in MDL 1553, strongly suggest that many litigation screenings were of workers with modest occupational exposures to asbestos (and silica). Most clinical studies, however, would likely be of occupationaly exposed groups that were thought to be intensely exposed to asbestos. Thus, it is not unlikely that the exposure levels of those workers in the clinical studies exceeds the exposure levels of those recruited for asbestos screenings.

To introduce the results of a review\textsuperscript{43} of these studies, I first discuss the uniquely high disease level of insulators\textsuperscript{44} and the effect that

\textsuperscript{42} Of the eighty-five studies reviewed, seventy-two were of populations exposed to asbestos. Fourteen of these studies were excluded because they did not meet certain criteria described below. Thus, the review includes fifty-eight studies of populations exposed to asbestos. There were thirteen studies of unexposed populations, of which eleven are included and two excluded.

\textsuperscript{43} The review I have undertaken is of studies determining the prevalence of radiographic evidence of fibrosis in populations exposed to asbestos. The definition of “prevalence” is the proportion of a population which has the condition of interest. It is a useful measure of chronic and irreversible conditions such as asbestosis. The prevalence can be expressed in any unit, depending on how rare or common the condition is. Common conditions are often expressed as percentages, while extremely rare conditions may be expressed as occurrences per million population members. Prevalence is a static measure and should be not considered a rate. I have not undertaken to do a meta-analysis of these studies because any increase in the statistical validity of the results would not appreciably add to the utility of the review for purposes of comparison to the results of litigation screenings. In a meta-analysis, the prevalence of lung opacities (P) is a random variable with a prevalence of P(1-P)/n. The pooled prevalence calculated would be a weighted average where weights assigned are the inverse of the variances. For a description of the procedures to be used in a meta-analysis, see H. Frumkin & J. Berlin, \textit{Asbestos Exposure and Gastrointestinal Malignancy: Review and Meta Analysis}, 14 AM. J. INDUS. MED. 79 (1988); V. Velanovich, \textit{Meta-Analysis for Combining Bayesian Probabilities}, 35 MED. HYPOTHESES 192 (1991).

\textsuperscript{44} Insulators apply insulation materials to pipes and ductworks, or other mechanical systems to help control and maintain temperature. They are primarily employed in the building trades doing construction insulation work but are also employed as insulation workers in shipyards and powerhouse construction and repair. I.J. Selikoff et al., \textit{The Occurrence of Asbestosis Among Insulation Workers in the United States}, 132 ANNALES N.Y. ACAD. SCI. 139, 141 (1965) [hereinafter Selikoff et al., 1965]. Some of the insulation materials used contained no asbestos. One of the asbestos-containing products used was magnesia block insulation which usually
five insulators’ studies have on the results of such a review.

A. Insulator Studies

Insulators have sustained the highest levels of asbestosis of any occupational group exposed to asbestos during the course of employment. This unfortunate distinction is a function of the dose-related nature of asbestosis: the development and severity of asbestos-induced lung disease is a function of the intensity of exposure (dose) and latency—the time between first exposure and disease manifestation. The latency period for asbestosis is at least 10 years but is mostly in the 20-30 year range, though it can be as long as 40 years. Most occupational exposures to asbestos-containing products of the duration and intensity to cause disease took place in the shipyards during World War II and in construction and certain industrial trades thereafter, peaking in the late 1960s to early 1970s and substantially lessening by the end of the 1970s. Of the more than 40 occupational groups exposed to asbestos dusts, insulators were typically exposed to structures that contained approximately 15% asbestos. Asbestos cement, another important product, generally had 15-20% asbestos content.

45 See Brickman, Asbestos Litigation, supra note 1, at 49 n.42 for a detailed explanation; see also Jill Ohar at el., Changing Patterns in Asbestos-Induced Lung Disease, 125 CHEST 744, 745 (2004) [hereinafter Ohar et al., 2004]; infra note 64. In clinical studies, latency is also used to mean the time between first exposure and when the study is done or the X-rays taken.

46 See Jeffrey M. Shea & Catherine M. Martinez, Pulmonary-Critical Care Associates of E. Tex., Asbestosis, http://www.pcca.net/Asbestosis.html (“There is a well-defined latency period of approximately 20 years or more between the initial exposure to asbestos and the development of asbestos related calcification and scarring.”); Kun-Il Kim et al., Imaging of Occupational Disease, 21 RADIOGRAPHICS 1371, 1379 (2001) (“Most workers in whom pulmonary fibrosis (asbestosis) develops have been exposed to high dust concentrations for a prolonged period. There is a definite dose-effect relationship. Disease usually occurs approximately 20 years following initial exposure.”).


48 There were 41 occupations listed in the Manville Trust data base: (1) Air Conditioning & heating installer, maintenance; (2) Asbestos miner, plant worker; (3) Asbestos removal, abatement; (4) Auto mechanic/bodywork; (5) Boilerworker, cleaner, inspector, engineer, repair; (6) Brake manufacturing, installer, repair; (7) Brick mason, layer, hod carrier; (8) Building maintenance, building engineer; (9) Building occupant, officeworker, clerical, professional; (10) Carpenter/woodworker/cabinet maker; (11) Chipper, grinder; (12) Custodian, janitor; (13) Electrician, electrical worker; (14) Engineer (chemical, mechanical etc.); (15) Factory worker (assembly line) non asbestos; (16) Family member, bystander; (17) Firefighter; (18) Furnace
the highest levels of asbestos in the workplace and for the longest periods of time. While they constituted a very small percentage (0.67%) of the population at risk to an asbestos-associated disease, because of occupational exposure,49 insulators’ risk of contracting a respiratory malignancy was 4-25 times that of other occupationally exposed groups.50

A number of the clinical studies reviewed include insulators among the subjects of the study, generally without separately identifying the results attributable solely to the insulators.51 Eight studies, however, worker, repair installer; (19) Glass Worker; (20) Heavy equipment operator (includes truck/forklift/crane); (21) Insulator, asbestos; (22) Laborer (construction, demolition, shipyard); (23) Longshoreman, dock-worker; (24) Machinist; (25) Millwright; (26) Painter; (27) Pipefitter—asbestos; (28) Pipefitter, steamfitter; (29) Plasterer, sheet-rock, drywall, joiner; (30) Plumber; (31) Railroad engineer, brakeman, carman, conductor, fireman; (32) Rigger; (33) Sandblaster; (34) Seaman—other than engine room; (35) Seaman—engine room only; (36) Sheetmetal worker; (37) Shipfitter; (38) Shipwright; (39) Steelworker, foundry, aluminum; (40) Warehouse Worker; (41) Welder, blacksmith. Claims Resolution Management Corp., 1995 Industry/Occupation Chart, http://www.claimsres.com/DocumentsMT.html (last visited Nov. 30, 2006).

49 Of an estimated total of 27,527,000 workers occupationally exposed to asbestos in the 1940-1979 period, 184,000 (0.67%) did insulation work. William J. Nicholson et al., Occupational Exposure to Asbestos: Population at Risk and Projected Mortality—1980-2030, 3 AM. J. INDUS. MED. 259, 283 tbl. XII (1982) [hereinafter Nicholson et al., 1982].

50 Insulators’ relative risk of contracting cancer after 25 years employment is substantially higher than that of other occupational groups because of their longer average employments in the trade and their exposure to higher concentrations of asbestos fibers. From 1942-1979, insulators’ average employment time ranged from 12.5 to 15.9 years, whereas other occupational groups’ employment durations were typically one quarter to one half that of insulators. Id. at 284 tbl. XII. Insulators also were exposed to substantially higher concentrations of asbestos fibers than other occupational groups. Id. at 286 tbl. XV. Those in the construction trades (not including insulators) have 15-25% of the risk of insulators of contracting cancer, utility services—30%, chemical plant and refinery maintenance workers—15%, and automobile maintenance workers—4%. Id. at 287 tbl. XVII. Taking into account both exposure levels and average duration of employment, the following relative population risks of contracting cancer were calculated:

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Risk</th>
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<tbody>
<tr>
<td>Insulators</td>
<td>46</td>
</tr>
<tr>
<td>Manufacturing</td>
<td>4.6</td>
</tr>
<tr>
<td>Utility Services</td>
<td>4.9</td>
</tr>
<tr>
<td>Shipyard employee</td>
<td>3.3</td>
</tr>
<tr>
<td>Construction</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Id. at 288.

were solely of insulators. The first and most prominent of these studies was published in 1965 by Dr. Irving Selikoff. This study evaluated members of the Insulation Workers Union and reported that of 1,117 insulators studied, 542 (48.5%) had asbestosis based on radiological changes only. A later re-reading of the films using ILO scoring reported that 422 (37.7%) exhibited radiographic changes graded 1/0 or greater. The Selikoff study was instrumental in informing both industry and workers that asbestos-related diseases were not only caused by asbestos exposures during the manufacture of asbestos-containing products but also when those products were used in the workplace.

Four other insulators’ studies also characterized by greater latency periods and higher concentrations of fiber exposures than that of most of those who participated in clinical studies of occupationally exposed workers, also showed high levels of radiographic evidence of fibrosis.  

Selikoff et al., 1965, supra note 44.

Id. In fact, the Selikoff study did not diagnose insulators but only measured the prevalence of radiographic findings of fibrosis. For the protocol for performing a diagnosis, see infra note 141.

In his published study, Selikoff stated that 542 (48.5%) of the 1,117 had asbestosis based on the sole criteria of radiological changes. Selikoff et al., 1965, supra note 44, at 144-45. The study did not use the ILO system because it had not yet been uniformly adopted. Twenty years later, these same X-rays were re-examined and graded on the ILO system and the results were that 422 (37.7%) of the 1,117 X-rays were graded 1/0 or higher. No diagnoses were undertaken. R. Lilis et al., Asbestosis: Interstitial Pulmonary Fibrosis and Pleural Fibrosis in a Cohort of Asbestos Insulation Workers: Influence of Cigarette Smoking, 10 AM. J. INDUS. MED. 459 (1986).

See Miller et al., Relationship of Pulmonary Function to Radiographic Interstitial Fibrosis in 2,611 Long-term Asbestos Insulators, 145 AM. REV. RESPIRATORY DISEASE 263 (1992) [hereinafter Miller et al., 1992]. The Miller study examined 2,611 X-rays, of which 1,557 (59.6%) had small and irregular opacities graded 1/0 or higher, thus showing asbestos-induced parenchymal abnormalities. Id. at 283-84. The elapsed time from first exposure averaged 35.5 years.

R. Lilis et al., Radiographic Abnormalities in a Large Group of Insulators with Long Term Asbestos Exposure: Effects of Duration From Onset of Exposure and Smoking, 20 AM. J. INDUS. MED. 1 (1991). This study examined 2,790 insulators, finding that 1,683 (60.3%) had opacities graded 1/0 or higher. Id. Of the 2,790 insulators examined, 86.8% had a latency of more than 30 years. The latency periods for this population were broken down as follows: 368 (13.2%) were first exposed less than 29 years before examination; 1,712 (61.3%) were first exposed 30-39 years before examination, and 710 (25.4%) were first exposed over 40 years before examination.

R.L. Murphy, Jr. et al., Effects of Low Concentrations of Asbestos: Clinical, Environmental, Radiographic and Epidemiologic Observations in Shipyard Pipe Coverers and Controls, 285 NEW ENG. J. MED. 1271 (1971). The Murphy Study found that, of 101 pipecoverers examined, 44 (43.56%) had opacities graded 1/0 or higher. In addition to the 101 pipecoverers, 94 pipefitters who were a control group also exposed to asbestos were examined. The results of the part of the examination dealing with pipefitters is included in the review of the other exposed workers. All 195 workers were employed at a New England shipyard in November 1965. The pipecoverers were employed at the yard for an average of 17.4 years. Of the 101 pipecoverers examined, 45 (44.55%) had been exposed more than 30 years, dating back as far as the 1920s. M.J. Campbell & J.H.M. Langlands, Analysis of a Follow-up Study: An Example from
A review of these five insulators’ studies indicates that of 6,790 insulators studied, 3,790 (55.8%) were found to have radiographic evidence of fibrosis graded as 1/0 or higher on the ILO scale.56

In addition to the five insulator studies discussed above, there have been three other insulator-only studies (including pipecoverers57); these found much lower levels of prevalence of radiographic evidence of fibrosis than did the five studies listed above.58 Of a total of 617 X-rays examined in these three studies, 100 (16.2%) were found to have opacities of 1/0 or greater. A review of all eight insulators’ studies indicates that of a total of 7,407 X-rays, 3,890 (52.5%) exhibited opacities of 1/0 or higher.

The purpose of the review of clinical studies undertaken for this

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56 Langlands et al., supra note 55.

57 I am including “pipecovers,” a separate occupational group listed in the statistics compiled by the Manville Trust, with insulators because they appear to do the same work as insulators. The number of pipecovers that filed claims with the Manville Trust through September 30, 2006 is 614. See infra note 60.

58 J. Bourbeau et al., The Relationship Between Respiratory Impairment and Asbestos-related Pleural Abnormality in an Active Workforce, 142 AM. REV. RESPIRATORY DISEASE 837 (1990) [hereinafter Bourbeau et al., 1990] (examining 110 X-rays, of which 11 (10%) showed parenchymal fibrosis—these opacities were always of a small irregular type, such as would be consistent with asbestosis); S.M Kennedy et al., Lung Function and Chest Radiograph Abnormalities Among Construction Insulators, 20 AM. J. INDUS. MED. 673 (1991); [hereinafter Kennedy 1991] (examining 88 X-rays, of which 16 (18.2%) showed parenchymal abnormalities); K.H. Kilburn et al., Interaction of Asbestos, Age, and Cigarette Smoking in Producing Radiographic Evidence of Diffuse Pulmonary Fibrosis, 80 AM. J. MED. 377 (1986) [hereinafter Kilburn et al., 1986] (examining 419 X-rays, of which 73 (17.4%) showed diffuse pulmonary fibrosis).

I am excluding a study done in 1946 of shipyard workers who had been heavily exposed to asbestos, which found that virtually none of those examined had lung profusions indicating the existence of disease, because it was done prematurely. W.E. Fleischer et al., A Healthy Survey of Pipe Covering Operations in Constructing Naval Vessels, 28 J. INDUS. HYGEINE & TOXICOLOGY 9 (1946) [hereinafter Fleischer et al., 1946]. The latency period for asbestosis is at least ten years. Agency for Toxic Substances & Disease Registry, Dep’t of Health & Human Servs., Center for Disease Control, Asbestos-Health Effects, (2006), available at http://www.atsdr.cdc.gov/asbestos/asbestos_health_effects. Of the 1,074 workers examined, only approximately 4.7% had been in the industry for more than ten years. Because the study was done prematurely, using the Fleischer results would skew the percentage of fibrosis among insulators downward and detract from the validity of the review.
Article is to determine the prevalence of fibrosis based on radiographic evidence found among a wide range of workers occupationally exposed to asbestos, and to compare those results with the prevalence found by the litigation doctors. Because of the substantial impact of the insulators’ studies on the results of the review of clinical studies, it is important to determine whether that impact is disproportionate to the point of diminishing the validity of the comparison.

In the clinical studies reviewed, insulators account for almost 9% of all occupationally exposed workers who were subjects of the studies. This is more than three and a half times the percentage that insulators represent of all those with known occupations who filed claims for compensation with the Manville Trust between 1992 and 2006. In that time period, insulators constituted 2.5% of the claimants with known occupations who filed claims with the Manville Trust.

Substantially all of the nonmalignant claims filed with the Manville Trust were generated by litigation screenings. Moreover, very few screened litigants with diagnoses of asbestosis failed to file claims with the Manville Trust. Accordingly, claim filings with the Manville Trust are a surrogate for the population of screened litigants.

Because insulators are substantially overrepresented in the clinical studies when compared to their percentage of filings with the Manville Trust and thus the population screened, it is necessary to adjust the results of the review to maintain comparability. Since insulators account for almost 9% of all occupationally exposed workers who were the subjects of the clinical studies but only 2.5% of the screened

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59 The five insulators’ studies with the greatest latency periods and length of exposure to and concentration of fibers exposures, see supra notes 54-55, alone account for 31.98% of the total number of findings of radiographic evidence of fibrosis (ILO 1/0 or greater) identified in the fifty-seven studies of exposed populations (3,790 of 11,851), and thus these studies would have a substantial impact on the outcome of a review of clinical studies of exposed workers’ levels of fibrosis based on X-ray readings.

60 Though insulators constituted only 0.67% of the total occupationally exposed population in the United States, see supra note 49, they account for 4.4% of those with identified occupations who filed claims for compensation with the Manville Trust between 1988 and 2006. See Claims Resolution Management Corp., Alleged Occupation by Summary Injury Chart, through September 30, 2006 (Nov. 21, 2006) (on file with author) [hereinafter Occupation Chart]. The Manville Trust has received 687,352 claims for compensation through September 30, 2006. Of these, 458,556 have identified occupations, of which 20,215 are insulators and pipecoverers (4.4%); 19,601 (4.27%) are listed as insulators and 614 (0.13%) are listed as pipecoverers. Id.

61 See supra note 13.

62 It is commonly understood by those with experience in asbestos litigation that at least until the Manville Trust changed its Trust Distribution Procedures, effective in mid-2004, the vast majority—at least 90%—of those seeking compensation for asbestos-related injuries filed claims with the Manville Trust.

63 As noted, in addition to the eight insulator studies, there were a number of other studies which included insulators but did not separate out the results for just the insulators. See supra
population, then to maintain the desired comparability, the results of the seven insulator studies should be discounted by 72.22%.64

B. Clinical Studies of Exposed Workers

I conducted a literature search to identify all published studies65 that: (1) sampled populations occupationally exposed to asbestos;66 (2)
administered pulmonary X-rays; (3) had doctors, whether or not certified by NIOSH as B Readers, grade the X-rays using the ILO classification system; and (4) identified the number of the X-rays graded as 1/0 or higher. Where a study re-evaluated the X-rays read for a previous study, I included only the re-readings. In addition to the eight insulators’ studies, I identified fifty other studies of occupationally exposed workers, which examined over twenty different occupational groups. The criteria listed led me to exclude of fourteen of the eight insulators' studies, I identified fifty other studies of occupationally exposed workers, which examined over twenty different occupational groups. The criteria listed led me to exclude of fourteen of the eight insulators' studies, I identified fifty other studies of occupationally exposed workers, which examined over twenty different occupational groups. The criteria listed led me to exclude of fourteen of the studies reviewed list the number of X-rays graded as 1/1 or higher but do not list the number of 1/0s, or list the 1/0s only if there are other indicia of asbestos exposure. I am nonetheless including these studies because doing so provides a more complete representation of the clinical studies while not compromising the validity of the review. Including these eight studies decreases the prevalence percentage found by the clinical studies from 11.75% to 11.56%. The eight studies are O. Metadilogkul & P. Supanachart, Occupational Asbestosis and Asbestos Related Diseases Among Workers Exposed To Asbestos, 1987, Thailand, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, supra note 51, at 331; Myint & Myint, supra note 51; P. Oksa et al., Parenchymal and Pleural Fibrosis in Construction Workers, 21 AM. J. INDUS. MED. 561 (1992) [hereinafter Oksa et al., 1992]; C.E. Rossiter & P.G. Harries, U.K. Naval Dockyards Asbestosis Study: Survey of the Sample Population Aged 50-59 Years, 36 BRIT. J. INDUS. MED. 281 (1979); C.E. Rossiter et al., Royal Naval Dockyards Asbestosis Research Project: Nine-Year Follow-Up Study of Men Exposed to Asbestos in Devonport Dockyard, 73 J. ROYAL. SOC'Y. MED. 337 (1980); G. Sheers et al., UK Naval Dockyards Asbestosis Study: Radiological Methods in the Surveillance of Workers Exposed to Asbestos, 35 BRIT. J. INDUS. MED. 195 (1978) [hereinafter Sheers et al., 1978]; M. Silberschmid et al., Chest Radiographs in Railroad Employees with Asbestos Exposure—A 5 year Follow-Up Using ILO 1980 Classification, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, supra note 51, at 381 [hereinafter “Silberschmid et. al.”]. See infra note 72 for more information on these studies.

This has the effect of increasing the number of fibroses found, presumably because of the longer latency period. In the Kagamimori 1997 study, the original number X-rayed was 4,919, of which 67 (1.4%) were found to have opacities of 1/0 or higher. S. Kagamimori et al., Studies on Changes in Categories for Pneumoconiosis X-ray Classification in Japanese Workers with Occupational Exposure to Mineral Dusts, in PROCEEDINGS OF NINTH INTERNATIONAL CONFERENCE ON OCCUPATIONAL RESPIRATORY DISEASES, Kyoto, Japan, 166-69 (1997). Nine years later, 3,024 of the original population were re-examined and the new X-rays showed 81 (2.7%) as having opacities of 1/0 or higher. For purposes of this computation, only the second study is included.

This is in addition to the eight insulators’ studies.

These studies examined boilermakers, tire workers, merchant marine seamen, construction workers, sheet metal workers, ironworkers, pipefitters, electricians, plumbers, cleaners, elevator construction workers, cement plant workers, laborers, welders, drywall construction workers, millwrights, insulators, ship repairmen, painters, building custodians, naval dockyard workers, mineral dust workers, textile workers, and factory workers, among other occupational groups. Also, in one study of an exposed population, the study group consisted of wives of shipyard workers. Kilburn et al., 1986, supra note 58.
seventy-two studies that were identified in the literature search.  

The result of a review of the fifty-eight studies (including the adjusted results of the review of eight insulators’ studies) is that of a total of 78,219 exposed workers’ X-rays, 9,042 (11.56%) were found to

71 H.E. Amandus et al., Significance of Irregular Small Opacities in Radiographs of Coalminers in the USA, 33 BRIT. J. INDUS. MED. 13 (1976). This study was excluded because in order to be eligible for the study, a person must have already been read as 1/0 or higher.


A.C. Friedman et al., Asbestos-Related Pleural Disease and Asbestosis: A Comparison of CT and Chest Radiography, 150 AM. J. ROENTGENOLOGY 269 (1988) [hereinafter, Friedman 1988]. This study of 60 men is excluded because the subjects were selected based on previous chest X-rays interpreted as indicating asbestos-related pleural and parenchymal disease or a malignancy.

H. Robin et al., Radiologic Changes after Cessation of Exposure Among Chrysotile Asbestos Miners in Italy, 330 ANNALS N.Y. ACAD. SCI. 157 (1979). This study is excluded because it was of asbestos miners.


L.C. Oliver et al., Asbestos-Related Radiographic Abnormalities In Public School Custodians, 6 TOXICOLOGY & INDUS. HEALTH 629 (1990) (examining the X-rays only for pleural plaques).

have fibroses graded as 1/0 or higher on the ILO scale. If the

72 Eight of the studies that have been included listed findings of radiographic evidence of fibrosis graded as 1/1 or higher but did not include those X-rays graded as 1/0. See supra note 67. In addition to the eight insulator studies, the fifty other studies reviewed are:

M. Albin et al., Chest X-ray Films From Construction Workers: International Labour Office (ILO 1980) Classification Compared With Routine Readings, 49 BRIT. J. INDUS. MED. 862 (1992) [hereinafter M. Albin et al., 1992] (identifying the number of 1/0s but not considering a reading of under 1/1 as indicating fibrosis). The study found that 20% (41 of 210) of the subjects within ILO profusion category 1/1 had a pneumoconiosis, but did not indicate whether any of the 41 pneumoconioses were caused by asbestos exposure. Id. at 864.

S. Barnhart et al., supra note 51.

G. Berry et al., Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory, 36 BRIT. J. INDUS. MED. 98 (1979) (studying asbestos textile factory workers working for at least ten years). An earlier 1968 study was of male workers with ten or more years of exposure after January 1, 1933, who were still working on June 30, 1966; the present study includes 89 men who by 1972 completed ten or more years. The results were that of 379 evaluated, 88 (23.2%) were found to have opacities graded 1/0 or higher.


C-R Chen et al., Occupational Exposure and Respiratory Morbidity Among Asbestos Workers in Taiwan, 91 J. FORMOSAN MED. ASSOC. 1138 (1992).


E.A. Gaensler & A.M. Goff, Asbestos-Related Disease in Crocidolite and Chrysotile Filter Paper Plants, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE supra note 51, at 397 (examining workers who were engaged in manufacturing of specialty and filter papers containing asbestos).

E.A. Gaensler et al., Radiographic Progression of Asbestosis With or Without Continued Exposure, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE supra note 51, at 386 [hereinafter Gaensler et al., Radiographic Progression] (examining the workers at six locations—two shipyards, three paper manufacturing plants, and one plant specializing in manufacturing insulation board containing asbestos).


J. Gitlin et al., Comparison of ‘B’ Readers’ Interpretations of Chest Radiographs for Asbestos Related Changes, 11 ACAD. RADIOLOGY. 843 (2004).


N. Hisanaga et al. Pleural Plaques and Irregular Opacities on Chest Radiographs Among Construction Workers, in PROCEEDINGS OF NINTH INTERNATIONAL CONFERENCE ON
OCCUPATIONAL RESPIRATORY DISEASES, Kyoto, Japan, 286-289 (1997).


Kagamimori et al., supra note 68, at 166-169 (1997).

Kilburn et al., 1986, supra note 58 (including several study groups—some exposed and some not—separately, and is being treated as separate studies for this analysis).


Miller, supra note 64.

Murphy et al., supra note 53.

Myint & Myint, supra note 67, at 375 (presenting the results of the X-rays as positive only when the interpretation was 1/1 or higher).

J. Ohar et al., Changing Patterns in Asbestos-Induced Lung Disease, 125 CHEST 744 (2004) [hereinafter Ohar et. al., 2004]. This study did not distinguish between 0/1 and 1/0. Thus, this review includes only the X-rays that were read 1/1 or higher. Of the 437 X-rays examined, 16% (70) had 1/1 or higher and 40% (175) were either 0/1 or 1/0.

Oksa et al., 1992, supra note 67 (presenting the number of X-rays graded as 1/1 or higher but did not list the number graded as 1/0).

Reger et al., 1990, supra note 35. This study is included although it was a re-reading of 439 X-rays that had been found positive for asbestos-related disease which had been the basis for filing legal claims for asbestos-related injury, and although the re-readings included all profusions of 0/1 and above.


Rossiter & Harries, supra note 66. This study included what the authors called “high exposure trades,” (referring to sprayers and laggers) and the results are presented separately for the high exposure trades (30.8% were found to have 1/1 or higher graded opacities) and everyone else (2.9% were found to have 1/1 or higher graded opacities.) For the review, the results are combined.

Rossiter et al., supra note 66.

C. Rubin & L. Ringenbach, The Use of Court Experts in Asbestos Litigation, 137 F.R.D. 35 (1991). As noted, this was not a clinical study but is included because it is a functional equivalent. See supra note 90 and accompanying text; see also infra note 153.

R. Saito et al., A Study On Asbestos-Associated Lung Diseases Among Former U.S. Naval Shipyard Workers, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES
insulators’ studies are included without adjustment, then of the total 83,568 X-rays reviewed, 11,851 (14.18%) were found to have fibroses graded as 1/0 or higher.

CONFERENCE, supra note 51, at 362 [hereinafter R. Saito et al., 1990] Saito et al. studied 248 former U.S. Naval shipyard workers and found that 232 (93.5%) had parenchymal fibrosis. The study found “[n]ot only small irregular opacities characteristic of asbestos exposure but also small nodular opacities . . . [which are probably caused by] welding, sandblasting and other dusty work in ship repair and/or building work. Therefore the development of parenchymal fibrosis was interpreted as combined profusion.” Id. at 362. A part of this study had an unexposed control group and the results of this group are separately included in the review of studies of unexposed populations.


Sheers et al., 1978, supra note 66. The focus of this study was to compare the methods of finding asbestos related abnormalities. It did not provide detail about the study group, exposure levels, or prevalence. It used various subgroups within itself to test positives read by one method against another. Finally, it identified small opacities as “positive” when graded as 1/1 or higher.

Silberschmid et al., supra note 66. This study presented the results of the X-ray readings by placing them into three categories: 0/0-1/0, 1/1-2/1, and 2/2-3/+ . Thus, included in the review are those X-rays that were read 1/1 or higher.

N.L. Sprince et al., Asbestos Related Disease in Plumbers and Pipefitters Employed in Building Construction, 27 J. OCCUPATIONAL MED. 771 (1985);


III. Disparities Between the Findings of Clinical Studies and Litigation Screenings

A. Understatement of the Degree of Disparity

The litigation B Readers’ 50-90% positive X-ray reading range for radiographic evidence of fibrosis graded as 1/0 or higher on the ILO scale is many multiples of the 11.56% percentage generated by a review of the clinical studies. This alone provides compelling evidence of systematically erroneous, if not fraudulent, medical reports by the comparative handful of B Readers and doctors employed by screening companies and plaintiffs’ lawyers. Moreover, this simple comparison understates the degree of disparity between the two sets of results.

1. The Effect of Differing Shapes and Locations of Opacities on X-Ray Readings

The clinical studies included in the review, grade the X-rays on the ILO scale and identify those X-rays with radiographic evidence of fibrosis, i.e., opacities or visible scarring of the lung of grade 1/0 or higher. The shape, size, and location of opacities, however, are important factors in the determination of the cause of the radiographic evidence of fibrosis.  

<table>
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<tr>
<th></th>
<th>N</th>
<th>≥ 1/0</th>
<th>%</th>
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<tbody>
<tr>
<td>58 studies (including eight adjusted insulator studies)</td>
<td>78,219</td>
<td>9,042</td>
<td>11.56%</td>
</tr>
<tr>
<td>58 studies (including eight insulator studies without adjustment)</td>
<td>83,568</td>
<td>11,851</td>
<td>14.18%</td>
</tr>
</tbody>
</table>

73 “The ILO system standardizes the interpretation of chest x-rays using descriptions of the size, shape, and profusion (i.e., degree or severity) of radiographic abnormalities (i.e., visible lung markings or scarring).” In re Silica Prods. Liab. Litig. (MDL 1553), 398 F. Supp. 2d 563, 591 (S.D. Tex. 2005). The ILO classification system describes both small and large opacities as...
higher were small and rounded, and located in the mid and upper zones of the lung, and the individual’s work history indicated substantial occupational exposures to sand dusts such as that experienced by sandblasters, and a medical examination of the individual yielded consistent results, the individual’s diagnosis would likely be silicosis. Opacities caused by asbestos exposure are primarily irregular and linear and appear mostly on both sides at the base and periphery of the lungs. Because some of the 58 clinical studies only grade the opacities and do not identify the shapes and locations of the opacities, and few of the studies actually state that the opacities were determined on the basis of parenchymal abnormalities. Int’l Labour Org., Guidelines for the Use of ILO International Classification of Radiographs of Pneumoconiosis (rev. ed. 2000). Small opacities are those up to 10mm in width. Id. Large opacities are those exceeding 10mm in width. Large opacities are also then categorized into three subcategories (A, B and C) by their specific size, profusion, and location in the lungs. Neither asbestosis nor silicosis, however, present as large opacities. Small opacities are classified by profusion, location in the lungs, shape, and size. Id. Profusion refers to the “concentration of small opacities in affected zones of the lung.” Id. “Affected zones” refers to the “zones in which the opacities are seen . . . . Each lung is divided into three zones (upper, middle, lower) by horizontal lines drawn at approximately one-third and two-thirds of the vertical distance between the lung apices and the domes of the diaphragm.” Id.

Small opacities can have an irregular or a rounded shape and each shape is subcategorized by the size of the opacities. Id. Small rounded opacities are classified in three categories by size and recorded with the letters p, q, and r; they are “defined by the appearances of the small opacities on the corresponding standard radiographs”: p-opacities are small rounded opacities with diameters up to about 1.5mm; q-opacities are small rounded opacities with diameters between 1.5mm up to about 3mm; r-opacities are small rounded opacities with diameters between 3mm up to about 10mm. Id. Small irregular opacities are recorded with the letters s, t, and u, and are also “defined by the appearances of the small opacities on the corresponding standard radiographs”: s-opacities are small irregular opacities with widths up to about 1.5mm; t-opacities are small irregular opacities with widths between about 1.5mm up to 3mm; u-opacities are small irregular opacities with widths between 3mm up to about 10mm. Id.

“[O]n a chest x-ray, silicosis presents with small, rounded opacities, in the upper ormid zones of the lungs. . . . By contrast, on a chest x-ray, asbestosis presents with irregular linear opacities, primarily at the bases and periphery of the lungs.” MDL 1553, 398 F. Supp. 2d at 594. “Asbestosis specifically refers to interstitial fibrosis caused by the deposition of asbestos fibers in the lung . . . . In its classic form, there is diffuse, bilateral, pale, firm fibrosis most severe in the peripheral zones of the lower lobes.” Am Thoracic Soc’y, supra note 17. Eventually, the opacities may spread to the middle and upper lung zones. Also, even though irregular opacities are primarily presented from exposure to asbestos, mixed irregular and rounded opacities can also often be seen. Id.

74 See supra note 73. If the individual was a coal miner, however, the diagnosis would likely be coal workers’ pneumoconiosis (“black lung” disease) instead of, or in addition to, silicosis. A. Oikonomou & N.L. Müller, Imaging of Pneumoconiosis, 15 Imaging 11 (2003). Both present similarly on radiographic manifestations as small, round opacities in the mid and upper zones of the lungs. Id. In a study of 6,166 coalminers, 801 (13%) X-rays were graded as 1/0. Of these, only 222 were irregular opacities, 455 were rounded opacities and 124 were mixed. See H.E. Amandus et al., Significance of Irregular Small Opacities in Radiographs of Coalminers in the USA, 33 Brit. J. Indus. Med. 13 (1976)

75 See supra note 73.
clinical or radiographic evidence to be consistent with asbestosis,\(^76\) at least some of the X-rays graded 1/0 or higher in these studies were based on opacities that may not have been consistent with asbestosis. By contrast, the litigation B Readers assert that they are identifying and grading only those opacities “consistent with asbestosis.” If the medical studies were to have limited identifying opacities to only those “consistent with asbestosis,” they would have found a lower prevalence of fibrosis due to asbestos exposure than their published results.

2. The Possibility of Over-Reading of Fibrosis in the Clinical Studies

According to medical literature, there are some conditions that manifest themselves on pulmonary X-rays that are not properly read as radiographic evidence of fibrosis but are similar enough to fibrosis to be easily misinterpreted as such.\(^77\) Some medical conditions may cause lung changes that can “mimic roentgenographically the specific fibrogenic dust entity or forms of immunologic occupational disease.”\(^78\) In addition, parenchymal abnormalities produced by aging and smoking have been postulated to be “indistinguishable from occupationally related pulmonary fibrosis.”\(^79\) Thus, it is possible that the medical studies may have overstated the number of X-rays with radiographic evidence of fibrosis graded as 1/0 or higher.

\(^{76}\) Of the 58 studies, 16 do not say anything about the size, shapes, or location of the opacities; 10 state only that they identified small opacities; 26 state that the opacities observed were of irregular shape; and only 6 of the studies state that the opacities are of irregular shape and located in the lower lung zones. Of the 16 studies that did not describe the opacities, 5 indicated that a radiologist had interpreted the X-rays as consistent with asbestosis (even though the bases for these interpretations are not set out in the studies). In addition, for 5 studies, the authors referred to those identified with radiographic evidence of fibrosis graded 1/0 or higher, as having asbestosis. No diagnoses, however, were undertaken in these studies and the use of the term “asbestosis” does not appear to be used in a medically rigorous manner.

\(^{77}\) Chest radiographic interpretations have been read as positive for fibrosis when, in fact, they were negative; the misinterpretation results from “increased basilar linear markings caused by emphysema or pleural changes that overlay the parenchyma.” A.C. Friedman et al., *Computed Tomography of Benign Pleural and Pulmonary Parenchymal Abnormalities Related to Asbestos Exposure*, 11 SEMINARS ULTRASOUND CT & MR 393, 399-400 (1990) [hereinafter Friedman, *Computed Tomography*]. Prominent vessels, chronic obstructive pulmonary disease, bronchiectasis, scarring from surgery, old tuberculosis, obscuration of the lung by plaques en face, and walls of bullae (emphysema) have also been misread as parenchymal asbestosis. See Friedman 1988, supra note 71, at 270-71.


Additionally, a study comparing techniques in diagnosing asbestos-related pleural disease and asbestosis concluded that on the basis of the study that “a positive diagnosis of pleural or parenchymal disease would be correct in only approximately 50% of patients.”\textsuperscript{80}

3. “Background” Prevalence of Fibrosis

A second reason why the clinical studies may overstate the number of X-rays with radiographic evidence of fibrosis graded 1/0 or higher due to asbestos exposure is that there is a “background” prevalence of small opacities in populations occupationally unexposed to asbestos or other mineral dusts. Of the thirteen studies published on the prevalence of small lung opacities in such populations, eleven are included and two are excluded from the review.\textsuperscript{81} These studies indicated a prevalence of

\textsuperscript{80} Friedman 1988, supra note 71, at 272. (evaluating the utility of the HRCT compared to chest X-rays in the diagnosis of asbestos-related disease); see infra note 154.

\textsuperscript{81} The eleven included studies are:

R.M. Castellan et al., \textit{Prevalence of Radiographic Appearance of Pneumoconiosis in an Unexposed Blue Collar Population}, 131 AM. REV. RESPIRATORY DISEASE 684 (1985) (examining 1,422 X-rays and finding opacities graded as 1/0 or higher in 3 (.21%).

D.M. Epstein et al., \textit{Application of ILO Classification to a Population without Industrial Exposure: Findings to be Differentiated from Pneumoconiosis}, 142 AM. J. ROENTGENOLOGY 53 (1984) (examining 200 X-rays and finding opacities graded as 1/0 or higher in 36 (18%).

J.R. Glover et. al., \textit{Effects of Exposure to Slate Dust in North Wales}, 37 BRIT. J. INDUS. MED. 152 (1980) (examining 402 X-rays and determining that there were opacities graded as 1/0 or higher in 39 (9.7%).

K. Jakobsson et al., \textit{supra} note 72 (examining 29 X-rays and finding that there were opacities graded as 1/0 or higher in 2 (6.8%). This study was separated for the purposes of the review because some of the population was exposed. The results of the study as it pertains to the exposed is included in the review of exposed populations.

S.M. Kennedy et al., \textit{Lung Function and Chest Radiograph Abnormalities among Construction Insulators}, 20 AM J. INDUS. MED. 673 (1991) (examining 149 unexposed in addition to its study of insulators, and finding 7 (4.7%) to have opacities graded 1/0 or higher).

K.H. Kilburn et al., \textit{Interaction of Asbestos, Age, and Cigarette Smoking in Producing Radiographic Evidence of Diffuse Pulmonary Fibrosis}, 80 AM. J. MED. 377 (1986) (examined 2,514 X-rays and finding opacities graded as 1/0 in 32 (1.27%). This study had five categories of populations. Two of them were unexposed and are included here with the unexposed studies, one was an insulator population and was included with the insulators’ studies, and two were of exposed populations and are included in the review of exposed populations.

A.J. Zitting et al., \textit{Radiographic Small Lung Opacities and Pleural Abnormalities as a Consequence of Asbestos Exposure in an Adult Population}, 21 SCANDINAVIAN J. WORK ENV’T. & HEALTH 470 (1995) (examining 3,494 X-rays and finding opacities graded as 1/0 or higher in 408 (11.7%). The population that was examined was classified as probably exposed, possibly exposed, and unlikely exposed; for the purposes of this review, the results were separated out. In the review of unexposed populations, only the results of the unlikely exposed are included. The other two categories are presented in the exposed populations above.

S. Cordier et al., \textit{Epidemiologic Investigation of Respiratory Effects Related to Environmental Exposure to Asbestos Inside Insulated Buildings}, 42 ARCHIVES OF ENVTL. HEALTH 303 (1987)
radiographic evidence of fibrosis graded as 1/0 or higher ranging from 0.21% to 57%. The approximate median prevalence was 6.8%. A review of the results of the eleven studies indicates a prevalence of radiographic evidence of fibrosis among these unexposed populations of 3.19% (3,547 of 111,127). Additionally, Swedish studies of white collar workers who were not occupationally exposed to asbestos indicated that 11% had a medium reading of 1/0 or greater and 5% had a reading of 1/1 or greater.

[hereinafter Cordier et al., 1987]. This study was of three groups: occupationally exposed; environmentally exposed (working in asbestos-insulated buildings for at least 15 years with no known occupational exposure); and nonexposed. For purposes of this cumulation, I am excluding only the occupationally exposed because the prevalence of fibrosis caused by working in buildings with asbestos insulation is extremely low. For this study, N=1108 and averaging the results of two B Readers, the number of X-rays graded 1/0 or higher was 174.

Alan M. Ducatman et al., “B-Readers” and Asbestos Medical Surveillance, 30 J. OCCUPATIONAL MED. 644 (1988) [hereinafter Ducatman et al., 1988]. For the Ducatman et al., 1988 study, N=105,029 and the number with X-rays of 1/0 or higher was 3778 (3.51%). I am excluding an outlier B Reader who was 5-100 times more likely to find 1/0 or higher than the other readers. Therefore, N=100,381 and the 1/0 and higher total is 2,799 (2.79%).

Bjorn Hilt et al., Asbestos-Related Radiographic Changes by ILO Classification of 10 x 10 cm Chest X-Rays in a Screening of the General Population, 37 J. OCCUPATIONAL & ENVTL. MED. 189 (1995) [hereinafter Hilt et al., 1995]. The Hilt et al., 1995 study describes a previous screening of 21,453 males aged 40 years or more in the county of Telemark, Norway. It was found that 18.1% had been occupationally exposed to asbestos and the prevalence rate was 0.4% (86). A sample of 1,388 of these X-rays was randomly selected for reevaluation. The results of the reevaluation were that 25 (1.8%) were found to have small opacities of 1/0 or higher. In this sample, 18% were found to have had occupational exposure. Neither study provides a means for excluding those occupationally exposed from the results. Somewhat arbitrarily, and in view of the relatively low number of opacities graded 1/0 or higher, I am including the results of the reevaluation in the review.

R. Saito et al., 1990, supra note 72 (finding that of the 40 person without occupational asbestos exposure, 22 (57%) had X-rays graded 1/0 or higher).

The two studies of unexposed populations that were excluded were: William Weiss, Cigarette Smoking and Diffuse Pulmonary Fibrosis, 99 AM. REV RESPIRATORY DISEASE 67 (1969) [hereinafter Weiss, 1969] and William Weiss, Cigarette Smoking and Diffuse Pulmonary Fibrosis, 14 ARCHIVES ENVTL. HEALTH 564 (1967) [hereinafter Weiss, 1967]. Both of these studies are excluded because they did not provide the results using the ILO classification system. The results of both of these studies showed a prevalence of diffuse pulmonary fibrosis in the same range as other studies of unexposed populations. The Weiss 1967 study examined 999 people and found fibrosis in 3.10% . The Weiss 1969 study examined 2,825 people and found fibrosis in 1.4%.

A meta-analysis of the first seven studies listed above found a prevalence of 5.3%. Meyer, Prevalence/Unexposed, supra note 79. The prevalence found in these seven studies ranged from 0.21% to 11.7%. Id. The prevalence was 11.3% in Europe and 1.6% in North America. A review of the results of the seven studies indicates a prevalence of 6.4% (527 of 8,210). The meta-analysis required that each study had at least two B Readers or their equivalent read the X-rays and grade then on the ILO scale. Because of this requirement, the meta-analysis omitted the Ducatman et al., 1988 study—the largest of all the studies—because the 23 B Readers did not read the same films. See id.

M. Albin et al., 1992, supra note 72, at 866.
A variety of reasons for the background prevalence are examined in the literature. One explanation is that increasing age and smoking each result in opacities that are indistinguishable from occupationally related pulmonary fibrosis.84

This background prevalence of fibrosis in the general population is likely to have resulted in elevated findings of radiographic evidence of fibrosis in the clinical studies of populations occupationally exposed to asbestos. No study has been done to attempt to identify the amount of misattribution of fibrosis to asbestos exposure in clinical studies.

One 1985 study suggests that the degree of misattribution may be significant. This study, looked at the routine admission chest X-rays of patients hospitalized in an urban university medical center who were not known to have any industrial exposure to asbestos.85 Out of 200 X-rays examined, 36 (18%) had profusion levels of 1/0 or greater, and 35 (17.5%) had profusions of 0/1.86 If the chest X-rays of the patients with profusions of 0/1 were read by the comparative handful of B Readers with all the financial incentives attendant in the litigation context to grade the X-rays as 1/0 or higher,87 it is likely that these B Readers would have read the 0/1s as 1/0s. On that basis,88 it would be plausible to conclude that a third or more of the adult population without occupational exposure to asbestos could be found to have lung opacities.

84 “Age and smoking habits have been postulated to produce radiographic parenchymal abnormalities in unexposed populations indistinguishable from occupationally related pulmonary fibrosis.” Meyer, Prevalence/Unexposed, supra note 79, at 405. One study showed a threefold increase in lung abnormalities in smokers when compared to nonsmokers. Id. at 408. Older workers have an increased prevalence of opacities which may be due to cumulative environmental exposures and, perhaps, age itself. Id.; see also, Weiss, 1969, supra note 81; Weiss, 1967, supra note 81 (studying 999 men and women who came to the survey unit of the Philadelphia Tuberculosis and Health Association for free chest X-rays and finding that the prevalence of “diffuse pulmonary fibrosis” showed a strong dose-response relationship to cigarette smoking). Of 527 current smokers, 23 (4.4%) were found to have “diffuse pulmonary fibrosis.” For men who smoked more than one pack a day for 20 years or more, the prevalence exceeded 20%. Id.; Anders J. Zitting, Prevalence of Radiographic Lung Opacities and Pleural Abnormalities in a Representative Adult Population Sample, 107 CHEST 126, 127 (1995) [hereinafter Zitting, Prevalence] (finding that a correlation exists between aging and the presence of fibrosis); see infra notes 161-170.


86 Id. at 54.

87 For discussion of the financial incentives of “entrepreneurial” B Readers to read X-rays as 1/0 or higher, see Brickman, Asbestos Litigation, supra note 1, at 90.

88 Conclusions based on the results of this study are subject to serious caveats. The sample was certainly not a representative one and the fact that all of the X-rays were of hospitalized patients injects another level of caution. Nonetheless, the study provides a useful, if anecdotal, insight into the prevalence of fibrosis among those not known to have been occupationally exposed to asbestos.
of 1/0 or higher on the basis of the standards used by the B Readers most selected to read X-rays obtained in the course of attorney-sponsored asbestos screenings. Even if the X-rays are read in a clinical rather than a litigation setting, another study concludes that one-quarter of men between 55 and 64 in the general population have lung abnormalities that register at least 1/0 on the ILO scale, and the prevalence of such X-ray readings continues to increase with age.89

B. Clinical Re-readings of Litigation B Readers’ Results

Beyond the review of the clinical studies, there is additional support for the conclusion that the B Readers most frequently selected by plaintiffs’ lawyers for litigation screenings are manufacturing B reads for money. In five clinical studies, a judicial proceeding, and an investigation undertaken by the American Bar Association, X-rays read as 1/0 or higher and found to be “consistent with asbestosis” were re-read by a panel of independent B Readers or otherwise analyzed. These studies and proceedings, summarized below, indicate error rates ranging from 60-97%.

1. In an aggregated asbestos litigation, U.S. District Court Judge Carl B. Rubin substituted impartial medical experts for the parties’ experts. The impartial experts found that only 10 (15%) of the 65 plaintiffs claiming to have asbestosis in the 1987-1990 proceeding, did in fact have asbestosis.90

2. In 1986, the United Rubber Workers’ International Union (URW) requested that NIOSH conduct an evaluation of the occurrence of pneumoconiosis among tire workers to determine if the union/industry-operated medical surveillance program, which failed to detect any excess asbestosis or other pneumoconiotic conditions among tire workers, had missed cases of asbestos-related disease.91 The basis for this concern was a very high rate of pneumoconiosis generated by asbestos screenings. Information distributed to tire workers by plaintiffs’ lawyers stated that at one screening location, 64% of those screened, tested positive for asbestosis, and at a second screening

89 Zitting, Prevalence, supra note 84, at 127.
90 Carl B. Rubin & Laura Ringerbach, The Use of Court Experts in Asbestos Litigation, 137 F.R.D. 35 (1991). It can be presumed that since the plaintiffs were claiming to have asbestosis, all had had their X-rays read as 1/0 or higher.
location, 94% tested positive for asbestosis.\textsuperscript{92} Focusing on workers with the greatest potential for disease, NIOSH had an independent panel evaluate 987 X-rays from the surveillance program of workers over forty years of age. The NIOSH panel found that only two, or 0.2%, showed physical changes consistent with the mildest form of asbestosis.\textsuperscript{93}

3. In a 1990 study of 439 tire workers who filed suit after litigation screenings, four medical professors and radiologists re-examined the plaintiffs’ X-rays and found that realistically, only eleven of the claimants (2.5%) had lung conditions consistent with asbestos exposure—a 97.5% error rate.\textsuperscript{94}

4. Doctors interviewed for a report for the American Bar Association Commission on Asbestos Litigation reported having “seen hundreds or even thousands of examples of over-reading of x-rays for litigation purposes.”\textsuperscript{95} One doctor reviewed the medical records of 15,000 people who had been diagnosed with asbestosis based solely on X-ray readings, and determined that “only 10% of the persons could validly be diagnosed with asbestosis.”\textsuperscript{96} “Another doctor reported a 62% error rate on review of X-ray screening results previously read as ‘consistent with asbestosis,’” and a third doctor reviewed 22,000 asbestos-related claims and “found a presumptive x-ray review error rate of up to 86% among five readers, none of whose results matched the general patterns in epidemiological studies.”\textsuperscript{97}

5. Between 1994 and 1995, the Manville Trust experienced huge increases in the number of claims by unimpaired people with non-malignant lung disease. In 1995, this spike spurred the Trust to institute a medical audit program, in which neutral academics analyzed and evaluated 5% of the claims submitted by each law firm during each payment cycle. The review process was intentionally designed “in favor of confirming the disease documented by the claimant and to give


\textsuperscript{93} JANCOVIC & REGER, 1989, \textit{supra} note 91, at 12-14.

\textsuperscript{94} Reger et al., 1990, \textit{supra} note 35. The Reger study did not indicate how many of the 439 active and retired tireworkers had been diagnosed with asbestosis—only that they had been diagnosed with a condition consistent with asbestos exposure which would include pleural abnormalities. Of the eleven subjects that he found actually had lung conditions consistent with asbestos exposure, approximately half may have been found in the study to have pleural abnormalities only. See \textit{id.} at 1089 In that case, only about half of the 11 subjects were diagnosed with asbestosis.

\textsuperscript{95} ABA REPORT, \textit{supra} note 11, at 13 (2003).

\textsuperscript{96} \textit{Id.}

\textsuperscript{97} \textit{Id.}
the benefit of any doubt to the claimant." 98 The results of the audit revealed that even by the extremely conservative audit criteria, there was a high medical audit failure rate, especially for the 1/0 asbestosis claims. 99 For example, analysis of asbestosis claims filed in 1996 revealed that over 66% of the claimants had either no disease at all, or had a less severe condition than alleged in the submission, 100 and that the ten physicians used most often by plaintiffs’ law firms had an average failure rate of 63%. 101 The audit was discontinued after plaintiffs’ lawyers strongly objected to its use. 102

6. In 2004, a study (Gitlin Study) compared 492 X reads of X-rays that were used to support asbestos lawsuits with a panel of six consultant B Readers’ interpretations of the same X-rays. 103 The consultant B Readers were completely blinded to the source of payments, source of X-rays, the attorneys involved, the status of films in litigation, the identity of the B Readers, the individuals’ names, and the results of their cumulative findings. All of the films originally came from plaintiffs’ counsel and had been filed in support of plaintiffs’ asbestos lawsuits.

While plaintiffs’ B Readers had found 95.9% of the 492 X-rays to

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100 See Letter from Mark E. Lederer, Manville Trust, to Elihu Inselbuch 2 (April 24, 1998) (on file with author).

101 Nine of these doctors had failure rates ranging from 50% to 70% while the tenth failed 36% of the time. Roger Parloff, Mass Tort Medicine Men, Am. Law, Jan. 3, 2003, at 98. It should be borne in mind that the X-ray review process was “intentionally designed . . . to operate in favor of confirming the disease documented by the claimant and to give the benefit of any doubt to the claimant.” Houser Affidavit, supra note 98, ¶ 14.

102 See Brickman, Asbestos Litigation, supra note 1, at 128-37.

103 The study was designed and conducted by two researchers: Dr. Joseph N. Gitlin, an associate professor at Johns Hopkins University, who designed and directed the National X-ray Exposure Studies in the United States for the U.S. Public Health Service; and Mr. Otha Linton, a senior executive of the American College of Radiology, where he managed the Task Force on Pneumoconiosis for NIOSH, and was involved in the development of the B Reader program. See Joseph N. Gitlin et al., Comparison of “B” Readers’ Interpretations of Chest Radiographs for Asbestos Related Changes, 11 ACAD. RADIOLOGY 843 (2004) [hereinafter Gitlin Study].

104 Id. at 1402. The B Readers on the panel included one who had consulted primarily for plaintiffs, two who consulted for plaintiffs and defendants, two who consulted primarily for defendants, and two who had no previous participation in reading films for litigation. The total is seven because one of the consultant B Readers died during the course of the study and was replaced. Affidavit of Joseph N. Gitlin DPH, In re Congoleum Corp., No. 03-51524 (Bankr. D.N.J. March 23, 2005).
have a profusion of 1/0 or higher on the ILO scale, and that these findings were “consistent with asbestosis,” the six consultant readers found that only 4.5% of the same X-rays had a profusion of 1/0 or higher. Even these readings did not mean that 4.5% of the 492 had asbestosis. Rather, the re-readings only indicated that 4.5% of the X-rays had small opacities of 1/0 or greater, which could have been the result of old age, obesity, smoking and more than one hundred other causes, including exposure to asbestos.

Based on a statistical analysis, the Gitlin Study determined that there was “a probability of less than 1 in 10,000 that the differences noted between initial and consultant readers are due to chance alone.”

Of the seven B Readers who accounted for a substantial majority of the initial 492 B reads that the Gitlin Study found to have a more than 90% error rate—two have refused to testify about their diagnoses and invoked their Fifth Amendment right against self-incrimination, and one recanted all of his asbestosis and silicosis diagnoses. These seven B Readers appear in a compilation by the Manville Trust of the top twenty-five doctors who authored medical reports in support of claims submitted to the Trust through December 31, 2005. In total, they account for a staggering 222,410 medical reports.

7. The Gitlin Study has been critized by various plaintiffs’ lawyers and others. The gist of the criticism is that (1) the X-rays used in the

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105 Gitlin Study, supra note 103, at 850.
106 The seven B Readers who account for a substantial majority of the initial 492 B reads are Dr. Dominic G. Gaziano, Dr. Ella A. Kazerooni, Dr. Jay T. Segarra, Dr. James W. Ballard, Dr. Phillip H. Lucas, Dr. Ray A. Harron, and Dr. Richard B. Levine.
107 Letter from the Law Firm of Leitman, Siegal & Payne, P.C., to Daniel J. Mulholland (Nov. 11, 2005) (on file with author) (indicating that Dr. James Ballard “will not be producing... [subpoenaed] documents in accordance with his rights under the Fifth Amendment to the United States Constitution’’); Defendants’ Brief In Support of Motion For An Evidentiary Hearing And Motion to Dismiss at 3, In re All Asbestos Cases, Special Docket 073958 (Ohio C.P. Feb. 3, 2006) (indicating that in addition to Dr. Ballard, Dr. Ray Harron was refusing to answer questions about the medical evidence he had provided on Fifth Amendment grounds); see also infra note 256.
108 Dr. Richard B. Levine filed an affidavit in MDL 875, in which he stated that he never diagnosed asbestosis or silicosis, Affidavit of Richard Levine ¶¶ 3, 6, 9, In re Asbestos Prods. Liab. Litig. (No. VI), Civ. Action No. MDL 875 (E.D. Pa. May 1, 2006), despite the fact that he provided over 22,000 medical reports in support of claims filed with the Manville Trust. See CRMC Response, supra note 19, at ques. 14(a) and 14(c).
109 CRMC Response, supra note 19.
110 See, e.g., Objection and Response of The Official Committee of Asbestos Claimants to the Motion of the Official Committee of Asbestos Property Damage Claimants at 15, In re Federal-Mogul Global, Inc., Case No. 01-10578 (RTL) (Bankr. D. Del. Sept. 21, 2004); Letters to the Editor (Dr. Kenneth D. Rosenman), 11 ACAD. RADIOLOGY 1396 (2004); Letters to the Editor (Drs. Alfred Franzblau and Brenda Gillespie, 11 ACAD. RADIOLOGY 1400 (2004).
Gitlin Study were not a representative random sample; (2) there was no control group; and (3) the film selection may have been biased. Dr. Gitlin has rebutted these criticisms. Moreover, the results of his study have been corroborated by a new re-reading study which used a random sample and a control group and which was undertaken in the course of a bankruptcy proceeding.

Despite the fact that in most asbestos-related bankruptcies, there are usually tens of thousands of pending nonmalignant claims, thus making a randomized sample for re-reading easily available, attempts by the debtor or commercial creditors to have such a re-reading have been vigorously opposed by plaintiffs’ lawyers and have mostly been turned down by bankruptcy courts. In a handful of bankruptcies, however, bankruptcy judges have allowed the debtor to conduct discovery of the pending claims including examining a sample of these claims. In the W.R. Grace bankruptcy, the court allowed the debtor to conduct discovery of pending claims including distributing Personal Injury Questionnaires to these claimants. According to the responses, “there were 5,438 claimants who alleged a non-mesothelioma malignancy caused by a Grace exposure and who were relying on X-ray evidence to support the attribution of their cancer to asbestos exposure.”

Two proportionate random samples of 500 X-rays each which met the criteria of the study designed by Dr. Daniel A. Henry


112 See, e.g., Owens-Corning v. Credit Suisse First Boston, 322 B.R. 719 (D. Del. 2005)

113 Order Re: Personal Injury Claim Estimation, In re USG Corp., No. 01-2094 (JFK) (D.Del. Oct. 21, 2005); Order Concerning Schedule for Motions to Compel Regarding the W.R. Grace Asbestos Personal Injury Questionnaire and Schedule for Supplementation of Questionnaire Responses, In re W.R. Grace & Co., No. 01-1139 (JFK) (Bankr. D. Del. Nov. 14, 2006) [hereinafter Order Concerning Schedule for Motions to Compel]; In re G-I Industries, Inc., Case No. 01-3035 (Bankr. D.N.J. Aug. 11, 2006) (rejecting the motion for appointment of a medical panel to review the medical evidence in the pending claims but allowing the debtor a limited period of time to conduct a proposed sampling of pending claims to include use of a “questionnaire”); see also Order Implementing G-I’s Claimant Questionnaire and Sampling Protocol, In re G-I Holdings, Inc., Case No. 01-3035 (Mar. 1, 2007) (containing the detailed Questionnaire to be filled out for the 2500 claims in the sample).

114 Order Concerning Schedule for Motions to Compel, supra note 113.

Because of overlapping claims in the two samples, the total number of study films was 807. These X-rays were read by three B Readers who were blinded as to the source of the X-rays, the purpose of the Study and the entity on whose behalf they were reading the X-rays. Of the 807 claimants, 471 had an X-ray and accompanying ILO reading that met the inclusion criteria and were selected for the comparison study.

The B Readers selected by plaintiffs’ lawyers had found that 383 of the 471 claimants (81.31%) had a profusion of 1/0 or greater on the ILO scale; this was eleven times more frequent than the majority readings of the study B Readers who reported profusions in 33 (about 7%) of these same claimants. The Henry Study, which used both a proportionate random sample and a control group, noted that its results are consistent with those of the Gitlin Study.

A number of the B Readers who had initially read the X-rays had also read X-rays that were reread in the Gitlin Study. The error rates (% over-read) for these B Readers are indicated in the chart below.

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116 Id. Dr. Henry is Director, Thoracic and Cardiac Radiology, Department of Radiology, Medical College of Virginia Hospitals.
117 Id. at 5.
118 Id. at 4.
119 Id. at 5-6.
120 A total of 47 control films, including 25 negative and 22 positive films, were selected. See id. at 7 & app. E. The Kappa statistic (which compares agreement against that which might be expected by chance), sensitivity (likelihood that a positive film will be correctly classified as positive), and specificity (likelihood that a negative film will be correctly classified as negative) were computed for the majority reading and for each of the 3 independent B Readers. Id. at 7. The Kappa statistic for the majority reading was 0.74 which was in the “substantial agreement” range. Id. at 8. The majority readings had similar high sensitivity and specificity, correctly classifying positive films as positive and negative films as negative more than 85% of the time. Id.
121 Id. at 8. The Gitlin Study found a virtually identical error rate (91%). Gitlin Study, supra note 103.
As noted in the chart above, Drs. James Ballard, Dominic Gaziano, Ray Harron, Philip Lucas and Jay Segarra had found between 87.8% and 97.5% of the X-rays they read as positive for asbestosis and had error rates that ranged from 80.48% to 100%.\footnote{Id.}

The Henry Study also analyzed the error rates of the law firms which had submitted the most films in the samples and their percentage of over-reads.\footnote{Id.} For eight of the top nine law firms ranked by number of claims included in the sample, the percentage of over-reads ranged from 71.3% to 93.33%.\footnote{Id. One law firm had only 35.82% of over-reads.}

Finally, though it is not a re-reading, one additional unpublished study produced results that are fully consistent with the very high error rates found by the re-readings discussed above.\footnote{An unpublished study of workers in power plants provides an additional basis for validating the very high error rates that clinical studies and medical experts report. Electricity generating power plants (powerhouses) have extensive amounts of thermal insulation. In older plants, the insulation is likely to include large amounts of asbestos-containing products. R.C. Browne, Health in Power Stations, 64 PROCEEDINGS ROYAL. SOC’Y. MED. 1075 (1971); Jack H. Fontaine & David M. Trayer, Asbestos Control in Steam Generating Plants, 36 AM. INDUS. HYGIENE. ASSOC. J. 126 (1975); R. Lazarus, Lung-Function Reference Values From Victorian Power-Industry Workmen, 2 MED. J. AUSTL. 121 (1982). The most intense asbestos exposures associated with powerhouses are realized by construction workers during construction, maintenance and the dismantling of powerhouses. See, e.g., In re Joint E. & S. Dists. Asbestos Litig: All Powerhouse Cases, No. NYAL-PH-8888, 1991 U.S. Dist. LEXIS 8401 (E.D.N.Y & S.D.N.Y. 1991) (describing 700 cases that were consolidated for trial, in which mostly construction workers were claiming injurious exposure to asbestos during the construction or repair of powerhouses). Workers employed in powerhouses, however, may spend large portions of their workday in close proximity to asbestos insulation and may be called upon to make occasional repairs. At least some of these powerhouse workers have brought suit, but there is no clear indication of how many of these claims were generated by litigation screenings. See In re N.Y. City Asbestos Litig., 142 F.R.D. 60 (E.D.N.Y. & S.D.N.Y. 1992) (attaching the report of a special master regarding the resolution of asbestos personal injury and wrongful death claims brought in federal and state courts, including approximately 1,000 state court claims that were}
In the table below, I summarize the error rates found by the five clinical studies, the one judicial proceeding where the court-appointed experts re-read the X-rays, and the ABA investigation.

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<th>Clinical Study</th>
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<td>Judge Rubin</td>
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<td>NIOSH</td>
<td>78%</td>
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<tr>
<td>Reger (tire workers)</td>
<td>97.5%</td>
</tr>
<tr>
<td>ABA</td>
<td>62-90%</td>
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<tr>
<td>Manville Trust</td>
<td>63%</td>
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<tr>
<td>Gitlin Study</td>
<td>91-92%</td>
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<tr>
<td>Henry Study</td>
<td>91.4%</td>
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part of the Powerhouse and Brooklyn Naval Yard consolidation). Several studies have been done of powerhouse workers. See, e.g., Fontaine & Trayer, supra; A. Hirsch et al., Asbestos Risk Among Full-Time Workers in an Electricity-Generating Power Station, 330 ANNALS N.Y. ACAD. SCI. 137 (1979); G. Cammarano et al., Cancer Mortality Among Workers in a Thermoelectric Power Plant, 10 SCANDINAVIAN J. WORK ENV’T. & HEALTH 259 (1984); G. Cammarano et al., Additional Follow-Up of Cancer Mortality among Workers in a Thermo-Electric Power Plant, 12 SCANDINAVIAN J. WORK ENV’T. & HEALTH 631 (1986); F. Forastiere et al., Respiratory Cancer Mortality Among Workers Employed in Thermoelectric Power Plants, 15 SCANDINAVIAN J. WORK ENV’T. & HEALTH 383 (1989); Y. Lerman et al., Asbestos Related Health Hazards among Power Plant Workers, 47 BRIT. J. INDUS. MED. 281 (1990); G. Petrelli et al., A Retrospective Cohort Mortality Study on Workers of Two Thermoelectric Power Plants: Fourteen-Year Follow-Up Results, 5 EUR. J. EPIDEMIOLOGY 87 (1989). However, these studies have either been limited to, or have also included, insulators whose asbestos exposure involved work prior to power plant employment. See Joseph M. Miller, M.D., Benign Exposure To Asbestos Among Power Plant Workers, at tbl. 4 (1990) (unpublished manuscript on file with author) [hereinafter Miller 1990]. For discussion of insulators’ high exposures and resulting disease prevalence, see supra notes 44-64 and accompanying text. To provide a valid comparison, a study of powerhouse workers whose lifespan of employment was confined to power plant operations was undertaken. This unpublished study identified 114 workers who had extensive work histories with an average of 23 years of exposure and a mean latency of 32 years. Id. at tbls. 2 & 4. The study found that none of the 114 workers had asbestosis and 95% had no impaired lung function. Id. at 3, 5-6. One of the two readers found 2 of the 114 with 1/0 readings and none with higher; the other found none with 1/0 or higher. Id. at tbl. 3. The study concluded that no cases of “definite asbestosis” were found. Id. at 5. Extensive exposure to asbestos was confirmed by the findings of circumscribed pleural plaques in 40-46% of those studied. Id. at 5. While this unpublished study did not re-read X-rays generated for litigation, it can be seen functionally as a re-reading on the assumption that whatever litigation screenings of the powerhouse workers that did take place produced the same rate of positive X-ray readings, 60-80%, that most litigation screenings, including re-reads, generate, and, the same rate of diagnoses of asbestosis of those screened positive, 80-90%.

128 The NIOSH study did not indicate whether the X-rays had previously been read by litigation B Readers. I am using the reported results of 64% and 94% positive for two screenings simply averaging them (79%), and comparing that to the 0.2 prevalence rate found by the NIOSH Study. See supra note 91.
C. The Disparity Between the Prevalence of Pleural Plaques in Litigation Screenings and Clinical Settings

Pleural plaques (pleural fibrosis), sometimes referred to in asbestos litigation as “pleural disease,” are deposits of collagen fibers on the linings (pleura) of the lung, usually detectable only by X-rays, 15-20 years after initial and substantial exposure to asbestos. As described in the medical literature, the vast majority of individuals found to have pleural plaques have no symptomatology or lung impairment. 129

Medical studies consistently show that, among those exposed to asbestos in a variety of settings, pleural plaques are two to three times more likely to be prevalent than pulmonary asbestosis.130 Consistent with the medical literature, pleural plaque claims accounted for the majority of nonmalignant asbestos claims in the 1980s.131

By the mid-1990s, however, the volume of 1/0 asbestosis claims exceeded pleural plaque claims by a substantial margin.132 This abrupt

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129 See, e.g., Victor L. Roggli, Fiber Analysis, in OCCUPATIONAL & ENVTL. MED. 255 (2d ed. 1992); VICTOR ROGGLI, DONALD GREENBERG & PHILLIP PRATT, PATHOLOGY OF ASBESTOS ASSOCIATED DISEASES 30 (1992). A severe form of pleural fibrosis can, however, be impairing. See Brickman, Asbestos Litigation, supra note 1, at 51-54, 60 for further description of pleural plaques and their role in asbestos litigation.

130 See Gunnar Hillerdale et al., Asbestos, Asbestosis, Pleural Plaque and Lung Cancer, 23 SCANDINAVIAN J. WORK ENV’T. &. HEALTH 93, 96 (1997) (“[I]n most investigations pleural plaques are the most common radiologists’ finding in persons exposed to asbestos.”); Irving Selikoff, Asbestosis: Interstitial Pulmonary Fibrosis and Pleural Fibrosis in a Cohort of Asbestos Insulation Workers: Influence of Cigarette Smoking, 10 AM. J. INDUS. MED. 459, 469 (1986) (concluding, based on a study of 1,117 insulation workers, that pleural changes (pleural plaques) were more common that pulmonary fibrosis (asbestosis) regardless of smoking history); see also Albert Miller et al., Spirometric Impairment in Long Term Insulators, 105 CHEST 175 (1994); Albert Miller et al., Relation of Spirometric Function To Radiographic Interstitial Fibrosis in Two Large Work Forces Exposed To Asbestos And Evaluation Of The ILO Profusion Score, 53 OCCUPATIONAL & ENVTL. MED. 808 (1996); Laura S. Welch et al., The National Sheet Metal Worker Asbestosis Disease Screening Program Radiographic Findings, 25 AM. J. INDUS. MED. 6345 (1994). In a study of men occupationally exposed to asbestos in naval dockyards, pleural abnormalities were found ten times more frequently than interstitial fibrosis. See Harries et al., supra note 71.

131 See In re Joint E. & S. Dist. Asbestos Litig., 129 B.R. 710, 934 (E.D.N.Y. & S.D.N.Y 1991), rev’d 982 F.2d 721 (2d Cir. 1992) (indicating that of the 136,250 claims pending against the Manville Trust, as of April 10, 1991, 54.4% were for pleural plaques and 30.7% were for asbestosis, for a ratio of 1.77 to 1); see also, Brickman, Administrative Alternative?, supra note 12, at 1861 (reviewing disease mix data, indicating that pleural plaques accounted for 45-60% of outstanding claims in the 1988-1991 period whereas asbestosis accounted for 25-37% of the claims).

132 In a study by Dr. Gary Friedman of 1,691 X-ray and pulmonary function reports involving claims against Owens Corning, see supra note 19, Dr. Friedman determined that none of the five
shift in X-ray readings by litigation doctors is accounted for by the global *Georgine* settlement in January 1993, which was later invalidated by both the Third Circuit and the U.S. Supreme Court. As part of that settlement, most of the leading plaintiffs’ lawyers, who exchanged upwards of $300 million in fees for settling their current inventories of asbestos claimants including pleural plaque claims, agreed that future pleural plaque claims would have no value (unless and until the claimants later manifested with an actual asbestos related disease).

Thereafter, B Readers regularly selected by these lawyers significantly diminished their findings of pleural plaques and instead found radiographic evidence of fibrosis graded as 1/0 or higher, which was “consistent with asbestosis.” Thus, between 1994 and 2002, in filings with the Manville Trust, claimants alleged “pleural disease” alone approximately 55,000 times, but more than five times that number of asbestosis claims (278,000). Moreover, the ratio of asbestosis to pleural plaque claims steadily increased in this period. These B Readers, and other doctors, then went on to diagnose the vast majority of these claimants as having asbestosis—a compensable disease. These new claimants, who were diagnosed with asbestosis, had worked alongside other claimants at identical work sites whose screening-generated X-rays these same B Readers had often previously read as showing “pleural disease” only and not asbestosis.

The substantial disparity between the ratio of pleural plaques to pulmonary asbestosis found in clinical settings (2:1 to 3:1) and the ratio generated by litigation screenings after the *Georgine* settlement (0.2:1) is further evidence of the speciousness of the medical reports generated by litigation screenings. In addition, the apparent fungibility between X-ray readings of pleural plaques and fibrosis consistent with asbestosis of the litigation doctors is itself at least circumstantial evidence that the X-ray readings of the litigation doctors are “manufactured for money.”

B Readers who accounted for 80% of the X-ray readings in the sample examined, had identified more “pleural only” cases than pulmonary asbestosis. In fact, of the 1,691 cases reviewed, only 124 “pleural only” cases were identified. Moreover, the ratio of pulmonary asbestosis to “pleural only” disease for the four B Readers in the sample accounting for 76.8% of the claims, was 47 to 1, whereas in the remaining reports submitted by over 40 other B Readers and physicians, the ratio of pulmonary asbestosis to “pleural only” disease was 2 to 1.

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134 *Amchem Prods., Inc. v. Windsor*, 521 U.S. 591 (1997), aff’d *g* 83 F. 3d 610 (3d Cir. 1996).
135 *Id.* at 601-04.
137 *Id.*
D. The Disparity Between Rates of Clinical Diagnoses of Asbestosis and Those Generated by Litigation Screenings

Just as the process of reading screening-generated X-rays is fundamentally flawed from the perspective of applicable medical protocols, so too is the process of diagnosing those whose X-rays have been read as indicating radiographic evidence of fibrosis. In a clinical setting, the diagnosis of asbestosis or other type of pneumonocosis follows a specific process. Because there are many possible causes of pulmonary fibrosis, the American Medical Association, the American Thoracic Society, NIOSH, the Association of Occupational and Environmental Clinics, and others have developed diagnostic protocols for occupational disease. The most critical task in the process of diagnosing the cause of pulmonary fibrosis is to “exclude alternative causes for the findings.” Based upon the testimony of prominent occupational medicine physicians, the American Bar Association Commission on Asbestos Litigation described the established protocol for diagnosing nonmalignant asbestos-related diseases:

Each of the doctors interviewed by the Commission independently stated that the diagnosis of asbestos-related pleural disease, and particularly asbestosis, requires assessment of a number of factors including the review of chest x-rays, pulmonary function tests, latency, and the taking of a complete occupational, exposure, medical and smoking history. Because many symptoms and findings are not specific to asbestos-related disease, this approach is necessary to enable a physician to exclude other more probable causes for various findings. This then enables the physician to support a conclusion that the patient’s medical condition is the result of asbestos exposure. These types of requirements are typical for assessment of disability or impairment under various legislative and regulatory systems, including Social Security, the Federal Employees Compensation Act (FECA), and state worker compensation programs.

The diagnostic process followed by litigation doctors falls well short of the requisite standards set by the medical profession. Judge Jack found that a critical part of the diagnostic process is the taking of a detailed occupational history by a trained professional—a task that

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138 See infra notes 161-170
139 See supra note 17.
140 Am. Thoracic Soc’y, supra note 17.
141 ABA REPORT, supra note 11, at 12.
requires thirty minutes or more. In litigation screenings, the occupational histories are taken by people with no medical training who have significant financial incentives to create a history that would support a diagnosis of asbestosis (or silicosis). Judge Jack concluded that “virtually all of these diagnoses fail to satisfy the minimum, medically acceptable criteria.” Judge Jack further found that the litigation doctors “simply ignored” the requirement that they consider and rule out other, more probable, causes of fibrosis. Indeed, she noted that to the extent that differential diagnoses were made at all in the course of litigation screenings, they were done by the medical stenographers who typed the medical reports by interpreting the boxes checked on the ILO forms by the litigation doctors.

It should come as no surprise then to learn that the disparity between diagnoses of asbestosis done in clinical studies and those done in the course of litigation screenings are even more pronounced than the disparities in the X-ray readings. I estimate that at least 80% of litigants whose screening-generated X-rays are graded as 1/0 or higher are then diagnosed with asbestosis “within a reasonable degree of medical certainty.” Only two of the clinical studies included in the review undertook to diagnose the causes of the radiographic evidence of fibrosis. Two other studies also provide relevant information.

The Koskinen Study of 18,943 Finnish workers occupationally exposed to asbestos in the construction, shipyard, and “asbestos industry” found that 2.8% (534) were found to have opacities of 1/0 or greater. Of these 534, 23.2% (124) were diagnosed with

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143 Id. at 622; see also sources cited in Brickman, Asbestos Litigation, supra n. 1, at 67 n.101.
144 MDL 1553, 398 F. Supp. 2d at 625.
145 Id. at 629.
146 Id. at 630.
147 See supra note 37; see also Brickman, Asbestos Litigation, supra note 1, at 86 n.164.
148 See Koskinen et al., supra note 72. The study was limited to those who fit the following criteria: individuals with at least ten years in construction, who had commenced work before 1980; individuals with one year in a shipyard, who had commenced work before 1980; or individuals with one year in the asbestos industry, who had commenced work before 1976. The mean year of onset of exposure for the entire group was 1960. The mean duration of employment was 26 years, and the average duration of asbestos exposure was 9 years. The number studied totaled 18,943. Of the total 4,133 individuals who screened positive, three-quarters were diagnosed with an occupational disease, of which 4% (124) were diagnosed with asbestosis. The criteria for testing positive were: (i) small irregular lung opacities clearly consistent with interstitial fibrosis (1/1); (ii) small irregular lung opacities indicating mild interstitial fibrosis (1/0) and findings consistent with unilateral or bilateral pleural plaques; (iii) findings indicating marked abnormalities of the visceral pleura not known to be caused by infection; and (iv) findings consistent with bilateral pleural plaques.
The Murphy Study found that of 195 workers exposed to asbestos in shipyards, a total of 65 (33.3%) had radiographic evidence of fibrosis graded as 1/0 or higher and of these, 18.5% (12 of 65) were diagnosed with asbestosis. Thus, of the number of workers studied, 6.15% were diagnosed with asbestosis. It is noteworthy that 101 of the 195 workers studied were pipecoverers who “prepared and applied insulating materials to machinery and pipes.” As previously indicated, the disease rates of pipecoverers and insulators have been far higher than that of other occupational groups with asbestos exposure. Of the 101 pipe coverers, 44 had X-ray readings of 1/0 or higher, and of these, 11 were diagnosed with asbestosis. Only one of the 94 pipefitters who were included as a control group was diagnosed with asbestosis.

In addition to these two clinical studies, in a consolidated asbestos trial, U.S. District Court Judge Carl B. Rubin substituted impartial medical experts for the parties’ medical experts. Sixty-five plaintiffs were claiming that they had asbestosis and presumably had X-rays graded at 1/0 or higher. The court appointed experts diagnosed ten of the 65 plaintiffs with asbestosis—a diagnosis rate of 15%.

One additional study sheds light on the diagnosis rate. This study was of English naval dockyard workers who had occupational exposure to asbestos and indicated that of the 3,856 workers studied, only 0.3% were diagnosed with asbestosis. However, because the study used more exacting diagnostic criteria—the effect of which could not be measured or estimated—I am not including it in my review of rates of clinical diagnoses of asbestosis.

Because of the way the data is presented, it is possible that the percentage that might have been diagnosed with asbestosis could have been somewhat higher or lower. The study only submitted for diagnosis those with “positive” X-ray readings. As defined in the study, that excluded the 0.5% (95) with 1/0 readings who did not have any pleural plaques. Id. at 11. Had these 95 workers been included in the group of 4,133 who were identified in the study as “positive,” it is possible that some of them would have been diagnosed with asbestosis, and thus the percentage could have been higher. In addition, the study counted as positive those with bilateral plural plaques and pleural thickening even if they had no opacities graded as 1/0 or higher. While it is unclear form the study, it may be that some of these 3,694 (of the 4,133) were among the 124 diagnoses with asbestosis. In that event, the percentage with X-ray readings of 1/0 and higher diagnosed with asbestosis would have been lower.

The criteria for the “epidemiologic diagnosis” that was done included the presence of at least three of five standardized clinical abnormalities commonly reported in persons with known asbestosis. Id. at 1274-75.

See supra note 90.

See P.G. Harris et al., supra note 70. This study of workers exposed to asbestos at four naval ports found that of 3,856 studied, 12 (0.3%) had “confirmed pulmonary fibrosis.”
A simple comparison of the diagnosis rates of 15%, 18.5%, and 23.2%, with the estimated 80% or higher rate generated by asbestos litigation screenings understates the degree of difference. As noted, the litigation screenings have generated a 50-90% positive rate for radiographic evidence of fibrosis and findings that these are “consistent with asbestosis,” as compared to the 11.76% rate found in the review of clinical studies, and the 2.8% prevalence rate found in the Koskinen Study. A more informative comparison is the percentages of those screened that were diagnosed with asbestosis. In the Koskinen Study, 0.65% (124 of 18,943) were diagnosed with asbestosis. Had the 18,943 occupationally exposed workers been recruited to attend asbestos litigation screenings, perhaps 9,500 (50%) to 13,200 (70%) or more would have been found to have radiographic evidence of fibrosis and, of these, 7,500 to 10,500 would have been diagnosed with asbestosis, compared to the 124 actually diagnosed in the clinical setting.

Other informative comparisons are those between the Koskinen Study and the results of the litigation screenings conducted by RTS, and criteria for “confirmed pulmonary fibrosis” consisted of an X-ray reading indicating fibrosis, a clinical examination of the chest (for basal rales), and measurement of pulmonary function. Thus, the study is using the term “confirmed pulmonary fibrosis” as the equivalent of a diagnosis of asbestosis. However, the study included a requirement of diminished lung function and the presence of basal rales but did not break down the results so that the effect of these additional criteria could be measured.

In a Swedish study of 210 construction workers exposed to fibrogenic dust such as asbestos and crystalline silica, and found to have a profusion of 1/1, only 41 (20%) were reported as showing a pneumonconiosis. M. Albin et al., 1992, supra note 72, at 864. The study did not provide diagnoses but, given the exposures to crystalline silica and presumably other occupational dusts, it is undoubtedly true that the number with asbestosis was less than the 20% determined to have a pneumonconiosis.

The Friedman 1988 study, supra note 71, is also relevant. It focused on the role of high resolution CT in the diagnosis of asbestosis. Sixty men, average age 58 years, with at least one year of occupational asbestos exposure, were studied. These sixty men were chosen because they already had X-rays read as indicating an asbestos-related abnormality. Of the 60, 55 had asbestos-related pleural disease with or without parenchymal asbestosis. The remaining 5 had: pleural disease and mesothelioma (2); interstitial lung disease (2); and interstitial lung disease with lung cancer (1). New X-rays were taken, which resulted in thirteen patients (22%) being diagnosed with pleural disease without parenchymal involvement, two patients (3%) diagnosed with parenchymal asbestosis without pleural disease, and nineteen patients (32%) being diagnosed with both asbestos-related pleural disease and parenchymal asbestosis. The diagnosis of asbestosis was reached, with the use of the HRCT, in 21 patients (35%). This diagnosis rate is elevated due to the limitation of the study to those who already had X-rays read as indicating asbestos-related abnormalities.

See supra note 148.

In the unpublished powerhouse study, see supra note 65, of 114 powerhouse workers who averaged 23 years of exposure, had a mean latency of 32 years, and of whom 40-46% had radiographic evidence of asbestos exposure, none were found to have asbestosis.
of the results of the review of clinical studies with RTS’s results. As noted, RTS doctors diagnosed 17,877 of the 32,119 persons (55.7%) screened by RTS—whose records had been analyzed in the course of MDL 875—as having asbestosis.\footnote{See supra note 37.} If we superimpose the results of the Koskinen Study of 18,943 occupationally exposed workers, then instead of 17,877 having been diagnosed with asbestosis by RTS doctors, the number would have been 209.

Additionally, if we superimpose the results of the review of clinical studies on the 32,119 persons screened by RTS whose records have been analyzed in the course of MDL 875, then approximately 3800 would have had their X-rays read as indicating evidence of fibrosis graded 1/0 or higher, and of these, approximately 950 or less would have been diagnosed with asbestosis. RTS doctors, however, diagnosed 17,877 of those RTS screened with asbestosis.\footnote{Id.}

The results of the Murphy and Koskinen Studies, as well as those of the review of clinical studies, are consistent with the medical literature that states that new manifestations of asbestosis largely ceased by 1990. Indeed, more than fifteen years ago, medical experts called asbestosis a “disappearing disease,”\footnote{Kevin Browne, Asbestos-Related Disorders, in OCCUPATIONAL LUNG DISORDERS 410 (3d ed. 1994).} and a condition that is “exceedingly rare.”\footnote{“We have not seen a single case of significant asbestosis with first exposure during the past 30 years.” E.A. Gaensler, P.J. Jederlinic & A. Churg, Idiopathic Pulmonary Fibrosis in Asbestos-Exposed Workers, 144 AM. REV. RESPIRATORY DISEASE 695, 695-96 (1991); E.A. Gaensler, Asbestos Exposure in Buildings, 13 CLINICAL CHEST MED. 231 (1992). In a study published in 1990 of workers engaged in manufacturing of specialty and filler papers containing asbestos, the authors concluded that “[t]his study confirmed our impression that asbestosis is a disappearing disease. Among persons first exposed before 1950, 47.6% had developed fibrosis … decreased to 18.0% for 1950-1959, and among those first exposed after 1959 only 2.0% had developed asbestosis.” Gaensler et al., Radiographic Progression, supra note 72, at 387; see D.M. Rosenberg, Asbestos Related Disorders A Realistic Perspective, 111 CHEST 1424 (1997); see also Letter from Dr. James Crapo, S. COMM. ON THE JUDICIARY, THE FAIRNESS IN ASBESTOS INJURY RESOLUTION ACT OF 2003, S. REP. NO. 108-118, at attach. A (2003).}

1. Other Causes of Fibrosis in Addition to Asbestos Exposure

The Koskininen Study indicates a falloff of approximately 75% from X-ray readings of radiographic evidence of fibrosis to diagnoses of asbestosis. A principal reason is that there are well over one hundred possible causes of radiographic evidence of fibrosis besides asbestos
exposure,\textsuperscript{161} including aging,\textsuperscript{162} smoking,\textsuperscript{163} obesity, and the use of certain medications.\textsuperscript{164} Some of the conditions that must be excluded as possible causes of radiographic evidence of fibrosis before a diagnosis of asbestosis can be made are collagen-vascular disease and sarcoid,\textsuperscript{165} cholesterol pneumonitis,\textsuperscript{166} parenchymal Hodgkin’s disease, rheumatoid lung\textsuperscript{167} as well as others.\textsuperscript{168} A condition called “idiopathic pulmonary fibrosis,”\textsuperscript{169} that is, fibrosis with no known cause, is indistinguishable on radiographs from the fibrosis produced by asbestos exposure and has been misread as asbestosis.\textsuperscript{170}

Accordingly, it is probable that the substantial disparity between the X-ray readings of the litigation doctors and the results of the clinical

\begin{footnotesize}
\begin{itemize}
  \item See Marvin I. Schwartz, Approach to the Understanding, Diagnosis and Management of Interstitial Lung Disease, in INTERSTITIAL LUNG DISEASE 1, 4-5, tbl. 1-1 (Marvin I. Schwartz & Talmadge E. King, eds. 1998); PULMONARY PATHOLOGY 647, 648-49 (D. Dail & S. Hammar, eds. 2d ed. 1994) ("More than 100 known causes of interstitial lung disease are recognized. . . . [M]ost patients with advanced pulmonary fibrosis, whose tissue samples do not meet the histological criteria for asbestosis . . . do not have asbestos-induced fibrosis, even though there may have been a history of exposure to asbestos.").
  \item See supra notes 84 and 89.
  \item Id. In the Weiss & Theodos study of workers in asbestos products manufacturing plants, Weiss concludes that “there is no doubt that cigarette smoking alone produces pulmonary interstitial fibrosis.” Weiss & Theodos, supra note 72, at 344. The authors go on to suggest that, especially in case of mild pulmonary disease, more research needs to be done in order to ascertain if the two causes (smoking and asbestos) are entirely separate or work synergistically to cause fibrosis. Id.
  \item In addition to aging, commonly found “conditions/diseases not related to asbestosis which appear as interstitial lung disease on X-rays include. . . smoking history, obesity, lupus, siliconosis, or numerous other medical conditions.” Affidavit of Dr. Robert Steiner re: Medical Standards of Care for Diagnosing Asbestos-Related Diseases, Motion For Case Mgmt Order Concerning Litig. Screenings at 3, In re Asbestos Prods. Liability Litig. (No.VI), No. MDL 875 (E.D. Pa. July 30, 2001); see also Tatsui Enomoto et al., Diabetes Mellitus May Increase Risk for Idiopathic Pulmonary Fibrosis, 123 CHEST 2007 (2003) (discussing the correlation between prevalence of idiopathic pulmonary fibrosis and age, smoking history, and lifestyle-related diseases, such as obesity and diabetes mellitus). Pulmonary fibrosis is also known to be caused by certain medications, radiation, connective tissue or collagen diseases, sarcoidosis—a disease characterized by the formation of granulomas (areas of inflammatory cells), which frequently affects the lungs—Farmer’s Lung, an allergic reaction to some organic substances, such as moldy hay, various environmental exposures, and sometimes genetic / familial history. Am. Lung Assoc., Interstitial Lung Disease and Pulmonary Fibrosis. Known Causes of Pulmonary Fibrosis, available at www.http://www.lungusa.org/site/pp.aspx?c=dvLUK90E#b=35436&printmode=1 (last visited Nov 21, 2006).
  \item Friedman, Computed Tomography, supra note 77, at 399-400, 401.
  \item See supra note 78.
  \item See supra note 77.
  \item Id.
  \item Id.
  \item Idiopathic pulmonary fibrosis, also known as cryptogenic fibrosing alveolitis, is a “chronic lung condition of uncertain etiology . . . characterized histologically by the presence of usual interstitial pneumonia, and often has typical radiological appearances.” O.J. Dempsey et al., Idiopathic Pulmonary Fibrosis: An Update, 99 Q.J. MED. 643 (2006).
  \item Friedman, Computed Tomography, supra note 77, at 399-400.
\end{itemize}
\end{footnotesize}
studies would be exceeded by the disparity between the diagnoses of asbestosis by the litigation doctors and the results that clinical studies would have produced had they, as did Koskinen, Murphy, and Judge Rubin, also undertaken to provide diagnoses.

E. The Disparity Between the Pandemic Outbreak of Asbestosis Filings in the Courts and the Number of Annual Hospitalizations Primarily Due to Asbestosis

Between 1990 and 2004, approximately 80% of the more than 470,000 claims of nonmalignant asbestos-related disease claims filed with the Manville Trust, which were mostly generated by litigation screenings, claimed asbestosis.171 The volume of these claims of asbestosis is inconsistent with the following: the medical literature which concludes that by 1990, new cases of asbestosis had largely disappeared;172 clinical re-readings of X-rays generated by litigation screenings, which indicate an error rate ranging from 60-97%;173 and the clinical studies reviewed in this Article, which indicate that, of those occupationally exposed to asbestos, approximately 3% would likely be diagnosed with asbestosis.174

The validity of the screening diagnoses is further undermined by survey data from the National Center for Health Statistics (NCHS), which annually conducts the National Hospital Discharge Survey (NHDS). This survey provides data on inpatient utilization of non-Federal, short-stay hospitals in the United States.175 To generate the data, the NCHS annually identifies approximately 500 hospitals which it surveys to collect a sampling of hospital discharge diagnoses.176

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171 Data for the specific 1990-2004 period on the number and types of nonmalignant claims are not available. The available data is that for the period 1988 through September 30, 2006, of the approximately 585,440 nonmalignant claims filed, approximately 465,200 claimants alleged asbestosis.

172 See supra note 160.

173 See supra notes 127-128.

174 See supra notes 148-156.


176 The design of the survey includes a three stage sampling. Since 1988, the sampling frame consists of approximately 6,000 hospitals listed in the SMG Hospital Market database. VITAL & HEALTH STATISTICS, ESTIMATES FROM TWO SURVEY DESIGNS: NATIONAL HOSPITAL DISCHARGE SURVEY, SERIES 13: DATA FROM THE NATIONAL HEALTH SURVEY, NO. 111, DHHS PUB. NO. (PHS) 92-1772 (1992) [hereinafter VITAL AND HEALTH NO. 111, 1992]. The first step is to assign each of the approximately 6,000 hospitals to a PSU (geographic sampling unit), and to
Accessing this data base, I have been able to determine the number of hospitalizations primarily due to asbestosis that took place in the same time period as the claim filings with the Manville Trust. In the 15-year period (1990-2004), surveys of the medical records of a random sample comprising approximately 4,500,000 patients discharged from non-federal hospitals, indicated that the total number of patients who were hospitalized primarily because of asbestosis was 57.\textsuperscript{177}

\textsuperscript{177} See E-mails from Karen A. Lees, MPH, Center for Disease Control & Prevention, National
Each surveyed medical record of a person discharged from a hospital can be assigned up to seven diagnostic codes using the ICD-9 codes. The first listed diagnosis is the principal diagnosis—the condition primarily responsible for causing the admission of the patient to the hospital.

The NHDS data is used to project the diagnoses of all discharged patients annually. Since 1988, approximately 300,000 records are pulled annually for sampling. This is approximately 1% of the about 30 million annual hospital discharges. The projections based on the 300,000 records are then published in an annual report. If a projection is based on less than 30 records, or has a relative standard error of more than 30 percent, it is not published because of the unreliability of the estimate. If the projection is based on 30-59 records, it is presented but is preceded by an asterisk (*) to indicate that it has a low reliability.

In each of the years 1990-2004, during which there were approximately 4,500,000 hospital discharge records included in the survey, asbestosis was never listed as the principal diagnosis more than
eight times; in some of the years, it was listed zero times. Because the number of patients discharged with a principal diagnosis of asbestosis was below 30 for each of the 15 years, the annual NHDS publication did not list any projections for asbestosis as a First Listed Diagnosis. Indeed, the ICD Code for asbestosis was simply omitted in each of the years 1990-2004 from the tables listing First Listed Diagnosis. By way of comparison, in the period 1990-2003, chronic obstructive pulmonary disease (COPD, ICD-9 Code 496) was projected to be the primary cause of almost 2,000,000 hospitalizations.

The NHDS survey also provides the number of times asbestosis was listed in a second through seventh position. Between 1990 and 2004, the number of diagnoses of asbestosis for each of first through seventh positions per year ranged from 0 to 41. In the 105 diagnosis

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185 The ICD-9-CM code for asbestosis is 501. AM. MED. ASS’N, INTERNATIONAL CLASSIFICATION OF DISEASES, NINTH REVISION, CLINICAL MODIFICATION, PHYSICIAN ICD-9-ICM 2005, at 565 (2004). In the period 1990-2004, the number of annual hospitalizations which were primarily due to asbestosis as generated by the sampling of about 500 hospitals, ranged from 0 to 8. See Emails from Karen A. Lees, MPH, supra note 177. For the 15 year period, the total of First Listed asbestosis diagnoses was 57. Id.


187 For 1990, for example, there were 13 diagnoses of asbestosis listed in second position, 17 in third position, 10 in fourth, 9 in fifth, 0 in sixth, and 1 in seventh, for a total of 50. See Chart, Asbestosis by Year and By Position, in E-mails from Karen A. Lees, MPH, supra note 177
positions in that time frame (7 positions for each of the 15 years), asbestosis diagnoses were 30 or higher on six occasions, ranging from 30 to 41. Projections based on these numbers would have been asterisked to indicate that because they were based on 30-59 records, they had a low reliability. Accordingly, though the NHDS publication does not separately list the number of projected hospital discharge diagnoses for each of positions two through seven, had it done so, it would have omitted asbestosis from those tables on 99 of the 105 projections and asterisked the six projections that would have been published.

In addition to tables listing the projected number of First Listed Diagnoses for all persons discharged from hospitals, the NHDS publication annually lists All-Listed Diagnoses. In the 15 year period, the total of All-Listed Diagnoses of asbestosis derived from the survey ranged from 53 to 158. Projections of the total number of All-Listed Diagnoses of asbestosis based on the survey data ranged from a low of 4,865 in 1990 to a high of 22,441 in 2002. As noted, only six of the 105 data cells had sufficient numbers of asbestosis discharges to generate publishable projections and these six projections would have had a “low validity.”

In addition to the annual NHDS publication, NIOSH periodically publishes the Work-Related Lung Disease Surveillance Report. A table in the report presents the estimated number of hospital discharges with a diagnosis of asbestosis for 1970 to 2000, which ranges from 300 to 20,000. These estimates are based on the NHDS All-Listed Diagnosis projections. A note at the bottom of the table states “NCHS recommends that, in statistical comparisons, estimates of less than 5,000 not be used and that estimates of 5,000 to 10,000 be used with caution.” For the year 2000, the NIOSH Report lists a projected nationwide estimate for discharges with an asbestosis diagnosis as 20,000. This projection is based on raw survey data of 133 discharges. That is, asbestosis was listed on 133 records in the first

(providing the raw and projected data for the number of diagnoses of asbestosis by position). The total for second through seventh positions ranged between 50-72 in the 1990-1995 period, increased to 89-116 in the 1996-1999 period, and increased again in the 2000-2004 period to 111-153. Id.

188 Id.
190 Id. at 15.
191 Id.
192 Id. The NHDS number is 20,223. Id.
through seventh position of the about 300,000 hospital record sample reviewed by NCHS in the year 2000.\textsuperscript{193} The raw survey data indicates that asbestosis as a diagnosis appeared in the following positions:

\begin{table}
\centering
\begin{tabular}{|c|c|}
\hline
Position & Number of Diagnoses of Asbestosis \\
\hline
1 & 6 \\
2 & 17 \\
3 & 18 \\
4 & 27 \\
5 & 23 \\
6 & 22 \\
7 & 20 \\
Total & 133 \\
\hline
\end{tabular}
\caption{Discharge Diagnoses of Asbestosis in the year 2000, in a Sample of Approximately 300,000 Hospital Discharges}
\end{table}

While none of the NHDS data regarding asbestosis as the primary cause of a hospitalization is sufficient to be the basis for projections of the number of national hospital discharges listing asbestosis as the primary cause, the All-Listed Diagnosis data, though averaging fewer than 100 diagnoses a year of asbestosis in each of the seven positions, has some statistical significance.

The definition of the Principal Diagnosis as the condition primarily responsible for causing the admission of the patient to the hospital is specific and the results of the survey indicating a very low number of hospitalizations primarily because of asbestosis is consistent with medical literature on the prevalence of asbestosis. The validity of the data and projections based on that data of the number of discharges in which asbestosis appears in second through seventh position, however,

\textsuperscript{193}In the year 2000, the projected number of patients discharged from hospitals with a diagnosis of asbestosis in any of the first through seventh positions was 20,223. The raw data on which this projection was based was 133 discharge diagnoses of asbestosis in the first through seventh positions. Since there were approximately 300,000 hospital records reviewed and up to seven diagnostic positions per record, a total of approximately 2,100,000 diagnoses were in the sample. A diagnosis of asbestosis in any one of the seven positions would exclude a diagnosis of asbestosis in any of the other six positions. Accordingly while asbestosis could have been one of 2,100,000 diagnoses 14.29\% of the time, the raw data indicated that asbestosis was a listed diagnosis 0.006\% of the time.
is open to question.194

The evidence set forth in this Article assessing the reliability of X-ray readings and diagnoses of asbestosis generated by litigation screenings may provide a plausible basis for questioning the validity of the results projected by the NHDS. The issue posed is whether the published hospital discharge diagnosis projections of asbestosis in the second through seventh positions represent medical judgments or are more a function of litigation screenings. Testing the hypothesis,  

194 The reliability of discharge diagnoses may be subject to a number of influences. See Notes of Interview of the Data Quality Manager at a Major Hospital who is in Charge of Discharge Coding, Nov. 2, 2006 (on file with the author). For example, the actual coding of diagnoses of discharged patients done by hospital personnel upon review of each patient’s medical chart may be affected by whether the diagnosis has associated procedures that are eligible for insurance coverage. See Annlouise R. Assaf et al., Possible Influence of the Prospective Payment System on the Arrangement of Discharge Diagnoses for Coronary Heart Disease, 329 NEW ENG. J. MED. 931 (1993) (finding that changes in the system used for hospital reimbursement may influence the assignment of discharge diagnostic codes, leading to the use of codes that result in higher reimbursement). The study focused on two states, Rhode Island and Massachusetts, because each in 1983 and 1985 respectively, changed from a fee-for-service method of payment to a system under which diagnosis-related groups (DRGs) were used to reimburse hospitals for the care of Medicare patients. The study compared the rates of hospital discharge diagnoses of various forms of coronary heart disease and determined that the frequency of assignment of codes for the acute forms of coronary heart disease (which provided higher reimbursement) rose from 35.2% to 48.4% among discharged patients with cardiac disease after the institution of the DRGs. The study found a trend away from discharge diagnoses with lower reimbursement towards those with higher levels of reimbursement for patients. See also Ark. Dep’t. of Health & Human Servs., Div. of Aging & Adult Servs., Arkansas Senior Medicare/Medicaid Patrol Manual at app. 3, (in collaboration with University of Arkansas at Little Rock), available at http://www.arkansas.gov/dhhs/aging/asmp.html (last visited Apr. 26, 2007) (describing how providers of medical services have a financial incentive to “upcode” (use codes that result in higher payments), or otherwise misrepresent what medical conditions are present). A review of the hospital records of 48 discharged patients in the period 1979-1982 who were diagnosed with extrinsic allergic alveolitis (EAA) indicates a basis for further caution. Based on published criteria for the diagnosis of EAA, only three cases (6%) could be classified as probable EAA, while 10 (22%) were possible cases, and 34 (73%) were not EAA. The study concluded that limitations were apparent in the accuracy of discharge coding and also in the accuracy of the physician’s diagnosis. Howard M. Kipen et al., Limitations of Hospital Discharge Diagnoses for Surveillance of Extrinsic Allergic Alevolitis, 17 AM. J. INDUS. MED. 701 (1990). The study noted, however, that because of the small number of hospital records obtained and reviewed, caution is warranted in generalizing the results. Id. at 705-06.

Another example of how unreliable diagnoses can show up on the abstracts collected by the NHDS is by repetition of an erroneous, never-substantiated diagnosis throughout the patient’s clinical record. See, e.g., Lawrence Martin, Pitfalls in Diagnosing Occupational Lung Disease for Purposes of Compensation—One Physician’s Perspective, www.lakesidpress.com/pulmonary/papers/pitfalls/pitfalls1-7.html (1997). Martin describes how one patient whose physician wrote “R/O [rule out] asbestosis” on a chest X-ray request form had an X-ray report generated which stated that the results were “not typical of asbestosis but cannot rule out that diagnosis.” When the patient was hospitalized, the diagnosis of asbestosis was placed in the record. A claim was made for asbestosis as the cause of death. However, asbestosis was never established, and a review of the records showed it traceable to the single X-ray report.
however, that public health data that is published annually listing asbestosis as a discharge diagnosis has been corrupted by litigation screenings would require a substantial and costly study.\(^{195}\)

A scenario that may result in the generation of misleading data is as follows. (1) The hospitalized patient who is discharged with a diagnosis of asbestosis appearing in the second through seventh position was one of over 700,000 who were recruited to participate in litigation screenings during the period 1988-2004. (2) One of the litigation B Readers read the X-ray taken at a screening as indicating radiographic evidence of fibrosis graded 1/0 or higher on the ILO scale and concluded that this finding was “consistent with asbestosis.” (3) That B Reader, or another litigation doctor, issued a diagnosis of asbestosis “within a reasonable degree of medical certainty.” (4) On that basis, the lawyer that sponsored the screening brought suit against scores of defendants and also claimed against a number of asbestos bankruptcy trusts. (5) Those claims generated a number of settlements grossing $60,000–$100,000 between 1990 and 2000, and a smaller amount thereafter, of which the litigant received about half. (6) Years later, the litigant is hospitalized for heart disease, pneumonia, COPD, or numerous other diseases.\(^{196}\) (7) As part of the admission procedure (or prior thereto, if hospitalized by his family doctor or a surgeon who is to operate), a medical history is taken. (8) The patient states that he was diagnosed with asbestosis (and, as confirmation, received compensation for his illness). (9) This information is recorded in the patient’s medical chart. (10) The chart is selected as one of those discharged patient’s charts to be sampled for the NHDS. (11) Thereafter, hospital or survey personnel go through the chart and assign ICD-9 codes. Finally, (12) the listing of asbestosis in the medical history part of the chart results in

\(^{195}\) Such a study could start by identifying a random sample of persons who participated in asbestos screenings in a given time period, e.g., 1990-2004, who were diagnosed with asbestosis by the litigation doctors. These individuals would then have to be contacted to determine whether they were hospitalized at a later point in time. For those that were hospitalized, their hospital records would have to be reviewed to see if an indication of asbestosis appears in their chart and, if so, what the basis was for that information. For example, was the information elicited from the patient in the course of taking a medical history where the patient was requested to list all diseases? And, if so, did the patient indicate how he learned that he had asbestosis? Finally, it would have to be determined whether, on the basis of the information in the chart, asbestosis would be one of seven listed diagnoses if that chart were included in the annual National Hospital Discharge Survey. In order for the study to have statistical significance, the size of the random sample at the front end would have to be quite substantial in order for back end cohorts to be of sufficient size.

\(^{196}\) Aside from an extensive study as set forth supra note 195, there is no way to determine the likelihood that those who were hospitalized and determined to have a discharge diagnosis of asbestosis had attended a screening and been diagnosed by one of the litigation doctors as having asbestosis.
ICD-9 code 501 being listed as one of the discharge diagnoses in second through seventh position.

F. The Disparity Between the Results of Pulmonary Function Tests Administered in Litigation Screenings and Clinical Settings

A battery of pulmonary function tests (PFTs), if administered correctly, can provide a more objective assessment of the extent of pulmonary fibrosis than can radiographic readings grading opacities on the standardized, but nonetheless somewhat subjective, ILO scale. My research indicates, however, that PFTs are being administered to generate false findings of impairment in order to materially increase the value of the claims. According to medical literature, on average,
few of those screened for asbestosis, whose X-rays are legitimately graded 1/0 and 1/1 on the ILO scale, suffer from lung impairment as measured on the basis of an FVC, FEV1, TLC or DLCO falling below 80% of the predicted value.\(^{200}\) This is so because, according to medical literature, lung impairment measured by the “below 80% standard” usually does not manifest until the interstitial fibrosis is severe enough

\(^{200}\) This is not to state that pulmonary function is not affected when there is evidence of radiographic asbestosis. Studies have shown that as profusion scores increase, spirometry and diffusion capacity decrease. See Albert Miller, Radiographic Readings for Asbestosis: Misuse of Science—Validation of the ILO Classification, 50 AM. J. INDUS. MED. 63 (2007). However, by the “below 80% of predicted value” standard, the studies cited below conclude that the averages for those with ILO readings of 1/0 and 1/1 did not fall below 80% of predicted value.

In a study of 2611 asbestos insulators—one of the largest reported populations occupationally exposed to asbestos in a single trade—none of the mean percentages of insulators with ILO scores of 1/0 (456, 17.5%) and 1/1 (627, 24%), fell below 80% of predicted value on FVC and FEV1/FVC tests. By the “below 80% of predicted value” measure, lung impairment was generally not found until ILO scores were 1/2 or higher. A. Miller et al., Relationship of Pulmonary Function to Radiographic Interstitial Fibrosis in 2,611 Long-term Asbestos Insulators, 145 AM. REV. RESPIRATORY. DISEASE. 263 (1992). Four studies co-authored by Dr. Jay T. Segarra relate ILO profusions with impairment as measured by pulmonary function tests:


Assuming the profusion levels of the 1,305 individuals in the “Two Groups of Building Trades Workers” study found to have “asbestos-related radiographic changes” were 1/0 and 1/1, then the abstracts of these studies indicate that of a total of 11,408 individuals tested for asbestos-related conditions, 48% (5517) were found to have ILO profusions of 1/0 or greater. (This is consistent with the conclusion set forth, supra note 36, analyzing Dr. Segarra’s responses to the subpoenas in the Ragsdale case). The average performance on each of the pulmonary function tests in each of the studies for those with ILO profusion of 1/0 and 1/1 was above 80% of predicted value. Only those with ILOs of 1/2 or higher were, on average, found to have lung impairment by the “below 80%” standard.
to be graded as 1/2 level on the ILO scale. Nonetheless, screening companies that administer PFTs frequently find lung impairment for most of those with X-rays that have been read as 1/0 and 1/1 on the ILO scale. A case in point. N&M provided records in the silica MDL indicating that it administered PFTs to the large majority of the individuals it screened. An examination of “tens of thousands of PFT records from N&M” indicates that “N&M’s testing methods produced positive results (i.e., purportedly showed impairment) in over 75% of the tests.” making those claims eligible for substantially higher compensation. While N&M’s doctors graded 80-95% of the X-rays taken at screenings and put into the silica MDL repository that have been examined, as 1/0 or greater, approximately 90% of N&M’s ILO readings were 1/0 and 1/1. According to the medical literature reviewed above, on average, few of the PFTs of those whose X-rays were graded 1/0 and 1/1 should have resulted in findings of lung

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201 Id.
203 Certain Defendant’s First Amended Supplemental Brief in Response to Plaintiff’s Challenge to the Constitutionality of Florida’s Asbestos and Silica Compensation Fairness Act, supra note 202, at 27.
204 See supra note 199.
205 See supra note 35.
206 Litigation B Readers read the vast majority of the X-rays which they find positive for fibrosis as 1/0 and 1/1. For example, in MDL 1553, Judge Jack discusses the rate of positive X-ray readings of Dr. Ballard, MDL 1553, 398 F. Supp. 2d at 610-11, Dr. Oaks, id. at 619-20, and overall for 6,510 B readings, of which more than 92% were graded 1/0 or 1/1 and less than 2% were graded 2/1 or higher. Id. at 629; See Certain Defendants’ Combined Motion and Brief to Exclude Diagnostic Materials Created by Respiratory Testing Services, Inc., and to Dismiss Claims of Plaintiffs Relying on Same at 8, In re Asbestos Prods. Liab. Litig. (No. VI), Case No. MDL 875 (E.D. Pa., April 3, 2007) (finding that over 93% of individuals screened by RTS and diagnosed for asbestosis had profusions of 1/0 or 1/1). However, because of the progressiveness of some fibroses, in clinical practice the ILO range is much broader with significant percentages of 1/2s, 2/1s and higher, especially among aging populations. See Transcript of Daubert Hearings at 80-86, MDL 1553, 398 F. Supp. 2d 563 (Feb. 18, 2005) (testimony of Dr. John E. Parker). Dr. W. Allen Oaks, who read X-rays for N&M, testified that among a large group of people with silicosis, one would expect to find a greater profusion among older people. However, for the 447 litigants’ X-rays that he read, which, according to Judge Jack, were of a fairly even distribution of people between 50 and 80 years of age, Dr. Oaks found 408 to be 1/0 and 39 to be 1/1. He did not find any with a profusion of greater than 1/1. Dr. John E. Parker, the former administrator of NIOSH’s B Reader program, called this consistency of profusion “stunning,” “def[y]ing all statistical logic and all medical and scientific evidence of what happens to the lung when it’s exposed to workday dust.” MDL 1553, 398 F. Supp. 2d at 619-20. Dr. Parker further stated that “this lack of variability suggests to me that readers are not being intellectually and scientifically honest in their classification.” Id.
impairment by the “below 80% standard.” Nonetheless, N&M-administered PFTs generated a 75% rate of lung impairment.

IV. THE SILICA MDL

Additional evidence on the reliability of X-ray readings done as part of litigation screenings and diagnoses of asbestosis is set forth in Judge Jack’s opinion in the silica MDL.\(^{207}\) In that opinion, Judge Jack substantially corroborated my conclusions regarding the elements of the illegitimate “entrepreneurial” model of asbestos claim generation, including the production of hundreds of thousands of unreliable medical reports. To be sure, Judge Jack’s findings were based on silicosis—an injury caused by exposure to silica.\(^{208}\) However, she was examining the identical “entrepreneurial” claim generation process, including some of the same screening enterprises and the same doctors who had engaged in the identical practices with regard to the generation of claims of asbestosis and the production of medical reports in support of those claims.\(^{209}\) In some cases, diagnoses of both asbestosis and silicosis were generated simultaneously by the same litigation doctor.\(^{210}\) on the
basis of a single X-ray and a cursory review of the individual’s occupational history, largely produced by the law firm that hired the screening company.

The MDL proceeding was the culmination of an “epidemic” of approximately 20,000 silicosis filings, mostly in state courts in Mississippi and Texas, beginning in 2002—an anomalous phenomenon, because as a result of government regulation and industry practice, there had been a 70% decline in the death rate from silicosis over the previous thirty years. The reasons for this “phantom epidemic” are twofold. First, the U.S. Senate began consideration of legislation to provide an administrative alternative to asbestos litigation, which would, inter alia, limit the recovery for non-malignant unimpaired asbestosis claims to medical monitoring expenses. Second, key states, most importantly, Mississippi and Texas, enacted substantial asbestos litigation reform. Worried about the future of claim generation and concerned that the end game had begun for asbestos litigation, some plaintiffs’ lawyers began directing some of the screening enterprises that they had hired, to screen hundreds of thousands of workers exposed to asbestos containing products, to instead screen for silicosis. These screening companies then abruptly shifted gears from ginning up asbestosis claims to silicosis claims.

When evidence surfaced that the X-ray readings and diagnoses of

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211 Morriss & Dudley, supra note 208, at 322-30.
212 Brickman, Silica/Asbestos Litigation, supra note 22, at 41.
214 In re Silica Prods. Liab. Litig. (MDL 1553), 398 F. Supp. 2d 563, 620 (S.D. Tex. 2005); Hearings on Asbestos: Mixed Dust and FELA Issues Before the S. Comm. on the Judiciary, 109th Cong. 8-9 (Feb. 2, 2005) (written statement of Lester Brickman at 8-9 quoting Heath Mason, co-owner of N&M, Inc., who testified that the reason his company changed from asbestos to silica screening is because of the “Hatch Bill”).
215 MDL 1553, 398 F. Supp. 2d at 620 (“One might also focus on the decline in measures in asbestos lawsuits, leaving a network of plaintiffs’ lawyers and screening companies scouting for a new means of support.”).
216 See id. at 597 (“[S]ometime around 2001, law firms began asking the companies to screen people for silicosis.” (citing Transcript of Daubert Hearing at 287, MDL 1553, 398 F. Supp.2d 563 (Feb. 17, 2005)). After N&M had begun screening for silica, Dr. Ray Harron diagnosed 99.69% of 6,350 screenings with abnormalities consistent with silicosis; however, prior to silicosis screening, Dr. Harron diagnosed 1,087 of the same MDL plaintiffs with abnormalities consistent with asbestosis, not silicosis. Id. at 607-08.

In short, when Dr. Harron first examined 1,807 Plaintiffs’ x-rays for asbestos litigation (virtually all done prior to 2000, when mass silica litigation was just a gleam in a lawyer’s eye), he found them all to be consistent only with asbestosis and not with silicosis. But upon re-examining these 1,807 MDL Plaintiffs’ x-rays for silica litigation, Dr. Harron found evidence of silicosis in every case.

Id. at 608
silicosis for the 10,000 claimants may have been fraudulently generated, Judge Jack presided over a Daubert hearing that she ordered to take place to test the reliability of the medical reports produced by a handful of litigation doctors.\textsuperscript{217} In addition, she permitted the defendants to undertake extensive discovery of the doctors and screening companies. Her actions were unprecedented in mass tort litigation. Indeed, most judges, out of reluctance to, in effect, put the tort system on trial, would not have permitted the defendants to conduct the extensive discovery that Judge Jack allowed.\textsuperscript{218} But for the fortuity of Judge Jack’s selection to preside over the MDL, the pervasive fraud that she uncovered would likely never have come to public attention.

Among the evidence of fraud that Judge Jack permitted to be introduced was the revelation that at least 60\% of the silicosis claimants had previously filed asbestosis claims\textsuperscript{219}—a phenomenon that become known as “retreading.”\textsuperscript{220} While it is medically possible for a claimant to have the dual diseases of asbestosis and silicosis, it is a “clinical rarity”\textsuperscript{221}—a medical euphemism for “virtually never.” Indeed, this dual disease phenomenon is so rare that most pulmonologists have

\begin{footnotesize}
\textsuperscript{217} See id.
\textsuperscript{218} Cf. Brickman, Asbestos Litigation, supra note 1, at 164 n.503. Consider, for example, Vicksburg, Mississippi Circuit Court Judge Isadore W. Patrick’s denial of a motion for sanctions against the former firm of O’Quinn, Laminack & Pirtle (now the O’Quinn Law Firm) for allegedly pursuing frivolous silicosis claims on behalf of clients and submitting allegedly unreliable diagnoses to support these claims that were the subject of Judge Jack’s report. Referring to the mass screening process, Judge Patrick held that:

\textit{[O’Quinn, Laminack & Pirtle] relied upon a nationally accepted method used in prior mass tort cases, i.e. mass screenings of persons who potentially may have had a silica claim, due to injuries incurred as a result of exposure to silica. . . . [T]hese mass screenings were conducted by a physician, Dr. Harron, who had obtained a national certification to do such screenings.}

McDuff v. Aearo, No. 02-101, 2006 WL 1970163, at *1 (Miss. Cir. Ct. June 27, 2006); see also Mary Alice Robbins, Mississippi Judge Declines to Sanction O’Quinn, Laminack & Pirtle, TEX. LAW., July 10, 2006, at 7-8. Notably, Judge Patrick did not take cognizance of the state of knowledge in the asbestos and silica litigation industry of Dr. Harron’s reputation for unreliability. See Brickman, Silica/Asbestos Litigation, supra note 22, at 42. Further the “nationally accepted method” of claim generation referred to by Judge Patrick had never been subjected to inquiry because no judge had ever permitted the wide ranging discovery that was required to uncover the fraudulent scheme. Judge Patrick’s ruling is on appeal to the Mississippi Supreme Court. See Silica Defendants Appeal Mississippi Sanctions, COURTROOM NEWS, Sept. 20, 2006, available at http://www.harrismartin.com/article_detail.cfm?articleid=7488.

\textsuperscript{219} MDL 1553, 398 F. Supp. 2d at 628.


\textsuperscript{221} MDL 1553, 398 F. Supp. 2d at 594-96 (collecting doctors’ testimony that, although it is theoretically possible, in their extensive pulmonary practice, none of them had ever seen such a case of dual disease).
\end{footnotesize}
never seen a single such case. “Retreading” was done by having B Readers re-read X-rays previously read as indicating radiographic evidence of fibrosis “consistent with asbestosis,” to generate claims of silicosis. In some cases, the same B Readers were contradicting their own prior readings.222

Other evidence of fraud that was uncovered in the unprecedented discovery permitted by Judge Jack—though only after she repeatedly threatened contempt citations for failure to provide records—was the percentage of “positive” findings of silicosis. As summarized by Judge Jack, over 92% of the 6,510 B reads produced as part of plaintiffs’ initial disclosures were positive.223 Dr. Ray Harron’s rates were simply off the chart with a 99.69% positive rate.224 Commenting on the “positives” rate achieved by N&M, Judge Jack observed:

Overall, N&M—a small Mississippi [screening] company operated without medical oversight—managed to generate the diagnoses for approximately 6,757 MDL Plaintiffs. To place this accomplishment in perspective, in just over two years, N&M found 400 times more silicosis cases than the Mayo Clinic (which sees 250,000 patients a year) treated during the same period.225

The testimony by doctors and screening companies and the records produced in response to subpoenas enforced by threats of contempt led Judge Jack to conclude that “it is apparent that truth and justice had very little to do with these diagnoses . . . . [Indeed] it is clear that the lawyers, doctors and screening companies were all willing participants” in a scheme to “manufacture . . . [diagnoses] for money.”226 “[E]ach lawyer had to know that he or she was filing at least some claims that falsely alleged silicosis.”227 This is the equivalent of a finding of fraud.

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222 Silica MDL Plaintiff Willie Jones was screened at least four times by Dr. Jay T. Segarra: (1) March 14, 2002; (2) September 9, 2002; (3) February 27, 2003; and (4) June 27, 2003. The first and third screenings resulted in silicosis diagnoses by Dr. Segarra, with, in Dr. Segarra’s words, “no radiographic evidence for pulmonary asbestosis.” The second and forth screenings resulted in wholly inconsistent diagnoses of “mixed dust pneumoconiosis (silicosis and asbestosis).” Defendants’ Motion for Production of Pulmonary Diagnoses and Evaluations at 4, In re Tex. State Silica Prods. Liab. Litig., Cause No. 2004-70000 (Tex. Dist. Ct. Apr. 3, 2007).

223 MDL 1553, 398 F. Supp. 2d at 629.
224 Id. at 607-08; see also supra note 217 and infra note 226.
225 MDL 1553, 398 F. Supp. 2d at 603 (citation to record omitted).
226 Id. at 635. Referring specifically to Dr. Ray Harron, who has done over 80,000 B-Reads for asbestos litigation, Judge Jack found that with regard to his silicosis diagnoses, “Dr. Harron [found] evidence of the disease he was currently being paid to find.” Id. at 577.
227 Id. at 636.
A. Dual Diagnoses and the Law Firm of O’Quinn, Laminack & Pirtle

The Law Firm of O’Quinn, Laminack & Pirtle (O’Quinn) was Lead Plaintiffs’ Counsel in the silica MDL, and represented over 2,100 plaintiffs in the proceeding.228 A defense counsel stated during the proceedings that 73% of one group of O’Quinn’s cases had previously filed asbestosis claims.229 In an August 22, 2005 exchange with Judge Jack, Richard Laminack attempted to respond to the overwhelming evidence presented in the MDL that most, if not all, of the dual disease claims were spurious and defend the integrity of his firm’s silicosis claims, and to justify the bona fides of his clients’ silica claims by arguing that though many of his clients had previously filed asbestosis claims, “the explanation on a lot of the cases is the asbestosis diagnosis is wrong.”230 When pressed about the asbestosis claims, Mr. Laminack responded, “I doubt the numbers, and I doubt the diagnosis.”231 Thus, he was contending that his clients were not dual disease claimants because their prior filings of asbestosis claims were based on invalid diagnoses.232

Consistent with this position, Laminack further stated that: “[the firm] never, never represented an asbestos claimant and then turned around and retread it as a silicosis claimant. We never, ever did that.”233 This is belied by the statement of two of the firm’s clients.234

Moreover, as set out below, at least some, if not most, of the asbestosis claims filings that were based upon diagnoses that Mr. Laminack opined were “wrong” were done by or for an affiliated law firm acting in

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228 STAFF OF H. COMM. ON ENERGY & COMMERCE, MEMORANDUM TO THE SUBCOMM. ON OVERSIGHT AND INVESTIGATIONS: 109TH CONG., OVERSIGHT AND INVESTIGATIONS HEARINGS: “THE SILICOSIS STORY: MASS TORT SCREENING AND THE PUBLIC HEALTH” FOURTH DAY OF HEARINGS (Comm. Print July 25, 2006) (on file with author). The Subcommittee on Oversight and Investigations had been investigating the issues presented by Judge Jack in MDL 1553, specifically examining doctors, screening companies, state regulators of radiological medicine, state medical boards, and law firms related to the MDL 1553 litigation, as a case study, to determine the public health issues arising from the use of mass tort screenings to identify claimants for a lawsuit. Id. at 2-3.


230 Id. at 62-63.

231 Id. at 64; see also Jack the Ripper, WALL ST. J., Aug. 31, 2005, at A8.


233 Transcript of Status Conference, supra note 229, at 58-59.

234 Two O’Quinn clients stated to the staff of the Subcommittee on Oversight and Investigations that they were first diagnosed with asbestosis and, some time later, received a letter from the firm telling them that they also had silicosis. MEMORANDUM FROM THE SUBCOMM. ON OVERSIGHT AND INVESTIGATIONS, supra note 228.
conjunction with the O’Quinn firm.

In testimony before the House Subcommittee on Oversight and Investigations, the O’Quinn firm repeated the assertion that it did not retread asbestos claims as silicosis claims and indeed “did not have an asbestos docket.”235 Joseph Gibson, an attorney with the O’Quinn firm, previously stated in an affidavit that he was “aware that some of our clients had Asbestosis diagnoses because during the time our plaintiffs were being tested for Silicosis, some plaintiffs were found to have X-ray findings that were consistent with Asbestosis.”236 He stated that this was the only exception to the O’Quinn firm’s general rule that the “law firm did not have in its possession any records relating to Asbestosis claims that its Silica MDL plaintiffs may or may not have had.”

When a firm sponsored litigation screening generated diagnoses of both asbestosis and silicosis for the same litigant, the firm referred the asbestosis claim to the Foster Law Firm, formerly known as Foster & Harssema,238 and shared in any fees generated by the asbestos case.239

The Foster Law Firm is located at 440 Louisiana, Suite 2100, Houston, Texas. The O’Quinn firm is located at 440 Louisiana, Suite 2300, Houston, Texas.240 The O’Quinn firm had participated in the creation of the Foster firm. It “initially financed the start-up of [the Foster] law firm” in 2001241 and two O’Quinn partners, Mr. O’Quinn and Mr. Laminack, were elected managers of the Foster firm.242

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235 Abel Manji, an attorney with the O’Quinn Law Firm who assumed responsibility for O’Quinn’s silicosis cases after joining the firm in May 2005, was designated to be a witness at the Oversight and Investigations Hearing on July 26, 2006. He testified that the O’Quinn Firm did not “engage in the practice of retreading old asbestos cases into new silicosis cases, in fact, the O’Quinn Firm did not have an asbestos docket.” The Silicosis Story: Mass Tort Screening and the Public Health: Hearings Before the Subcommittee on Oversight and Investigations of the H. Comm. on Energy and Commerce, 109th Cong., at 384 (2006) (testimony of Abel Manji) [hereinafter Hearings].

236 Affidavit of Joseph Gibson, MDL 1553, 398 F. Supp. 2d 563 (Mar. 9, 2005.)
237 Id.
238 Id. Gibson further stated that the O’Quinn Law Firm had handled some asbestosis cases directly, but the vast majority were referred to Ryan Foster. Id.
239 Hearings, supra note 235, at 423-24 (testimony of Richard Laminack). Laminack testified that the O’Quinn Firm and the Foster Firm had a “referral arrangement,” whereby the O’Quinn Firm earned a referral fee for every successful asbestosis claim they sent to the Foster firm. Id.
241 Hearings, supra note 235, at 423 (testimony of Richard Laminack).
242 Id. In response to a question from Rep. Walden, “[a]nd, are you an officer, or director, or have you ever been, of the Foster Law Firm?,” Laminack answered:

Well, when it was originally set up, it was set up to have three managers, I was
2002 to 2005, two of three managers and directors of the Foster firm were members of the O’Quinn firm, including variously Mr. Laminack, Mr. O’Quinn, and Mr. Pirtle.\textsuperscript{243}

The relationship between the O’Quinn and Foster law firms is made manifest by the process the firms followed in generating litigants. For example, both the O’Quinn and Foster firms hired N&M, Inc.\textsuperscript{244} to perform screenings. These screenings for the firms generated one X-ray and one physical examination per litigant.\textsuperscript{245} N&M hired Dr. Ray Harron\textsuperscript{246} to read the X-rays and perform the diagnosing.\textsuperscript{247} Dr. Harron’s typical X-ray impression read “bilateral interstitial fibrosis consistent with asbestosis, silicosis and coal workers pneumoconiosis.”\textsuperscript{248} Under instructions from the O’Quinn firm, where there were dual diagnoses, Dr. Harron then prepared two separate letters, one stating a diagnosis of asbestosis and the other of silicosis.\textsuperscript{249}
The asbestosis diagnosis letter was sent to the Foster firm and the silicosis diagnosis letter was sent to the O’Quinn firm. As noted, the O’Quinn firm shared in the fees generated by the asbestosis claim. As further noted, Laminack had testified that the O’Quinn firm’s silicosis claims were genuine even where there also had been a diagnosis of asbestosis for the same claimant because “the asbestosis diagnosis is wrong.”

V. THE REFUSAL TO PROVIDE SCREENING RECORDS AS EVIDENCE OF PREDETERMINED PERCENTAGES OF POSITIVE X-RAY READINGS AND DIAGNOSES

The evidence reviewed in this Article, including (1) the prevalence of radiographic findings of fibrosis and diagnoses of asbestosis found by litigation screenings as compared to clinical studies, (2) clinical re-readings of litigation B Readers’ prevalence percentages, (3) the number of annual hospitalizations primarily because of asbestosis, and (4) the results of pulmonary function tests administered by screening companies, leads inexorably to the identical conclusion reached by Judge Jack in the silica MDL: the medical reports are manufactured for money.

Moreover, the B Readers, diagnosing doctors and screening companies involved in litigation screenings appear to have predetermined percentages of “positive” findings irrespective of the X-rays or files they are reviewing or PFT tests they are administering. Indeed, this appears to be the “product” they are selling to lawyers. If a fairly significant fact to leave out of a diagnoses letter? [Mr. Laminack:] Well, with all due respect congressman, what you are looking at is a partial document, the letter your looking at was attached to a package of four documents that included the exact findings from the B-read and the exact medical history, and in the case where there was a dual diagnosis, that information was clearly stated in the B-read information and in the medical history. So, if the implication is that somebody was trying to hide the fact, that’s simply not true. That letter, the package contained all the details of the dual diagnosis.”

Laminack stated that the O’Quinn Firm insisted that there be two letters separating the diagnoses because “our firm doesn’t handle asbestos cases.”

Id.

250 Id. Heath Mason explained that the same law firm “had two sets of lawyers... for this particular thing—one to handle their silica exposure, one to handle their asbestos exposure.” Transcript of Daubert Hearings at 400, MDL 1553, 398 F. Supp. 2d 563 (Feb. 17, 2005).

251 See supra note 239.

252 See supra note 230.

253 There is also evidence that law firms have “signature” percentages of positive X-ray readings and diagnosis that they demand that doctors and screening companies adhere to. In the audit undertaken by the Manville Trust, see Brickman, Asbestos Litigation, supra note 1, at 128, the failure rate of a given B Reader often varied significantly depending on which law firms were employing the B Reader. See Houser Affidavit, supra note 98, ¶ 27. In fact, biostatisticians from
such a determination were to be made, it could be “smoking gun” evidence of fraud that would not only subject these doctors’ findings to challenge, but also expose them and the screening companies to possible criminal prosecution. If “signature” percentages of fibrosis and asbestosis were the actual product that doctors and screening companies were selling to lawyers, we would expect that these doctors and screening companies would go to great lengths to avoid disclosing information that would enable computation of their positive rates of finding radiographic evidence of fibrosis and diagnosing asbestosis. This may explain why, outside of the silica MDL, where Judge Jack utilized the full powers of her office to overcome resistance to the production of the subpoenaed records, and MDL 875 where Judge Giles has allowed some discovery of RTS’s record, B Readers, and other doctors and screening company representatives who are deposed and subpoenaed to produce records of all of their X-ray readings, diagnoses, and PFT tests, and not just those for the litigants in that case—records which would enable a determination of their total percent “positives”—move to quash subpoenas for these records and otherwise simply refuse to comply. In addition, leading plaintiffs’ law firms, understanding

Pennsylvania State University and the University of Pennsylvania, who were commissioned by the Manville Trust to assist with the analysis of the audit data, concluded that the identity of the particular law firm that submitted any given claim was a “strikingly significant predictor” of whether that claim would fail the audit, and that those findings exhibited “huge levels of statistical significance.” Localio Report, supra note 99, at 18.

254 See, e.g., Response and Brief in Support of Response of Jay Segarra, M.D., to Defendants’ Combined Motion and Brief to Quash Response to Subpoena to Jay Segarra, M.D. and Combined Motion and Brief in Support of Motion of Jay Segarra, M.D., to Quash or, in the Alternative, Modify Subpoena to Jay Segarra, M.D., In re Asbestos Prods. Liab. Litig. (No. VI), MDL No. 875 (E.D. Pa. Sept. 13, 2006). This motion requested that the court modify defendants’ subpoena for the records of all of Dr. Segarra’s X-ray readings and diagnoses done for asbestos litigation purposes, which defendants argue is needed “because analysis of Dr. Segarra’s pattern and practice will help the Court to determine whether his diagnoses . . . are reliable,” id. at 2, acknowledging that he has been the primary diagnosing doctor for 23,200 asbestos claims submitted to the Manville Trust, id. at 3-4, asserting that “[t]he one thing the Defendants do not have [and cannot have] are copies of Dr. Segarra’s negative reports,” id. at 4 (emphasis in original), and seeking to limit the subpoena to just the diagnoses in the cases before the MDL court. Letter from Daniel J. Mulholland, Forman Perry Watkins Krutz & Tardy LLP, to X.M. Frascogna, Jr., Special Master, Fairley v. Pulmosan Safety Equip. Co., Civ. Action No. CI-2004-001-SI (Miss. Cir. Ct. Feb. 8, 2006) (detailing Dr. Jay Segarra’s repeated and adamant refusals in one matter to produce subpoenaed data that he acknowledged that he kept that would allow calculation of his percent “positives”).

Other litigation doctors similarly refuse to provide subpoenaed records that would allow calculation of their percent positives. See, e.g., Defendants’ Brief in Response to Dr. Schonfeld’s Opposition to Motion for Evidentiary Hearing, In re All Asbestos Cases Special Docket No. 073958 (Ohio C.P. Feb. 3, 2006) (detailing Dr. Schonfeld’s opposition to the defendants’ motion for an evidentiary hearing concerning the sufficiency of the medical evidentiary support offered by the plaintiffs pursuant to the court’s prior case management order dealing with 35,000 pending
what is at stake, vigorously oppose efforts to subpoena the records of the litigation doctors. Some of the litigation doctors as well as two screening company principals have pled their Fifth Amendment right against self-incrimination as a basis for refusing to testify and produce records. The implications of doctors refusing to testify about their X-asbestos cases.) Dr. Schonfeld argues, \textit{inter alia}, that his production of certain requested documents would violate the Health Insurance Portability and Accountability Act (HIPAA). Dr. Schonfeld has previously testified that the persons he examines, however, are not his “patients,” that he provides no treatment or follow-up care, and that he is not their doctor. See also Certain Defendants’ Combined Motion and Brief to Compel Dr. Alvin J. Schonfeld’s Response to Subpoena, \textit{In re Asbestos Prods. Liab. Litig.}, (No. VI), MDL No. 875 (E.D. Pa. Oct. 27, 2006) (detailing the extensive history of Dr. Schonfeld’s opposition to producing his records pursuant to a prior court order authorizing discovery into the screening process); Motion to Quash Deposition Subpoena, or in the Alternative, Motion for Protective Order and Memorandum of Law in Support Thereof at 3-4, \textit{In re Deposition Subpoena Served upon James W. Ballard M.D., Lawrence v. Chesteron, Case No. CIV-2000-73-2} (Ala. Cir. Ct. Feb. 16, 2007) (seeking to quash a subpoena for Dr. Ballard’s testimony because it imposes a burden on him, as he will have to otherwise invoke his Fifth Amendment right against self-incrimination, which “could be the subject of adverse comment throughout further \textit{[civil] proceedings},” and thus reduce the commercial value of Dr. Ballard’s diagnoses, and acknowledging that Dr. Ballard is believed to be a subject of the grand jury investigation being conducted by the U.S. Attorney for the Southern District of New York); Additional Brief in Opposition to Dr. Ballard’s Motion to Quash the W.R. Grace Subpoena, \textit{In re Deposition Subpoena Served Upon James W. Ballard, M.D., In re W.R. Grace & Co., 315 B.R. 353} (Bankr. D. Del. Feb. 26, 2006) (No. 01-1139 (JFK)); see also Brickman, \textit{Asbestos Litigation}, supra note 1, at 84-86. 255 See, e.g., Plaintiffs’ Motion to Quash the Subpoenas Served by Forman Perry Upon Various Diagnosing Physicians and Entities, \textit{In re Asbestos Products Liability Litigation} (No. VI), MDL Docket No. 875 (E.D. Pa. March 23, 2007); Response to Defendants’ Motion for Production of Pulmonary Diagnoses and Evaluations, \textit{In re: Texas State Silica Prod. Liab.}, Master Docket No. 2004-7000 (Tex. Dist. Ct. Apr. 13, 2007). 256 Doctors Ray Harron, Andrew Harron and James Ballard, between them responsible for more than 4,000 diagnoses of silicosis, were subpoenaed to appear before the House Energy and Commerce Subcommittee on Oversight and Investigations; each invoked their Fifth Amendment rights in declining to respond to this question asked by Subcommittee Chairman Ed Whitfield: “Will you certify that each of these diagnoses and all others that you made in this litigation are accurate and made pursuant to all medical practices, standards and ethics?” House Committee on Energy and Commerce, Press Release, \textit{Doctors Refuse to Testify at Silicosis Hearing; Others Recount Diagnoses ‘Manufactured for Money,’} Mar. 9, 2006, available at http://republicans.energycommerce.house.gov/108/News/03092006.1810.htm. In addition, Dr. Todd Coulter, who was responsible for 237 diagnoses in MDL 1553, all done for Occupational Diagnostics, a screening company, “took the Fifth” and declined to testify before the House subcommittee. \textit{Hearings, supra note 235, at 436} (testimony of Dr. H. Todd Coulter); see also \textit{Silicosis Clam-Up, WALL ST. J.,} Mar. 13, 2006, at A18; \textit{supra} note 107. Dr. James W. Ballard also invoked his Fifth Amendment privilege and refused to answer a variety of questions about his medical opinion in a civil proceeding. See Deposition of James W. Ballard, \textit{In re W.R. Grace & Co., 315 B.R. 353} (Bankr. D. Del. Feb. 22, 2007) (Civ. Action No. 01-1139); see also \textit{supra} note 107. Charles Foster, the owner of Respiratory Testing Services, also “took the Fifth” before the House Subcommittee concerning the MDL 1553 silica cases. \textit{Hearings, supra note 235, at 264} (testimony of Charles Foster), and did so again during the entirety of his deposition on asbestos claims, in the W.R. Grace bankruptcy. See Deposition of Charles Foster at 8, \textit{In re W.R. Grace & Co., 315 B.R. 353} (Oct. 27, 2006). Health Mason, the co-owner of N&M, Inc., the screening company that accounted for the bulk of the silicosis claims that were included in the
ray readings and diagnoses on the grounds that that testimony may tend to incriminate them notwithstanding, the Fifth Amendment protection does not generally extend to doctors’ and screening company’s records. While it remains uncertain whether all of the records that have been subpoenaed will be produced, it is of critical importance for a full and final determination of whether hundreds of thousands of diagnoses have been “manufactured for money,” that these records be preserved.

silica MDL, see supra notes 35 and 226, invoked his Fifth Amendment privilege against self-incrimination in response to each substantive question posed to him. See Deposition of Charlie Health Mason, In re W.R. Grace et al., In re W.R. Grace & Co. 315 B.R. 353 (Feb. 27, 2007).

Unlike in criminal cases, a witness “taking the Fifth” in a civil proceeding can give rise to a negative inference that the answer would be disadvantageous. See, e.g., Baxter v. Palmigiano, 425 U.S. 308, 318 (1976); Libuti v. United States, 107 F.3d 110, 120-25 (2d. Cir. 1997). In addition, the invocation of the Fifth Amendment by the principals of two screening companies, N&M and RTS, has significant implications for the admissibility of the medical reports of the litigation doctors that these two screening companies used. N&M and RTS have accounted for approximately 60,000-70,000 asbestos litigants. The doctors they hired to read the X-rays and provide diagnoses include Ray Harron, Andrew Harron, George Martindale, Jay Segarra, Walter Allen Oaks, Jose Roman-Candelaria, Paul Venizelos, Dominic Gaziano, Robert Altmeyer and Alvin Schonfeld. Just six of these doctors (Harron, Segarra, Venizelos, Gaziano, Altmeyer, and Schonfeld) have accounted for 206,794 medical reports submitted to the Manville Trust. See CRMC Response, supra note 19, at ques. 14(a) and 14(c). These litigation doctors relied on the X-rays and in many cases, the PFTs that the screening companies administered to provide their diagnoses. Since the administrators of those X-rays and PFTs have “taken the fifth” with regard to all matters relating to their screening practices, that raises the issues of whether the medical reports can be properly authenticated for purposes of admission and whether the methodologies used by these doctors are reliable. See Fed. R. Evid 702 & 703, and their state counterparts.

U.S. District Court Judge James Giles, presiding over MDL 875, has reached a similar conclusion, finding that the medical reports generated by asbestos litigation screenings “lack reliability and accountability” and are “inherently suspicious as to their reliability.” See supra note 8.

The privilege is to protect against compulsory incrimination through one’s own testimony or personal records. . . The privilege may not be based on incrimination resulting from the contents or nature of the thing demanded. (Moreover, records normally kept or required to be maintained by law or under professional rules are not privileged.). The U.S. Supreme Court has held that “[i]t is also clear that the Fifth Amendment does not independently proscribe the compelled production of every sort of incriminating evidence but applies only when the accused is compelled to make a Testimonial Communication that is incriminating.” Fisher v. United States, 425 U.S. 391, 406 (1976); see also United States v. Hubell, 530 U.S. 27 (2000) (stating that a person cannot avoid producing subpoenaed documents merely because they contained incriminating evidence and defining communications that are “testimonial” in character and therefore are protected). The issue of whether the Fifth Amendment protection against self-incrimination extends to records is complex and the very limited discussion in this footnote is not being offered as anything more than an introductory note.

See Certain Defendants’ Emergency Motion For Temporary Restraining Order And Any Other Relief The Court Deems Proper, In re Asbestos Prods. Liab. Litig. (No. VI), MDL Docket No. 875 (E.D. Pa. Jan. 9, 2007) (seeking an order prohibiting Dr. Alvin Schonfeld from continuing to periodically destroy his records). It is undoubtedly the case that copies of at least most of the records of screenings including X-rays ILO reports and diagnoses are in the
CONCLUSION

A review of the evidence emerging from a search of the files in the depository created by Judge Jack for documentary evidence obtained during the course of discovery in the silica MDL, as well as other evidence, permits an assessment of the reliability of X-rays readings and diagnoses of asbestosis and silicosis generated in the course of litigation screenings. Litigation screenings have accounted for substantially all of the 585,000 nonmalignant claims filed with the Manville Trust between 1988 and 2006. Under the illegitimate “entrepreneurial” model, a comparative handful of doctors, numbering approximately 25, have accounted for the majority of the hundreds of thousands of medical reports generated by litigation screenings.

Perhaps the single most important finding presented is the rate of positive readings of X-rays by these litigation doctors. On the basis of the evidence reviewed in this Article, I estimate that the litigation doctors read 50%-90% of the X-rays generated by litigation screenings as indicating radiographic evidence of fibrosis graded 1/0 or higher on the ILO scale, which they find are “consistent with asbestosis.” In addition, I estimate that 80% or more of this group are then diagnosed with asbestosis “within a reasonable degree of medical certainty.” Because “failed” X-rays and diagnoses are reread and rediagnosed by other litigation doctors, it is likely that the actual rates of positive X-ray readings and diagnoses are higher.

A review of clinical studies indicates that the prevalence of radiographic evidence of fibrosis in populations occupationally exposed to asbestos is approximately 11.56%. A number of reasons are advanced for why this prevalence range may overstate the percentage of radiographic findings fibroses identified in the clinical studies. Even if the clinical studies’ prevalence range is not discounted for overreading, the prevalence range cannot be directly compared to that of the litigation doctors. There are more than 100 possible causes of radiographic evidence of fibrosis other than exposure to asbestos, including old age, obesity, and smoking. Moreover, most of the clinical studies did not specifically find that the opacities that they graded as 1/0 or higher were “consistent with asbestosis.”

Two clinical studies and one court ordered “study” indicate that 15-23% of those occupationally exposed workers identified as having radiographic evidence of fibrosis were diagnosed with asbestosis. Litigation doctors, however, diagnose 80% or more of those with X-
rays graded at 1/0 or higher with asbestosis. Even this simple comparison does not fully capture the degree of disparity. If litigation doctors had screened the 18,943 Finnish workers occupationally exposed to asbestos that were the subject of one of the clinical studies, they would likely have diagnosed approximately 7,500 to 10,500 with asbestosis, compared to the 124 actually diagnosed with asbestosis in the clinical setting, and compared to the approximately 560 that the review of the clinical studies suggests.

Further evidence of the unreliability of the medical reports generated by litigation screenings is set forth in a review of seven clinical studies or their equivalent, which re-read X-rays initially read by litigation doctors as 1/0 or higher. Included in the seven studies is the Henry Study which confirmed the results of the Gitlin Study, finding that the litigation B Readers’ error rate was approximately 91%. In toto, the seven clinical re-readings or their equivalent indicated error rates for the initial readings ranging from 60-97%.

Another comparison, which affords considerable insight into the validity of the medical reports generated by litigation screenings, is the ratio of pleural plaques to pulmonary fibrosis found in clinical studies (2:1 to 3:1) versus the 0.2:1 ratio found in litigation screenings after a global settlement significantly reduced the value of future pleural plaque claims.

Evidence that the litigation doctors have a predetermined percentage of positive X-ray readings and diagnoses which they do not wish to disclose has also been reviewed. This includes a detailed description of the repeated refusals of several of the litigation doctors to provide subpoenaed records including all of the medical reports they issued for persons who were recruited to attend litigation screenings. Providing these records, in some cases, would enable their percentage of positive X-ray readings and diagnoses to be determined. These refusals are circumstantial evidence that their medical reports are at least suspect if not fraudulent. So too is the invocation of the Fifth Amendment by four of the litigation doctors as the basis for refusing to testify about their diagnoses and communications with screening company principals. Charles Foster, head of the RTS screening company and Health Mason, head of the N&M screening company, also invoked the Fifth Amendment and refused to testify in civil proceedings about the screenings they conducted. These two screening companies have accounted for 60,000-70,000 asbestos claims and have principally used ten of the most prolific litigation B Readers to read X-rays and

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260 See supra note 121.
issue diagnoses.\textsuperscript{261}

Pulmonary function test results have also been reviewed. PFTs done in the course of litigation screenings are often under the supervision of one of the litigation doctors or for their use in issuing diagnoses. A comparison of PFT results generated by litigation screenings and the results of clinical studies indicates an even greater disparity than that between clinical studies and the litigation doctors’ X-ray readings and diagnoses of asbestosis.

The reliability of the prevalence of radiographic evidence of fibrosis and of asbestosis found by the litigation doctors is further undermined by medical literature which states that by 1990, new cases of asbestosis had largely disappeared and by data assembled by the National Center for Health Statistics for the National Hospital Discharge Survey (NHDS). The evidence reviewed is that in the 15-year period between 1990 and 2004, the NHDS examined an approximately 1% sample of hospital discharges, amounting to approximately 4,500,000 hospital discharge records. It found that of this number, a total of 57 patients had been hospitalized primarily because of asbestosis. Because this number, which ranged from 0 to 8 for each of the 15 years, is so small, the annual NHDS does not list any projections for asbestosis as a “First Listed Diagnoses.”

Finally, I summarized some of the evidence that U.S. District Court Judge Janis Jack reviewed in her detailed opinion in the silica MDL. Judge Jack’s findings of a “phantom silicosis epidemic” and the methods of generating false medical reports largely corroborated my own findings that I had published a year earlier with regard to asbestos litigation. Judge Jack found that the litigation doctors in the silica MDL had graded over 92% of the 6,510 B reads produced as part of the plaintiffs’ initial disclosures as positive. Among the evidence that led Judge Jack to conclude that virtually all of the medical reports were unreliable was the revelation that 60-70% of the silicosis claimants had previously filed asbestosis claims. Medical literature and testimony is that such a dual disease is a “clinical rarity” and virtually never seen by practicing pulmonologists. Retreading asbestosis claims as silicosis is not only evidence that the diagnoses of silicosis were unreliable but also that diagnoses of asbestosis in these same cases were equally unreliable.

Judge Jack’s conclusion is unprecedented in the annals of judicial decision-making:

[I]t is apparent that truth and justice had very little to do with these diagnoses. . . . [Indeed,] it is clear that the lawyers, doctors and

\textsuperscript{261} See supra note 254.
screening companies were all willing participants... [in a] scheme... to manufacture... [diagnoses] for money.\textsuperscript{262}

The evidence reviewed in this Article indicates that Judge Jack’s findings with respect to silica litigation, applies with at least equal force to nonmalignant asbestos litigation: the diagnoses are mostly manufactured for money.

APPENDIX
APPENDIX

85 clinical studies were identified for the purposes of this review; 16 were ultimately excluded. Of the 69 included, 11 were of unexposed populations and 58 were of exposed populations. Of the 58 exposed studies, 8 were of insulators.

A. A Review of 58 Clinical Studies of Exposed Populations

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>STUDY</th>
<th>STUDY GROUP &amp; EXPOSURE DETAILS</th>
<th>N</th>
<th>I/0 OR HIGHER</th>
<th>OPACITY TYPE; LANGUAGE USED</th>
<th>DETAIL</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. Albin, et al.</td>
<td>Chest X-ray Films From Construction Workers: International Labour Office (ILO 1980) Classification Compared With Routine Readings, 49 BRIT. J. INDUS. MED. 862 (1992)</td>
<td>Construction workers</td>
<td>484</td>
<td>258 (53.5%)</td>
<td>Profusion of small opacities</td>
<td>The authors studied the extent of agreement between ILO classifications and clinical readings of chest X-rays of construction workers. While the number of 1/0 readings are identified, the study does not consider a reading of under 1/1 as indicating fibrosis. The X-rays were read by a panel of 15 readers. Of the 210 subjects found to be in the 1/1 category, only 41 (20%) were found to have pneumoconiosis. Thus, 80% of those graded 1/1 were found not to have any pneumoconiosis.</td>
</tr>
<tr>
<td>S. Barnhart et al.</td>
<td>The CARET Asbestos-Exposed Cohort: Baseline Characteristics and Comparison to Other Asbestos-Exposed Cohorts, 32 AM. J. INDUS. MED. 573 (1998)</td>
<td>Individuals with a mean latency of 35 years and a mean of 27 years asbestos exposure</td>
<td>4,060</td>
<td>1,583 (38.9%)</td>
<td>Small irregular opacities</td>
<td>To be included in the study the subjects had to be: (1) 45-69 yrs. old; (2) smokers who quit within 15 years before the study; and (3) exposed to asbestos beginning at least 15 years before the study. Asbestos exposure was defined as having (a) worked in a trade known to be at high risk of asbestos exposure</td>
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for a minimum of 5 yrs. at least 10 yrs. previously; or (b) a chest x-ray that was read as indicating small irregular opacities graded 1/0 or higher, or pleural thickening, or calcifications, and an occupational history consistent with substantial asbestos exposure. Of those studied, 34% were eligible because of a high risk trade; 21% were eligible only because of a positive radiograph; 44% were eligible because of both. The X-rays were read by one reader.


Asbestos textile factory workers working for at least 10 years 379 88 (23.2%) Small opacities The study averaged scores of 4 readers; if the average was halfway between two adjacent categories, the reading was rounded downwards.


Insulators who were working in 1990; subjects were all 35 to 55 years old 110 11 (10%) Small irregular opacities, such as would be consistent with asbestosis. Parenchymal fibrosis The X-rays were read by two B Readers.


Elevator construction workers, averaging 27 years on the job 91 0 N/A Though no parenchymal abnormalities were found, 20 (22%) of those studied had pleural abnormalities. The X-rays were read by three B Readers.

M.J. Campbell  Analysis of a Follow-up Study, An Example from Information not provided directly in this 171 84 (49.1%) N/A The original study, which was conducted between 1965 and 1966, had 252
<table>
<thead>
<tr>
<th>Authors</th>
<th>Title</th>
<th>Follow-up Study Details</th>
<th>X-ray Grading System Used</th>
<th>Subjects, But Had Not Used the ILO System to Classify X-ray Abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.H.M. Langlands</td>
<td><em>Asbestos-Exposed Insulation Workers</em>, 8 SCANDINAVIAN J. WORK, ENV'T. &amp; HEALTH 43 (1982)</td>
<td>Follow-up study; original study detailed that of the 252 studied in 1965-1966, 37% were insulators for more than 20 years; in the follow-up study, these men had been insulators for greater than 30 years, and a larger percentage were already working for more than 20 years</td>
<td>N/A</td>
<td>This study demonstrated that a reduction in FVC and FEV&lt;sub&gt;1&lt;/sub&gt;, which were associated with an increased cumulative dose of exposure, preceded clinical and radiographic abnormalities.</td>
</tr>
<tr>
<td>C-R Chen et al.</td>
<td>Occupational Exposure and Respiratory Morbidity Among Asbestos Workers in Taiwan, 91 J. FORMOSAN MED. ASSOC. 1138 (1992)</td>
<td>Workers in 35 asbestos-related factories in Taiwan; 21 were involved in manufacturing asbestos cement; 10 were involved in friction material; 1 was involved in textiles; 1 was involved in insulation; mean age was 41.6 years; average time of exposure was 8.1 years</td>
<td>459</td>
<td>Each X-ray was read by three chest physicians. The study found no cases of asbestos-related lung disease. “This study demonstrated that a reduction in FVC and FEV&lt;sub&gt;1&lt;/sub&gt;, which were associated with an increased cumulative dose of exposure, preceded clinical and radiographic abnormalities.” 91 J. FORMOSAN MED. ASSOC. at 1141.</td>
</tr>
<tr>
<td>S. Cordier et al.</td>
<td>Epidemiologic Investigation of Respiratory Effects Related to Environmental Exposure to Asbestos Inside Insulated Buildings, 42 ARCHIVES ENVTL. HEALTH 303 (1987)</td>
<td>Electricians, plumbers, cleaners, working in direct contact with asbestos from insulated pipes and closets; out of 224 individuals studied, 146 had more than</td>
<td>224</td>
<td>The study included exposed and unexposed populations, which were separated for this analysis. One of the two readers graded 31 X-rays with an ILO reading of 1/0 or higher, and the other found 36. The average of the two was used, rounding up.</td>
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<tr>
<td>Author(s)</td>
<td>Title</td>
<td>Reference</td>
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<tr>
<td>G. L. Delclos et al.</td>
<td><em>Interobserver Variability Using the ILO (1980) Classification in Subjects Referred for Compensation Evaluation</em>, in <em>Proceedings of the VII Int'l Pneumoconioses Conference</em>, DHHS (NIOSH) Pub. No. 90-108, at 960-64 (1990)</td>
<td>Of those included, 417 had asbestos exposure, 52 had asbestos and silica exposure; the average time since first exposure to asbestos was 32.6 years, and average time in the trade was 27.9 years</td>
<td>10 years pass since their first exposure; 42 had 5-9 years pass; 27 had 0-4 yrs. pass; in 9 instances, the time since first exposure was unknown</td>
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<tr>
<td>J. M. Dement et al.</td>
<td><em>Surveillance of Respiratory Diseases Among Construction and Trade Workers at Department of Energy Nuclear Sites</em>, 43 Am. J. Indus. Med. 559 (2003)</td>
<td>Workers at the DOE. 17+ categories of construction and trade workers; most were pipefitters, electricians, laborers, and carpenters; average time in trade was not available for all the workers; for those for whom the data existed, the average was 26.4 years in the trade</td>
<td>There were three subsets of workers divided by site, and each site had its own B Reader.</td>
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<td>R.Y. Demers et al.</td>
<td><em>Asbestos-Related Pulmonary Disease in Boilermakers/Welders who averaged 18 years</em></td>
<td>Boilermakers/Welders who averaged 18 years</td>
<td>All of the interstitial abnormalities were of the type consistent with exposure to asbestos.</td>
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<td>In this study, two Readers each read independently. The authors included...</td>
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<td>A. Fischbein et al.</td>
<td>Drywall Construction and Asbestos Exposure</td>
<td>40 AM. INDUS. HYGEINE ASSOC. 402 (1979)</td>
<td>Drywall construction trade workers; 19 workers had 0-9 years since the onset of exposure, 28 workers had 10-14 years, 31 workers had 15-19 years, 26 workers had 20-24 years, 10 workers had over 26 years; thus, 83.3% were in the 10+ category. Readings were abnormal if irregular opacities were present (s or t ≥ 1/0). Five readers read the results, which were recorded once a consensus was reached. Results were separated out by years of exposure: (1) 0-9: 5 positive; (2) 10-19: 22 positive; (3) 20-35: 18 positive. The authors note that the &quot;prevalence of asbestosis in this population of drywall tapers is similar to that found by others among asbestos insulation workers.&quot; 40 AM. INDUS. HYGEINE ASSOC. at 407 (footnote omitted).</td>
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<tr>
<td>A. Fischbein et al.</td>
<td>Respiratory Findings Among Ironworkers: Results From a Clinical Survey in the New York Metropolitan Area and Identification of Health Hazards From Asbestos</td>
<td>48 BRIT. J. INDUS. MED. 404 (1991)</td>
<td>Ironworkers employed at construction sites, but not routinely using asbestos products; 62.3% had been in the trade for over 20 years; 22.8% had been in the trade 10-20 years; 14.4% had been in the trade for under 10 years, making the average time in trade 22.9 years; mean latency was 25.7 years. Of the 62 individuals with readings of 1/0 or higher, (1) 17 had readings of 1/1 or higher; (2) 5 had concomitant radiographic features consistent with emphysema; (3) 33 also exhibited pleural changes; (4) 60 had worked for 20 years or more. The &quot;[r]adiographic abnormalities . . . [were] consistent with asbestos associated effects.&quot; 48 BRIT. J. INDUS. MED. at 409.</td>
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<td>E.A. Gaensler &amp; A.M. Goff</td>
<td>Asbestos-Related Disease in Crocidolite and Chrysotile Filter Paper Plants, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 397 (1990)</td>
<td>Workers at two small, old paper mills engaged in the manufacture of specialty and filter papers (Mixing asbestos with cellulose, making cigarette filters); 67 persons exposed to chrysotile; 136 persons exposed to crocidolite</td>
<td>203</td>
<td>77 (37.9%)</td>
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<td>E.A. Gaensler et al.</td>
<td>Radiographic Progression of Asbestosis With or Without Continued Exposure, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 386 (1990)</td>
<td>Six sets of workers were studied: shipyard; filter paper manufacturing plant; gasket manufacturing plant; electrical insulation manufacturing plant; insulation board manufacturing plant</td>
<td>1,764</td>
<td>254 (14.4%)</td>
</tr>
<tr>
<td>M. Garcia-Closas et al.</td>
<td>Asbestos-Related Diseases in Construction Carpenters, 27 AM. J. INDUS. MED. 115 (1995)</td>
<td>Construction carpenters (506); Millwrights (55); other jobs such as welding, painting, and ship repair; average time in trade for the 20 individuals with X-rays graded at 1/0 or higher</td>
<td>631</td>
<td>20 (3.2%)</td>
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<td>Description</td>
<td>Methodology</td>
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<td>J. Gitlin et al.</td>
<td><em>Comparison of ‘B’ Readers’ Interpretations of Chest Radiographs for Asbestos Related Changes</em>, 11 ACAD. RADIOLOGY 843 (2004)</td>
<td>Chest radiographs previously interpreted by physicians retained by attorneys representing persons alleging respiratory changes due to exposure to asbestos</td>
<td>492 readings graded at 1/0 or higher</td>
<td>Small opacities; parenchymal abnormality</td>
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<tr>
<td>B. Hilt et al.</td>
<td><em>Chest Radiographs in Subjects with Asbestos-Related Abnormalities: Comparison Between ILO Categorizations and Clinical Reading</em>, 21 AM. J. INDUS. MED. 855 (1992)</td>
<td>Exposed in non-traditionally recognized groups; out of the 84 with X-rays graded at 1/0 or higher, the most exposure occurred at electrochemical industries maintenance jobs; for men with lung fibrosis 1/0 or higher, the mean time since their first asbestos exposure was 43.5 years, and the mean duration of exposure was 4.4 years</td>
<td>430 readings graded at 1/0 or higher</td>
<td>Irregular opacities, most prevalent in the middle and lower fields; lung fibrosis; asbestos-related radiographic changes</td>
</tr>
<tr>
<td>N. Hisanaga et al.</td>
<td><em>Pleural Plaques and Irregular Opacities on Chest Radiographs Among Construction Workers: Carpenters, Plasterers, Electricians, Steel-Workers</em>, 6,864 readings</td>
<td></td>
<td>6,864 readings graded at 1/0 or higher</td>
<td>32 showed irregular opacities, and 51 showed pleural plaques</td>
</tr>
</tbody>
</table>

Evaluation of medical and occupational history was conducted to determine if the radiological changes were to be regarded as asbestos-related. Results were based on a consensus of one B Reader and one radiologist reading side by side. This study reclassified X-rays previously read as having a condition consistent with an asbestiform mineral exposure, using the ILO system.
<table>
<thead>
<tr>
<th>Source</th>
<th>Description</th>
<th>Workers/Exposure</th>
<th>X-rays</th>
<th>Results</th>
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<tbody>
<tr>
<td>J.M. Hughes &amp; H. Weill</td>
<td>Pulmonary Fibrosis as a Determinant of Asbestos-Induced Lung Cancer in a Population of Asbestos Cement Workers in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 370 (1990)</td>
<td>Workers in two New Orleans asbestos cement plants; mean age of those studied was 45 years</td>
<td>839</td>
<td>79 (9%); Small opacities</td>
</tr>
<tr>
<td>K. Jakobsson et al.</td>
<td>Radiologic Changes in Asbestos Cement Workers, 52 OCCUPATIONAL &amp; ENVTL. MED. 20 (1995)</td>
<td>Blue collar asbestos cement plant workers; median time since first exposure was 23.5 years; median time on the job was 19.7 years; Median year of start of employment was 1951 (range 1920-1967)</td>
<td>174</td>
<td>36 (20%); Small irregular opacities, “s” or “t”; parenchymal abnormality</td>
</tr>
<tr>
<td>J. Jankovic &amp; R. Reger</td>
<td>Health Hazard Evaluation Report- United Rubber Workers’ International Union, NIOSH Investigation, MHETA 87-017-1949 (1989)</td>
<td>Tire workers, part of the United Rubber Workers’ International Union; all study participants were over 40 yrs. old.</td>
<td>987</td>
<td>2 (0.20%); Small opacities irregular in shape and predominantly in the lower lung zones</td>
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<td>Author(s)</td>
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<td>S. Kagamimori et al.</td>
<td>Studies on Changes in Categories for Pneumoconiosis X-ray Classification in Japanese Workers With Occupational Exposure to Mineral Dusts, Proceedings of Ninth Int’l Conference on Occupational Respiratory Diseases, Kyoto, Japan, 166-169 (1997)</td>
<td>Mineral dust workers in Japan</td>
<td>3,024</td>
<td>81 (2.68%)</td>
</tr>
<tr>
<td>S.M. Kennedy et al.</td>
<td>Lung Function and Chest Radiograph Abnormalities Among Construction Insulators, 20 Am. J. Indus. Med. 673 (1991)</td>
<td>Current and retired construction insulators, aged: 50 years or older</td>
<td>88</td>
<td>16 (18.2%)</td>
</tr>
<tr>
<td>K.H. Kilburn et al.</td>
<td>Interaction of Asbestos, Age, and Cigarette Smoking in Producing Radiographic Evidence of Diffuse Pulmonary Fibrosis, 80 Am. J. Med. 377 (1986)</td>
<td>Wives of shipyard workers; the workers had been employed at the shipyard for twenty years or more, and this was probably their initial contact with asbestos; the women had only in-the-home exposure to asbestos brought in by their husbands</td>
<td>269</td>
<td>19 (7.06%)</td>
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<td>Authors</td>
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<td>K.H. Kilburn et al.</td>
<td><em>Interaction of Asbestos, Age, and Cigarette Smoking in Producing Radiographic Evidence of Diffuse Pulmonary Fibrosis</em>, 80 AM. J. MED. 377 (1986)</td>
<td>Male shipyard workers; 20 years or more from initial shipyard employment and probable initial contact with asbestos; recruited in 1981; of the 32 aged 71-85, 20 (62.5%) had 1/0 or higher. This study separated the numbers also by those who ever smoked and never smoked. For all age groups except the oldest (71-85), a higher percent of smokers had X-rays graded at 1/0 or higher than those who had never smoked. The term “parenchymal asbestosis” is used interchangeably with “diffuse pulmonary fibrosis.”</td>
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<tr>
<td>K.H. Kilburn &amp; R. Warshaw</td>
<td><em>Airway Obstruction in Asbestosis Studied in Shipyard Workers</em>, in <em>PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE</em>, DHHS (NIOSH) PUB. NO. 90-108, at 408 (1990)</td>
<td>Boilermakers employed mostly in ship repair and some in new ship construction; of those studied, the mean age was 52.5 years; the mean amount of time of exposure to asbestos was 27.3 years; to be eligible for the study, the individual had to have been exposed for at least 15 years. The study refers to ILO readings of 1/0 or higher as asbestosis.</td>
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<tr>
<td>K. Koskinen et al.</td>
<td><em>Radiographic Abnormalities Among Finnish Construction, Shipyard and Asbestos Industry Workers</em>, 24 SCANDINAVIAN J. WORK ENV'T &amp; HEALTH 109 (1998)</td>
<td>17,937 construction workers; 456 shipyard workers; 550 asbestos industry workers; study was limited to those employed for at least 10 years; in construction and com-... Small irregular lung opacities indicative of interstitial pulmonary fibrosis. The criteria for testing positive was (i) opacities clearly consistent with interstitial fibrosis (1/1); (ii) opacities indicating mild interstitial fibrosis (1/0), and findings consistent with unilateral or bilateral pleural plaques; (iii) findings indicating marked abnormalities of the visceral pleura not known to be caused...</td>
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<td>J. Lefante et al.</td>
<td>An Analysis of X-ray Reader Agreement: Do Five Readers Significantly Increase Reader Classification Reliability Over That of Three Readers? in PROCEEDINGS OF THE VIIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 482 (1990)</td>
<td>Workers employed in the manufacture of man-made fibers; mean age was 41 (ranging from 19 to 76)</td>
<td>1168</td>
<td>19 (1.62%)</td>
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<tr>
<td>S.M. Levin &amp; I.J. Selikoff</td>
<td>Radiological Abnormalities and Asbestos Exposure Among Custodians of the New York City Board of Education, 31 ANNALS. N.Y. ACAD. SCI. 653 (1991)</td>
<td>Custodians of New York City Board of Education; 66% had begun custodial work before 1965; examinations were done between 1985 and 1987;</td>
<td>660</td>
<td>105 (16%)</td>
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<td>Study</td>
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<td>R. Lillis et al.</td>
<td>Radiographic Abnormalities in Asbestos Insulators: Effects of Duration From Onset of Exposure and Smoking, Relationships of Dyspnea With Parenchymal and Pleural Fibrosis, 20 Am. J. Indus. Med. 1 (1991)</td>
<td>Active and retired asbestos insulators; 86.8% had 30 years or more from onset of asbestos exposure; testing was performed in 19 cities between 1981 and 1983</td>
<td>2,790 Active and retired asbestos insulators; 86.8% had 30 years or more from onset of asbestos exposure; testing was performed in 19 cities between 1981 and 1983. Small irregular opacities indicating the presence of interstitial pulmonary fibrosis. Study also showed that cigarette smoking contributes to the prevalence and severity of interstitial fibrosis.</td>
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<td>O. Metadilogkul &amp; P. Supanachart</td>
<td>Occupational Asbestosis and Asbestos Related Diseases Among Workers Exposed To Asbestos, 1987, Thailand, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSIS CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 331 (1990)</td>
<td>All workers in the 24 factories registered by Ministry of Industry that used asbestos in a production process</td>
<td>All films were read by the same B Reader (Dr. Lilis).</td>
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<td>A. Miller et al.</td>
<td>Relationship of Pulmonary Function to Radiographic Interstitial Fibrosis in 2,611 Long-term Asbestos Insulators, 145 Am. Rev.</td>
<td>Insulators who were enrolled in the union on January 1, 1967 and who had at least 30 yrs from onset of exposure</td>
<td>2611 Insulators who were enrolled in the union on January 1, 1967 and who had at least 30 yrs from onset of exposure. Small irregular opacities, consistent with interstitial pulmonary fibrosis. All films were read by the same B Reader (Dr. Lilis).</td>
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<td>Study</td>
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<td>J. Miller</td>
<td>Benign Exposure to Asbestos Among Power Plant Workers</td>
<td>1990</td>
<td>Unpublished manuscript on file with author</td>
<td>No definite cases of asbestosis; none of the initial/subsequent films showed small irregular opacities more than 1/0</td>
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<td>R.L. Murphy Jr. et al.</td>
<td>Effects of Low Concentrations of Asbestos. Clinical, Environmental, Radiographic and Epidemiologic Observations in Shipyard Pipe Coverers and Controls</td>
<td>1971</td>
<td>45 of those studied (44.55%) had been exposed for more than 30 years</td>
<td>Results were coded by number: 1=none, 2=abnormal not consistent w/asbestosis, 3=questionably consistent with asbestosis, 4=consistent with slight asbestosis, 5=consistent with moderately advanced asbestosis, and 6=consistent with advanced asbestosis. When the ILO system was developed, the authors added a footnote of how to convert their readings to the ILO categories: 1=0/0; 3=0/1; 4=1/0, 1/1, 1/2; 5=2/1, 2/2, 2/3; and 6=3/2, 3/3, 3/4.</td>
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<tr>
<td>R.L. Murphy Jr. et al.</td>
<td>Effects of Low Concentrations of Asbestos. Clinical, Environmental, Radiographic and Epidemiologic Observations in Shipyard Pipe Coverers and Controls</td>
<td>1971</td>
<td>21 (22.34%)</td>
<td>The X-rays were read by three readers.</td>
</tr>
<tr>
<td>R.T. Myint &amp; S. Myint</td>
<td>Small Airway Impairment Findings at the Screening of Asbestos workers in different trades—sheet</td>
<td>1981-1983</td>
<td>183 (29%) were 1/1 or</td>
<td>This study included 70 insulators but the results could not be segregated for use</td>
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<td>Study</td>
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<td>639 Asbestos Workers with Exposure History of 20 yrs., in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 375 (1990)</td>
<td>metal workers, pipefitters, insulators, boilermakers, bricklayers, iron workers and others; to be included, subjects had to have been exposed for 20 or more years.</td>
<td>higher results as “incidence of asbestosis in chest X-ray profusion between 1/1-3/3.” in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES CONFERENCE, at 375</td>
<td>in the “insulator” section. This study did not identify the number of X-rays read as 1/0. Results support the “synergistic action” of cigarette smoking and asbestos exposure.</td>
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<tr>
<td>J. Ohar et al.</td>
<td>Changing Patterns in Asbestos-Induced Lung Disease, 125 CHEST 744 (2004)</td>
<td>Entry criteria was a documented workplace asbestos exposure, latency of more than 10 years and an abnormal chest X-ray consistent with the history of asbestos exposure</td>
<td>3,383</td>
<td>312 (9.2%) were 1/1 or higher</td>
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<tr>
<td>P. Oksa et al.</td>
<td>Parenchymal and Pleural Fibrosis in Construction Workers, 21 AM. J. INDUS. MED. 561 (1992)</td>
<td>Construction workers; of the 437 studied, 81% verified exposure to asbestos; the average duration of exposure was 3.7 years</td>
<td>437</td>
<td>70 (16%) were 1/1 or higher</td>
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<td>Author(s)</td>
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<td>R.B. Reger et al.</td>
<td><em>Cases of Alleged Asbestos-Related Disease: A Radiologic Re-evaluation</em>, 32 J. OCCUPATIONAL MED. 1088 (1990)</td>
<td>Tireworkers; previously designated as having a condition consistent with an asbestiform mineral exposure</td>
<td>439</td>
<td>8</td>
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<tr>
<td>A.Z. Roszkay et al.</td>
<td><em>Respiratory Health in Asbestos-Exposed Ironworkers</em>, 29 AM. J. INDUS. MED. 459 (1996)</td>
<td>Asbestos-exposed ironworkers in Michigan; average length of time since joining the union was 24.5 years. (ranging from 0.8 to 51.7 years); calendar years, in trade spanned from 1937 to 1990</td>
<td>547</td>
<td>38</td>
</tr>
<tr>
<td>L. Rosenstock et al.</td>
<td><em>The Relation Among Pulmonary Function, Chest Roentgenographic Abnormalities, and Smoking Status in an Asbestos-Exposed Cohort</em>, 138 AM.</td>
<td>Plumbers, pipefitters, welders, steamfitters, refrigeration, and others; the mean age among participants was 42.1 years; the</td>
<td>681</td>
<td>132</td>
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<td>Study on Asbestos Exposure and Other Dusty Work</td>
<td>Small Nodule and Other Asbestos Exposure and Other Dusty Work</td>
<td>Trade Duration in yrs.</td>
<td>Small Nodule and Other Asbestos Exposure and Other Dusty Work</td>
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<td>Shoot reads 1/3 or higher because these were read independently by 5 readers.</td>
<td>Shipyard workers—mostly sprayers, laggers, asbestos storemen, and masons using asbestos cement; small subgroup of Devonport (U.K.) workers; about one-half worked in high exposure trades</td>
<td>Shipyard workers in Devonport</td>
<td>73 J. ROYAL. SOC'Y. MED. 337 (1980)</td>
<td>73 J. ROYAL. SOC'Y. MED. 337 (1980)</td>
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<td>The court appointed ten medical experts to review the plaintiffs' medical records including re-reading the X-rays.</td>
<td>Though the X-ray readings were not uniform, the reason for nonuniform results was not clear.</td>
<td>The court's experts submitted diagnoses of asbestosis and did not list X-ray readings.</td>
<td>137 F.R.D. 35 (1991)</td>
<td>137 F.R.D. 35 (1991)</td>
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<td>This study did not identify the number of X-rays read as 1/0. High exposure trades were sprayers and laggers.</td>
<td>This study did not identify the number of X-rays read as 1/0. High exposure trades were sprayers and laggers.</td>
<td>This study did not identify the number of X-rays read as 1/0. High exposure trades were sprayers and laggers.</td>
<td>610 CARDOZO LAW REVIEW Vol. 29:2</td>
<td>610 CARDOZO LAW REVIEW Vol. 29:2</td>
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<td>Study</td>
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<td>D.A. Schwartz et al.</td>
<td>Asbestos-Induced Pleural Fibrosis and Impaired Lung Function, 141 AM. REV. RESPIRATORY DISEASE, 321 (1990)</td>
<td>Sheet metal workers, employed for a minimum of 25 years as of 1986; average time in trade was 32.7 years</td>
<td>Defined asbestos as a “profusion of 1/0 or greater”</td>
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<tr>
<td>I.J. Selikoff et al.</td>
<td>Asbestotic Radiological Abnormalities Among United States Merchant Marine Seamen, 47 BRIT. J. INDUS. MED. 292 (1990)</td>
<td>Merchant marine seamen; X-rays taken between 1985 and 1987</td>
<td>The onset of exposure had occurred before 1939 in almost 11% of those studied. Of these individuals, 49.5% had started employment between 1940 and 1949, and only 8.1% had started in 1970 or later.</td>
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<td>I. Selikoff &amp; R. Lilis</td>
<td>Radiological Abnormalities Among Sheet-Metal Workers in the Construction Industry in the United States and Canada: Relationship to Asbestos Exposure, ARCHIVES ENV'T'L. HEALTH, 30 (1991)</td>
<td>Sheet-metal workers in the construction industry; X-rays taken between 1986 and 1987; employed for at least 35 years; mean duration from onset of asbestos exposure was 39.5 years</td>
<td>Small irregular opacities in the lung parenchyma</td>
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<td>I.J. Selikoff et al.</td>
<td>The Occurrence of Asbestosis Among Insulation Workers from local unions in</td>
<td>Insulation workers from local unions in</td>
<td>Radiological change as the Of those studied who had 40 or more years since the onset of exposure, there</td>
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<td>Study Title</td>
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<td>Workers in the United States, 132 ANNALS N. Y. ACAD. SCI. 139 (1965)</td>
<td>New York and New Jersey; study was conducted in 1963; of the 1,117 individuals studied, 18.26% had 35 years or more pass since onset of exposure.</td>
<td>sole criteria of evidence of pulmonary asbestosis was an abnormal X-ray in 94.2%. Among those with 0 to 9 years since onset, the percent of X-ray readings that were abnormal was 10.4%. No ILO system was used. This review uses the results of a later study which reread these X-rays using the ILO system.</td>
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<tr>
<td>G. Sheers et al.</td>
<td>Male naval dockyard workers in the United Kingdom; a stratified sample was drawn in favor of those more heavily exposed.</td>
<td>674, 20 (3%) were 1/1 or higher</td>
<td>Asbestos-related abnormality; parenchymal fibrosis; small opacities Read by five Readers. This study did not identify the number of X-rays read as 1/0.</td>
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<tr>
<td>M. Silberschmid et al.</td>
<td>Railway employees with exposure to asbestos; more than 40 years since first exposure for 32% of those studied; more than 30 yrs. for 47% of those studied; the mean age was 65.1 years; 72% were between 60 and 70 yrs. old.</td>
<td>175, 21 (12%) were 1/1 or higher</td>
<td>N/A This study was done twice, in 1981 and 1986. The 1986 results are used because the operative criteria stated in the review are that, in such cases, the reevaluation of the same populations will be used. This study did not identify the number of X-rays read as 1/0. The X-rays were read by two readers.</td>
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<tr>
<td>N.L. Sprince et al.</td>
<td>Plumbers and pipefitters employed in building construction; mean number of years of employment was 24.3</td>
<td>153, 12 (7.8%) Parenchymal abnormality, defined as small irregular opacities</td>
<td>Seven of those individuals studied had readings of 1/0 and 5 had readings of 1/1. The X-rays were read by one reader.</td>
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<td>Plumbers and pipefitters in a New York union chapter; about 26.4% had less than 15 years latency (years since first exposure), 30.5% had 16-25 years latency, 24% had 26-35 years latency; and 19% had less than 35 yrs</td>
<td>343</td>
<td>42 (12.2%)</td>
<td>Opacities of size “s” or “t” and profusion 1/0 or greater; parenchymal change consistent with possible asbestosis</td>
<td>Of the 797 individuals invited to participate; 343 accepted. Those who did not respond tended to be older, retired, and living out of state—thus the “study group represented a younger...subset.” <em>Proceedings of the VIIth International Pneumoconioses Conference</em>, at 334; the X-rays were read by one B Reader.</td>
</tr>
<tr>
<td>William Weiss &amp; Peter A. Theodos</td>
<td>Pleuropulmonary Disease Among Asbestos Workers in Relation to Smoking and Type of Exposure, 20 J. Occupational Med. 341 (1978)</td>
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<tr>
<td>Employees in two asbestos manufacturing plants aged 40 and over; 42 (43%) have been working for more than 30 years</td>
<td>98</td>
<td>20 (20%)</td>
<td>The types of irregular opacities were limited to “s” and “t” in ILO classification</td>
<td>Almost all the cases of X-ray abnormalities typical of asbestosis were found in men over 40 years old. The X-rays were read jointly and classified by consensus.</td>
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<tr>
<td>Sheet metal workers first employed in the trade at least 20 years before the study, examined starting in 1986; The average time worked in the industry was 32.8 years, and the average time of being a sheet metal worker was 35 yrs; among those with over 40 years since entering the trade, 17.3% had X-rays graded at 1/0 or greater</td>
<td>9,605</td>
<td>1,178 (12.3%)</td>
<td>Small opacities; Parenchymal abnormalities; asbestos-related abnormalities</td>
<td>Each X-ray was read by one reader.</td>
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</table>

Representative sample of Finnish adult population 3,601 506 (14.05%) N/A The X-rays were read by two readers. Based on a survey, the group was divided into three categories: (1) probably exposed, (2) possibly exposed, and (3) unlikely exposed. Those in this study are limited to those included in categories (1) and (2). Category (3) is included in the review of unexposed populations.

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>STUDY</th>
<th>STUDY GROUP &amp; EXPOSURE DETAILS</th>
<th>N</th>
<th>I/0 OR HIGHER</th>
<th>OPACITY TYPE: LANGUAGE USED</th>
<th>DETAIL</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.E. Amandus et al.</td>
<td>Significance of Irregular Small Opacities in radiographs of Coalminers in the USA, 33 BRIT. J. INDUS. MED. 13 (1976)</td>
<td>Coalminers</td>
<td>6,166</td>
<td>801 (13%)</td>
<td>N/A</td>
<td>The authors studied coalminers who had no asbestos exposure. This study is not included in the exposed review because miners are excluded, and it is not included in the unexposed review because of intensive exposure to coal dust and possibly silica. To be eligible for the study, the subject had to have a previous X-ray that was read as showing small opacities 1/0 or greater. Those with either massive fibrosis or with a clear X-ray were excluded.</td>
</tr>
<tr>
<td>H. Anton-Culver et al.</td>
<td>An Epidemiologic Study of Asbestos-Related Chest X-ray Changes to Identify Work Areas of High Risk in Shipyard workers employed in a West Coast shipyard, with a median age of 49 years;</td>
<td>3,903 515 abnrml. N/A</td>
<td></td>
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<td></td>
<td>The 515 individuals in the first category include multiple pleural plaques, markedly increased bilateral pleural thickening and interstitial disease.</td>
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<tr>
<td>Study</td>
<td>Description</td>
<td>Median Age</td>
<td>Median Duration</td>
<td>Diagnosis</td>
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<td>a Shipyard Population, 4</td>
<td>Men with a history of occupational exposure to asbestos; at least one year of occupational exposure to asbestos; 55 of the 60 included had asbestos related pleural disease, the others were suspected of having mesothelioma, asthma from exposure, and lung cancer</td>
<td>33 yrs</td>
<td>13 yrs</td>
<td>(1) probably asbestos related (13.2%); (2) possibly asbestos related (7.5%); (3) probably not related to asbestos exposure (12%); and (4) no abnormality (67.3%).</td>
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<tr>
<td>A.C. Friedman et al.</td>
<td>This study of 60 men is excluded because the subjects were selected based on previous chest X-rays interpreted as indicating asbestos-related pleural and parenchymal disease or a malignancy. This study diagnosed 21 of the 60 subjects with asbestosis; 19 were diagnosed with parenchymal asbestosis and pleural disease, and 2 had parenchymal asbestosis without pleural disease.</td>
<td>60</td>
<td>N/A</td>
<td>N/A</td>
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<tr>
<td>P.G. Harries et al.</td>
<td>This study is excluded because it does not provide ILO scores though it does list the number found with “confirmed pulmonary fibrosis.” However, this determination is based on X-ray readings, lung function testing, and clinical examinations. Based on this criteria, of 3,856 tested, 12 (0.3%) were found to have the condition listed that appears to be the equivalent of a diagnosis of asbestosis.</td>
<td>3,856</td>
<td>N/A</td>
<td>N/A</td>
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<tr>
<td>Author(s)</td>
<td>Title</td>
<td>Study Details</td>
<td>Workers</td>
<td>N/A</td>
<td>N/A</td>
<td>Remarks</td>
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<tr>
<td>S. Kagamimori et al.</td>
<td>Studies on Changes in Categories for Pneumoconiosis X-ray Classification in Japanese Workers With Occupational Exposure to Mineral Dusts, Proceedings of Ninth International Conference on Occupational Respiratory Diseases, Kyoto, Japan at 166-69 (1997) (reproducing original 1986 study)</td>
<td>Workers exposed to mineral dusts in Japan</td>
<td>4,959</td>
<td>67 (1.35%)</td>
<td>N/A</td>
<td>These were evaluated by an official panel on pneumoconiosis. This part of the study is excluded because the results of the re-evaluation which indicate a higher prevalence of fibrosis is being used in the review.</td>
</tr>
<tr>
<td>Langlands, J.H.M. et al.</td>
<td>Insulation Workers in Belfast. 2. Morbidity in Men Still at Work, 28 BRIT. J. INDUS. MED. 217 (1971)</td>
<td>Insulators</td>
<td>162</td>
<td>N/A</td>
<td>N/A</td>
<td>This study did not use the ILO classification system. The authors evaluated the causes of deaths as the subjects died. Some of the subjects of this study were re-evaluated in Campbell 1982, which is included, supra.</td>
</tr>
<tr>
<td>Liddell, F.D.K. et al.</td>
<td>Radiological Changes and Fibre Exposure in Chrysotile Workers Aged 60-69 Yrs. at Thetford Mines, 26 ANNALS OCCUPATIONAL HYGIENE 889 (1982)</td>
<td>Asbestos mine workers aged 60 years or older</td>
<td>515</td>
<td>136 (26.4%)</td>
<td>N/A</td>
<td>This study is excluded because the subjects were not using asbestos containing products, were not in proximity to others using such products, and were not involved in the manufacturing of asbestos products. In addition, miners are excluded from this review.</td>
</tr>
<tr>
<td>McMillan, G.H.G. et al.</td>
<td>Effects of Smoking on Attack Rates of Pulmonary and Pleural Lesions Related to Exposure to Asbestos Dust, 37 BRIT. J. INDUS. MED. 268 (1980)</td>
<td>Shipyard workers, in the U.K. dockyards</td>
<td>1731</td>
<td>N/A</td>
<td>N/A</td>
<td>This study did not use the ILO classification system.</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Reference</td>
<td>Participants</td>
<td>Number</td>
<td>Percentage</td>
<td>Description</td>
<td>Notes</td>
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<tr>
<td>E.R.A. Merewether et al.</td>
<td>Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry, HM Stationery Office, London, UK (1930)</td>
<td>Textile workers manufacturing insulating materials from practically pure asbestos. Of the 363 examined, 21 had been employed for more than 20 years, 28 for between 15 and 19 years, 84 for between 10 and 14 years, and 230 for less than 10 years</td>
<td>133</td>
<td>62 (46.6%)</td>
<td>Radiological signs of diffuse fibrosis</td>
<td>This study was published in 1930, before there was an ILO system. It examined 363 workers, but only 133 had an X-ray done. Of the 363 studied, 95 (26.17%) showed a definite fibrosis due to asbestos dust. The data for the 133 that had an X-ray is not available.</td>
</tr>
<tr>
<td>L.C. Oliver et al.</td>
<td>Asbestos-Related Radiographic Abnormalities In Public School Custodians, 6 TOXICOLOGY INDUS. HEALTH 629 (1990)</td>
<td>Public school custodians</td>
<td>120</td>
<td>N/A</td>
<td>N/A</td>
<td>This study examined X-rays only for pleural plaques.</td>
</tr>
<tr>
<td>Pearle, J.L.</td>
<td>Smoking and Duration of Asbestos Exposure in the Production of Functional and Roentgenographic Abnormalities In Shipyard Workers, 24 J. OCCUPATIONAL MED. 37 (1982)</td>
<td>Shipyard workers</td>
<td>131</td>
<td>N/A</td>
<td>N/A</td>
<td>This study did not use the ILO classification system to describe its results.</td>
</tr>
<tr>
<td>H. Robin et al.</td>
<td>Clinical, Radiological and Functional Abnormalities Among Workers of an Asbestos-Cement Factory, in PROCEEDINGS OF THE VIITH INTERNATIONAL PNEUMOCONIOSES</td>
<td>Workers in a factory in France mainly producing fibrocement pipes and roofing components</td>
<td>92</td>
<td>N/A</td>
<td>N/A</td>
<td>This study does not present results of the X-ray readings. The study does provide PFT results, and states that five persons were found to have pulmonary fibrosis, nine with benign pleural thickening and 57 with associated pulmonary fibrosis and nonmalignant</td>
</tr>
</tbody>
</table>
pleural changes.

Rubino, G.F. et al. Radiologic Changes After Cessation of Exposure Among Chrysotile Asbestos Miners in Italy, 330 ANNALS N.Y. ACAD. SCI. 157 (1979) Chrysotile Miners 56 21 (37.5%) N/A Miners are excluded from this review. In addition, the subjects were not using asbestos-containing products, in proximity of others using such products, or in the manufacturing of asbestos products.

William Weiss Cigarette Smoking, Asbestos, and Pulmonary Fibrosis, 104 AM. REV. RESPIRATORY DISEASE 223 (1971) Asbestos textile mill workers 100 36 (36%) N/A This study is excluded because it did not use the ILO classification system to record the results. Instead, the term “pulmonary fibrosis” is used to characterize the results.

C. A Review of 11 Clinical Studies of Unexposed Populations

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>STUDY</th>
<th>STUDY GROUP &amp; EXPOSURE DETAILS</th>
<th>N</th>
<th>1/0 OR HIGHER</th>
<th>OPACITY TYPE: LANGUAGE USED</th>
<th>DETAIL</th>
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<tbody>
<tr>
<td>S. Cordier et al.</td>
<td>Epidemiologic Investigation of Respiratory Effects Related to Environmental Exposure to Asbestos In-</td>
<td>Three groups: occupationally exposed; environmentally exposed (working in asbestos-</td>
<td>1108</td>
<td>174 (15.7%)</td>
<td>N/A</td>
<td>For purposes of this review, I have excluded only the occupationally exposed subjects because the prevalence of fibrosis caused by working in build-</td>
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<tr>
<td>Study Description</td>
<td>Participants</td>
<td>Findings</td>
<td>Notes</td>
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<tr>
<td>Insulated Buildings, 42 ARCHIVES ENVTL. HEALTH 303 (1987)</td>
<td>insulated buildings for at least 15 years with no known occupational exposure; and nonexposed</td>
<td>findings with asbestos insulation is extremely low. The study recorded results as an average of two B Readers.</td>
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<tr>
<td>Alan M. Ducatman et al. 'Readers' and Asbestos Medical Surveillance, 30 J. OCCUPATIONAL MED. 644 (1988)</td>
<td>US Navy Employees</td>
<td>105,029 2799 (2.8%)</td>
<td>In this study, the number of individuals studied was 105,029, and the number with X-rays read as 1/0 or higher was 3778 (3.51%). I am excluding an outlier B Reader who was 5-100 times more likely to find that X-rays were 1/0 or higher than the other readers. This study was excluded from the Meyer's meta-analysis of seven studies because the 23 B Readers did not read the same films; instead each B Reader read films ranging in number from 1,777 to 5,779 which were not reread by other readers.</td>
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<tr>
<td>D.M. Epstein et al. Application of ILO Classification to a Population Without Industrial Exposure: Findings to be Differentiated from Pneumococcosis, 142 Am. J. ROENTGENOLOGY 53 (1984)</td>
<td>Adults admitted to a university medical center, Philadelphia</td>
<td>200 36 (18%)</td>
<td>In Meyer's meta-analysis (see text, supra note 79), he excludes from the 36 with readings of 1/0 or higher, ten individuals who had medical conditions that caused their fibroses and four that may have been exposed. However, while he adjusts the 1/0 or higher number, he does not adjust N. I am including all the readings that were 1/0 or higher. Each X-ray was read by 2 readers.</td>
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<tr>
<td>J.R. Glover et al. Effects of Exposure to Slate Dust in North Wales.</td>
<td>Men chosen from electoral rolls</td>
<td>402 39 (9.7%)</td>
<td>The group studied was divided into those exposed to slate (silica) and</td>
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<td>Study</td>
<td>Title</td>
<td>Methods</td>
<td>Findings</td>
<td>X-rays Read By</td>
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<tr>
<td>Bjorn Hilt et al.</td>
<td>Asbestos-Related Radiographic Changes by ILO Classification of 10 x 10 cm Chest X-rays in a Screening of the General Population, 37 J. Envtl. Med. 189 (1995)</td>
<td>This study describes a previous screening of 21,453 males aged 40 or older in the county of Telemark, Norway</td>
<td>Those unexposed. Only the unexposed population is used in this review. This study also correlated opacities with smoking. The X-rays were read by three readers.</td>
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<tr>
<td>S.M. Kennedy et al.</td>
<td>Lung Function and Chest Radiograph Abnormalities Among Construction Insulators, 20 Am. J. Indus. Med. 673 (1991)</td>
<td>Employed bus mechanics and retired grain and civic workers</td>
<td>This study is used in the insulator section. There were two separate groups. The X-rays were read independently by two readers.</td>
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<tr>
<td>K. Jacobsson et al.</td>
<td>Radiological Changes in Asbestos Cement Workers, 52 Occupational Envtl. Med. 20 (1995)</td>
<td>White collar workers from an asbestos cement plant, in Sweden</td>
<td>Part of this study is separated out and included in the exposed review, because it also examined 174 blue collar workers. X-rays were read independently by five readers.</td>
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<tr>
<td>Authors</td>
<td>Title</td>
<td>Study Details</td>
<td>Population Size</td>
<td>Radiographic Findings</td>
<td>Comments</td>
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<tr>
<td>K.H. Kilburn et al.</td>
<td>Interaction of Asbestos, Age, and Cigarette Smoking in Producing Radiographic Evidence of Diffuse Pulmonary Fibrosis, 80 AM. J. MED. 377 (1986)</td>
<td>Samples of population in Michigan and sample of census tract in California</td>
<td>2514</td>
<td>32 (1.27%) Irregular opacities; diffuse pulmonary fibrosis; lung changes typical of asbestosis.</td>
<td>This study had five categories of populations: two were unexposed; one was an insulator study; and two were exposed. Only the two unexposed groups are included here. There were three physicians used to interpret the X-rays and an arithmetic average was used.</td>
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<tr>
<td>R. Saito et al.</td>
<td>A Study On Asbestos-Associated Lung Diseases Among Former U.S. Naval Shipyard Workers, in PROCEEDINGS OF THE VIIth INTERNATIONAL PNEUMOCONIOSES CONFERENCE, DHHS (NIOSH) PUB. NO. 90-108, at 362 (1990)</td>
<td>This was an unexposed control group in the study of US Naval yard workers in Japan; mean age of 67.2 years</td>
<td>40</td>
<td>22 (57%) Small irregular opacities and small nodular opacities</td>
<td>No explanation is offered for the relatively high prevalence of pneumoconiosis in the control group of persons not occupationally exposed to asbestos.</td>
<td></td>
</tr>
<tr>
<td>A.J. Zitting et al.</td>
<td>Radiographic Small Lung Opacities and Pleural Abnormalities as a Consequence of Asbestos Exposure in an Adult Population, 21 SCANDINAVIAN J. WORK ENV'T. &amp; HEALTH 470 (1995)</td>
<td>Representative sample of adult Finnish population</td>
<td>3,494</td>
<td>408 (11.7%)</td>
<td>Two radiologists read the X-rays. Based on a survey, the group was divided into three categories: (1) probably exposed, (2) possibly exposed, and (3) unlikely exposed. Those included in this review are limited to category (3).</td>
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</table>
### D. Clinical Studies of Unexposed Populations Excluded from the Review

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>STUDY</th>
<th>STUDY GROUP &amp; EXPOSURE DETAILS</th>
<th>N</th>
<th>I/I0 OR HIGHER</th>
<th>OPACITY TYPE; LANGUAGE USED</th>
<th>DETAIL</th>
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</thead>
<tbody>
<tr>
<td>William Weiss</td>
<td><em>Cigarette Smoking and Diffuse Pulmonary Fibrosis, 99 AM. REV. RESPIRATORY DISEASE 67 (1969)</em></td>
<td>Screening offered to all adults who came to the Philadelphia Tuberculosis and Health Association’s Central X-ray Unit and had a private physician</td>
<td>2,825</td>
<td>40 (1.4%)</td>
<td>N/A</td>
<td>This study is excluded because the results are not provided using the ILO classification system. Instead, the term “pulmonary fibrosis” is used. The study found that the prevalence of diffuse pulmonary fibrosis was three times higher in cigarette smokers than in non-smokers.</td>
</tr>
<tr>
<td>William Weiss</td>
<td><em>Cigarette Smoking and Diffuse Pulmonary Fibrosis, 14 ARCHIVES ENVTL. HEALTH 564 (1967)</em></td>
<td>Adults undergoing routine chest photofluorograms at the Philadelphia Tuberculosis and Health Association, between March 11, 1966 and April 12, 1966</td>
<td>999</td>
<td>31 (3.1%)</td>
<td>N/A</td>
<td>This study is excluded because the results are not provided using the ILO classification system. Instead, the term “pulmonary fibrosis” is used.</td>
</tr>
</tbody>
</table>