Comment

The Dark Side of Workers’ Compensation: Burdens and Benefits in Occupational Disease Coverage

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It has long been recognized that occupational disability can occur as easily from continued exposure to unhealthful conditions as it can from sudden injury; but that recognition has not prevented the imposition of legal proof requirements that compensation claimants and the medical profession are not in a position to discharge. The author here examines the results of these legal requirements, the nature of the overall problem, and possible means of assuring that occupationally disabled workers receive the compensation to which they are entitled.

More attention has been focused on health and safety conditions in American workplaces in the past few years than at any other time since the turn of the century. Popular writings have dramatized working conditions and called for reform.¹ Recent federal legislation has placed new duties on employers.² However, most of the debate has centered on the prevention of unsafe and unhealthy working conditions; the adequacy of programs directed towards the consequences of workplace hazards has largely escaped public scrutiny.

The operation of the workers' compensation system generates little of the publicity of such recent occupational health issues as threshold limit values for airborne contaminants or warrantless workplace in-

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spections by OSHA. Workers' compensation law raises no such direct, public issue regarding the rights of workers pitted against the prerogatives of corporate employers. It does not regulate work practices. Despite its low profile, the workers' compensation system substantially affects the security of millions of workers. Its subject is the disabled individual worker, who seeks to enforce his or her rights in an individualized legal proceeding long after the work injury or toxic exposure has occurred. Workers' compensation benefits are often the sole source to which disabled workers can turn for aid.

The relative obscurity of workers' compensation law has masked its failure to adequately protect victims of occupational diseases. Because of inherent flaws in the process by which the law determines the link between a worker's condition and his or her employment, probably the majority of workers with occupational diseases are excluded from coverage. As a federal study group reported in 1977:

[N]ot many victims of work-related disease receive workers' compensation. Only two percent of the cases in a survey of closed claims . . . were occupational disease cases (including heart attack cases) . . . .

About 30,000 new occupational disease cases are now being compensated annually — less than half the estimated number of occupational disease fatalities. Moreover, a substantial proportion of the cases receiving workers' compensation for occupational disease are for short term and often non-severe conditions such as dermatitis.3

This comment examines the shortcomings of occupational disease coverage which result from the law's reliance on medical expertise to identify and diagnose disease causation. The burden of medical uncertainty and lack of knowledge is borne by two classes of workers: those disabled by occupational exposures not yet known to be toxic, and those with occupational diseases difficult to differentiate from clinically similar non-occupational counterparts. The obstacles faced by workers in the former category are treated below by examining the methods by which diseases are classified as occupational in origin.

3. Policy Group of the Interdepartmental Workers' Compensation Task Force, Workers' Compensation: Is There a Better Way? 6 (1977) (This document is listed in Monthly Catalog of United States Government Publications, October 1977, at 134). A survey of health conditions of 908 workers in small industries in Oregon and Washington found that 31% of medical conditions of the workers were of "probable occupational origin." The authors thought this to be a conservative estimate. The identified diseases were not necessarily disabling. Only 3% of the cases were also found in workers' compensation claims files. Moreover, most of the disease cases in the workers' compensation files were acute conditions. D. Discher, G. Kleinman & F. Foster, Pilot Study for Development of an Occupational Disease Surveillance Method, (Dep't of Health, Education and Welfare Publication No. (NIOSH) 75-162, 1975). In Great Britain, a study of foundry workers estimated that between 2500 and 7700 workers with lung disease were potentially eligible for benefits, but an average of only 47 a year were awarded compensation. McCallum, Respiratory Disease in Foundrymen, 29 Brit. J. Indus. Med. 341-43 (1972).
Barriers to recovery for the latter group are analyzed by a case study of byssinosis, a disabling respiratory disease of textile workers.

Because medical knowledge of occupational disease is poorly developed, the law's assignment to the disabled worker of the burden of proof is both unfair and contrary to the social purposes of workers' compensation. In some cases, workers should be given the benefit of a statutory presumption that their condition is caused by exposure to a workplace contaminant. The presumption approach, however, is of limited usefulness for the worker suffering from diseases not yet determined to be occupationally induced. In these cases, the workers' compensation system cannot offer satisfactory protection to the disease victim.

I

OCCUPATIONAL DISEASE COVERAGE UNDER WORKERS' COMPENSATION

A. Historical Development

Since the heyday of industrial capitalism in the late 1800's, government has taken faltering steps to bring workplace hazards under control. Though some early safety laws attempted to reduce the number of industrial accidents, most of the legislation enacted simply expanded the legal rights of workers injured on the job. Prior common law held the employer liable only for personal negligent acts; the employer was free from liability if an injury to a worker was caused by a "fellow servant."4 Between 1890 and 1910, state legislatures enacted employer's liability laws limiting the employer's traditional defenses — injury by fellow-servant, assumption of risk, and contributory negligence5 — but statutory reform of the common law was far from complete.6 The results of the rules for work injury, even as modified, were scandalous. For example, the United States Steel Corporation lost

4. The rule was announced by Chief Justice Shaw in Farwell v. Boston & W. R.R. Corp., 45 Mass. 49, 59 (1842):

Where several persons are employed in the conduct of one common enterprise or undertaking, and the safety of each depends much on the care and skill with which each other shall perform his appropriate duty, each is an observer of the conduct of the others, can give notice of any misconduct, incapacity, or neglect of duty, and leave the service, if the common employer will not take such precautions, and employ such agents as the safety of the whole party may require. By these means, the safety of each will be much more effectually secured, than could be done by a resort to the common employer for indemnity in case of loss by the negligence of each other. Regarding it in this light, it is the ordinary case of one sustaining an injury in the course of his own employment, in which he must bear the loss himself, or seek his remedy, if he have any, against the actual wrong-doer.

5. The history of employer's liability legislation is traced in W. DODD, ADMINISTRATION OF WORKMEN'S COMPENSATION, 11-16 (1936).

6. Id.; see, e.g., Act of March 6, 1907, ch. 97, 1907 Cal. Stats. (amending Civil Code § 1970, now repealed. Current version at CAL. LABOR CODE §§ 2803, 2804 (West 1971)) (contributory
only six verdicts to employees between 1906 and 1912.\footnote{7}

The first state workers' compensation law was passed in New York in 1910.\footnote{8} Legislation followed soon after in most other states. The commonly accepted purpose of workers' compensation was to eliminate the negligence concept for work injuries, instead treating accidents as part of the cost of production of commodities.\footnote{9} Workers' compensation was considered to be humanitarian legislation, enacted to recognize what was thought to be an inevitable cost of private enterprise:

The evil [that the law] seeks to remedy is one that calls loudly for action. Accidents to workmen engaged in the industries enumerated in it are all but inevitable . . . no matter how carefully laws for the prevention of accidents in such industries may be framed, or how rigidly they may be enforced . . . . Heretofore these losses have been borne by the injured workmen themselves, by their dependents, or by the state at large. It was the belief of the Legislature that they should be borne by the industries causing them, or, perhaps more accurately, by the consumers of the products of such industries.\footnote{10}

Early statutes did not cover occupational diseases, which also were not successfully handled under the common law.\footnote{11} Coverage for occupational disease was resisted in part by arguments that workers assumed the risk of unhealthy jobs and that diseases were frequently attributable to the carelessness or poor hygiene of the worker.\footnote{12} Coverage was gradually extended to occupational diseases by statute\footnote{13} or through judicial construction of work accident statutes.\footnote{14} Disease claims generally were grafted onto the existing procedure for compensating injuries.

Occupational diseases first attracted special attention in the 1930's, when legislatures, industry, and insurers were faced with widespread public concern over silicosis. A flurry of activity was produced by publicity surrounding a disaster at Gauley Bridge, West Virginia, negligence defense maintained, fellow servant and assumption of risk rules modified but not eliminated); No. 329, 1907 Pa. Laws (fellow servant rule only eliminated).

\footnote{7} D. Brody, Steelworkers in America, 92 (1960).


\footnote{9} In re Hotel Bond Co., 89 Conn. 143, 93 A. 245, 247 (1915); Mackin v. Detroit-Timkin Axle Co., 187 Mich. 8, 153 N.W. 49, 51 (1915).

\footnote{10} State v. Clausen, 65 Wash. 156, 117 P. 1101, 1113 (1911).


\footnote{12} See, e.g., In re Hurle, 217 Mass. 223, 104 N.E. 336 (1914); but see Adams v. Acme White Lead & Color Works, 182 Mich. 157, 148 N.W. 485 (1914).
where 476 tunnel workers died and 1500 were disabled from dust exposures as short as nine months. The impact of subsequent silicosis legislation, however, was greatly diminished by various technical provisions designed to reduce the liability of employers.

The restrictive spirit of silicosis legislation was matched by legal obstacles to recovery for other diseases. Until the past few years, most states only compensated for diseases specifically listed by statute. Besides the obvious purpose of limiting the liability of employers, schedule coverage supposedly was intended to curb the discretion of judges and administrators by excluding coverage of diseases for which medical knowledge was then insufficient. Schedule coverage was enacted over objections that such specification was unfair to workers. Other provisions designed to restrict eligibility for occupational disease compensation are interspersed throughout the statutes.

While no exhaustive detailing is attempted here, the following examples illustrate the restrictive tenor of state occupational disease statutes. In Arkansas, the worker must prove the causation of disease by "clear and convincing" evidence. In many states, eligibility depends on whether the disability or death occurred within one year after the last injurious exposure to a workplace hazard. In others, disability or death from certain diseases is presumed not to be due to work exposure unless, within the past ten years, the worker was exposed for a two year period within the state and five years in all. South Carolina, a major

16. The following laws were common: no partial disability allowed for silicosis claims (19 S.C. ACTS § 72-255 (1962), now modified S.C. CODE § 42-11-60 (Supp. 1977)); statutes of limitation running from last dust exposure rather than from discovery of the disease (ALA. CODE tit. 25 § 25-5-147 (1975); N.C. GEN. STAT. § 97-58(a) (1972)); no compensation because of "wilful self-exposure" if uncomfortable safety gear is not worn (W. VA. CODE § 23-4-2 (1978)); a sliding scale of benefits which in the first few years paid awards significantly below those applicable to other compensation claims (N.Y. LAWS ch. 887 (4-A) (1936); GA. CODE ANN. § 114-815 (1973)); and liability limited to those workers who had been employed a certain minimum number of years by the last employer (ARIZ. REV. STAT. ANN. § 23-901.02 (Supp. 1977)) or who had worked a certain number of years within the state (GA. CODE ANN. § 114-814 (1973)).
17. At the last legislative session, Tennessee dropped its former schedule which was limited to twelve diseases. TENN. CODE ANN. § 50-1101 (Supp. 1977) (amending TENN. CODE ANN. § 50-1101 (1977)). In 1971, North Carolina revised its list to include twenty-eight named diseases. N.C. GEN. STAT. § 97-53 (1972).
18. See PENNSYLVANIA COMMISSION ON COMPENSATION FOR OCCUPATIONAL DISEASE, OCCUPATIONAL DISEASE COMPENSATION: REPORT TO THE GOVERNOR, at 18, 19, 27-28 (1934).
19. "Many workers are sacrificed under the present system of waiting until an occupational disease becomes fairly widespread before an attempt is made to provide a remedy and to grant compensation to the injured workers or their surviving dependents." R. GOLDBERG, OCCUPATIONAL DISEASES IN RELATION TO COMPENSATION AND HEALTH INSURANCE, 170 (1931).
textile-producing state, recently dropped a requirement that workers be exposed to harmful substances for at least one year, but added a specific provision that byssinosis claimants must have been exposed to dust in their employment for at least seven years.  

B. Basic Problems in Statutory Coverage

Perhaps the most significant limitation on coverage of occupational diseases is the statutory definition of disease itself. Some states provide for broad inclusion by defining "injury" to mean diseases "arising out of the employment." Twenty states cover only diseases which are "peculiar" to the worker's occupation. Several others exclude "ordinary diseases of life" to which the general public is exposed, regardless of whether work exposures can also cause them. The basic work-relation test poses significant problems for victims of diseases of undetermined origin and for claimants whose occupational disorders are difficult to distinguish from non-occupational conditions with similar clinical symptoms. The "peculiar risk" and "ordinary diseases of life" requirements add an unnecessary eligibility factor which often results in the elimination of valid claims for reasons unrelated to the compensatory purposes of occupational disease law.

Legal reforms can ameliorate the position of the occupational disease claimant. The "peculiar risk" and "ordinary diseases of life" tests, and other restrictions not consistent with the fair compensation of disease victims, can be eliminated by state or federal legislation. But legal redrafting, without more, will not substantially upgrade the adequacy of coverage. Rather, the basic obstacle to equitable protection of occupational disease victims is the fact that the workers' compensation system currently relies almost exclusively on medical evidence to determine the occupational causation of disease. After elaborating on this central role of medicine, the next section illustrates that there are inherent limits to occupational disease coverage if the law continues to resolve claims through medical testimony.

27. See, e.g., Kelly-Springfield Tire Co. v. Roland, 197 Md. 354, 79 A.2d 153 (1951) (claim for work-related asthma rejected when there was no proof that asthma had ever been contracted in either of the employer's rubber plants); Morrow v. Memorial Mission Hosp., 21 N.C. App. 299, 204 S.E.2d 543 (1974) (hospital mechanic cut hands fixing toilet, claim denied in part because no evidence was introduced to show that infectious hepatitis was peculiar to occupation).
C. Proof of Causation in Occupational Disease Claims

Determining the interrelation of a claimant’s disability and occupation is not a factually technical or complex matter in the majority of workers’ compensation cases. In a typical injury case, it is undisputed that a broken toe resulting from a tumbling crate is the consequence of a work event. Disputes instead center on whether the injury is one “arising out of and in the course of employment”—a test requiring legal, not technical, judgments. Expert medical evidence, when necessary, is used to evaluate the extent of disability, rather than to aid the fact-finder in determining the relationship of the injury to the work.

Legal and factual difficulties increase as the injury loses its close relation in time and space to the employment. Hernias, back injuries, heart disease, and mental disorder all present difficulties in ascertaining the extent to which the claimant’s work contributed to the injury. Such injuries commonly occur outside of the employment environment as manifestations of physical degeneration or mental stress. The distinction between occupational and non-occupational contributing factors is a difficult one, often requiring resort to expert medical opinion. However, where the injury followed closely in time after an identifiable traumatic work event, courts are not as rigid about requiring medical evidence to support the claim.29

The onset of most occupational diseases cannot be linked to a traumatic event. Instead, the claimant must demonstrate a causal relation between occupational exposure and disease to establish eligibility for compensation.30 For most disease claims, occupational causation of disease must be supported by expert medical evidence.31 Statistical evidence that the claimant’s occupation is associated with a disproportionally high incidence of a particular disease, without testimony about the individual claimant’s condition, may be insufficient proof to support a claim.32

Under existing compensation law, then, the outcome of occupational disease claims rests in the hands of medical witnesses. Courts and compensation boards consistently deny claims where medical experts know too little of the diagnosed condition to testify to its causal origin. For instance, compensation was refused where existing medi-

cal knowledge could not determine the causes of sarcoidosis, a form of muscle cancer, or whether exposure to benzene or xylene causes leukemia.

In limited situations, benefits have been awarded despite the absence of medical testimony linking an occupational exposure or traumatic event to the onset of disease. In *Volk v. Birdseye Division*, a cannery worker was awarded benefits for an eye disorder of unknown origin, based upon proof that a large mass of broccoli dusted with an unidentified powder struck the claimant’s eye. The court was satisfied by what it termed a “common-sense” inference that the impact of the dusted broccoli was connected with the immediate development of the eye disorder. Similarly, the claimant in *Valente v. Bourne Mills* received compensation for removal of her breast after it was struck by a flying object at work. No medical evidence was introduced to connect the disorder with the traumatic event. The court held that reasonable inferences from undisputed and unimpeached lay testimony could support the claim. The court disregarded a rule requiring medical evidence of a causal connection between the accident and the disease:

> [I]f literally followed [the rule] would turn a compensation case into a clinic where doctors seek to determine the “diagnosis” of a patient’s ailment and the “pathological nature” of that condition according to the more exacting norms of medical science. The application of so strict a rule to establish the required causal relationship to establish the required causal relationship in the field of law, where the ultimate objective is the attainment of substantial justice according to the remedial purposes and provisions of the act, would cast an unfair burden upon a person injured by accident.

The court admitted that medical evidence was necessary to support claims where there is a “seeming absence of connection between a particular accident and a claimed resulting injury.” The *Valente* and *Volk* cases are notable for the presence of an identifiable workplace incident and a close temporal sequence between the event and disease. The courts felt justified in overlooking the absence of medical testimony when a reasonable inference of causation could be drawn.

34. City of Jacksonville Police Dep’t v. Hobbs, 246 So.2d 561 (Fla. 1971).
38. 77 R.I. 274, 75 A.2d 191 (1950).
39. 75 A.2d at 194.
40. *Id*.
41. Courts commonly uphold awards for heart attacks when a work exertion occurred which could trigger the seizure. A. LARSON, THE LAW OF WORKMEN’S COMPENSATION § 38. The causal inference drawn from the fact that attack follows work-related exertion has been questioned by a committee of the American Heart Association, which states that work-related factors
Most occupational disease claimants will not be assisted by such an approach to proof of causation. Courts reject inferences derived from non-medical testimony when disease allegedly is caused not by a traumatic event but by years of gradual, continuous exposure to an occupational contaminant. The court in *Miller v. National Cabinet Co.*,\(^{42}\) refused to draw an inference of occupational causation without supporting medical evidence, ruling that "there comes a point even in legal thinking where the relationship of cause and effect becomes too attenuated to be regarded."\(^{43}\) The court indicated that claims for diseases of unknown cause could not rest on non-medical evidence without proof of a traumatic event followed by disease in the same spot, especially where there was a lapse in time between the ceasing of work and the discovery of disease. The court, in less than temperate terms, rejected the notion that occupational disease claims could be approved when evidence of causation was unclear:

[C]ourts will not in all instances demand scientific demonstration of cause and effect relationships but will insist only on practical probability . . . [But using the contention that inferences may be drawn which are unsupported by medical evidence of causation] as a fulcrum to establish a rule that a question of fact arises whenever a medical expert testifies in measured terms that an asserted cause of a disease is possible . . . is erroneous. Otherwise for so long as the causes of a disease — like cancer — are unknown to science, everyone contracting the disease could secure medical testimony that it is "possible" that the disease is contracted from a wide variety of causes, choosing in each instance the particular possibility having the greatest promise of holding liable some responsible defendant. [Unless proof of occupational causation is required,] for so long as the causes of the disease are unknown to medical science, the claimant or plaintiff can always recover—since no one can prove that the disease had other causes. This is a perversion of the normal rule that the disease must have resulted from the occupation and that the burden of proving causation is upon the party asserting it. The law does not intend that the less that is known about a disease the greater shall be the opportunity of recovery in court.\(^{44}\)

Isolated cases show more sympathy to the position of a claimant whose disease is of unknown or unascertainable origin.\(^{45}\)

\[^{42}\text{8 N.Y.2d 277, 168 N.E.2d 811 (1960).}\]
\[^{43}\text{168 N.E.2d at 816.}\]
\[^{44}\text{168 N.E.2d at 817, 818.}\]
\[^{45}\text{See McAllister v. Workmen's Comp. App. Board, 69 Cal.2d 408, 445 P.2d 313 (1968), in which the court reversed a decision rejecting the claim of a firefighter that his lung cancer was caused by smoke inhalation, not cigarette smoking. The defendant introduced no rebuttal evi-}\]
Shortcomings in medical knowledge do not generally alter the requirement that the claimant provide medical proof of causation. The practical result is that the burden of awaiting advances in medical sophistication is inextricably bound to the worker's burden of proving every element of a compensation claim.

II
MEDICAL LIMITS ON LEGAL CLAIMS

A. Problems in Proving Work-Relatedness

The law's reliance on medicine to determine causation limits the effectiveness of occupational disease compensation on two levels. First, the existence of an occupational disease must be established before the condition of a disabled worker can be linked to an employment exposure. Until the origin of a disease is identified, the worker's disability will be attributed to a non-occupational cause or an unknown source. The practical effect of tying compensation to the pace of medical research is to place upon the worker the entire cost of undiscovered occupational disease.

Second, even if a disease is known to be caused by a workplace substance and is highly prevalent, the diseased claimant will be unsuccessful unless the disease can be reliably diagnosed and differentiated from related non-occupational conditions. Medical knowledge that a disease exists is not necessarily useful at the level of individual diagnosis. Doctors might readily diagnose a certain disease, such as lung cancer, but be unable to confirm its occupational origin. Alternately, physicians may lack reliable clinical diagnostic tests to distinguish an occupational disorder from a common disease which duplicates the symptoms of the occupational disease. In either case, although the toxic effects of a workplace exposure are well documented, medicine cannot reliably determine the condition of the individual claimant.

B. Identification of Occupational Diseases

In 1910, an advocate of worker's compensation coverage for occupational diseases offered that "no very serious effects of industry have been observed except in the case of a comparatively small number of
dence on the toxicity of the pollutants inhaled by the claimant. The court took the following approach to the problem of identifying causation of disease:
Future scientific developments will tell us more about lung cancer. Ultimately it may be possible to pinpoint with certainty the cause of each case of the disease. But the Legislature did not contemplate years of *damnum absque injuria* pending such scientific certainty. Accordingly, we and the Workmen's Compensation Appeals Board are bound to uphold a claim in which the proof of industrial causation is reasonably probable, although not certain or "convincing." We must do so even though the exact causal mechanism is unclear or even unknown.

445 P.2d at 319. McAllister has not been followed by courts outside of California.
particular trades.  

Such optimism, based on the scant knowledge of industrial disease of the day, is no longer possible. The risk of infectious disease which was so great in the 19th century has been brought under control by improved public health measures. But that risk has been replaced by a drastic increase in exposure to new physical and chemical substances in the workplace. Dangers from coal mining and rock quarrying have been recognized for decades; only recently have health hazards been acknowledged in more routine factory work. Exposure to asbestos in mining, manufacture, and construction has been shown to produce not only asbestosis but also lung cancer and mesothelioma. Rubber and coke-oven workers are exposed to a greatly increased risk of contracting a variety of cancers. Polyvinyl chloride, once blithely considered safe by industry, is now known to cause liver cancer. Workers exposed to another widely used chemical, bis-ether, face a risk nine times greater than the general population of developing lung cancer.

The federal government has estimated that workers are exposed to 3,000 new chemicals a year. Estimates of those contracting occupational diseases vary from 390,000 to 500,000 per year. The impact on health of new industrial substances, singly or in combination, is virtually unknown. Of the 20,000 harmful substances on the government's Toxic Substance List, exposure standards have been set for only five hundred. Besides the continual introduction of new chemicals into the workplace, modern industrial practices have not necessarily diminished the risk of exposure. For instance, the rising incidence of byssinosis among textile workers has been attributed to the picking of cotton by machines and the increasing mechanization within the

54. J. STELLMAN & B. DAUM, WORK IS DANGEROUS TO YOUR HEALTH (1973).
Health hazards in the workplace have gone undiscovered for a variety of reasons. Medical surveillance of workers, monitoring systems to measure levels of contaminants, and systems to detect the early onset of disease have all been absent from American industry. Investigation of industrial contaminants has been hampered as well by the intransigence of many employers, who have nearly exclusive access to medical data on employees and the composition of the chemicals used in their manufacturing process. In notable instances, industries have obstructed the examination of employees and attempts to register contaminant levels. The recognition of berylliosis was delayed when many companies refused to reveal the number of workers exposed to beryllium in their operations. Researchers have also been excluded from several textile mills in the recent past.

The most important obstacle to identification of occupational causation is the nature of the diseases themselves. With few exceptions, the signs and symptoms of occupational diseases mimic those of common diseases. Moreover, individual physicians are in a poor position to recognize environmental causes of disease. Identification of occupational diseases requires a knowledge of toxicology which most general practitioners do not possess. Physicians in general are trained to diagnose and provide treatment. A concern for prevention — which calls for an understanding of the environmental or occupational causes of disease — is not a high priority among doctors. Often, little importance is placed on defining the origin of the patient's condition.

59. N. Ashford, supra n. 53, at 408.
60. The attempts of one asbestos manufacturer to deter investigation are detailed in P. Brodeur, The Expendable Americans (1974).
63. R. Schilling, Occupational Health Practice 29 (1973). For example, nausea, vomiting, or respiratory symptoms caused by exposure to chemicals are seldom distinct in nature from such symptoms produced by non-occupational sources. Dernehl, Chemical Exposure, Three Case Histories, 34th Congress on Occupational Health, Occupational Medicine Symposium, at VI-3 (1975). Recognition of non-fibrotic occupational lung disease has been delayed because clinical findings in such patients are quite similar to those caused by non-occupational conditions. Selikoff, Widening Perspectives of Occupational Lung Disease, 2 Preventive Med. 412 (1973).
64. A. Bouhuys, supra n. 48, at 275; Tabershaw, Acceptability of Risks to the Health of Workers, 18 J. Occup. Med. 674 (1976).
65. R. Schilling, supra n. 63, at 29.
66. Tabershaw, supra n. 64, at 675.
67. Id. See also, Lee, Comments and Perspectives, Multiple Factors in the Causation of Environmentally Induced Diseases 224 (Fogarty Int'l Cen. Proc. No. 12 (1972).
Even if the treating physician is motivated to investigate causal factors, available techniques to him limit his capacity to do so. The doctor must often rely on the patient's description of the environmental factors to which he or she has been exposed.68 The worker, who is often uninformed of the invisible work hazards to which he or she is subject, may be unable to provide necessary information.69 While lung function tests will reveal lung impairment, they usually will not identify the source of the problem; patients with no industrial exposure too often show similar patterns.70 In practice, the physician's effort to ascertain occupational information is generally not extensive.71 "The likelihood [that the doctor will note] the precise type of work performed and the chemical, solvent, and physical stresses to which the patient was exposed is not high."72

Because of the difficulty in distinguishing occupational diseases by other means, epidemiological research is often the sole method available to identify the occupational causation of diseases to which the general population also is susceptible.73 Epidemiology determines cause of disease by investigating the association between incidence of disease and exposure to specific industrial contaminants.74

Epidemiological "cohort" studies compare the condition of large numbers of exposed workers with a control group not exposed to the contaminant. An estimate of the risk of contracting disease through workplace exposure can be made by comparing the health record of the two groups over a period of time. "Case-control" studies take an opposite approach. In such studies, the two groups are those with and those without a particular disease. The frequency of exposure to an industrial contaminant is then calculated from each group. While cohort studies are more reliable, they take longer to conduct and are more expensive than case-control studies. Moreover, case-control studies are especially useful for investigating less common diseases. Unfortunately, such studies often rely on past work, and medical records which

68. A. BOUHUYS, supra n. 48, at 275.
69. [An occupational questionnaire] is often complicated and difficult to take. The worker may have forgotten dates of employment and dates of short, but critical, work periods. His own beliefs about the causes of his disease may even make him forget to mention his work or deny its importance. Through ignorance, fear of losing a job, advice from fellow workers, a lawyer, or his family, some patients may also be reluctant to reveal their work exposures.

70. McKerrow & Gilson, Lung Function and its Measurement in Industrial Pulmonary Disease, MODERN TRENDS IN OCCUPATIONAL HEALTH 33, 46 (1960); Trieff & Corrigan, supra n. 50, at 123.
71. Trieff & Corrigan, supra n. 50, at 133.
72. Id., at 110.
73. R. SCHILLING, supra n. 63, at 409; L. CRALLEY & P. ATKINS, INDUSTRIAL ENVIRONMENTAL HEALTH 1 (2nd ed. 1975).
74. R. SCHILLING, supra n. 63, at 169.
are incomplete or collected for other purposes. Other epidemiological methods, such as proportional mortality studies, are used to obtain indications of excess hazards associated with work.

Epidemiology reveals statistical associations between incidence of disease and exposure to contaminants, which are then checked by laboratory experiment. Therefore, epidemiology necessarily is a study of probabilities. The major challenge for epidemiologists is to determine when an observed relationship between exposure and disease is merely a statistical association and when it is causal. Three factors are important to the decision: 1) the relationship in time between the exposure and illness; 2) the strength of the association; and 3) whether the observed connection is consonant with existing medical knowledge and evidence of the distribution of the disease among the general population. In the absence of direct experiment to identify the responsible causal agent, determining cause is "neither easy nor objective."

Epidemiology has established the occupational origin of several conditions of previously unknown etiology. Continued investigation will undoubtedly result in the continued identification of harmful occupational contaminants. Epidemiological advances have aided workers in many industries by compelling preventive measures and providing the basis for compensation claims. It would be a mistake, however, to allow progress in occupational disease law to await the results of epidemiological research. The detection of many industrial diseases is probably beyond the reach of epidemiology. The expense and time involved in such studies limit the number of workplaces and industries which can be profitably examined; not even a strong public health effort could keep up with the continual introduction of untested substances into the workplace. Effective epidemiological investigation is also restricted to the more potent carcinogens and other contaminants. If a toxic agent is relatively weak, the increased incidence of disease in the exposed group over that of the control group may be undetectable. Detection of less potent toxic agents requires large study populations, which precludes studies in smaller industries.

The most significant limit on the efficacy of epidemiology is the fact that many occupational diseases do not arise until thirty years or longer after initial exposure. This passage of time can obscure the

76. For a general discussion of epidemiological methods, see R. Schilling, supra n. 63, at 169-89; B. MacMahon & T. Pugh, Epidemiology: Principles and Methods (1970).
77. B. MacMahon & T. Pugh, supra n. 76, at 21.
78. Id.
79. B. Dinman, supra n. 75, at 10.
80. Selikoff, supra n. 63, at 427.
81. Hueper, Occupational and Environmental Cancers of the Respiratory System, 3 Recent Results in Cancer Research 27 (1966); Mancuso, supra n. 56, at 22.
discovery of causal relationships, as workers quit work where they were exposed or leave the industry altogether. Studies which do not allow for long latency periods may produce "false negative" results, where disease is not detected due to premature collection of data. The present danger associated with workplace exposure can only be estimated from epidemiological studies, since current studies of diseases with long latency periods are actually measuring the result of workplace exposures of twenty or thirty years ago. Current exposure levels may be different, and substances used in production may have been discontinued or newly introduced. Conversely, studies conducted today will not yield results for decades for the many diseases whose onset is delayed.

Another problem is that epidemiology is most effective when used to study substances in isolation. This allows experimentation with and identification of specific hazards. For industries where exposure is limited to a particular contaminant, such as cotton dust, causal inferences will be strong. But for many industries, such as rubber manufacture, exposure to hundreds of chemicals is quite common. This simultaneous exposure reduces the ability of the epidemiological method to isolate occupational causation in these industries, since neither the exposure nor the disease produced by it are clearly identifiable. "Mixed" exposures may also hamper diagnosis of occupational disease in the individual worker as well as the threshold identification of a hazardous workplace exposure. Irving Selikoff, a prominent investigator of asbestos, has suggested some implications of mixed exposures for the diagnosis of disease:

There has been increasing realization that the reason why "classical" clinical and x-ray findings of specific pneumoconioses are not always seen, is that complex rather than "pure" exposures are common or, in some circumstances, probably the rule. An important condition in which this is true is foundry workers' lung disease, where crystalline silica, silicates, metal dusts, and high temperatures may all combine to modify the effects to be expected from any one of these agents. When we add to these the additional powerful influence of cigarette smoking, the blurring of diagnostic outlines can be appreciated. Very little is known of the number of individuals who might be affected by mixed exposures, and still less is known of the pathogenetic implications of the interactions among two or more dusts simultaneously present in human lung. They, too, form an area of inquiry of considerable interest, since environmental lung disease is likely to be far more frequently

82. Selikoff, supra n. 63, at 427.
83. Mancuso, supra n. 55, at 22.
84. Selikoff, supra n. 63, at 430.
85. Mancuso, supra n. 55, at 22.
86. Selikoff, supra n. 63, at 413.
associated with the presence in lung of a variety of substances rather than one or two. 87

C. The Diagnosis of Occupational Disease

The significance for workers' compensation law of a gap in medical knowledge is not exhausted by demonstrating the medical problems in identifying occupational causes of disease. Medical obstacles to recovery are equally great to a disabled worker suffering from an occupational disease for which diagnosis is difficult. If the suspected occupational disease is also an “ordinary disease of life,” such as liver cancer caused by polyvinyl chloride, the claimant not only confronts a legal barrier 88 but also the necessity of showing that his or her disease is in fact caused by the work exposure.

If the suspected disease is peculiar to a specific work exposure, such as asbestosis, silicosis, byssinosis, or berylliosis, there is no need to show a causal link since there is no non-occupational counterpart. The problem instead is to prove, for example, that the condition is actually byssinosis or berylliosis instead of a non-occupational lung disorder. That element of the medical proof is not necessary in the case of occupational carcinomas, for which the identity, if not the cause, is easily determined.

In either case, epidemiological evidence of the presence or prevalence of the occupational disease in working groups is of little assistance in an individual claim. The task of epidemiology is largely fulfilled when an increased prevalence of disease is demonstrated in an exposed group as compared to a control group; 89 detailed techniques useful at the individual level may not be developed. Similarly, the prevention and hazard-identification purposes of epidemiology, which are directed towards the implementation of measures to control the hazard, do not require the development of detailed clinical diagnostic techniques required for individual workers. 90

Finally, in the case of common diseases which can have occupational origins, such as lung cancer, and occupational diseases such as byssinosis, which have no non-occupational counterpart but are difficult to distinguish from other diseases, a particular case cannot be assigned occupational origin merely because there is knowledge that such diseases exist among workers. 91 The following sections examine byssi-

88. See notes 24 through 27 and discussion in text.
89. A. BOUHUYS, supra n. 48, at 275.
90. Id.
91. Recent medical evidence underscores the prevalence of occupational disease. For instance, rubber tire workers exposed to workplace toxins for at least ten years are reported to have
nosis to illustrate the difficulties in diagnosing occupational diseases which mimic the symptoms of related non-occupational conditions.

III

BYSSINOSIS: PROBLEMS IN DIAGNOSING OCCUPATIONAL DISEASE

A. The History of Byssinosis

Byssinosis is a prime example of a major occupational disease which was virtually unrecognized in the United States until recently. In 1705, Ramazzini noted that workers preparing flax and hemp for spinning suffered from coughs and "asthma" from dust. In Great Britain, textile workers' union representatives complained of chronic bronchitis and emphysema among workers in the carding rooms as early as 1908. A government study performed in the early 1920's found that British cardroom workers died much more frequently from respiratory diseases than did the general population. In 1927, the British Home Secretary appointed a committee to investigate the relationship between dust in textile mill cardrooms and disease.

Following intensive study, byssinosis became a compensable disease in Great Britain in 1941. As ventilation and engineering controls became more common in English mills, byssinosis was thought to be vanishing; this proved untrue. Chronic bronchitis remained highly prevalent among mill workers. A yearly loss of lung capacity (FEV₁) four times that of a control group. Fine & Peters, Respiratory Morbidity in Rubber Workers, 31 ARCHIVES OF ENVT'L HEALTH 5-14 (1976). Rubber curing room workers had an increased risk of contracting lung cancer, and those exposed to solvents were susceptible to lymphatic leukemia. McMichael, supra n. 49. Top-side coke-oven workers are seven times more likely to develop lung cancer than non-exposed individuals. Lloyd, supra n. 49. Workers exposed to bis-ether run a nine-fold risk of lung cancer. Lemen, supra n. 51. But cancers and diminished lung capacity are also common to the general population. Absent a distinctive element separating occupationally-induced cancers and other diseases from their non-occupational twins, the disabled worker's claim will fail.

Take, for example, the matter of whether dust exposure can cause bronchitis. In 1966, a British study concluded that exposure to dust is not a significant variable in determining the prevalence of bronchitis in miners. Medical Research Council, Chronic Bronchitis and Occupation, 1 BRIT. MED. J. 101 (1966). A current text on lung diseases concludes that some occupational exposures can cause bronchitis. W. MORGAN & A. SEATON, OCCUPATIONAL LUNG DISORDERS 265-73 (1975). The authors further contend that "[t]he symptoms of bronchitis, namely, persistent cough and phlegm, when produced by occupational exposure are no different from those seen in subjects suffering from chronic bronchitis in the absence of significant occupational exposure." Id. at 267. If "occupational bronchitis" does exist, the number of disabled workers excluded from benefits by legal definition and medical uncertainty is compounded.

92. B. RAMAZZINI, A TREATISE OF THE DISEASES OF TRADESMEN (1705).
94. Id. at 77.
95. Id. at iv.
prevalent in mill towns. Occupational mortality statistics in the late 1940's indicated that textile workers were at high risk for heart disease. Since byssinotics often die from right heart failure, the misinterpretation of mill workers' health probably stemmed from physicians' observations of only the most advanced cases in the hospitals. Subsequent epidemiological studies in the 1950's found that two-thirds of cardroom workers handling coarse cotton had byssinosis to some degree.

Despite a half-century of accumulated knowledge in Great Britain, the widespread existence of byssinosis was not acknowledged in the United States until 1961. United States Public Health Service studies in the 1920's and 1930's found that a disproportionately high number of textile mill workers suffered from respiratory illness. Since no questions were asked about symptoms on the first day of the work week, the studies failed to relate the high rate of lung disease to cotton dust. The medical literature in the 1940's periodically reported isolated cases of byssinosis. But not until 1961, after two British researchers conducted the first modern epidemiological study in the United States, was byssinosis identified as a disease common to American textile workers. Since then, scores of studies have been performed, frequently over the active opposition of the textile industry. The incidence of byssinosis of various grades has ranged from 25% to 41% of cardroom workers and 12% to 22% of spinners.

The most current and extensive research indicates that at least 35,000 current and retired mill workers have chronic, disabling byssinosis. The study compared the lung function and respiratory symptoms of several hundred mill workers with a similar number of "controls" who had no contact with cotton dust. Taking into account

98. HAMILTON & HARDY, supra n. 69, at 465.
99. J. MORRIS, supra n. 95, at 197.
100. Schilling, Byssinosis in Cotton and Other Textile Workers, LANCET 261, 319 (1956).
102. Id.
104. McKerrow & Schilling, supra n. 99.
105. Dr. Arend Bouhuys, an early byssinosis researcher, was refused permission to study workers by every textile mill owner in Georgia. He finally conducted his research in a textile mill within a federal penitentiary in Atlanta. Another doctor, based in North Carolina, failed to gain permission to enter mills in that state for several years. The mill owners told him that "there was no evidence of a disease, and that we should mind our own business." Washington Post, January 1, 1978, at B-4.
106. Harris, Merchant, Kilburn, & Hamilton, supra n. 94.
non-work related factors such as sex, age, and smoking habits, cotton dust exposure was the most significant variable explaining respiratory symptoms in textile mill workers. It was also isolated as the cause of poor lung function among mill workers as compared with controls.108

B. Chronic Byssinosis: Diagnosis by Questionnaire

1. General Method

Despite recent advances in epidemiological knowledge, the diagnosis of chronic byssinosis in individual cases remains problematic. Recent medical research has refined techniques for detecting individual acute cases of byssinosis. Such methods are useful for determining whether workers at risk of developing disabling byssinosis should be transferred to less dusty areas of mills.109 However, reliable diagnostic procedures are still unavailable for chronic byssinosis. Since workers’ compensation provides only for economic incapacity, not medical impairment, the diagnosis of chronic rather than acute byssinosis is the real test of the adequacy of compensation law.

The diagnostic problems of chronic byssinosis are compounded by the range of similarities between byssinosis and other lung disorders. Byssinosis is only distinguished from non-occupational lung diseases by its distinctive pattern of initial symptoms. Chest tightness and other symptoms appear upon the exposure to cotton dust on the first working day after a weekend or vacation. Symptoms may disappear after the end of the shift. Those workers who react more severely to dust report chest tightness on the second workday and occasionally on succeeding days. Those with the most advanced conditions experience continued chest tightness regardless of dust exposure. Continued exposure over a working lifetime can produce a disabling shortness of breath.110 Doctors have few tests by which chronic byssinosis can be distinguished from such common lung diseases as chronic bronchitis and emphysema. Byssinosis cannot be identified by chest x-ray, because it does not alter the structure or visual appearance of the lungs.111 If any abnormalities are observed upon physical examination, they are indistinguishable from alterations caused by bronchitis or emphysema.112 Autopsies of those with byssinosis reveal changes only in the airways,

108. While smoking contributed to an excessive loss of lung function, smoking habits could not explain the difference in lung function between textile workers and the control group. Id. at 182.


111. Schilling, Byssinosis, CLINICAL ASPECTS OF INHALED PARTICLES 70 (1972).

112. A. BOUHUYS, supra n. 48, at 418.
which is consistent with chronic bronchitis or asthmatic bronchitis.\textsuperscript{113} Most importantly, the symptoms of chronic byssinosis cannot be distinguished from those of chronic bronchitis and emphysema in a clinical examination.\textsuperscript{114}

The problems of diagnosing chronic byssinosis become more clear upon comparison to the manner in which acute byssinosis is identified. Two procedures are available by which acute byssinosis can be reliably diagnosed. The diagnostic tool first used for byssinosis is a questionnaire administered to the worker, covering occupational history, respiratory symptoms, and the standard elements of a medical history.\textsuperscript{115} The work history elicits information about every job in the textile mill which the worker has held, including the number of years spent in each. Questions are asked about the history and intensity of cough, phlegm production, shortness of breath, and chest tightness. The worker is asked to describe any present respiratory problems, including any particularly severe chest tightness or shortness of breath.

Diagnosis of byssinosis by questionnaire hinges on the worker's response to that question. Since symptoms of other lung diseases are more stable throughout the week, a response that symptoms are more severe on the first work day of the week identifies cotton dust exposure as the source of the disease. If the worker so responds, byssinosis is diagnosed and a grade of severity is assigned. The grade is determined by the frequency of "Monday" symptoms and their occurrence during the remainder of the work week.\textsuperscript{116}

The occupational questionnaire has been supplemented by the use of a number of lung function tests. Such tests, performed by blowing into a spirometer,\textsuperscript{117} measure lung capacity and efficiency of ventilation. One test in particular, the FEV\textsubscript{1},\textsuperscript{118} has been used to measure the lung function of textile workers. A worker's FEV\textsubscript{1} is measured prior to the first working day of the week and again at the end of that workshift. If the second FEV\textsubscript{1} reading is significantly below the first, the worker has acute but possibly reversible byssinosis.\textsuperscript{119} According to at least one lung specialist, the drop in FEV\textsubscript{1} over a workshift is sufficient

\begin{itemize}
    \item \textsuperscript{113} Edwards, Macartney, Rooke, & Ward, \textit{The Pathology of the Lung in Byssinotics}, 30 \textit{THORAX} 612-23 (1975).
    \item \textsuperscript{114} W. Morgan & A. Seaton, \textit{supra} n. 89, at 278.
    \item \textsuperscript{115} A standard byssinosis questionnaire is reproduced in 17 \textit{J. OCCUP. MED.} 462-66 (1975).
    \item \textsuperscript{116} A. Bouhuys, \textit{supra} n. 48, at 417. A similar grading system is based on the results of lung function tests. \textit{Id.}, at 436.
    \item \textsuperscript{117} A spirometer measures air capacity of the lungs. \textit{Taber's Cyclopedic Medical Dictionary} at S-81 (13th ed. 1977).
    \item \textsuperscript{118} The FEV\textsubscript{1} test measures the volume of air expelled in the first second of a forced expiration following maximal inhalation. Make, \textit{Medical Surveillance for Occupational Respiratory Diseases}, 17 \textit{J. OCCUP. MED.} 519, 520-22 (1975).
    \item \textsuperscript{119} W. Morgan & A. Seaton, \textit{supra} n. 89, at 279; A. Bouhuys, \textit{supra} n. 48, at 417.
\end{itemize}
to diagnose acute byssinosis, even where there has been a negative finding based on the occupational questionnaire. Others allow only that a decrement in FEV$_1$ over a workshift confirms a positive diagnosis based on the questionnaire.

The use of both diagnostic tools can produce a reliable diagnosis of acute byssinosis. The validity of spirometric tests depends on patient cooperation and correct procedure. But if properly performed, lung function tests offer objective evidence of dust-related lung impairment to complement the necessarily subjective questionnaire approach.

The textile worker asserting a claim of chronic disabling byssinosis, however, does not benefit from the recent progress in the diagnosis of acute byssinosis. Workers' compensation claimants commonly have left work years before normal retirement age due to disabling lung disease. Doctors hesitate to subject such workers to the dust exposure sufficient to take workshift FEV$_1$ tests because they are so short of breath. Indeed, chronic byssinotics often show no workshift changes in lung performance measured by lung function tests. An FEV$_1$ or other lung function test can measure the extent of permanent lung impairment at any time. But such tests, without a link to actual dust exposure, cannot pinpoint the cause of the worker's impairment.

The diagnosis of chronic byssinosis might be improved if more were known about the relationship between acute and chronic byssinosis. Medical studies have reported that chronic impairment is much more likely to occur among workers who previously were acute reactors. For current claimants and those in the near future, there is little possibility of confirming the diagnosis of chronic byssinosis through workshift FEV$_1$ tests taken in earlier years. Information on individual workers' acute dust reactions is not generally available; reliable tests and medical surveillance of workers by mills are rather recent develop-
ments. Due to the absence of the relatively objective FEV$_1$ evidence, the questionnaire is the only practical tool for diagnosing chronic byssinosis.\footnote{128}

2. \textit{Weakness of the Questionnaire Method of Diagnosis}

The necessary reliance on the questionnaire for the diagnosis of chronic byssinosis significantly reduces the fairness to diseased claimants of conventional workers' compensation law. Diagnostic methods such as lung function tests and chest x-rays offer a relatively objective basis for interpretation by a physician. In comparison, a diagnosis based solely on a questionnaire is completely subjective. The reliability of an occupational questionnaire itself turns on subjective factors: the worker's perception of his or her sensations; the worker's description of those sensations to the examiner; the perceptions of the examiner; and the interpretations by the examining physician based on all the information received from the worker.\footnote{129} The examiner's technique therefore assumes great importance.\footnote{130} The sections below elaborate on how some of these factors interact to cast serious doubt on the validity of diagnosis by questionnaire.

\textit{a. The Examiner's Ability to Interview}

First, the reliability of the questionnaire rests in large part on whether the examiner is able to elicit truthful and complete responses from the worker. The physician must be aware of the potential health hazards created by workplace exposure to chemicals and dust. Physicians commonly neglect to inquire sufficiently into a patient's occupational history.\footnote{131} The diagnosis of occupational lung disease in particular is difficult because of the need to know details of the production processes at the worker's factory and type of the agent present in the specific work environment.\footnote{132} The doctor must possess enough knowledge of textile production and byssinosis to know both what questions to ask and what the answers reveal.\footnote{133}

Even assuming sensitivity of the physician to occupational diseases, certain pitfalls plague history-taking in general. Studies have identified predispositions of physicians to find or not find abnormal

\footnote{128} R. Parkes, \textit{supra} n. 126, at 438. Diagnosis of chronic byssinosis is even more problematic if the worker has chronic bronchitis as well. \textit{Id.}, at 439.

\footnote{129} A. Feinstein, \textit{Clinical Judgment} 312 (1967).

\footnote{130} R. Parkes, \textit{supra} n. 126, at 438.

\footnote{131} Trieff & Corrigan, \textit{supra} n. 50, at 110.


\footnote{133} For instance, the doctor's evaluation of the worker's exposure to dust depends on his or her knowledge that a card room is dustier than a finishing room. Make, \textit{supra} n. 118, at 519.
The expectation that a disease will act in a typical way may prevent the examiner from recognizing atypical cases. This may be a special problem in the diagnosis of byssinosis, where such importance is placed on the sequence of symptoms over a working life.

The attitude, approach, and vocabulary used by the examiner are also crucial. One byssinosis researcher claims that the prevalence of byssinosis in surveys can be altered significantly merely by the tone and emphasis of the examiner’s questions. Additionally, the social, political, and medical predispositions of the interviewer may prevent accurate exchange of information, or affect the doctor’s own interpretation of the claimant’s condition.

The importance of the examiner’s ability to elicit accurate information is underscored by the fact that it is virtually unassailable in a compensation proceeding. Though proof of bias would invalidate medical testimony, the interviewing skill of the physician, as a discrete element of technical competence, is a difficult matter to question. Moreover, errors in history-taking would be difficult to detect, since no witnesses are usually present during such an examination.

b. Observer Variability

A second factor which diminishes the validity of questionnaires is the problem of variance in diagnosis among several persons examining a single patient. Equity in compensation law is furthered when different physicians, confronted with a single case, reach conclusions consistent with each other. But where different examiners reach different results, it is difficult to determine whether the disagreement is based upon the actual signs and symptoms of the patient or is the result of some other inconsistency among the observers.

Several studies have shown that observer variability in the administration of questionnaires is a major error factor in diagnosis. In a

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135. A. Feinstein, supra n. 129, at 309; Selkoff, supra n. 87, at 203.
136. A. Feinstein, supra n. 129, at 313.
137. W. Morgan, personal communication with the author.
138. The studies on observer variability are used to test the validity of prevalence data generated by questionnaires in epidemiological surveys. The setting in which questionnaires are administered in surveys differs from that involving clinical diagnosis of a single patient. Schilling maintains that variability will be less where there is more than one opportunity for observation. Schilling, supra n. 132. The implicit assumption is that there will be little variability among experts in the diagnosis of byssinosis. However, specialists who perform examinations for byssinosis for compensation claimants commonly see patients only once, and the number of specialists is small. Workers may be diverted from the compensation examination process altogether by the erroneous diagnoses of the family physicians they originally consulted.
study of observers who administered questionnaires for chronic bronchitis, the range of identification of breathlessness went from a low of 17% of all cases by one doctor to a high of 38% by another, and that of phlegm from 21% to 34%. The causes of the discrepancies were investigated. Over 60% of the disagreement was attributed to the observer, only 20% to different replies by the subject, and the remainder to differences in interpreting borderline answers. Another study, conducted to measure the prevalence of bronchitis as determined by a history of cough and sputum, concluded that "the recorded answers to several questions commonly asked in taking clinical histories were not reproducible from one observer to another, so they could not be considered objective scientific observations."

Observer variability has been found to exist as well in byssinosis examiners. Schilling found that two observers, using the standard questionnaire, disagreed on 24% of byssinosis diagnoses. The discrepancy was attributed to the examiners' bias towards or against finding abnormality, which in turn influenced the workers in their replies. While such variability does not invalidate statistical integrity in a long-term epidemiological study, variability is a critical factor in the diagnosis of individual patients. It is questionable whether such a high degree of divergence in medical opinion is acceptable in compensation proceedings, where determination of causation so summarily assigns medical costs among the parties.

c. The Worker's Ability to Convey Accurate Information

The third feature which limits the usefulness of questionnaires in compensation proceedings is their dependence on the accuracy of the worker's response to questioning. The validity of the byssinosis questionnaire in particular places prime importance on reliable responses by workers, since a diagnosis of byssinosis by this method depends upon a finding of chest tightness on the first day of the workweek. Even a truthful recounting of present symptoms is less important than an accurate memory. Since chronic byssinotics experience constant chest tightness, diagnosis hinges on the memory of chest pains on certain isolated days many years ago. Researchers have recognized the memory problem of disabled older workers:

In its final stages, byssinosis cannot be distinguished from nonoccupational chronic bronchitis and emphysema except for the past history of

141. Schilling, supra n. 134.
142. R. PARKES, supra n. 126, at 437.
chest tightness characteristically worse at the beginning of the working week. The textile worker with byssinosis often forgets his early symptoms and is diagnosed as suffering from non-occupational chronic respiratory disease.\footnote{143. Schilling, supra n. 109, at 72.}

The potential for mis-diagnosis of workers with poor memories is not the only deficiency that results from the use of the questionnaire without benefit of lung function evidence. As noted above,\footnote{144. See notes 117 through 121 and discussion in text.} acute byssinosis can be diagnosed by evidence of a reduction in FEV$_1$ over the first workshift of the week regardless of responses to the questionnaire. But the questionnaire method, which is based only on obvious symptoms, cannot detect byssinotic workers who were impaired by dust but did not experience noticeable chest tightness. Such workers are regularly found in epidemiological studies.\footnote{145. A. Bouhuys, personal communication with the author.}

Surveys have suggested that responses of workers to questions are poorly reproducible. The accuracy of responses has been tested by interviewing workers twice to see if the same answer to the same question is given to both questionnaires.\footnote{146. Fairbairn, Wood, & Fletcher, supra n. 139, at 186. After an initial survey of textile workers, a researcher conducted a repeat survey nine months later. Of 34 persons complaining of chest tightness on the first workshift of the week, only fifteen positively answered at both interviews. Braun, Jurgiel, Kaschak, & Babyak, Prevalence of Respiratory Signs and Symptoms Among U.S. Cotton Textile Workers, 15 J. OCCUP. MED. 414-19 (1973). However, the time of year in which the questionnaire is taken may affect the results. Respiratory symptoms have been found to be more severe and ventilatory capacity lower in winter than in summer. R. SCHILLING, supra n. 63, at 193.}

The phenomenon Schilling reported\footnote{147. See note 141 and discussion in text.} and the inherent defects of history-taking are probably responsible for the discrepancies that appear. Such low duplication of responses further dilute the value of the questionnaire as an accurate diagnostic tool. “While there is little doubt that Schilling’s modification of the MRC questionnaire is a useful means of quantifying the prevalence of byssinosis in epidemiological studies, as a diagnostic aid in the individual it is of much less value.”\footnote{148. Morgan, supra n. 122.}

3. Work-relatedness, Medical Techniques, and Byssinosis

The above discussion questions the wisdom of legal recourse to medical techniques to identify the occupational causation of cases of byssinosis. The byssinosis questionnaire appears incapable of accurately determining the existence of chronic byssinosis. Unfortunately,
most of its inaccuracies result in a diagnosis that byssinosis is absent, when it may actually have been contracted.

It may be the nature of present diagnostic knowledge that no methods are available which can perfectly differentiate similar diseases from one another. Ideally, the claims of two workers, both of whom are impaired and have a history of dust exposure, should vary in result only because the origins of their disease are different. That ideal is diminished to the extent that diagnostic techniques are inaccurate. Perhaps workers' compensation law should not demand unreasonable exactitude when medical expertise may be helpful in resolving the legal issue of causation. At some point, however, medical diagnosis will be so imprecise that the law irrationally excludes workers from coverage by relying on it. Has far from the ideal of error-free diagnosis can the law go without violating its purpose to recompense workers for occupationally-induced conditions?

Medical evidence is most useful to the workers' compensation process when the presence of an occupational disease can be detected by a foolproof method. The ideal method would exclude all possible explanations for the disease except the presence of a factor pointing unmistakably to occupational causation. But medical testimony is of little assistance when no reliable test is available to diagnose occupational disease. There, the distinction of the condition from a non-occupational disease must be based on speculation.

It is accepted that physicians may differ in the application of their medical judgment to a particular case. For many conditions, the disease process is sufficiently understood and the technology which detects it reliable enough that professional differences are based on rational interpretation rather than conjecture. In such cases, disputes among expert physicians as to the importance of particular data ultimately further the purpose of classifying the condition as occupational.

The professional judgment of physicians merits consideration only insofar as their opinions are based on reliable procedures. A diagnostic method should satisfy three tests before it is considered sufficiently accurate to be used in compensation proceedings concerning occupational disease. First, the procedure must actually measure what

149. For example, shadows more than one centimeter in diameter on the chest x-ray of a coal miner are clear evidence of progressive massive fibrosis unless there are definite signs of other lung disease. W. MORGAN & A. SEATON, supra n. 91, at 193.

150. For example, questions may exist about the meaning of the cloud on the x-ray, or whether the level of toxin revealed by a blood test is sufficiently high to cause the disorder.

151. For example, a dispute over the interpretation of an x-ray shadow or toxin level would be meaningless if x-ray technology could not reproduce an image with requisite clarity, or if repeated tests of the same blood produced different levels of toxin.
it purports to; it must be able to isolate and report the factor sought from it, and reject other variables which might also explain the result. Second, the mechanism which produces the data must be internally reproducible — able to replicate its results within a tolerable limit of deviation. Third, the result of the test should be capable of duplication by other physicians; this is the "observer variability" factor discussed above. A degree of such variability is acceptable in epidemiological research, since the different judgments of observers evens out statistically when a large population is studied. No such correction of variability is available to benefit the individual claimant, who will see only one or two doctors.

The diagnostic method used to detect chronic byssinosis fails all three criteria of reliability. The occupational questionnaire is most often the sole diagnostic procedure available to distinguish byssinosis from such presumably non-occupational conditions as chronic bronchitis and emphysema. We have seen how the evidence suggests that the answers to the questionnaire are not reproducible by repeat interviewing, and that the degree of observer variability in the questionnaire process is high. But the most significant failing of the questionnaire is that it does not reliably measure what it is intended to. Many variables, extraneous to the presence of chest tightness on the first work day of the week, can confound the outcome of the process. Weakness of memory of the claimant, the attitudes, perceptions, and technical knowledge of the examining physician, and defects in communication between them all interfere with the accurate exchange of essential information.

By relying on the questionnaire to decide the legal issue of causation, the worker's compensation system excludes two classes of diseased workers from benefits: those whose workshift chest tightness was undetected by the physician taking the history, and those who suffered from acute byssinosis without experiencing the classic symptoms of workshift chest tightness. Workers in these two categories are no less deserving of compensation than other diseased workers, yet continued reliance on the questionnaire method of diagnosis assures that their claims will fail.

For byssinosis and a myriad of other diseases of occupational origin, medical knowledge is not sufficient to reliably distinguish occupational disorders from non-occupational conditions which are clinically

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152. A test which analyzed the amount of lead in blood by the shade of green produced by dropping a unit of blood into a solution would be meaningless, for instance, if the chemical properties of arsenic also turned the solution green.

153. R. SCHILLING, supra n. 63, at 194.

154. Recent research indicates that chronic bronchitis might also have occupational origins. See note 91.
identical. The diagnostic method to which the law turns in byssinosis cases is too uncertain to justify the claim of the workers' compensation system that it separates claims on the basis of work-relatedness. Although some byssinotic workers are compensated, general statistics for occupational disease\textsuperscript{155} indicate that the majority are not.

IV

LEGAL BURDEN, SOCIAL DEBT

That so many workers fail to discharge their legal burden has little to do with legitimate distinctions about the nature of their individual disabilities. Rather, it is directly related to the shortcomings of medical knowledge. In this context, workers suffering from diseases not yet linked to work exposure and workers who have occupational diseases which medicine cannot reliably diagnose are in a similar position. All must wait upon the progress of medical research, as an inextricable result of their burden of proof. Such a result can be tragic to the individuals involved. These twin burdens belie the social purposes of workers' compensation by transferring costs properly attributable from industry to individual workers generally unable to bear them.

Assigning the costs of a worker's disability is ultimately a legal, not a medical, issue. As long as the law requires that distinctions be made on the basis of work-relatedness, some method must be devised to determine the cause of a claimant's disease. Current medical techniques cannot satisfactorily resolve the work-relatedness issue for byssinosis and many other diseases of occupational origin.

Once it is concluded that knowledge is presently insufficient to resolve a legal issue, the law must turn to some other mode of analysis for making decisions. The theory of workers' compensation is that injuries and diseases arising as a part of production are properly attributable to the employer and consumers of the product, not to the individual worker. In a real sense, those who work in a contaminated environment sacrifice their health to the profit of employers who, for whatever reason, do not provide a healthy workplace. Short of quitting, workers have no control over whether they will contract a disease.

Responsibility for the initial research and surveillance necessary to identify hazards and for reduction of contaminant levels should rest with industry. Compensation itself is little solace for the suffering and reduction of life span caused by occupational diseases. To the extent that disabled workers remain uncompensated for diseases arising from employment, the cost-allocation purposes of workers' compensation are unfulfilled.

\textsuperscript{155} See notes 3 and 91.
These policy considerations, taken in conjunction with the impos-
sibility of scientific proof, justify the shift of the burden of proof to the
employer in workers’ compensation cases. The next section focuses on
how such a shift has been implemented for one disease, and why it
should be considered for others.

V
TOWARD FAIR COMPENSATION OF OCCUPATIONAL DISEASE
VICTIMS

A. Existence of Occupational Disease by Statutory Presumption

Triggered by a 1968 West Virginia coal mine explosion which
killed 78 miners, thousands of mine workers organized to fight for
workers’ compensation coverage for “black lung disease,” or coal
worker’s pneumoconiosis (CWP). Their activity culminated in a three-
week strike which closed down nearly all of West Virginia’s mines and
resulted in a revamping of that state’s compensation law for CWP.156
Fourteen months after the explosion, Congress enacted the Federal
Coal Mine Health and Safety Act of 1969.157 That Act and its amend-
ing legislation, the Black Lung Benefits Act of 1972,158 fundamentally
altered the role of medicine in occupational disease claims. Instead of
deferring to the medical profession, Congress found that the available
diagnostic methods for CWP produced results inconsistent with the un-
derlying social purposes of workers’ compensation.

In effect, federal black lung legislation has replaced medical crite-
ria for linking disability to work exposure with specially designed legal
standards for determining occupational causation of CWP. The 1969
Act contained three presumptions which aided the claimant. First, it is
rebuttably presumed that a miner with CWP who worked over ten
years in an underground coal mine contracted CWP as a result of his
work.159 Second, it is rebuttably presumed that a miner’s death was
attributable to CWP if he died from a respiratory disease after having
worked in underground coal mines for more than ten years.160 Third,
the Act created an irrebuttable presumption that a miner with compli-
cated CWP, defined in the statute, is totally disabled due to CWP.161

If complicated CWP is diagnosed, therefore, the miner is relieved
of the burden of proving both occupational causation of the condition
and the extent of disability. The regulations construing the 1969 Act

156. The efforts of coal miners to secure workers’ compensation for black lung disease are
discussed in B. HUMEN, DEATH IN THE MINES 92-152 (1971).
also set out the required medical procedures for diagnosing the more common form of the disease, "simple" CWP. Diagnosis was to be based on a chest x-ray, autopsy, or biopsy;\textsuperscript{162} other possible diagnostic methods were excluded. Since biopsies were prohibited for solely compensation purposes, the claims of living miners turned on the results of the chest x-ray.

Despite their novelty, the presumptions (with one exception) did not substantially aid miners. Because of the severity of the condition and relative ease of diagnosis, miners with "complicated" CWP least needed the aid offered by that presumption. The living claimant with "simple" CWP still carried the burden of proving the existence of the disease and the resulting disability. That burden was restricted by the regulations' limitation on acceptable diagnostic procedures. While the presumption for causation of CWP triggered after ten years in the mines did alter the conventional allocation of burden of proof, it probably had little practical effect. Proof of the existence of dusts and fumes in the workplace, easily obtained by workers in such a notoriously dusty industry, is generally sufficient to implicate employment as the cause of an occupational disease.\textsuperscript{163}

The administration of the 1969 Act soon came under attack for its high rate of denied claims. As of March 1972, more than half of the claims filed had been denied. Of those denied, 62% were rejected because of the claimant's failure to provide positive x-ray evidence of CWP.\textsuperscript{164} Following several hearings in coal mining areas, amendments to the 1969 Act were drafted and enacted as the Black Lung Benefits Act of 1972.\textsuperscript{165}

In the 1972 Act, Congress made two basic changes in the claims procedure for simple CWP. No longer could any claim be denied solely on the basis of a negative x-ray.\textsuperscript{166} Claims are to be decided instead on the basis of other medical tests, a medical history, the opinion of the miner's physician, affidavits from lay persons, and other "relevant evidence."\textsuperscript{167} The other important 1972 amendment was the creation of a rebuttable presumption for those whose conditions had not been diagnosed as complicated CWP.\textsuperscript{168} If a miner or one claiming under him can show at least fifteen years of underground work and

\begin{itemize}
  \item \textsuperscript{162} 20 C.F.R. § 410.404(a) (1970).
  \item \textsuperscript{163} A. Larson, supra n. 41, § 41.50.
  \item \textsuperscript{165} P.L. 92-303, 86 Stat. 150 (1972).
  \item \textsuperscript{166} 30 U.S.C. § 923(b) (Supp. V 1975).
  \item \textsuperscript{167} 30 U.S.C. § 923(b) (Supp. V 1975).
  \item \textsuperscript{168} 30 U.S.C. § 921(c)(4) (Supp. V 1975).
\end{itemize}
total disability due to respiratory or pulmonary impairment, it is presumed that his disability is or was due to CWP.

Many members of the medical community believed that the modifications in the 1972 Act were scientifically unsupportable. Most experts contended that the diagnosis and progression of CWP could only be determined by chest x-ray. Some asserted that simple CWP was associated with only slight abnormalities in lung function which, considered in isolation from the effects of other diseases, could not produce respiratory impairment.

But other experts convinced Congress that the medical evidence did not all point in one direction. Testimony was presented which indicated that the non-existence of CWP could not be determined merely on the basis of a negative chest x-ray. One study cited to Congress found that x-rays, when compared with autopsies of the same miners, yielded erroneous diagnoses in 25% of the cases examined. Other evidence suggested that emphysema can cloud x-rays and prevent detection of CWP by x-ray alone. It was hypothesized that inadequate training and facilities in mining areas resulted in x-rays of poor quality.

Congress disposed of the clashing medical evidence by "resolve[ing] doubts in favor of the disabled miner... [i]n the absence of definitive medi-

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172. Id., at 12.

173. Id.


175. A frequently cited problem was that of x-ray film quality. In processing claims under the black lung program, physicians depended on x-rays taken at several locations around the country. Not all were equipped to adhere to rigorous radiological standards. Reger, Smith, Kibelstis, & Morgan, supra n. 169. Over-exposure of the film results in an under-estimation of the progression of CWP. Reger's study found that 24% of the films were rated mildly defective to very poor.

Observer variability in the interpretation of films is another error factor. One study found disagreements on the categorization of CWP among different observers ranging from 18.8% to 33.2% of the time. Despite such substantial differences, the authors insist that variability did not invalidate the use of radiography in the diagnosis of CWP. Reger & Morgan, On the Factors Influencing Consistency in the Radiologic Diagnosis of Pneumoconiosis, 102 AM. REV. RESP. DISEASES 905-15 (1970). Another study designed to formulate recommendations on the administration of the black lung program found that positive diagnoses of CWP varied from 57% to 94% of all cases. Morgan & Donner, supra n. 134.
cal conclusions. 176

The statutory presumption of causation preserves the work-rela-
tion concept of workers' compensation law. The causal link between
disease and employment is established legislatively on an aggregate
level, subject to disproof in individual cases. The causal inference is
now based on two factors which are unrelated to a determination of the
origin of an individual miner's condition: the period of dust exposure
of the miner, and the degree of the miner's physical impairment. Taken
by themselves, neither factor has been considered significant in com-
ensation law. The fifteen-year requirement is a measure of
probability among a class of workers. It does not identify that a partic-
ular claimant has suffered from that exposure. Such statistical evi-
dence would clearly be immaterial in a claim brought under state
compensation law. Similarly, evidence of medical impairment alone,
without proof of its source or nature, would not support a typical com-
ensation claim.

Nevertheless, each factor is related to the probabilities of occupa-
tional origin of the individual claimant's condition. The fifteen-year
minimum exposure period is a recognition that coal miners suffer a
statistically greater chance of contracting CWP after an extended pe-
riod of time spent working underground. The dust exposure require-
ment is the legal criterion of causation which supports the placement of
the risk of liability on the employer. By requiring evidence of totally
disabling respiratory impairment, the presumption maintains the cen-
tral element of compensation law — economic disability.

The presumption does not abandon what guidance medicine can
offer. Medical evidence still can be submitted either to show that the
claimant's respiratory disability is non-occupational, or that the envi-
ronment in which the particular miner worked was not sufficiently
dusty to cause a totally disabling condition. But the burden of produc-
ing and proving such rebuttal evidence is now on the employer. This
apportionment of the burden is appropriate; the probability that occu-
pational exposure causes or contributes to disability should preclude
imposing all the consequences of medical uncertainty on the stricken
worker. A particular employer's aggregate loss to employees under
such a presumption will approximate the epidemiological probability
that the employer was the cause of the individual worker's ailment.
Thus, the legal presumption approach can be defended on both epide-
miological and social policy grounds, since the compensation principle
justifies distribution of the human costs of production where medical
evidence has revealed a prevalence of occupational disease in an indus-
try.

B. Use of Legal Criteria to Determine Occupational Cause

The statutory presumptions embodied in the Black Lung Acts reflect Congress' unwillingness to base the determination of cause for compensation purposes on medical grounds alone. The presumptions are based upon evidence that existing diagnostic tests cannot distinguish CWP from non-occupational disorders with a sufficient degree of reliability. Medicine had demonstrated an association between years of exposure to coal dust and the probability of a miner contracting CWP, but, for individual miners with a record of over fifteen years dust exposure, medical expertise no longer determines causation. The Black Lung presumptions represent the instatement of legal tests for resolving a legal issue — how to properly allocate the costs of a worker's disability.

The shift to legal criteria for determining causation is laudable. The most persuasive reason for designing new causal tests is the incapacity of medicine to discover and diagnose occupational diseases with the accuracy presently required by the workers' compensation system. Medical research has only begun to identify the subtle effect on health of work exposures. So long as diseased workers are compelled by law to advance medical proof of causation, claimants in industries which have yet to be intensively investigated for toxic exposures will be excluded from coverage.

Even assuming the success of increased efforts by epidemiologists to identify hazardous workplace exposures, the law's coverage will not be necessarily improved. Unless the newly discovered occupational disease has signs or symptoms which allow it to be distinguished, the diseased worker is no better off. No longer is it assumed that there is but one cause for each disease: "[I]dentical disease manifestations—or at least manifestations that cannot be distinguished on the basis of present knowledge—may be associated with quite different basic constellations of etiologic factors."177

The new scientific awareness of the "multiple-factor etiology" concept of disease directly contradicts the law's current assumption that the occupational causation of diseases can be scientifically detected in an acceptable number of cases. In short, the law asks of medicine something it cannot deliver. An English respiratory disease specialist has reached a similar conclusion:

It might be expected that increase in knowledge would make it easier to devise an ideal compensation scheme, but in pneumoconiosis the reverse has occurred . . . . Recent work has also emphasized the importance of other factors, such as smoking and atmospheric pollution, in producing the bronchitis, with the result that even in large groups of

177. MacMahon, supra n. 47, at 6.
subjects it is difficult to determine with certainty what part of the disability is occupational and what is due to these other factors. In the individual it is virtually impossible except in extreme cases, and as a result it becomes difficult to operate satisfactorily an industrial injuries benefit scheme based on the premise that in the majority of claims it will be possible to decide with reasonable certainty in the individual that the injury has been caused by an identifiable industrial hazard. . . . A reconsideration of the whole problem is needed. The separation of industrial and non-industrial respiratory disability is no longer realistic in most cases, and it is, perhaps, more logical to provide adequate benefits for those who are severely disabled from whatever cause.178

Tests do exist for some diseases by which the effect of work exposures can be determined. The law, however, cannot be satisfied by the fact that recourse to medicine to determine causation in some instances is fair. Instead, fairness demands knowledge as to whether or not the diagnostic techniques can accurately detect all such cases of occupational disease. Diagnostic techniques which yield a substantial number of "false negative" results screen from coverage workers who actually have the disease. Byssinosis falls within this category. While chronic byssinosis can be diagnosed by questionnaire, the existence of weaknesses in the diagnostic method means that an untold number of workers with chronic byssinosis are falsely diagnosed as having chronic bronchitis. The law’s assertion that it fairly decides claims cannot survive diagnostic tests that fail to produce rational and consistent results.

The shift to legal methods of determining disease causation can also be supported on a more theoretical ground. Law and medicine pursue different objectives in deciding the cause of disease. Even within medicine, treating physicians may approach the concept of cause differently than epidemiologists, who are more concerned with prevention.179 The physician treating a diseased worker may be either untrained180 or unconcerned181 about determining cause. The practitioner’s interest is the mechanism of the body which results in signs and symptoms. This more narrow concern may produce a different concept of significance than that possessed by an epidemiologist, who in-

179. B. MacMahon & T. Pugh, supra n. 76, at 26.
180. Tabershaw, supra n. 64; Trieff & Corrigan, supra n. 50.
181. To the extent that the basic causes [of the condition] are buried in the irretrievable past, and particularly where the pattern varies with the individual, [the doctor] may well feel that a precise definition of etiology, on which disease classification tends to be based, is not necessary, or even possible. (Under existing compensation laws and practices, of course, the fixation of "blame" for a person's disease assumes large legal importance, and its establishment will continue to plague all concerned.) Lee, supra n. 67, at 224.
vestigates causal events on the environmental rather than physiological level. While the epidemiologist is responsible for identifying occupation-related causal factors, it is the practitioner to whom the worker must currently turn for testimony.

The law requires sufficient evidence before it will place liability for disease on the employer. It calls for social judgments, not technical ones. When law turns to medicine to resolve the causation issue, the medical expert necessarily uses medical standards of proof in reaching a conclusion. Strict standards for identifying causal relationships are justified in medicine, since research and treatment may require precise knowledge. But because of the differing objectives of law and medicine, the law should not use the same standard to allocate the costs of disease that doctors use for experimental and therapeutic purposes. The consequences of employing a strict medical standard of probability is to confer on employers the benefits of medical caution, while burdening disabled workers with the costs of medical uncertainty.

VI
CONCLUSION

There has been growing awareness, at least on the national level, of the unfairness to workers of relying exclusively on medicine to detect occupational disease. The National Commission on State Workmen’s Compensation Laws acknowledged the difficulties in diagnosing disease, but evaded the issue by recommending the use of a specialized “disability evaluation unit” to determine questions of causation. The proposed National Workers’ Compensation Act of 1975 left the question of disease diagnosis to the administrative process, although it did authorize the use of presumptions. The follow-up group to the National Commission recommended the use of epidemiological evidence to help establish the work relationship of a claimant’s condition. Admission of epidemiological knowledge to shift to the employer the burden of showing that a claimant’s condition did not arise out of employment would be a major advancement.

The use of statutory presumptions is a necessary step to secure to diseased workers the benefits of workers’ compensation. Their use is appropriate for diseases whose origin cannot be reliably diagnosed and for which epidemiological research has identified work exposure as a probable cause of disability. Care must be taken, however, not to per-

183. REPORT OF THE NATIONAL COMMISSION ON STATE WORKMEN’S COMPENSATION LAWS 51 (1972).
185. POLICY GROUP report, supra n. 3, at 12.
mit rebuttal evidence based on the same inaccurate diagnostic information which established the need for a presumption. For instance, a presumption that the respiratory disability of a textile mill worker with a history of exposure to cotton dust should not be rebutted by the fact that he or she did not report Monday chest tightness.

But even presumptions are of limited usefulness. They cannot aid workers suffering from diseases not yet determined to be occupationally induced. Since they are based on separate factual determinations for each disease, there is also the danger of spotty coverage like that which existed under the former "schedule" disease coverage of state statutes.

It is important not to lose sight of what is at stake in reforming occupational disease coverage. Workers' compensation awards at best offer the disabled worker limited benefits to ward off destitution. Increasing use of statutory presumptions and other methods to resolve occupational disease claims would result in compensation for many more workers. Reformers should anticipate objections similar to those directed against the black lung program, decrying the cost of more thorough occupational disease coverage and the conversion of workers' compensation into a "general health insurance program."

Such arguments miss the point. Because of the inequity of present occupational disease laws, workers have borne costs of hazardous and unhealthy workplaces which are properly attributable to employers who fail to eliminate such hazards. The law's reliance on medicine to determine causation has created only a pretense of statutory protection for the victims of occupational disease. Revision of the law's treatment of occupational disease, either within or outside the workers' compensation system, is essential to end the rule of the doctrine of *damnum absque injuria* in our workplaces.

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