

**LINDQUIST v. CITY OF JERSEY CITY FIRE
DEPARTMENT**

**Richard LINDQUIST, Petitioner-Appellant, v. CITY OF JERSEY CITY FIRE
DEPARTMENT, Respondent-Respondent.**

Argued Sept. 23, 2002. -- February 11, 2003

Thomas W. Polaski, Freehold, argued the cause for appellant (Gary P. Sarlo, attorney). John H. Geaney, Mt. Laurel, argued the cause for respondent (Capehart & Scatchard, attorneys).

The opinion of the Court was delivered by

The issue raised in this appeal is whether petitioner's employment as a fireman for approximately twenty-three years caused or contributed to his development of pulmonary emphysema within the meaning of the occupational disease provisions of the Workers' Compensation Act. Resolution of that issue requires us to decide how much workplace contribution is enough to trigger employer responsibility. The Judge of Compensation found that petitioner's occupational exposure materially contributed to the development of emphysema. The Appellate Division reversed, finding that the evidence was insufficient to establish medical causation between the employment and the emphysema. We disagree and reverse.

I.

Petitioner Richard Lindquist was employed as a full-time paid fireman with the City of Jersey City Fire Department from July 1972 until his retirement in January 1995. He was promoted to the rank of captain in 1979. Petitioner testified that during the first ten years of his employment, he responded to "30 to 60 large fires per year," "small one-room" fires, car fires, and "dump" fires. When he began his job in 1972, each firefighter was given a self-contained breathing apparatus, "but it was just very new and people didn't seem to use it until 1982." Although petitioner was exposed to "heavy smoke" for up to forty-five minutes to an hour and a half during larger fires, he frequently did not use the apparatus. In respect of smaller fires, petitioner described the duration of exposure to smoke as follows:

Well, I guess basically outdoor fires you try to stay upwind if you can, but a lot of times they are grass fires that-down where Liberty State Park is now and the Liberty Science used to be, it is all grass area, which still has pretty much been an abandoned grass area

that many times will catch on fire and the whole area would be lit, and you would be completely engulfed in smoke at the time, you couldn't get away from it.

Some of the fires involved burning chemicals, plastics, household cleaners, and propane.

In 1982, respondent distributed to all firefighters a protective device known as a Scott mask. The mask provided oxygen or "purified air" to the user. Petitioner testified that the Scott mask "did a pretty good job" of preventing fumes from entering the breathing passages. Nonetheless, he still inhaled smoke that seeped into the mask. He also frequently removed the mask to give orders to other firefighters and to clear moisture that accumulated on the mask.

From 1986 to 1992, petitioner was assigned to supervise the Hazardous Materials Unit of the fire department. During that time, petitioner responded to both residential and industrial fires. The burning items consisted of plastics and chemicals, causing "much more toxic smoke than the '70s and '60s." After 1992, petitioner returned to his position as captain.

Prior to commencing employment with respondent, petitioner had not experienced any problems with breathing or with his eyes, nose, or throat. He testified that during his tenure with the fire department, he was taken to the hospital numerous times. On other occasions he was administered oxygen at the scene of a fire. He suffered from shortness of breath, heart palpitations, and weakness. After responding to a toxic dump fire in 1991, petitioner and "most of the other [firefighters]" were sent to the hospital to test for arterial blood gases. Although not hospitalized, petitioner experienced throat irritation, rapid breathing, and dizziness.

Petitioner retired in 1995 at the age of forty-seven, due in part to an early buyout offer and in part to health considerations. At the time of his retirement, petitioner was less able to perform his responsibilities as a firefighter, and in particular as captain, because his energy and normal breathing capacity gradually had diminished. According to petitioner, he developed a "post nasal drip which would result in phlegm and coughing" that was "pretty constant" during his employment years. Now, those symptoms occur "[m]aybe two to three times a week." He also suffers from dry eyes and shortness of breath and is no longer able to play basketball with his son or take long walks with his wife. He is able to walk only one quarter to one half of a mile "before [he begins] breathing heavily." He cannot perform yard work or house work, such as "building sheds, [and] putting [together] decks," without some difficulty. In 1995 or 1996, petitioner began seeing a physician who prescribed treatment with a bronchodilator one or two times per week. The treatment relieves petitioner's symptoms "almost instantly."

Petitioner smoked approximately three-fourths of a pack of cigarettes per day for twenty-two years, stopping in 1992 or 1994. During the 1970s, he had a second job driving an oil truck. From 1982 through 1989, petitioner worked in residential construction in addition to his employment with the City of Jersey City. Shortly after leaving the fire department, petitioner became employed as a school bus driver.

Dr. Bernard Eisenstein testified on petitioner's behalf. Dr. Eisenstein specializes in heart and lung medicine and is Board Certified in internal medicine. He performed a complete examination of petitioner on January 16, 1995, to evaluate his pulmonary disability. In addition to the physical examination, Dr. Eisenstein performed a chest x-ray, and pulmonary function studies. The physical examination was "essentially negative, except [for] some areas of expiratory wheezing in the thorax." However, the chest x-ray was "abnormal [and] revealed increased bronchovascular markings with large lung volume, which . is compatible with emphysema." The doctor explained that petitioner's x-ray revealed "a hyperinflation . [indicating] . [that petitioner] has too much air in there [and] expiratory obstruction." The results of the pulmonary function studies were "only a little abnormal."

Based on those tests, Dr. Eisenstein concluded that petitioner suffered from "chronic obstructive pulmonary disease in the form of emphysema." He attributed petitioner's condition primarily to occupational exposure as a firefighter to fire, smoke, hazardous waste, combustion, and secondarily to cigarette smoking. However, he was unable to allocate an exact percentage to each cause. Specifically, Dr. Eisenstein stated:

Firefighters get bronchitis, firefighters get emphysema, and in my experience of many years you can't tell looking at an x-ray that this is due to cigarettes and this is due to work because of an occupation . In other words, emphysema can be caused by many things, as I said, so there's nothing characteristic as an occupation by a fireman, and they get a certain x-ray. There are certain changes seen in smoke inhalation, but he also can have these changes due to his exposures.

The doctor concluded that, "based upon a reasonable degree of medical probability," petitioner suffered "30 percent of partial total" permanent disability. On cross-examination, Dr. Eisenstein admitted that he could not cite any studies in which non-smoking firefighters developed emphysema.

In response to Dr. Eisenstein's testimony, respondent presented the testimony of Dr. Douglas Hutt. Dr. Hutt is Board Certified in internal, pulmonary, and critical care medicine. During his examination of petitioner on December 19, 1996, petitioner informed Dr. Hutt that his primary symptom was a post-nasal drip that began one year after he retired from the fire department. Petitioner also told the doctor that "he really wasn't very short of breath and could do all of his normal [activities] including normal walking and even walking upstairs and even doing some exercise including some mild jogging." However, petitioner advised Dr. Hutt that he noticed "a subtle difference in his ability to exercise and he really wasn't sure if this was related to some underlying medical condition or breathing problem or possibly because he was just getting older." Dr. Hutt further testified that petitioner "did not think this was a major problem at the time" of the examination. He noted that petitioner "did not remember any long term symptoms that he had after any of the . exposures to any of the [] bad fires." Additionally, petitioner told Dr. Hutt that "he smoked about three quarters of a pack [of cigarettes] a day for [twenty-two] years and that . his family pressured him to stop smoking about five years before [the] interview, but he did not stop because he was

having breathing problems.” He also told Dr. Hutt that his brother and sister suffer from allergies and that his grandfather died from emphysema.

Dr. Hutt performed a complete physical examination of petitioner including a chest x-ray, which revealed that petitioner's lungs were over-inflated and “that the lung fields themselves are very, very big.” Finally, Dr. Hutt performed a complete set of pulmonary function tests. The results showed that petitioner “had some mild airflow obstruction,” that he suffers from “air trapping,” which means that petitioner retains almost three and a half liters of air in his lungs after a complete exhale, and that his diffusing capacity—the ability of the lungs to transfer oxygen from the air to the bloodstream—was moderately to severely reduced. Dr. Hutt stated that petitioner's reduced diffusing capacity “correlates with destruction of lung tissue, lung injury, possibly scarring between the alveoli, which are the small air sacs, and the capillaries, which are small blood vessels” and “almost always [indicates] lung injury.”

Based on the physical examination and the diagnostic testing, Dr. Hutt concluded that petitioner suffers from emphysema caused by petitioner's cigarette smoking. According to the doctor, “even though only [twenty percent] of people that smoke cigarettes actually get emphysema, that number is [between seventy and eighty percent] higher if you have relatives that smoke cigarettes and get emphysema which is true in this patient's family in his grandfather.” He concluded that petitioner suffered “approximately [thirty percent] pulmonary impairment.”

Dr. Hutt acknowledged that many studies show that firefighters suffer from air flow obstruction and chronic bronchitis; however, he stated that “many of these studies . did not account for cigarette smoking” and “[n]one of the studies that [he had] seen . demonstrated conclusively or in any way that firefighters are [at] a greater risk for developing emphysema.” Thus he concluded that

[s]ince there's no data to suggest firefighters with exposure on the job can develop emphysema, the patient definitely has emphysema, he has a smoking history, which is clearly the most common cause of emphysema, and there's a strong family history of emphysema which increases your risk of developing emphysema from cigarette smoking, I believe that within a reasonable degree of medical probability that the patient's emphysema is related to his cigarette smoking.

According to the doctor, out of approximately “a hundred” studies concerning firefighters and lung disease in general, none address emphysema but rather deal with air flow obstruction, chronic bronchitis, and other “more serious diseases.” He stated that he had not “seen [studies] that specifically mention emphysema as an increased risk when you factor out cigarette smoking in firefighters.” Dr. Hutt further testified that one significant difference between exposure to firsthand cigarette smoke and occasional exposure to smoke from fires is that cigarette smoking continuously exposes the airways to smoke, whereas occupational smoke exposure to the airways during fires is sporadic.

In response to questions by the court, Dr. Hutt stated that “chemical exposures aren't generally felt to cause emphysema.” He admitted, however, that he could not say “with 100% certainty . that some of the exposure that [petitioner] might have had on his job may [not] have in some small way contributed to the development of the lung disease.”

The Judge of Compensation concluded that “petitioner's occupation[al] disease is due in a material degree to the occupational exposures described” during the trial. The judge also determined that petitioner had suffered an “appreciable impairment of [his] ability to carry on the ordinary pursuits of his retirement lifestyle.” The judge awarded petitioner a disability of thirty percent for emphysema.

On appeal, the Appellate Division reversed in an unpublished opinion, concluding that “the evidence of the causal connection between petitioner's employment and his emphysema is insufficient to sustain the award.” The court noted that the primary requirement of N.J.S.A. 34:15-31 is that “petitioner's disease be caused, to a material degree, by conditions in the workplace.” The second requirement of the statute, according to the panel, was “that the conditions contributing to the compensable disease must be characteristic of or peculiar to a particular trade.” The court concluded that the testimony of Dr. Eisenstein that “ ‘exposure to hazardous wastes played a significant role in causing [petitioner's] emphysema, and the cigarettes . played a less dominant role,’ ” and his inability to assign a percentage to each cause, was “insufficient to show that petitioner's work exposure exceeded the exposure caused by his smoking cigarettes.” (Emphasis added). The court also observed that Dr. Eisenstein relied “solely on petitioner's general characterizations of his work exposures over the years and not on any existing medical, epidemiological or scientific studies establishing causation.”

The court determined that petitioner's argument that given his testimony and Dr. Eisenstein's testimony, “ ‘respondent should have to come forward with definite medical evidence specifically excluding development of emphysema to one's work as a firefighter,’ ” impermissibly “shift[ed] the burden of proof to respondent.” In the court's view, “petitioner . bears the burden of showing by a preponderance of the evidence that his emphysema is caused in material degree by the conditions of his work environment.”

We granted petitioner's petition for certification, 171 N.J. 442, 794 A.2d 181 (2002), and now reverse.

II.

Petitioner argues that the Appellate Division exceeded the scope of its appellate review and ignored testimony in the record that provided an evidentiary basis to support medical causation. He also contends that, absent any contrary legislative history, N.J.S.A. 34:15-43.2 should be interpreted as creating a presumption that his pulmonary disease arose from his employment as a paid firefighter. By implication, petitioner argues that if the presumption applies, his cigarette smoking and the death of his grandfather from emphysema did not rebut the presumption that petitioner's emphysema is causally related to his exposure as a firefighter. Finally, petitioner contends that the higher standard

adopted in *Fiore v. Consolidated Freightways*, 140 N.J. 452, 659 A.2d 436 (1995), with respect to dual causation should be limited to cardiovascular injuries and was applied improperly by the Appellate Division.

III.

Preliminarily, the answers to all the issues presented must be informed by certain well-established general principles. When our Workers' Compensation Act (Act), N.J.S.A. 34:15-1 to -128, originally was enacted in 1911, L. 1911 c. 95, it provided no coverage for occupational diseases. See *Hichens v. Magnus Metal Co.*, 35 N.J.L.J. 327 (1912). The Act was amended thirteen years later to include occupational diseases. L. 1924, c. 124. Even then, only nine specifically enumerated diseases were covered: “anthrax, lead poisoning, mercury poisoning, arsenic poisoning, phosphorus poisoning, poisoning from all homologues and derivatives of benzene, wood alcohol poisoning, chrome poisoning, and caisson disease.” *Millison v. E.I. du Pont de Nemours & Co.*, 101 N.J. 161, 175, 501 A.2d 505 (1985). Eventually, the Act was amended to “replace[] its limited list of specific-named occupational diseases with a definitional phrase, ‘compensable occupational disease.’” *Ibid.* (quoting L. 1949, c. 29). “The current definition of compensable occupational disease is [contained] in N.J.S.A. 34:15-31[(a)].” *Millison*, *supra*, 101 N.J. at 175, 501 A.2d 505. That subsection defines “compensable occupational disease [s]” as those diseases established by a preponderance of the credible evidence to have arisen “out of and in the course of employment, which are due in a material degree to causes and conditions which are or were characteristic of or peculiar to a particular trade, occupation, process or place of employment.” N.J.S.A. 34:15-31a (emphasis added). “Material degree” means “a degree [substantially] greater than de minimis.” *Dwyer v. Ford Motor Co.*, 36 N.J. 487, 493-94, 178 A.2d 161 (1962); see also N.J.S.A. 34:15-7.2 (defining material degree as “an appreciable degree or a degree substantially greater than de minimis”).

Consequently, the history of occupational disease coverage under the Act has evolved from providing no coverage at all, to providing coverage for nine specifically enumerated diseases, to the current statutory approach of providing a general definition of compensable occupational diseases. That history “suggests either that the occupational disease risks of the workplace are too numerous to list separately or that in the future employees may contract occupational diseases, as yet unknown, that should nonetheless be compensated under the terms of the Act.” *Millison*, *supra*, 101 N.J. at 176, 501 A.2d 505.

Another principle of general application is that the Act “involved a historic trade-off whereby employees relinquished their rights to pursue common-law remedies in exchange for automatic entitlement to certain, but reduced benefits whenever they suffered injuries by [compensable] accident.” *Id.* at 174, 501 A.2d 505. See generally Richard A. Epstein, *The Historical Origins and Economic Structure of Workers' Compensation Law*, 16 Ga. L.Rev. 775 (1982) (discussing the stages of development of workers' compensation systems); Arthur Larson, *The Nature and Origins of Workmen's Compensation*, 37 Cornell L.Q. 206 (1952) (describing history and characteristics of

workers' compensation). “[T]he quid pro quo . was that employees would receive assurance of relatively swift and certain compensation payments, but would relinquish their rights to pursue a potentially larger recovery in a common-law action.” Millison, *supra*, 101 N.J. at 174, 501 A.2d 505. That concept is sometimes referred to as the social compromise theory because of both the gain and the loss experienced by employees and employers alike. Ellen R. Peirce and Terry Morehead Dworkin, *Workers' Compensation and Occupational Disease: A Return to Original Intent*, 67 *Or. L.Rev.* 649, 653 (1988).

Consequently, when the Division of Workers' Compensation and appellate courts are called upon to decide whether a particular occupational disease is causally related to a particular employment, they should utilize the original bargain rationale for workers' compensation and ergonomics to assist with the determination. Ergonomics refers to the applied science for the workplace that is broadly defined as “[t]he science relating to man and his work.” *Stasior v. National R.R. Passenger Corp.*, 19 F.Supp.2d 835, 847 (N.D.Ill.1998) (quoting *Dorland's Illustrated Medical Dictionary* 574 (28th ed.1994)). Ergonomics and other applicable principles should be the lens through which the determination of whether emphysema is an occupational disease that is related to a firefighter's exposure should be viewed.

Still another well-established principle is “the social policy of liberally construing the Act,” which is social legislation designed “to implement the legislative policy of affording coverage to as many workers as possible.” *Brower v. ICT Group*, 164 N.J. 367, 373, 753 A.2d 1045 (2000) (citing *Saunderlin v. E.I. DuPont Co.*, 102 N.J. 402, 419, 508 A.2d 1095 (1986)); *Secor v. Penn Serv. Garage*, 19 N.J. 315, 319, 117 A.2d 12 (1955). The same doctrine is applicable whether the claim involves an accidental injury or an occupational disease, or whether the focus is on a well-established or a modern health condition. But the bargain and the occupational disease statutory history contemplate that what constitutes a compensable occupational disease will be affected by many social and industrial factors that vary across time. At the very heart of the “original bargain . [is the notion that the burden of proof on claimants] would be easier . under workers' compensation laws than under common law.” Jason M. Solomon, Note, *Fulfilling The Bargain: How the Science of Ergonomics Can Inform the Laws of Workers' Compensation*, 101 *Colum. L.Rev.* 1140, 1156 (2001). Nonetheless, the doctrine of liberal construction does not extend to “‘the evaluation of credibility, or of weight or sufficiency of evidence.’” *Oszmanski v. Bergen Point Brass Foundry, Inc.*, 95 N.J.Super. 92, 95, 230 A.2d 151 (App.Div.1967) (quoting *Page v. Federated Metals Div.* 71 N.J.Super. 59, 63, 176 A.2d 290 (App.Div.1961), certif. denied, 38 N.J. 302, 184 A.2d 418 (1962)), certif. denied, 51 N.J. 181, 51 N.J. 181, 238 A.2d 468 (1968) (emphasis omitted).

It is also well-established that a successful petitioner in workers' compensation generally must prove both legal and medical causation when those issues are contested. “[T]he question of whether an injury occurred ‘by accident’ is interwoven with the issue of medical causation.” *Joy v. Florence Pipe Foundry Co.*, 64 N.J.Super. 13, 21, 165 A.2d 191 (App.Div.1960) (citing 1 *Larson, Workmen's Compensation Law*, §§ 38.82,

38.83 at 563-67), certif. denied, 34 N.J. 67, 167 A.2d 55 (1961). To establish causation in an occupational disease case, an employee must satisfy a two-part test by producing evidence to establish both (a) legal causation, and (b) medical causation. *City of Tuscaloosa v. Howard*, 55 Ala.App. 701, 318 So.2d 729 (Civ.App.1975), overruled on other grounds, *Gunter v. Borden, Inc.*, 680 So.2d 276 (Ala.Civ.App.1996); *In re Briggs*, 138 N.H. 623, 645 A.2d 655, 659 (1994). Medical causation means the injury is a physical or emotional consequence of work exposure. Stated another way, proof of medical causation means proof that the disability was actually caused by the work-related event. *Hone v. J.F. Shea Co.*, 728 P.2d 1008, 1011 (Utah 1986). Proof of legal causation means proof that the injury is work connected. *Kasper v. Board of Trustees of Teachers' Pension and Annuity Fund*, 164 N.J. 564, 591, 754 A.2d 525 (2000) (Coleman, J., concurring); *In re Lockheed Martin Corp.*, 147 N.H. 322, 786 A.2d 872, 874-75 (2001).

It is sufficient in New Jersey to prove that the exposure to a risk or danger in the workplace was in fact a contributing cause of the injury. That means proof that the work related activities probably caused or contributed to the employee's disabling injury as a matter of medical fact. *Coleman v. Cycle Transformer Corp.*, 105 N.J. 285, 290-91, 520 A.2d 1341 (1986). Direct causation is not required; proof establishing that the exposure caused the activation, acceleration or exacerbation of disabling symptoms is sufficient. See *W. Page Keeton et al., Prosser and Keeton on the Law of Torts* § 268 (5th ed.1984). As one commentator has observed, “[t]he legal question is how much workplace contribution will be enough to trigger the employer's liability under workers' compensation.” *Jordan Yospe, Note and Comment, U.S. Industries v. Director: “Claim” Versus “Condition” in the Analysis of Workers' Compensation Cases*, 12 *Am. J.L. & Med.* 273, 275 (1986). See also *Douglas Danner & Elliot L. Sagall, Medicolegal Causation: A Source of Professional Misunderstanding*, 3 *Am. J.L. & Med.* 303, 304-06 (1977).

Another important principle is that the Rules of Evidence do not apply to workers' compensation proceedings. N.J.S.A. 34:15-56 provides that when a Judge of Compensation decides a contested case, he or she “shall not be bound by the rules of evidence.” *Ibid.* Recently, we stated:

Although the Rules of Evidence do not control the admission of evidence in workers' compensation proceedings, it is well-settled that a judge of compensation's determination must be based on competent evidence. *Gilligan v. International Paper Co.*, 24 N.J. 230, 236, 131 A.2d 503 (1957); *Andricsak [v. National Fireproofing Corp.]*, 3 N.J. [466,] 471, 70 A.2d 750 [(1950)]; *Helminsky [v. Ford Motor Co.]*, 111 N.J.L. [369,] 373, 168 A. 420 [(E. & A.1933)]; *Friese [v. Nagle Packing Co.]*, 110 N.J.L. [588,] 588, 166 A. 307 [(E. & A.1933)]; *Gunter [v. Fischer Scientific Am.]*, 193 N.J.Super.688, 691, 475 A.2d 671 [(App.Div.1984)]; *Fagan v. Newark*, 78 N.J.Super. 294, 307-08, 188 A.2d 427 (App.Div.1963). The purpose of not requiring strict compliance with the Rules of Evidence is to simplify the nature of proofs that can be offered in workers' compensation proceedings. *Gunter, supra*, 193 N.J.Super. at 691, 475 A.2d 671. Viewed in that context, the real issue presented is not whether evidence was admitted in violation of the

Rules of Evidence, but whether there is substantial credible evidence in the record to support the judgment when the proofs are considered as a whole. *Szumski v. Dale Boat Yards, Inc.*, 48 N.J. 401, 410, 226 A.2d 11, cert. denied, 387 U.S. 944, 87 [S.Ct.] 2077, 18 [L. Ed.2d] 1331 (1967); *Close v. Kordulak Bros.*, 44 N.J. 589, 599, 210 A.2d 753 (1965); *Goyden v. State Judiciary, Superior Court of New Jersey*, 256 N.J.Super. 438, 446, 607 A.2d 651 (App.Div.1991), aff'd o.b., 128 N.J. 54, 607 A.2d 622 (1992); *Manzo v. Amalgamated Indus. Union Local 76B*, 241 N.J.Super. 604, 609, 575 A.2d 903 (App.Div.), certif. denied, 122 N.J. 372, 585 A.2d 379 (1990). Due regard must be given to the opportunity of the one who heard the witnesses to judge their credibility. *Szumski*, supra, 48 N.J. at 410, 226 A.2d 11; *Close*, supra, 44 N.J. at 599, 210 A.2d 753; *Goyden*, supra, 256 N.J.Super. at 446, 607 A.2d 651.

[*Reinhart v. E.I. DuPont De Nemours*, 147 N.J. 156, 163-64, 685 A.2d 1301 (1996).]

Because the Rules of Evidence do not apply to these workers' compensation proceedings, respondent did not seek a N.J.R.E. 104 hearing to challenge the admissibility of petitioner's expert's testimony with respect to medical causation. The Judge of Compensation found that expert's testimony to be credible and found petitioner's emphysema to be causally related to his employment exposure. Respondent has not waived its right to assert that the determination of the Judge of Compensation went so wide off the mark that a mistake must have been made that is correctable through appellate review.

In workers' compensation proceedings, as in the area of toxic tort litigation involving multiple causations or long-term exposure to toxic substances, it may not be possible scientifically for an injured person to prove decisively the medical cause of the injury. Faced with the need to accommodate the goals of our tort system when the scientific community was in disagreement, this Court adopted a less restrictive standard in *Rubanick v. Witco Chemical Corp.*, 125 N.J. 421, 593 A.2d 733 (1991), for the admissibility of scientific evidence. That same standard is to be used in weighing the credibility of opinion evidence presented by experts. Given that the only significant distinctions between *Rubanick* and the present case are the forum and the quantum of damages recoverable, the *Rubanick* standard governing the admissibility and reliability of medical causation evidence should be applied in workers' compensation cases as well. See, e.g., *Kemp ex rel. Wright v. State*, 174 N.J. 412, 809 A.2d 77 (2002) (applying *Rubanick* in non-toxic tort case).

Rubanick, supra, dealt with occupational exposure to PCBs during the course of the plaintiff's employment with the defendant. 125 N.J. at 425, 593 A.2d 733. The Court acknowledged that our Rules of Evidence governing the admissibility of expert evidence, formerly N.J.R.E. 19 and N.J.R.E. 56(2), now N.J.R.E. 702 and N.J.R.E. 703, are the starting point for determining whether the conventional general acceptance test of reliability should be the standard for the "admissibility of expert testimony relating to new or developing theories of causation in toxic-tort litigation." *Rubanick*, supra, 125 N.J. at 432, 593 A.2d 733. Because "scientific knowledge [was] not 'at a state of the art such that an expert's testimony could be sufficiently reliable,'" the Court "fashion[ed] a

broader standard for assessing the reliability of such evidence in [toxic-tort] litigation.” Id. at 432-33, 593 A.2d 733 (quoting *State v. Kelly*, 97 N.J. 178, 208, 478 A.2d 364 (1984)). The Court recognized that the need for a broader standard was due in part to “the extraordinary and unique burdens facing plaintiffs who seek to prove causation in toxic-tort litigation” and to “the extremely high level of proof required before scientists will accept a new theory.” Id. at 433, 593 A.2d 733. Consequently, in adopting a less restrictive standard, the Court held that

in toxic-tort litigation, a scientific theory of causation that has not yet reached general acceptance may be found to be sufficiently reliable if it is based on a sound, adequately-founded scientific methodology involving data and information of the type reasonably relied on by experts in the scientific field. The evidence of such scientific knowledge must be proffered by an expert who is sufficiently qualified by education, knowledge, training, and experience in the specific field of science. The expert must possess a demonstrated professional capability to assess the scientific significance of underlying data and information, to apply the scientific methodology, and to explain the bases for the opinion reached.

[Id. at 449, 593 A.2d 733.]

Finally, we turn to the appropriate standard of appellate review of determinations made in workers' compensation cases. “Courts generally give ‘substantial deference’ to administrative determinations.” *Earl v. Johnson & Johnson*, 158 N.J. 155, 161, 728 A.2d 820 (1999) (quoting *R & R Marketing, L.L.C. v. Brown-Forman Corp.*, 158 N.J. 170, 175, 729 A.2d 1 (1999)). In workers' compensation cases, the scope of appellate review is limited to “whether the findings made could reasonably have been reached on sufficient credible evidence present in the record, considering the proofs as a whole, with due regard to the opportunity of the one who heard the witnesses to judge of their credibility.” *Close*, supra, 44 N.J. at 599, 210 A.2d 753 (citation and quotation marks omitted). Deference must be accorded the factual findings and legal determinations made by the Judge of Compensation unless they are “‘manifestly unsupported by or inconsistent with competent relevant and reasonably credible evidence as to offend the interests of justice.’” *Perez v. Monmouth Cable Vision*, 278 N.J.Super. 275, 282, 650 A.2d 1025 (App.Div.1994) (quoting *Rova Farms Resort v. Investors Ins. Co.*, 65 N.J. 474, 484, 323 A.2d 495 (1974)), certif. denied, 140 N.J. 277, 658 A.2d 301 (1995).

The petitioner has the burden to demonstrate by a preponderance of the evidence that his or her environmental exposure while fighting fires was a substantial contributing cause of his or her occupational disease. Such a petitioner is not required to “prove that the nexus between the disease and the place of employment is certain.” *Magaw v. Middletown Bd. of Educ.*, 323 N.J.Super. 1, 11, 731 A.2d 1196 (App.Div.), certif. denied, 162 N.J. 485, 744 A.2d 1208 (1999); *Laffey v. Jersey City*, 289 N.J.Super. 292, 303, 673 A.2d 838 (App.Div.), certif. denied, 146 N.J. 500, 683 A.2d 202 (1996).

IV.

First, we address whether the standard articulated in *Fiore* for deciding occupational heart-attack cases applies to this case. We agree with petitioner that the discussion in *Fiore* with respect to dual causes of cardiovascular injuries requiring a petitioner to prove that his or her work exposure exceeded the exposure caused by personal factors such as cigarette smoking does not apply to non-heart cases such as this pulmonary case. See *Hellwig v. J.F. Rast & Co.*, 110 N.J. 37, 48, 538 A.2d 1243 (1988) (tracing the progression of cardiac law that led to the enactment of N.J.S.A. 34:15-7.2 in 1979 and also stating that “the Legislature plainly expressed its intention to modify the holding in *Dwyer*”). In *Fiore*, the Court acknowledged that it was articulating a “standard to apply in a dual-causation case involving an occupational disease that allegedly has caused coronary-artery disease and an angina attack.” *Fiore*, supra, 140 N.J. at 464, 659 A.2d 436. A higher standard was adopted for occupational heart cases because N.J.S.A. 34:15-7.2 was enacted to increase a petitioner’s burden beyond that previously required by *Dwyer*, supra, 36 N.J. 487, 178 A.2d 161. Under *Dwyer*, there was an “assumption that employers take their employees as they find them” and that “‘ordinary work effort or strain’” was sufficient to satisfy the material-degree contribution requirement. *Fiore*, supra, 140 N.J. at 466-67, 659 A.2d 436 (quoting *Dwyer*, supra, 36 N.J. at 493, 178 A.2d 161). *Dwyer* defined material contribution to mean “some employment exertion capable medically of helping the attack-of furthering its progress.” 36 N.J. at 493-94, 178 A.2d 161. To make certain that the higher standard required by N.J.S.A. 34:15-7.2 was limited to cardiovascular and cerebrovascular cases, we stated that “a petitioner asserting an occupational heart-disease claim must show that the work exposure exceeds the exposure caused by the petitioner’s personal-risk factors.” *Fiore*, supra, 140 N.J. at 473, 659 A.2d 436 (emphasis added). Because *Fiore* does not apply to pulmonary cases, the Appellate Division should not have applied its holding here.

The controlling test to be applied in this case is whether the work exposure substantially contributed to the development or aggravation of emphysema. Petitioner had the burden to demonstrate by a preponderance of the evidence that his environmental exposure while fighting fires was a substantial contributing cause or aggravation of his emphysema. To satisfy that obligation, he was not required to prove that his work exposure exceeded the exposure caused by smoking cigarettes. Nor was he required to “prove that the nexus between the disease and the place of employment is certain” because that would violate the preponderance of the evidence standard. *Magaw*, supra, 323 N.J.Super. at 11, 731 A.2d 1196; *Laffey*, supra, 289 N.J.Super. at 303, 673 A.2d 838.

In a case such as this one in which petitioner concedes that his personal risk factor played a significant role in developing emphysema, the Legislature has provided some relief to employers. When there are dual causes of an injury or disease, such as cigarette smoking and employment exposure, a 1979 amendment to the Act, L. 1979, c. 283, effective January 10, 1980, codified as N.J.S.A. 34:15-12(d), requires a credit to “be given [to] the employer or the employer’s insurance carrier for the previous loss of function and the burden of proof in such matter shall rest on the employer.” *Ibid.* The purpose of that amendment was to ameliorate the effect of prior law that an employer takes an employee as he finds the employee. Although that theory still pertains, the amendment permits a credit, regardless of whether or not the previous loss was work-

related, “to encourage [the] hiring [of] workers with pre-existing disabilities.” *Field v. Johns-Manville Sales Corp.*, 209 N.J.Super. 528, 530-31, 507 A.2d 1209 (App.Div.), certif. denied, 105 N.J. 531, 523 A.2d 172 (1986); *Abdullah v. S.B. Thomas, Inc.*, 190 N.J.Super. 26, 29-32, 461 A.2d 1179 (App.Div.1983).

V.

Next, we address whether the statutory presumption contained in N.J.S.A. 34:15-43.2 applies to this case involving, as it does, a member of a paid fire department. That statute provides:

[a]ny condition or impairment of health of any member of a volunteer fire department caused by any disease of the respiratory system shall be held and presumed to be an occupational disease unless the contrary be made to appear in rebuttal by satisfactory proof; providing

(a) Such disease develops or first manifests itself during a period while such member is an active member of such department; and

(b) Said member, upon entering said volunteer fire service, has or shall have undergone a medical examination, which examination failed or fails to disclose the presence of such disease or diseases; and

(c) Such disease develops or first manifests itself within 90 days from the event medically determined to be the cause thereof.

Any present member who did not undergo a medical examination upon entering said volunteer fire service, may undergo such examination within 180 days after the effective date of this act and in the event such examination does not disclose the presence of such disease or diseases, he shall thereafter be entitled to the benefits of this act.

[Ibid.]

There is no legislative history indicating the Legislature's intent when enacting the statute in 1964. Members of volunteer fire companies were already, at the time, covered by workers' compensation laws. N.J.S.A. 34:15-43. Paid firefighters as public employees were also covered by the Act. *Ibid.* Because we can find no plausible reason the Legislature would have intended a difference when voluntary and paid firefighters sustained the same pulmonary conditions after fighting the same fire together, we hold that the presumption applies to paid firefighters as well. Further support for concluding that the presumption should not be restricted to volunteer firefighters is the fact that in 1987, the Legislature enacted a rebuttable presumption that cardiovascular and cerebrovascular injury or death to paid and volunteer firefighters sustained while performing fire suppression or medical emergency functions are compensable. L. 1987, c. 382, codified as N.J.S.A. 34:15-7.3. As will be shown later, at least thirty states have

some form of a presumption for firefighters. If we have misperceived the Legislature's intent, we invite the Legislature to reconsider this matter.

The record does not fully inform us whether the statutory preconditions for the presumption have been met in this case. Assuming, however, that the presumption applies to this case and that subsections (a), (b) and (c) can be satisfied, the existence of such a rebuttable presumption is not dispositive of this appeal. To rebut the presumption, respondent presented evidence that petitioner smoked and had a family history of emphysema. Because the determination of whether respondent rebutted the presumption is intertwined with whether petitioner sustained his burden of proof on causation, we resolve both issues in petitioner's favor in section VI of this opinion.

Other jurisdictions have addressed the issue of statutory presumptions in favor of the firefighter in one form or another. For example, when Oregon adopted a disputable (rebuttable) presumption that firefighters' heart and pulmonary conditions are related to the employment, the legislature "intended to give relief to firefighters because statistical studies indicated firefighters were much more likely to suffer from heart and lung diseases due to exposure to smoke and gases under strenuous conditions." *Wright v. State Accident Ins. Fund*, 289 Or. 323, 613 P.2d 755, 758 (1980). Similarly, when the State of Washington created a presumption, it "recognized that fire fighters as a class have a higher rate of respiratory disease than the general public." Wash. Rev.Code § 51.32.185 Legislative Findings, 1987 c. 515.

As noted in the Appendix to this opinion, our research discloses that thirty states have adopted a presumption that a firefighter's pulmonary disease or condition is work related, generally following more than five years of such employment. Many states also require firefighters to have passed a medical examination at the beginning of their employment finding them free of pulmonary disease if they wish to invoke the presumption. Seventeen of those states' presumptions relate to pension, special disability funds, and retirements. Thirteen states, including New Jersey, have presumptions covering workers' compensation claims. At least one state legislature has been motivated by statistical studies indicating that firefighters are much more likely to suffer from heart and lung diseases due to exposure to smoke and gases under strenuous conditions. Other legislatures have reached the same result without reference to studies, stating that firefighters are "required to work in the midst of and are subject to heavy smoke fumes, and carcinogenic, poisonous, toxic or chemical gases from fires." 40 Ill. Comp. Stat. Ann. 5/4-110.1. The fact that so many states have created various forms of presumptions of compensability for specified occupational conditions, suggests "(1) that a generalized causative link exists between the injury or disease and the workplace and (2) that such a causative link will be difficult to prove." See Solomon, *supra*, at 1174 n. 105 (quoting Joseph La Dou, M.D., et al., *Cumulative Injury or Disease Claims: An Attempt to Define Employers' Liability for Workers' Compensation*, 6 Am. J.L. & Med. 1, 21 (1980)).

Because so many state legislatures have enacted various presumptions respecting the connection between firefighting and pulmonary and heart conditions, almost "a national

consensus emerges regarding” the reliability of that proposition. *Hamilton Amusement Ctr. v. Verniero*, 156 N.J. 254, 270, 716 A.2d 1137 (1998).

VI.

A.

We now consider whether petitioner's emphysema is medically related to his work exposure. The starting point is a statement of what that condition entails in general and a review of some pertinent studies, some of which focused on pulmonary conditions in firefighters. Emphysema is a “[c]hronic obstructive pulmonary disease (COPD), also called chronic obstructive lung disease[. It] is a term that is used for two closely related diseases of the respiratory system: chronic bronchitis and emphysema. In many patients these diseases occur together.” Div. of Lung Diseases & Office of Prevention, Educ. & Control, Nat'l Insts. of Health, Pub. No. 95-2020, *Chronic Obstructive Pulmonary Disease 1* (3d prtg.1995) (emphasis omitted) (hereinafter *Nat'l Insts. of Health*).

Chronic bronchitis, one of the two major diseases of the lung grouped under COPD, is diagnosed when a patient has excessive airway mucus secretion leading to a persistent, productive cough. An individual is considered to have chronic bronchitis if cough and sputum are present on most days for a minimum of 3 months for at least 2 successive years or for 6 months during 1 year. In chronic bronchitis, there also may be narrowing of the large and small airways making it more difficult to move air in and out of the lungs. An estimated 12.1 million Americans have chronic bronchitis.

In emphysema there is permanent destruction of the alveoli, the tiny elastic air sacs of the lung, because of irreversible destruction of a protein in the lung called elastin that is important for maintaining the strength of the alveolar walls. The loss of elastin also causes collapse or narrowing of the smallest air passages, called bronchioles, which in turn limits airflow out of the lung. The number of individuals with emphysema in the U.S. is estimated to be 2 million.

In the general population, emphysema usually develops in older individuals with a long smoking history. However, there is also a form of emphysema that runs in families. People with familial emphysema have a hereditary deficiency of a blood component, alpha-1-protease inhibitor, also called alpha-1-antitrypsin (AAT). The number of Americans with this genetic deficiency is quite small, probably no more than 70,000. It is estimated that 1 in 3,000 newborns have a genetic deficiency of AAT, and 1 to 3 percent of all cases of emphysema are due to AAT deficiency.

The destruction of elastin that occurs in emphysema is believed to result from an imbalance between two proteins in the lung—an enzyme called elastase which breaks down elastin, and AAT which inhibits elastase. In the normal individual, there is enough AAT to protect elastin so that abnormal elastin destruction does not occur. However, when there is a genetic deficiency of AAT, the activity of the elastase is not

inhibited and elastin degradation occurs unchecked. If individuals with a severe genetic deficiency of alpha-1-protease inhibitor smoke, they usually have symptoms of COPD by the time they reach early middle age. Deficiency of alpha-1-protease inhibitor can be detected by blood tests available through hospital laboratories. People from families in which relatives have developed emphysema in their thirties and forties should be tested for AAT deficiency. If a deficiency is found, it is critical for these people not to smoke.

Some scientists believe that nonfamilial emphysema, usually called “smoker's emphysema,” also results from an imbalance between elastin-degrading enzymes and their inhibitors. The elastase-AAT imbalance is thought to be a result of the effects of smoking, rather than inherited as in familial emphysema. Some evidence for this theory comes from studies on the effect of tobacco smoke on lung cells. These studies showed that tobacco smoke stimulates excess release of elastase from cells normally found in the lung. The inhaled smoke also stimulates more elastase-producing cells to migrate to the lung which in turn causes the release of even more elastase. To make matters worse, oxidants found in cigarette smoke inactivate a significant portion of the elastase inhibitors that are present, thereby decreasing the amount of active antielastase available for protecting the lung and further upsetting the elastase-antielastase balance.

Scientists believe that, in addition to smoking-related processes, there must be other factors that cause emphysema in the general population since only 15 to 20 percent of smokers develop emphysema. The nature and role of these other factors in smokers' emphysema are not yet clear.

[Id. at 2-4 (emphasis omitted).]

Although “[c]igarette smoking is the most important risk factor for COPD . [o]ther risk factors include age, heredity, exposure to air pollution at work and in the environment.” Id. at 1 (emphasis added). That means the National Institutes of Health has recognized that exposure to air pollutants at work can cause both chronic bronchitis and emphysema. Furthermore, “[s]cientists believe that, in addition to smoke-related processes, there must be other factors that cause emphysema in the general population since only 15 to 20 percent of smokers develop emphysema.” Id. at 4.

Not only is it well known that industrial pollutants at work can cause or contribute to the development of emphysema, courts in New Jersey have awarded workers' compensation for emphysema since the current general definition of occupational diseases became effective January 1, 1950. The first set of such cases was *Masko v. Barnett Foundry & Mach. Co.*, 53 N.J.Super. 414, 423, 147 A.2d 579 (App.Div.), certif. denied, 29 N.J. 464, 149 A.2d 859 (1959), and *Bucuk v. Edward A. Zusi Brass Foundry*, 49 N.J.Super. 187, 193, 139 A.2d 436 (App.Div.), certif. denied, 27 N.J. 398, 143 A.2d 9 (1958), awarding compensation for emphysema and silicosis caused by pulmonary irritants.

That same trend was continued into the next three decades. In *Ort v. Taylor-Wharton Co.*, 47 N.J. 198, 201, 219 A.2d 866 (1966), the petitioner was awarded thirty percent of total permanent disability for pneumoconiosis and emphysema caused by exposure to

foundry dust. Thereafter, he returned to work for the same employer in the same industrial environment that caused the pneumoconiosis to render him totally and permanently disabled. *Id.* at 202, 219 A.2d 866. In *Taylor by Taylor v. Engelhard Indus.*, 230 N.J.Super. 245, 247, 553 A.2d 361 (App.Div.1989), Solomon Taylor “was awarded [fifty-five percent] of total permanent disability for chronic bronchitis and pulmonary emphysema.” Those conditions were caused by the petitioner’s “expos[ure] to smoke, dust, fumes and other pulmonary irritants.” *Ibid.* In *Gierman v. M & H Mach. Co.*, 213 N.J.Super. 105, 107, 516 A.2d 632 (App.Div.1986), Howard Gierman was awarded total and permanent compensation “as the result of his occupational exposure to pulmonary irritants while working for respondent. The disability was characterized as chronic, obstructive pulmonary disease including emphysema.” *Ibid.* In *Brooks v. Bethlehem Steel Co.*, 66 N.J.Super. 135, 137, 168 A.2d 670 (App.Div.), *certif. denied*, 36 N.J. 29, 174 A.2d 657 (1961), compensation was awarded based on the petitioner’s exposure to smoke and fumes caused by his job as a burner-welder at the steel yard. Finally, another case finding emphysema related to an undescribed employment is *Vohta v. Bogue Elec. Mfg. Co.*, 60 N.J.Super. 169, 172, 158 A.2d 536 (App.Div.), *certif. denied*, 32 N.J. 353, 160 A.2d 849 (1960).

Although none of those cases involved firefighters, there are good reasons to infer that many of the workers in those cases were smokers. In *Dewey v. R.J. Reynolds Tobacco Co.*, 121 N.J. 69, 99-100, 577 A.2d 1239 (1990), we noted that tobacco manufacturers have been highly successful in getting people to smoke and that their advertisements have targeted certain groups such as minorities. A United States National Health Survey in 1970 revealed that “[r]egardless of industry, smoking is most prevalent among those jobs which are also likely to be most exposed to irritating and toxic dusts and fumes.” T. Sterling and J. Weinkam, *The Confounding of Occupation and Smoking and Its Consequences*, 30 *Soc. Sci. & Med.* 457, 459 (1990). The point to be made is that some smokers have been awarded workers’ compensation in New Jersey for emphysema based on their occupational exposure.

B.

Predictably, the expert witnesses who testified for the parties disagreed over whether petitioner’s occupational exposure contributed to his emphysema. Both experts were found to be qualified to render expert opinions under N.J.R.E. 702 by virtue of their “knowledge, skill, experience, training, or education.” *Ibid.*

Dr. Eisenstein agreed that emphysema can be caused exclusively by smoking cigarettes, from fighting fires and inhaling the smoke, fumes, gases, and heat alone or a combination of smoking cigarettes and occupational exposure. His opinion of causal relationship to employment was based on his many years of experience in examining firefighters, knowledge of pulmonary diseases, and special training. He stated that a physician cannot look at an x-ray and determine whether a firefighter’s emphysema was caused by occupational exposure because a fireman’s exposure is so varied. Based on his experience in examining firefighters, Dr. Eisenstein concluded that petitioner’s

emphysema is due to his work plus his smoking. The question of which percentage caused which, as I said, it's hard to say and I would say in this case because the smoking wasn't that much, a pack a day, and his exposure is significant industrial or occupational, I believe the material percentage of disability comes from his work and a lesser percentage from cigarettes.

Dr. Eisenstein testified that he based his opinion of medical causation on the detailed facts concerning the exposure that involved hazardous dump fires, medical history, the x-ray and pulmonary testing, and his own experience in the field for nearly fifty years, forty years of which were spent examining and testifying in workers' compensation cases, and his training of many years. He stated that he could point to no study done on firefighters who are non-smokers and who had emphysema.

In contrast, Dr. Hutt testified that the emphysema was caused by petitioner's cigarette smoking and family history that revealed that his grandfather died of emphysema. He stated:

Since there's no data to suggest that fire fighters with exposure on the job can develop emphysema, the patient definitely has emphysema, he has a smoking history, which is clearly the most common cause of emphysema, and there's a strong family history of emphysema which increases your risk of developing emphysema from cigarette smoking, I believe that within a reasonable degree of medical probability that the patient's emphysema is related to his cigarette smoking.

Well, when you're looking at someone that has lung disease and has exposure which can cause disease, you have to take into account what is cause and effect. It's clear that, and I believe everyone knows this, that emphysema is by and large caused by cigarette smoking although other things have occasionally been reported to cause emphysema, particularly congenital abnormalities and some enzymes or a disease called alpha-antitrypsin deficiency which may be responsible for up to five percent or maybe even more of cases of emphysema. There are probably some other exposures although it's not well-established in the literature that can lead to emphysema. The patient clearly has emphysema, and we know that even though only 20% of people that smoke cigarettes actually get emphysema, that number is significantly higher if you have relatives that smoke cigarettes and get emphysema which is true in this patient's family in his grandfather.

Dr. Hutt also testified that although he has read many unspecified studies on lung diseases that included firefighters, none dealt with firefighting and emphysema. He stated that although many of the studies have indicated that firefighters can get lung scarring and lung restriction from firefighting, "I haven't seen [studies] that specifically mention emphysema as an increased risk when you factor out cigarette smoking in firefighters." He was aware that postmortem and pathologic studies have shown "that cigarette smoke is by far the most common cause of the development of emphysema."

He testified that it is the frequent inhalation of smoke that affects certain enzymes, which causes emphysema. He was unaware of any studies linking emphysema to any smoke except cigarette smoke.

Dr. Hutt admitted that he was aware of “many studies which show that firefighters have worse airflow obstruction and chronic bronchitis . [and] may be at somewhat higher risk.” He concluded that, absent any studies that show that firefighters’ “exposure on the job can develop emphysema,” and in view of the fact that petitioner smoked and his grandfather had died of emphysema, petitioner’s emphysema is related to smoking.

C.

When, as in this case, studies of firefighters and other groups have been utilized to assist experts with the medical causation issue within the Rubanick context, consideration of some or all of those studies would be useful to a reviewing court. Although the numerous studies Dr. Hutt stated that he utilized in arriving at his opinion in this matter were never identified in the record and have not been made part of the appellate record before us, our independent research has uncovered many studies in this field. We have examined some of the articles presumably reviewed by Dr. Hutt. In any event, we take judicial notice of the studies uncovered in our research. See N.J.R.E. 201(b)(3), N.J.R.E. 202(b); *Planned Parenthood of Cent. New Jersey v. Farmer*, 165 N.J. 609, 640 n. 10, 762 A.2d 620 (2000); N.J.R.E. 803(c)(18); *Bird v. Somerset Hills Country Club*, 309 N.J.Super. 517, 523-24, 707 A.2d 1033 (App.Div.), certif. denied, 154 N.J. 609, 713 A.2d 500 (1998). See also *In re Jobes*, 108 N.J. 394, 418 n. 11, 529 A.2d 434 (1987) (indicating Court’s use of recent surveys to assist it in determining who should serve as surrogate decision-maker for irreversibly vegetative patient). Indeed, the studies our research uncovered are not nearly as one-sided as was suggested by Dr. Hutt. He conceded as much in his subsequent testimony presented in the companion case of *Culbert v. City of Jersey City*, 175 N.J. 286, 290-91, 814 A.2d 1094, 1096-97 (2003), also decided today.

A mortality survey of firefighters in urban Alberta between 1927 and 1987 showed no link between firefighting and chronic pulmonary disease. Tee L. Guidotti, M.D., M.P.H., *Mortality of Urban Firefighters in Alberta, 1927-1987*, 23 *Am. J. Indus. Med.* 921, 921 (1993). However, there are other studies to the contrary. The “healthy worker effect,” whereby sick workers leave employment and are not included in studies, complicates most studies of disease in firefighters. To reduce that effect, two studies were performed comparing mortality in firefighters and police officers. Because the socioeconomic background, smoking habits, and health requirements of these groups are similar, any increase in lung disease among firefighters is likely to have been caused by their employment. Paul A. Demers et al., *Mortality Among Firefighters From Three Northwestern United States Cities*, 49 *British J. Indus. Med.* 664, 668-69 (1992). As will be shown later, both groups of researchers found such an increase. Elizabeth Feuer, M.D., M.P.H., & Kenneth Rosenman, M.D., *Mortality in Police and Firefighters in New Jersey*, 9 *Am. J. Indus. Med.* 517, 526 (1986); Linda Rosenstock et al., *Respiratory Mortality Among Firefighters*, 47 *British J. Indus. Med.* 462, 464 (1990).

The Demers study is a follow-up of the Rosenstock study, published two years later. It found a smaller increase in the risk of non-malignant respiratory disease for firefighters than previously thought, but nonetheless concluded that “a raised risk of emphysema was found among firefighters compared with both United States white men and police.” Demers, *supra*, at 668-69. Those studies contain a predictable list of limitations, such as small sample size, difficulty in tracking subjects after retirement, vague death certificates, and inability to determine the amount and chemical content of smoke exposure. Demers, *supra*, at 669; Feuer, *supra*, at 526. Those studies comparing populations of healthy workers, similar in all relevant respects except fire smoke exposure, present the strongest scientific support for the proposition that firefighting is a significant cause of lung disease. Additional studies support that conclusion.

In an endeavor to circumvent some of the limitations surrounding the studies of Alberta firemen, a group of thirty-nine Boston firefighters was studied in 1976 for six weeks as they performed fire suppression functions. A.W. Musk et al., *Pulmonary Function in Firefighters: Acute Changes in Ventilatory Capacity and Their Correlates*, 36 *British J. Indus. Med.* 29, 29 (1979). Twenty-two of them were current smokers and seventeen were non-smokers, including eight ex-smokers. *Id.* at 30. The purpose of the study “was to determine the extent to which acute changes in pulmonary function occur during routine firefighting duty and to relate any changes to symptoms and to indices of smoke exposure.” *Id.* at 29. Each firefighter was equipped with a device to capture air samples while fighting the fires “in a mixed residential, retail and light industrial area.” *Id.* at 29-30. The study involved 137 fires, and “[t]he median time between cessation of exposure to smoke and the performance of pulmonary function tests was 29.0 minutes.” *Id.* at 30. The study revealed that although none “of the firefighters [sought] medical attention for smoke inhalation, [there were] acute changes in pulmonary function . in more than 30% of fire attendances.” *Id.* at 32. “This study shows that, as a result of his occupation, the respiratory system of the firefighter is subject to frequent episodes of acute irritation . [that] may produce cumulative damage manifested by an excessive yearly decline in FEV 1.0” (forced expiratory volume in one second). *Id.* at 29, 33. The study revealed that the impact of firefighting on the respiratory system of non-smokers was more severe than on smokers: “[c]urrent non-smokers showed a tendency to a greater mean decline in [forced expiratory volume in one second] on exposure to heavy or moderate smoke than smokers.” *Id.* at 31.

A follow-up study of the Boston Fire Department concluded that the previous finding that occupational smoke exposure apparently caused a reduction in ventilatory capacity of firefighters “can no longer be seen.” A.W. Musk et al., *Pulmonary Function in Firefighters: A Six-Year Follow-up in the Boston Fire Department*, 3 *Am. J. Indus. Med.* 3, 8 (1982). That conclusion, however, was contradicted four years later based on a study of firefighters in the San Francisco Fire Department. D. Sheppard, M.D., et al., *Acute Effects of Routine Firefighting on Lung Function*, 9 *Am. J. Indus. Med.* 333, 333 (1986). The San Francisco study concluded “that routine firefighting is associated with a high incidence of acute decrements in lung function.” *Ibid.* Thus, the results of the Sheppard study are “similar to [those] reported by Musk and coworkers [1979] from a study of Boston firefighters.” *Id.* at 337. According to the San Francisco study, “[t]he

results of the [Sheppard] study suggest that, despite the attention paid to personal respiratory protection since the study of Musk et al., [firefighters'] protection remains inadequate to prevent the common occurrence of acute decrements in lung functions.” Id. at 338.

The most recent study was conducted of New York City firefighters exposed to a variety of inhaled materials during and after the collapse of the World Trade Center on September 11, 2001. David J. Prezant, M.D., et al., Cough and Bronchial Responsiveness In Firefighters at the World Trade Center Site, 347 *New Eng. J. Med.* 806 (2002). That study “evaluated clinical features . of 332 firefighters in whom severe cough developed after exposure[as well as] the prevalence and severity of bronchial hyperreactivity in firefighters without severe cough[, and] classified [them] according to the level of exposure.” Id. at 806. Of the firefighters with severe cough, only three percent were smokers, and twenty percent were ex-smokers. Id. at 809. The study revealed that the majority of these firefighters had symptoms of dyspnea (shortness of breath usually associated with serious disease of the heart or lungs), gastroesophageal reflux disease and nasal congestion. Id. at 806. Chest x-rays showed no changes in ninety-six percent of the 332 firefighters in the study group. Ibid. However, bronchial hyperactivity was found in a significant number of firefighters without severe cough who had high level to moderate level exposure. Ibid. Significantly, the study revealed that even when “radiographic or physiological evidence of parenchymal lung disease was uncommon, . high resolution CT . provide[d] evidence of air trapping . [that] could be due to asthma, bronchitis, emphysema, or bronchiolitis.” Id. at 814. In other words, the study revealed that airway obstruction in the form of emphysema can occur in firefighters despite normal chest x-rays and virtually normal forced vital capacity and normal forced expiratory volume studies. Id. at 814.

D.

As noted above, appellate review of the Judge of Compensation's decision by this Court and the Appellate Division is limited to whether the judge's decision “could reasonably have been reached on sufficient credible evidence present in the record . [while giving] due regard to the compensation judge's expertise and ability to evaluate witness credibility.” *Magaw, supra*, 323 N.J.Super. at 15, 731 A.2d 1196 (citing *Close v. Kordulak Bros., supra*, 44 N.J. at 599, 210 A.2d 753). The Appellate Division held that petitioner had not presented sufficient credible evidence of causation to support the decision of the Judge of Compensation. Although the Appellate Division improperly applied the higher *Fiore* standard, our application of the correct standard leads us to conclude that sufficient credible evidence existed to support the Judge of Compensation's decision that petitioner had proven, by a preponderance of the evidence, that his employment as a firefighter was a material cause of his emphysema. See *Wiggins v. Port Auth.*, 276 N.J.Super. 636, 638-39, 648 A.2d 743 (App.Div.1994).

This Court has recognized for many years that the Act is “humane social legislation designed to place the cost of work-connected injury upon the employer who may readily provide for it as an operating expense.” *Tocci v. Tessler & Weiss, Inc.*, 28 N.J. 582,

586, 147 A.2d 783 (1959). Hence, the broad language in the occupational disease statute represents a “conscientious endeavor to maintain a liberally just line between those [occupational diseases] which may be said to have had some work connection and those which may be said to have been unrelated to the employment.” *Id.* at 587, 147 A.2d 783. “Because of the pernicious nature of occupational diseases, the Legislature has been solicitous of workers who suffer from these ailments.” *Earl v. Johnson & Johnson*, 158 N.J. 155, 166, 728 A.2d 820 (1999). Similarly, this Court should be solicitous of firefighters who have demonstrated a substantial likelihood that their fire suppression duties have contributed to the development of emphysema.

Petitioner's expert, Dr. Eisenstein, expressed the view that firefighting, rather than cigarette smoking, was the dominant cause of petitioner's emphysema. He based that opinion on his years of experience evaluating pulmonary disease in patients including firefighters. However, the Appellate Division held that “Dr. Eisenstein has asserted a causal relationship without credible foundation” because he did not cite any scientific studies demonstrating that firefighting causes emphysema.

It is well established that “[t]he absence of any objective medical or scientific evidence establishing a causal link between petitioner's place of employment and a claimed occupational disease will usually be fatal to the petitioner's workers' compensation case.” *Magaw*, *supra*, 323 N.J.Super. at 13, 731 A.2d 1196 (citing *Wiggins*, *supra*, 276 N.J.Super. at 644-45, 648 A.2d 743). However, courts must not penalize workers suffering from diseases for which science has not yet clearly established causation. As this Court stated in the toxic tort context, “[t]here are areas in which [the] judicial need for certain facts equals or exceeds the scientific community's ability to establish them. Many cases present issues with respect to which courts have been forced intuitively to make assumptions on the basis of available knowledge.” *Rubanick*, *supra*, 125 N.J. at 437, 593 A.2d 733 (quoting Justice Alan B. Handler, *The Judicial Pursuit of Knowledge: Truth and/or Justice*, 41 *Rutgers L.Rev.* 1, 26 (1988) (alterations in original)). Former Chief Justice Weintraub provided the answer to the kind of dilemma presented in this type of case when he stated:

When the possibility of causal connection is accepted, we cannot deny relief in all cases simply because science is unable decisively to dissipate the blur between possibility and probability. In such circumstances judges must do the best they can, with the hope their decisions square with the truth, and with a willingness to consider in succeeding cases whatever contribution scientific advances may offer.

[*Dwyer*, *supra*, 36 N.J. at 516, 178 A.2d 161 (Weintraub, C.J., concurring).]

More than a possibility of causal connection exists in this case. Although we do not relax the requirement that petitioner must prove his case by a preponderance of the evidence, and that his evidence must be scientifically reliable, we must examine the evidence in light of science's inability to provide conclusive answers to every question of causation. “While courts obviously do not wish to decide cases based on discredited science or medicine, the judicial system does not have the leisure to defer decision until

proper and definitive scientific or medical studies are available.” Magaw, *supra*, 323 N.J.Super. at 14, 731 A.2d 1196.

In this case, it is true that petitioner's expert did not cite any scientific studies to support his conclusion. Respondent's expert, Dr. Hutt, testified that he had read about one hundred unspecified studies concerning firefighters and lung disease, none of which established a causal link between firefighting and emphysema. However, our independent review of articles addressing firefighting and lung disease confirmed that some evidence to the contrary exists.

Science has not conclusively proven Dr. Eisenstein's opinion that firefighting can be the primary cause of emphysema in a firefighter who smokes. However, “this [is] not a case where the absence of medical evidence leads to an inference that the proposed medical theory was completely improbable or outlandish.” Magaw, *supra*, 323 N.J.Super. at 14, 731 A.2d 1196. Scientific data has verified what common sense suggests, that acute smoke exposure can damage the lungs. See *supra* Part VI.C. (discussing Boston, San Francisco, and World Trade Center studies). Some articles also have noted a correlation between firefighting and an increased risk of emphysema, but studies simply have not been performed that would conclusively establish causation, perhaps because of the difficulty in designing such a long-term, multi-variable study. See *supra* Part VI.C. (discussing Demers, Feuer, and Rosenstock studies).

Additionally, some data indicates that it is unusual for someone of petitioner's age and smoking history to contract emphysema. One article confirms that “[c]igarette smoking is the primary cause of COPD,” but also states that “[t]he risk of COPD is strongly associated with the intensity and duration of smoking and is unusual in patients younger than 45 years or in those with [less than] a 40-pack-year history of smoking.”¹ Jay H. Ryu, M.D., & Paul D. Scanlon, M.D., *Obstructive Lung Diseases: COPD, Asthma, and Many Imitators*, 76 *Mayo Clinic Proc.* 1144, 1144 (2001) (citing Sharon E. Straus, M.D., et al., *The Accuracy of Patient History, Wheezing, and Laryngeal Measurements in Diagnosing Obstructive Airway Disease*, 283 *JAMA* 1853, 1856 (2000)). Petitioner was diagnosed with emphysema at age forty-seven, making him relatively young for an emphysema patient. More significantly, based on the Judge of Compensation's finding that petitioner smoked three-fourths of a pack per day for twenty-two years, his pack-year history was only sixteen and a half years. According to the Ryu and Scanlon article, that level of past smoking is far below that expected in an emphysema patient. This strongly suggests that some other factors were materially responsible for causing petitioner's emphysema, although other literature indicates that emphysema can develop more quickly. See *Nat'l Insts. of Health, supra*, at 9.

Dr. Hutt suggested that petitioner's family history could account for his emphysema, and studies do indicate that “familial factors” can increase the risk. See *Nat'l Insts. of Health, supra*, at 4; Ryu & Scanlon, *supra*, at 1145. However, the “only known genetic abnormality that leads to COPD,” called alpha-1-antitrypsin deficiency, causes only about one percent of COPD cases. *Ibid.* But see *Nat'l Insts. of Health, supra*, at 3 (estimating one to three percent of emphysema cases are caused by this genetic

deficiency). Alpha-1-antitrypsin deficiency is detectable by a blood test that apparently was not performed on petitioner. *Ibid.* We therefore do not know the extent to which petitioner's family history contributed to his emphysema.

We find that enough scientific data exists in support of petitioner's case to allow a Judge of Compensation to find in petitioner's favor. Given the current level of scientific knowledge about emphysema, we find that Dr. Eisenstein's testimony was "not a subjective guess or mere possibility." *Magaw, supra*, 323 N.J.Super. at 15, 731 A.2d 1196. We find that sufficient credible evidence exists to support the Judge of Compensation's decision granting petitioner disability benefits. That conclusion is compelled by the principles that the Act represents social legislation, and is to be interpreted to expand rather than limit coverage, and that under the social compromise theory it is intended that a petitioner's burden of proof be lighter than in a common-law tort action. The conclusion is further compelled by the fact that the studies reveal that although smoking is the most significant risk factor, some other causal factors must exist because no more than twenty percent of smokers contract emphysema. *Nat'l Insts. of Health, supra*, at 4. Both experts testified that industrial exposure can cause emphysema and that the signs and symptoms have the same manifestation regardless of whether they are caused by cigarette smoking, industrial exposure, or a combination of exposures. We reemphasize that it is not necessary for petitioner to prove that firefighting was the most significant cause of his disease. Rather, he need only show that his employment exposure contributed in a material degree to the development of his emphysema. We hold that there is sufficient scientific evidence to support the Judge of Compensation's conclusion that petitioner sustained his burden of proof. For the same reasons, the presumption in favor of compensability has not been rebutted.

VII.

The judgment of the Appellate Division is reversed, and the judgment of the Division of Workers' Compensation is reinstated.

APPENDIX

(A) States with presumption for both workers' compensation and pension, retirement or disability plans:

Louisiana: *La.Rev.Stat. Ann. § 33:2581* (required five years' service, statute pertains to municipalities with more than thirteen thousand persons but also applies to workers' compensation; see *McKenzie v. City of Bossier City*, 585 So.2d 1229, 1233 (La.Ct.App.1991). See *La. Const. art. X, part II, § 16* (population requirement)).

Maine: *Me.Rev.Stat. Ann. tit. 5, § 18511* (state retirement system, requires 2 years' service, disease developed within 6 months of fighting a fire, and firefighter has been granted workers' compensation benefits); *Me.Rev.Stat. Ann. tit. 39-A, § 328* (workers' compensation, same conditions).

Oklahoma: Okla. Stat. tit. 11, § 49-110(A) (firefighters' pension & retirement system; medical exam; Supreme Court applied to workers' compensation in a heart disease case, Johnson v. Woodward, 38 P.3d 218 (Okla.2001)).

Virginia: Va.Code Ann. § 27-40.1 (firefighter's relief, requires medical exam); Va.Code Ann. § 65.2-402 (workers' compensation, requires medical exam).

(B) States with presumption for workers' compensation only:

Maryland: Md.Code Ann., Lab. & Empl. § 9-503.

Michigan: Mich. Comp. Laws Ann. § 418.405.

Nevada: Nev.Rev.Stat. Ann. § 617.455 (medical exam, conclusive with five years' service).

New Hampshire: N.H.Rev.Stat. Ann. § 281-A:17 (medical exam for volunteer or call firefighters, ends one month after sixty-fifth birthday).

New Jersey: N.J.S.A. 34:15-43.2.

North Dakota: N.D. Cent.Code § 65-01-15.1 (medical exam, five years' service). Ends no more than five years after retirement depending on length of service. Does not apply if firefighter has used tobacco within past two years: N.D. Cent.Code § 65-01-15.

Oregon: Or.Rev.Stat. § 656.802(4) (five years' service, medical exam).

South Carolina: S.C.Code Ann. § 42-11-30 (medical exam, must have joined fire dept. before thirty-seventh birthday, condition must have developed while “fighting a fire or within twenty-four hours from the date of last service in such activity”).

Washington: Wash. Rev.Code Ann. § 51.32.185 (ends no more than sixty months after employment ceases; will not apply to tobacco users after July 1, 2003).

(C) States with presumption for pension, retirement or disability plan only:

Alabama: Ala.Code § 11-43-144 (municipal corp. law, medical exam, three years' service).

Colorado: Colo.Rev.Stat. Ann. § 31-30.5-705 (firefighter's pension fund; cities over 100,000 population only, five years' service, “old hires” only). (For smaller cities, must show causation: Colo.Rev.Stat. Ann. § 31-30.5-703).

Georgia: Ga.Code Ann. § 47-7-102 (firefighter's pension fund; requires 5 years' fund membership and “total and permanent disability” resulting in firefighter's leaving employment, more stringent criteria for volunteers).

Hawaii: Haw.Rev.Stat. Ann. § 88-79(b) (public employees' retirement plan; medical exam).

Iowa: Iowa Code § 411.6(5)(c) (firefighters' retirement system; requires medical exam).

Kansas: Kan. Stat. Ann. § 74-4952 (firemen's retirement plan, generally requires five years' uninterrupted service).

Kentucky: Ky.Rev.Stat. Ann. § 79.080(7) (local gov't. employees' benefit plan, requires medical exam and five years' service).

Massachusetts: Mass. Gen. Laws ch. 32, § 94A (civil service retirement; medical exam).

Missouri: Mo. Ann. Stat. §§ 87.005, 87.006 (firemen's retirement and relief system; medical exam, five years' service; workers' compensation statute requires proof of "direct causal relationship", Mo. Ann. Stat. § 287.067).

Nebraska: Neb.Rev.Stat. § 18-1723 (pension and retirement; five years' service).

New York: N.Y. Gen. Mun. Law § 207-q; N.Y. Retire. & Soc. Sec. Law § 363-f (retirement and municipal laws; medical exam, enacted 2002, effective Sept. 11, 2001 through June 30, 2004).

Ohio: Ohio Rev.Code Ann. § 742.38(D)(3) (medical exam); workers' compensation statute requires causation and total disability, Ohio Rev.Code Ann. § 4123.68(W).

Rhode Island: R.I. Gen. Laws § 45-19-16 (firefighters' relief law; allows towns to create presumption, medical exam, excludes partial disability).

South Dakota: S.D. Codified Laws § 9-16-45 (city retirement systems; medical exam).

Tennessee: Tenn.Code Ann. § 7-51-201(b)(1) (local government employee compensation, medical exam).

Texas: Tex.Rev.Civ. Stat. Ann. Art. 6243e.2(1), 6243e.3 (city pensions; six years' service, slightly different rules for different sized cities).

Wisconsin: Wis. Stat. Ann. §§ 40.65(2); 891.45 (applies to public employee's trust fund and firefighter's pension; requires five years' service and medical exam).

(D) States whose "presumption" requires firefighter to show causation:

Idaho: Idaho Code § 72-438 (workers' compensation; requires lung disease to "result" from firefighting, allows presumption for cancer).

Illinois: 40 Ill. Comp. Stat. Ann. 5/4-110.1 (firefighters' pension fund for cities under 500,000 population; requires 5 years' service, medical exam for hires after 1971); 40 Ill. Comp. Stat. Ann. 5/6-112, 5/6-151.1 (firemen's annuity & benefit fund for cities over 500,000 population; requires medical exam and ten years' service, disease must "arise solely [from] employment").

Pennsylvania: 77 Pa. Stat. Ann. §§ 27.1(c), 1208 (workers' compensation, four years' service).

Utah: Utah Code Ann. § 49-16-102(6)(a) (firefighters' retirement act, no cases interpreting statute, appears to require causation and five years' service).

(E) States without any statutory presumption regarding COPD-type disease:

Alaska

Arizona

Arkansas

California

Connecticut

Delaware

Florida

Indiana

Minnesota

Mississippi

Montana (Mont.Code Ann. § 39-71-119: causation-based presumption eliminated in 1987).

New Mexico

North Carolina

Vermont

West Virginia

Wyoming

FOOTNOTES

[1.](#) According to Dr. Hutt's testimony, “[p]ack/years basically means the average number of packs someone has smoked over what period of time. So if someone smokes, you know, a pack a day for 20 years that will be 20-pack years.”

COLEMAN, J.

For reversing and reinstating-Chief Justice PORITZ and Justices COLEMAN, LONG, VERNIERO, LaVECCHIA, ZAZZALI, and ALBIN-7.Opposed-None.