

IN THE CIRCUIT COURT OF THE THIRTEENTH JUDICIAL CIRCUIT
IN AND FOR HILLSBOROUGH COUNTY, FLORIDA
CIVIL DIVISION

RALPH DONSON

Plaintiff,

v.

AIR AND LIQUID SYSTEMS, INC.,

As Successor-in-Interest to Buffalo Pumps, Inc.;

AURORA PUMP COMPANY;

CARRIER CORP.;

CBS CORPORATION, f/k/a VIACOM, INC.,

successor by merger to CBS CORPORATION,

f/k/a WESTINGHOUSE ELECTRIC

CORPORATION, Individually and as

Successor-in-Interest to BF

STURTEVANT CO;

CRANE CO., Individually and as

Successor-in-Interest to DEMING PUMP CO.,

COCHRANE CORPORATION and

CHAPMAN VALVE CO.;

DRAVO CORPORATION;

ELECTROLUX HOME PRODUCTS, INC.,

Individually and as Successor-in-Interest to

COPE-S-VULCAN, INC.;

FOSTER WHEELER ENERGY CORPORATION;

GENERAL ELECTRIC COMPANY;

G.G. OF FLORIDA INC. F/K/A HIGBEE;

GOULDS PUMPS, INC.;

IMO INDUSTRIES, INC., Individually and as

Successor-in-Interest to DELAVAL

TURBINE, INC.;

INGERSOLL-RAND COMPANY, Individually

and as Successor to TERRY STEAM

TURBINE COMPANY;

ITT CORPORATION, as Successor-in-Interest to

FOSTER ENGINEERING;

JOHN CRANE INC.;

JOHNSON CONTROLS, INC., as

Successor-in-Interest to YORK

INTERNATIONAL, INC.;

METROPOLITAN LIFE INSURANCE CO.;

STRAHMAN VALVES, INC.;

Case No.: New Case

Division: Z

VELAN VALVE CORP.;)
WARREN PUMPS, L.L.C.;)
)
Defendants.)

COMPLAINT

COMES NOW the Plaintiff, RALPH DONSON by and through the undersigned attorneys, and for his causes of action against the Defendants, states as follows:

GENERAL ALLEGATIONS AS TO ALL COUNTS

1. This is an action for damages in excess of Fifteen Thousand (\$15,000.00) Dollars.

2. The Plaintiff, RALPH DONSON is a citizen of the State of Florida, and resides at 7301 Fourwind Ln., Spring Hill, FL, 34606.

3. The Plaintiff, RALPH DONSON, served in the U.S. Navy from about 1970-76 at A-School in Great Lakes, IL, aboard the USS Wasp, USS Davis, and in Charleston navy shipyard in 1975, and again during dates in 1980 up to November at Charleston navy shipyard. He also worked at Factory Mutual in Florham Park, New Jersey in about 1978-79. Throughout these timeframes and at these locations, Mr. Donson routinely and according to his job duties as a Boiler Tender removed and replaced asbestos gaskets, block, refractory, castable, pipe covering, insulating cement, packing and/or spray from valves, boilers, pumps, and/or other miscellaneous machinery, and/or worked near others who did. Mr. Donson also utilized asbestos gloves and blankets and removed and/or replaced asbestos pipe and tile, and/or worked near others who did. Mr. Donson cut, scraped, chipped, mixed, pulled, and/or sawed these items, and/or worked near others who did. Mr. Donson, as well as others in his vicinity, routinely, and according to their job duties, performed these tasks. These processes, including sweeping up after each job, created asbestos dust that Mr. Donson regularly inhaled.

4. During the course of his employment at the location mentioned above, Mr. Donson was exposed to and inhaled, ingested or otherwise absorbed large amounts of asbestos fibers emanating from certain products he was working with and around, which were manufactured, sold, distributed, installed or promoted by the following Defendants: AIR AND LIQUID SYSTEMS, INC. as Successor-in-Interest to BUFFALO PUMPS, INC.; AURORA PUMP COMPANY; CARRIER CORP.; CBS CORPORATION, f/k/a VIACOM, INC., Successor by merger to CBS CORPORATION, f/k/a WESTINGHOUSE ELECTRIC CORPORATION, Individually and as Successor-in-Interest to BF STURTEVANT CO.; CRANE CO., Individually and as Successor-in-Interest to DEMING PUMP CO., COCHRANE CORP, and CHAPMAN VALVE CO.; DRAVO CORP.; ELECTROLUX HOME PRODUCTS, INC., Individually and as Successor-in-Interest to COPES-VULCAN, INC.; FLOWSERVE US INC., Individually and as Successor-in-Interest to BW/IP INC., f/k/a Borg Warner Industrial Products Inc., a successor to Borg Warner Corp.; FOSTER WHEELER ENERGY CORP.; GENERAL ELECTRIC COMPANY; G.G. OF FLORIDA INC F/K/A HIGBEE; GOULDS PUMPS, INC.; IMO INDUSTRIES, INC., Individually and as Successor-in-Interest to DELAVAL TURBINE, INC.; INGERSOLL-RAND COMPANY, Individually and as Successor to TERRY STEAM TURBINE COMPANY; ITT CORPORATION, as Successor-in-Interest to FOSTER ENGINEERING; JOHN CRANE INC.; JOHNSON CONTROLS, INC., as Successor-in-Interest to YORK INTERNATIONAL, INC.; METROPOLITAN LIFE INSURANCE CO.; STRAHMAN VALVES, INC.; VELAN VALVE CORP.; and WARREN PUMPS, LLC.

5. Defendant Air and Liquid Systems, Inc. is a foreign corporation organized in Pennsylvania and authorized to do business in the State of Florida. With respect to this

Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Buffalo pumps' insulation, packing, and gaskets.

6. Defendant Air and Liquid Systems, Inc. may be served by and through its registered agent National Registered Agents, Inc., 116 Pine St., 3rd Floor, Suite 320, Harrisburg, PA, 17101.

7. Defendant Aurora Pump Company is a foreign corporation organized in North Carolina and authorized to do business in the State of Florida. Plaintiff alleges exposure to friable asbestos emanating from at minimum Aurora pumps' insulation, packing, and gaskets.

8. Defendant Aurora Pump Company may be served by and through its registered agent Lynette Jones, 13515 Ballantyne Corporate Place, Charlotte, NC, 28277.

9. Defendant Carrier Corporation is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. Plaintiff alleges exposure to friable asbestos emanating from at minimum Carrier A/C systems/compressors' insulation and gaskets.

10. Defendant Carrier Corporation may be served by and through its registered agent CT Corporation System, 1200 S. Pine Island Road, Plantation, FL 33324.

11. Defendant CBS Corporation is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Westinghouse turbines and pumps' insulation, packing, and/or gaskets.

12. Defendant CBS Corporation may be served by and through its registered agent The Prentice-Hall Corporation System, Inc., 1201 Hays Street, Tallahassee, FL 32301.

13. Defendant Crane Co. is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Crane/Chapman valves, Deming/Cochrane/Chapman pumps', Crane steam traps insulation, packing, and/or gaskets.

14. Defendant Crane Co. may be served by and through its registered agent CT Corporation System, 1200 S. Pine Island Road, Plantation, FL 33324.

15. Defendant Dravo Corporation is a foreign corporation organized in the State of Pennsylvania and authorized to do business in the State of Florida. Plaintiff alleges exposure to friable asbestos emanating from at minimum Dravo pumps' insulation, packing, and gaskets.

16. Defendant Dravo Corporation may be served by and through its registered agent National Registered Agents, Inc., 116 Pine Street, Suite 320, Harrisburg, Pennsylvania, 17101.

17. Defendant Electrolux Home Products, Inc. is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Copes Vulcan desuperheaters' insulation and gaskets.

18. Defendant Electrolux Home Products, Inc. may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

19. Defendant Foster Wheeler Energy Corp. is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Foster Wheeler pumps' insulation, packing, and gaskets.

20. Defendant Foster Wheeler Energy Corp. may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

21. Defendant General Electric Company is a foreign corporation organized in the State of New York and authorized to do business in the State of Florida. Plaintiff alleges exposure to friable asbestos emanating from at minimum General Electric turbines' insulation and gaskets.

22. Defendant General Electric Company may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

23. Defendant G.G. of Florida Inc. is a Florida corporation incorporated in the State of New York. Plaintiff alleges exposure to friable asbestos emanating from Higby gaskets.

24. Defendant G.G. of Florida Inc. may be served by and through certified mail, return receipt requested at P.O. Box 37327, Jacksonville, FL, 32236.

25. Defendant Goulds Pumps, Inc. is a foreign corporation organized in Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Goulds pumps' insulation, packing, and gaskets.

26. Defendant Goulds Pumps, Inc. may be served by and through its registered agent The Corporation Trust Company, Corporation Trust Center, 1209 Orange Street, Wilmington, Delaware, 19801.

27. Defendant IMO Industries Inc. is a foreign corporation organized in the State of Delaware authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum DeLaval pumps' insulation, packing, and gaskets.

28. Defendant IMO Industries Inc. may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

29. Defendant Ingersoll-Rand Company is a foreign corporation organized in the State of New Jersey and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Ingersoll-Rand pumps' insulation, packing, and gaskets.

30. Defendant Ingersoll-Rand Company may be served by and through its registered agent Corporation Service Company, 1201 Hays Street, Tallahassee, FL 32301.

31. Defendant ITT Corporation is a foreign corporation organized in the State of Indiana and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Foster Engineering valves' insulation, packing, and gaskets.

32. Defendant ITT Corporation may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

33. Defendant John Crane Inc. is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum John Crane packing and gaskets.

34. Defendant John Crane Inc. may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

35. Defendant Johnson Controls, Inc. is a foreign corporation organized in the State of Wisconsin and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum York A/C systems and/or compressors insulation and gaskets.

36. Defendant Johnson Controls, Inc. may be served by and through its registered agent CT Corporation System, 1200 South Pine Island Road, Plantation, FL 33324.

37. Defendant Metropolitan Life Insurance Company is a foreign corporation organized in the State of New York and authorized to do business in the State of Florida.

38. Defendant Metropolitan Life Insurance Company may be served by and through its registered agent Chief Financial Officer, 200 E. Gaines Street, Tallahassee, FL 32399.

39. Defendant Strahman Valves, Inc. is a foreign corporation organized in the State of Pennsylvania and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Strahman valves' insulation, packing, and gaskets.

40. Defendant Strahman Valves, Inc. may be served by and through its registered agent Robert Jordan, 2801 Baglyos Circle, Bethlehem, PA, 18020.

41. Defendant Velan Valve Corp. is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Velan valves' insulation, packing, and gaskets.

42. Defendant Velan Valve Corp. may be served by and through its registered agent Corporation Service Company, 2711 Centerville Road, Ste. 400, Wilmington, DE, 19808.

43. Defendant Warren Pumps LLC is a foreign corporation organized in the State of Delaware and authorized to do business in the State of Florida. With respect to this Defendant, Plaintiff alleges exposure to friable asbestos emanating from at minimum Warren pumps' insulation, packing, and gaskets.

44. Defendant Warren Pumps LLC may be served by and through its registered agent The Corporation Trust Company, Corporation Trust Center, 1209 Orange Street, Wilmington, Delaware, 19801.

COUNT I
NEGLIGENCE

45. Plaintiff restates and re-alleges paragraphs one (1) through forty-four (44) above as though fully set forth herein as Count I of his Complaint and also states as follows:

46. At all times herein set forth, the Defendants' products were being employed in the manner and for the purposes for which they were intended.

47. Mr. Donson's exposure to and inhalation, ingestion or absorption of the asbestos fibers emanating from the above-mentioned products and related work duties was completely foreseeable and could or should have been anticipated by the Defendants.

48. The Defendants knew or should have known that the asbestos fibers contained in their products had a toxic, poisonous, and highly deleterious effect upon the health of persons inhaling, ingesting or otherwise absorbing them.

49. That on or around 7/8/16, Mr. Donson learned that he had Asbestosis, and at a subsequent time learned said disease was wrongfully caused. *See* Report of Dr. Modh, attached hereto.

50. At all times relevant to this action, the Defendants had a duty to exercise reasonable care and caution for the safety of Mr. Donson and others working with and around the products of the Defendants containing asbestos.

51. The Defendants failed to exercise ordinary care and caution for the safety of Mr. Donson in one or more of the following respects:

(a) Included asbestos in their products, even though it was completely

foreseeable and could or should have been anticipated that persons such as Mr. Donson working with or around them would inhale, ingest or otherwise absorb great amounts of that asbestos;

- (b) Included asbestos in their products when the Defendants knew or should have known that said asbestos fibers would have a toxic, poisonous and highly deleterious effect upon the health of persons inhaling, ingesting or otherwise absorbing them;
- (c) Included asbestos in their products when adequate substitutes for the asbestos in them was available;
- (d) Failed to provide any or adequate warnings to persons working with and around the products of the dangers of inhaling, ingesting or otherwise absorbing the asbestos fibers contained in them;
- (e) Failed to provide any or adequate instructions concerning the safe methods of working with and around the products, including specific instructions on how to avoid inhaling, ingesting or otherwise absorbing the asbestos fibers in them;
- (f) Failed to conduct tests on the asbestos-containing products manufactured, sold, delivered or installed by the Defendants in order to determine the hazards to which workers such as the Plaintiff might be exposed while working with the products.

52. That as a direct and proximate result of one or more of the foregoing acts or omissions on the part of the Defendants, Mr. Donson was exposed to and inhaled, ingested or otherwise absorbed great amounts of asbestos fibers causing Mr. Donson to develop Asbestosis, which disabled and disfigured Mr. Donson. The Plaintiff has been compelled to expend and become liable for large sums of monies for hospital, medical and other health care services necessary for the treatment of the Mr. Donson's asbestos-induced disease and conditions; Mr. Donson experienced great physical pain and mental anguish as a result of the inhalation, ingestion and absorption of said asbestos fibers; that as a further result of the Mr. Donson's asbestos-induced disease and conditions, and Mr. Donson was hindered and prevented from pursuing his normal course of employment, thereby losing large sums of money which otherwise would accrue to him and his estate.

WHEREFORE, Plaintiff demands judgment for compensatory damages against the Defendants in a sum in excess of FIFTEEN THOUSAND (\$15,000.00) DOLLARS, and the costs of these proceedings, which will fairly and reasonably compensate for the Plaintiff's injuries.

COUNT II
STRICT LIABILITY

53. Plaintiff restates and re-alleges paragraphs one (1) through forty-four (44) above as though fully set forth herein as Count II of his Complaint and also states as follows:

54. At the time Defendants used, installed, manufactured, sold and distributed the asbestos-containing products to which Mr. Donson was exposed, said products were in a defective condition and unreasonably dangerous in that:

- (a) Said products contained friable asbestos fibers as a constituent substance;
- (b) Said fibers were highly toxic, deleterious, poisonous and harmful to the health of Mr. Donson and others similarly situated; and
- (c) Said products were not accompanied by any warning/instructions or by inadequate warning/instructions advising of the danger of exposure to asbestos or of precautions to be employed in the use of asbestos-containing products.

55. Defendants' defective products reached the point of Mr. Donson's exposure in substantially the same condition as when defectively designed, used, manufactured, distributed, and sold.

56. At all times relevant to this action, said products were used in the manner and environment intended, and in the manner reasonably foreseeable and anticipated by the Defendants.

57. Defendants are strictly liable to Plaintiff for all injuries and damages that were contracted as a direct and proximal result of Mr. Donson's exposure to asbestos from said

products.

58. Asbestos and asbestos-containing products are inherently dangerous and as such, the Defendants, who made or sold asbestos or the equipment, processes or other things necessary for its use, are strictly liable to Plaintiff for all injuries and damages which were contracted thereby.

59. All Defendants who assisted, directly or indirectly, in the leasing or licensing of asbestos, asbestos-containing products and all equipment necessary for their use are strictly liable to Plaintiff for all the injuries and damages which were contracted thereby.

60. The handling of asbestos packages, installation, removal and use of asbestos and asbestos-containing products is an ultra-hazardous activity, and Defendants, who assisted directly or indirectly in this, are strictly liable for the Plaintiff's injuries which were caused thereby.

61. The Defendant-manufacturers and suppliers warranted the asbestos products for their intended purpose and use. Defendants violated this warranty, as the product was neither packaged nor provided in a method proper for its intended use; and Defendants are strictly liable to the Plaintiff for all injuries caused thereby.

62. That as a direct and proximate result of one or more of the foregoing acts or omissions on the part of the Defendants, Mr. Donson was exposed to and inhaled, ingested or otherwise absorbed great amounts of asbestos fibers causing Mr. Donson to develop Asbestosis, which disabled and disfigured Mr. Donson. The Plaintiff has been compelled to expend and become liable for large sums of monies for hospital, medical and other health care services necessary for the treatment of the Mr. Donson's asbestos-induced disease and conditions; Mr. Donson experienced great physical pain and mental anguish as a result of the inhalation,

ingestion and absorption of said asbestos fibers; that as a further result of the Mr. Donson's asbestos-induced disease and conditions, and Mr. Donson was hindered and prevented from pursuing his normal course of employment, thereby losing large sums of money which otherwise would accrue to him and his estate.

WHEREFORE, Plaintiff demands judgment for compensatory damages against the Defendants in a sum in excess of FIFTEEN THOUSAND (\$15,000.00) DOLLARS, and the costs of these proceedings, which will fairly and reasonably compensate for Plaintiff's injuries.

COUNT III:
FRAUDULENT INDUCEMENT

63. Plaintiff re-alleges the allegations set forth in paragraphs one (1) through forty-four (44) above, as if set forth herein.

64. Defendants, by acts omission, made false statements concerning the safety of their asbestos-containing products. Specifically, as alleged members of the asbestos industry Defendants failed to provide warnings of the dangers of asbestos associated and/or integrated into their products. *See Attached Expert Report of Dr. David Rosner.*

65. Defendants knew at the time Plaintiff's exposure to their products that asbestos was unsafe.

66. Defendants' acts of omission intentionally and/or negligently caused Plaintiff to be induced by their omission such that Plaintiff was exposed to asbestos emanating from Defendants' products.

67. In the course of his work, Plaintiff reasonably and justifiably relied on these omissions by Defendants.

68. As a direct and proximate result of Plaintiff's reliance on the omissions by Defendants, Plaintiff has suffered damages.

WHEREFORE, Plaintiff demands judgment for compensatory damages against the Defendants in a sum in excess of FIFTEEN THOUSAND (\$15,000.00) DOLLARS, and the costs of these proceedings, which will fairly and reasonably compensate for the Plaintiff's injuries.

DEMAND FOR JURY TRIAL

Plaintiff respectfully demands a trial by jury for all issues so triable in this action.

DATED this 29th day of September 2016.

Respectfully submitted,

/s/ Ben A. Vinson, Jr.

BEN A. VINSON, JR.

Florida Bar No. 0095227

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Attorney for Plaintiff

Expert Report
of
Gerald Markowitz PhD and David Rosner PhD
November 9, 2011

Introduction:

We have been asked to conduct a historical review of the relevant medical, scientific, industrial hygiene and industrial literature, as well as other relevant publically available information and provide our expert opinions, expressed to a reasonable degree of certainty, in answer to the following questions:

1. When was it known and therefore knowable that breathing dust containing asbestos could cause disease?
2. When was it known and therefore knowable that breathing dust containing asbestos could cause cancer?
3. Who was known to be at risk for contracting asbestos-caused diseases and when was that known?

Background, Qualifications and Experience:

David Rosner is Ronald H. Lauterstein Professor of Sociomedical Sciences History at Columbia University and Co-Director of the Center for the History of Public Health at Columbia's Mailman School of Public Health. An elected member of the National Academy of Sciences' Institute of Medicine, he received his BA from CCNY, his MPH from the University of Massachusetts and his PhD from Harvard in the History of Science. Until moving to Columbia in 1998, he was University Distinguished Professor of History at the City University of New York. In addition to numerous grants, he has been a Guggenheim Fellow, a recipient of a Robert Wood Johnson Investigator Award, a National Endowment for the Humanities Fellow and a Josiah Macy Fellow. He has been awarded the Distinguished Scholar's Prize from the City University, the Viseltear Prize for Outstanding Work in the History of Public Health from the APHA and the Distinguished Alumnus Award from the University of Massachusetts. He has also been honored at the Awards Dinner of the New York Committee on Occupational Safety and Health and he and Gerald Markowitz have been awarded the Upton Sinclair Memorial Lectureship "For Outstanding Occupational Health, Safety, and Environmental Journalism by the American Industrial Hygiene Association." He is author and editor of eleven books including A Once Charitable Enterprise (Cambridge University Press, 1982, 2004; Princeton University Press, 1987), "Hives of Sickness," Epidemics and Public Health in New York City (Rutgers University Press, 1995), and Health Care in America: Essays in Social History (with Susan Reverby). In addition, he has co-authored and edited with Gerald Markowitz numerous books and articles, including Deadly Dust: Silicosis and the Politics of Occupational Disease in Twentieth Century America, (Princeton University Press, 1991; 1994; University of Michigan, 2005), Children, Race, and Power: Kenneth and Mamie Clarks' Northside Center, (University Press of Virginia, 1996; Routledge Press, 2001); Dying for Work, (Indiana University Press, 1987) and "Slaves of the Depression," Workers' Letters About Life on the Job, (Cornell University Press, 1987). He and Gerald

Markowitz have authored Deceit and Denial: The Deadly Politics of Industrial Pollution (University of California Press/Milbank, 2002) and Are We Ready? Public Health Since 9/11 (University of California Press/ Milbank , 2006). In 2008 he edited The Contested Boundaries of Public Health, (with James Colgrove and Gerald Markowitz) from Rutgers University Press in 2008. His newest book (with Gerald Markowitz), titled Lead Wars, (University of California Press, forthcoming) details the recent conflicts at Johns Hopkins over studies of children placed in homes with low level lead exposure.

Gerald Markowitz, PhD is University Distinguished Professor of History at the John Jay College and the City University Graduate Center of the City University of New York. At CUNY he is also on the faculty of the PhD Program in Public Health. He is also Adjunct Professor of Sociomedical Sciences at Columbia University's Mailman School of Public Health. He received his BA from Earlham College and his MA and PhD from the University of Wisconsin. He is the recipient of a Robert Wood Johnson Investigator Award, a National Endowment for the Humanities Research Grant two grants from the National Science Foundation and others from the Milbank Memorial Fund. He has been awarded the Visellear Prize for Outstanding Work in the History of Public Health from the APHA. He has also been honored at the Awards Dinner of the New York Committee on Occupational Safety and Health and he and David Rosner have been awarded the Upton Sinclair Memorial Lectureship "For Outstanding Occupational Health, Safety, and Environmental Journalism by the American Industrial Hygiene Association. He is author and editor of eleven books including, with David Rosner, Deadly Dust: Silicosis and the Politics of Occupational Disease in Twentieth Century America, (Princeton University Press, 1991;1994; University of Michigan, 2005), Children, Race, and Power: Kenneth and Mamie Clarks' Northside Center, (University Press of Virginia, 1996; Routledge Press, 2001); Dying for Work, (Indiana University Press, 1987) and "Slaves of the Depression," Workers' Letters About Life on the Job, (Cornell University Press, 1987), Deceit and Denial: The Deadly Politics of Industrial Pollution (University of California Press/Milbank, 2002) and Are We Ready? Public Health Since 9/11 (University of California Press/ Milbank , 2006). In 2008 he edited The Contested Boundaries of Public Health, (with James Colgrove and David Rosner) from Rutgers University Press in 2008. His newest book (with David Rosner), titled Lead Wars, (University of California Press, forthcoming) details the recent conflicts at Johns Hopkins over studies of children placed in homes with low level lead exposure.

Our qualifications are more fully laid out in Exhibits A and B which contain our CVs.

Opinions and Bases for Opinions

Our opinions to a reasonable degree of certainty are:

1. In the early 1900s it was known that breathing asbestos dust was hazardous and by 1930 it was well recognized.
2. Beginning in the 1930s there was a suspected link between breathing asbestos dust and cancer and by the mid-1940s it was acknowledged by many medical researchers. It was confirmed by the epidemiological studies of Sir Richard Doll and by publications in JAMA and elsewhere by 1955.
3. It was known in the early 1900s through the 1930s and 1940s that the people at risk were the people inhaling asbestos.

The bases for these opinions are expressed below. We have reviewed and relied upon the references cited throughout this report as well as those contained in Appendix C, the Annotated Bibliography.

We reserve the right to supplement this report upon review of additional information or if asked to answer other questions.

There is an enormous literature that documents the relationship between asbestos exposure and disease. While some have traced this knowledge back to the ancients, the modern literature can be safely said to date to the beginning of the last century. Factory reports in England from the first decade of the 20th century note the poor health of weavers and others in manufacturing establishments using asbestos fibers. By the teens insurance industry statisticians noted the high mortality of these workers and in the late 1920s and early 1930s the medical community became acutely aware of clinical reports and major studies of asbestos workers who suffered what became known as asbestosis. By this time it was known that workers other than those directly handling asbestos were at risk for asbestosis. Bystanders and others who breathed dust containing asbestos

fibers also contracted the disease. Methods for reducing the risk of asbestos disease, including educating the workers and industrial engineering controls, were known. By the middle of the Depression exposure to asbestos was reported to be associated with lung cancer risks and by the mid- to- late 1940s this association was confirmed by many case studies that appeared in the medical literature and by reports in medical texts that made the link explicit. By the mid-1950s epidemiological studies confirmed this relationship.

While lung cancer was the first of the tumors associated with asbestos exposure other, rarer tumors in other parts of the body began to be identified in the 1950s and 1960s. By the mid 1960s mesotheliomas were identified and confirmed in many case reports and surveys of workers and by-standers. The range of diseases and cancers associated with asbestos exposure were widely acknowledged and documented by the 1960s.

Asbestos-related diseases in the early 1900s were rooted in work and factory production in the growing industrial economies of Britain, the United States and other industrial societies. In this new industrial environment where large portions of the workforce were now working indoors, in pits or underground, in jobs that entailed blasting, drilling, carding, weaving, cutting, and other dust-producing processes, medical researchers and clinicians, insurance actuaries and government officials observed that these dusts endangered the workforce. The variety of lung conditions produced by the "dusty trades" as they became known were called the "pneumoconioses," i.e. dust-diseases of the lungs, and were all characterized by shortness of breath (dyspnea), long latency periods, irreversible symptoms and increasingly severe disability often resulting in death years or decades after exposure. Initially, the major focus was on silica dusts produced in the sandblasting, mining, granite cutting, foundry and construction trades. Silica dust produced an

unusual disease of the lungs, silicosis, which appeared years – even decades – after exposure and provided the medical and research community a new model of chronic lung disease.

In 1898 the Annual Report of the Chief Inspector of Factories and Workshops in Britain noted “the evil effects of asbestos dust” in her examination of the “dusty occupations.” It stood out “on account of their easily demonstrated danger to the health of the workers, and because of the ascertained cases of injury to bronchial tubes and lungs medically attributed to the employment of the sufferers.” The Lady Inspector singled out “asbestos sifting and carding, silk opening” and noted that there were few precautions taken “to subdue the dust [and] no sort of ventilation being applied.” The link between inadequate ventilation, unsafe working conditions and dust were henceforth identified as of particular concern. In this early report the dangers from asbestos dust were ascribed to “the sharp, glass-like jagged nature of the particles [which when they] remain suspended in the air of a room, in any quantity ... have been found to be injurious, as might have been expected.”¹ The following year the Inspectors noted that the “jacketing for pipes, involving use of asbestos” was injurious to the workforce.² Also in the early century these factory inspectors suggested that the prevention of disease could be attained by improved ventilation and engineering controls.³ In Britain, the most distinguished medical personnel remarked that asbestos had been identified as dangerous by ancient cultures.⁴ The first clinical case of asbestos disease was documented in 1907 Dr. H. Montague Murray who reported on a 33

¹ Report of the Lady Inspectors, Annual Report of the Chief Inspector of Factories and Workshops for the Year 1898, “Part II – Reports,” London, 1899, pp. 171-172.

² Report of the Lady Inspectors, Annual Report of the Chief Inspector of Factories and Workshops for the Year 1899, London, [1900], p.264.

³ Great Britain Factory Department, Lady Inspectors, Annual Report of the Chief inspector of Factories and Workshops for the Year, 1906, London, His Majesty’s Stationary Office, 1907, p. 220.

year old carding room worker who had developed fibrosis of the lungs.⁵ In the second decade of the twentieth century the Labour Department in Canada investigated “the health of those employed in the asbestos industry.” They cited “One of the oldest medical practitioners in Thetford [who] expressed the view that the asbestos dust floating in the atmosphere of the cobbing room had a weakening effect on the lungs of those employed.” They suggested the use of engineering controls and respirators to keep the dust down.⁶

The British studies and reports on the dangers of asbestos dust were widely available in the United States when H.K. Pancoast, T.G. Miller and H.R.M. Landis published the first “Roentgenologic Study of the Effects of Dust Inhalation Upon the Lungs,” in the Transactions of the Association of American Physicians in 1917. In the discussion of their oral presentation at the meeting a Dr. Kober said, that “persons exposed to the habitual inhalation of dust, sooner or later would develop some chronic inflammation of the respiratory system.”⁷

The dangers from asbestos were well documented by Frederick Hoffman, the Vice President and actuary of the Prudential Life Insurance company in a major report produced in 1918 for the U.S. Bureau of Labor Statistics on “Mortality from Respiratory Disease in Dusty Trades (Inorganic Dusts).” This report detailed the variety of mineral dusts that potentially posed a threat to the workforce and gathered the existing statistics from the Prudential records of mortality. He remarked on the variety of occupational groups that were at risk of asbestos disease

⁴ Oliver, Thomas, Dangerous Trades, New York: E.P. Dutton, 1902, p.25.

⁵ Murray, H. Montague, MD., “Report of the Departmental Committee On Compensation for Industrial Diseases,” HMSO, Cd. 3495, p. 14; Cd. 3496, Minutes of Evidence, p. 127.

⁶ “Effects of Asbestos Dust on Workers’ Health in Asbestos Mines and Factories,” Labour Gazette (Can.), 12 (1912), 761-762.

including workers who covered pipes, furnaces, plastered walls, used asbestos in roofing, insulated electrical equipment, and engaged in the construction of storage rooms of refrigerating plants.⁸ Asbestos workers were seen by the insurance industry as being at particular risk for early death and thus it was “the practice of American and Canadian life insurance companies [that] asbestos workers are generally declined on account of the assumed health-injurious conditions of the industry.”⁹ Hoffman quoted from the 1910 Annual Report of Chief Inspector of Factories and Workshops, which included a report by Collis on the asbestos industry that stressed the need for dust protection for the workforce because of the deaths of asbestos workers. Hoffman also quoted Collis who noted that it was not only those who directly produced asbestos dust who were at risk of disease but also by-standers in the vicinity: “Women who sew the mattresses into sections with asbestos threads worked close to the man who beat the mattresses and of necessity inhaled the dust.”¹⁰ Hoffman concluded that there was an “urgent need for extensive investigation of health effects of asbestos manufacture.”¹¹

In the same year of Hoffman’s study academic medical researchers began to perform more sophisticated surveys of the workforce, using the new technology of the x-ray to identify pathological changes in the lungs of workers exposed to dusts, including asbestos. Pancoast and

⁷ Pancoast, H.K., T.G. Miller, and H.R.M. Landis, “A Roentgenologic Study of the Effects of Dust Inhalation upon the Lungs,” Transactions of the Association of American Physicians, 32 (1917), 97-108.

⁸ Hoffman, F. L. “Mortality from Respiratory Disease in Dusty Trades (Inorganic Dusts),” Bulletin, U.S. Bureau of Labor Statistics, #231, U.S. Bureau of labor, Washington DC, pp. 176-180, 1918.

⁹ Hoffman, F. L. “Mortality from Respiratory Disease in Dusty Trades (Inorganic Dusts),” Bulletin, U.S. Bureau of Labor Statistics, #231, U.S. Bureau of labor, Washington DC, p.178, 1918.

¹⁰ Hoffman, F. L. “Mortality from Respiratory Disease in Dusty Trades (Inorganic Dusts),” Bulletin, U.S. Bureau of Labor Statistics, #231, U.S. Bureau of labor, Washington DC, p.179, 1918.

¹¹ Hoffman, F. L. “Mortality from Respiratory Disease in Dusty Trades (Inorganic Dusts),” Bulletin, U.S. Bureau of Labor Statistics, #231, U.S. Bureau of labor, Washington DC, p.180, 1918.

his colleagues published a survey of 137 workers including 15 asbestos workers. They described the three stages of pneumoconiosis and noted that the second stage “is characterized by a more or less uniformly arranged mottling throughout the lung structure due to depositions of dust in the lymph structures, cells, and fibrous tissue interspaces, with the addition of a certain amount of localized fibrosis....It occurs comparatively early in coal miners, cement workers and certain metal grinders, somewhat later in potters and asbestos workers....”¹² Animal experiments reported in the British Medical Journal, an esteemed journal that was available internationally, confirmed that asbestos dust caused fibrosis.¹³ More animal experiments would be performed in the coming years and decades.¹⁴

Evidence accrued that documented the long latency of asbestos-related diseases. In 1925, Pancoast and his colleagues used the x-ray to show that of 17 asbestos workers, two “showed first stage changes and the other 15 definite second stage appearances.” The men who had worked longest at their jobs (17 and 14 years) “showed very definite diffuse, ‘soft’ spots throughout both lungs and another showed about the same appearance after fourteen years’ occupation. Very slight nodular shadows were found in one man after only two years’ occupation.”¹⁵ Cooke and his co-investigators used microscopy to look at the cells of workers in an asbestos factory where “a very

¹² Pancoast, Henry K., T. Grier Miller and Henry R.M. Landis, “A Roentogologic Study of the Effects of Dust Upon the Lungs,” American Journal of Roentgenology, 5 (1918), 129, 136.

¹³ Cooke, W. E. “Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust,” The British Medical Journal, 2, No. 3317, (July 26, 1924), 147.

¹⁴ Stewart, M. J., “Asbestosis Bodies in the Lungs of Guinea Pigs After three to Five Months Exposure in an Asbestos factory,” Journal of Pathology and Bacteriology, 33 (1930), 848; Schuste, Norah, “Pulmonary Asbestosis in a Dog,” Journal of Pathology and Bacteriology, 34 (1931), 751-757.

¹⁵ Pancoast, Henry K. and Eugene P Pendergrass, “A Review of our Present Knowledge of Pneumoconiosis based upon Roentgenologic Studies, with Notes on the Pathology of the Condition,” The American Journal of Roentgenology and Radium Therapy, 14 (November 1925), 419-420. See also, Pancoast, Henry K. and Eugene P Pendergrass, Pneumoconiosis (Silicosis): A Roentgenological Study, New York: Paul B. Hoeber, Inc, 1926, p.155.

considerable amount of dust is generated.” They concluded that asbestos dust produced a unique pathology in the lungs of these workers and that they had “never seen anything parallel to this in pneumoconiosis due to other dusts....”¹⁶ This British research was reported on by the most widely distributed medical journal in the United States, the Journal of the American Medical Association, (JAMA), in an article titled “Pneumoconiosis Caused by Asbestos Dust.”¹⁷ S. Roodhouse Gloyne, in an article titled, “The Presence of the Asbestos Fibre in the Lesions of Asbestos Workers,” remarked that the term “asbestos bodies” should replace the then-current term, “curious bodies” since it was clear that asbestos was the cause of the pathological change.¹⁸ Thomas Oliver’s report on “Pulmonary Asbestosis in its Clinical Aspects,” was published in the American Journal of Industrial Hygiene in 1927 (as well as in the British Medical Journal) and JAMA also reported on it.¹⁹

The attention to asbestosis continued to accrue in the American medical literature in the late 1920s. An editorial in the widely distributed JAMA remarked on Cooke’s 1924 report, noting that “many other cases seem to have occurred” and that while they hoped that factory conditions were improving, “asbestosis, because of its dangers and its unique pathologic features, deserves

¹⁶ Cooke, W. E. “Pulmonary Asbestosis,” The British Medical Journal, 2, No. 3491, (December 3, 1927), 1024-25. See also, Cooke, W. E. and C. F. Hill, “Pneumokoniosis Due to Asbestos Dust,” Journal of the Royal Microscopical Society, Series 3, Vol. 7 (1927), 232-238.

¹⁷ “Pneumoconiosis Caused by Asbestos Dust,” JAMA, 89 (July 23, 1927), 304.

¹⁸ Gloyne, S. Roodhouse, “The Presence of the Asbestos Fibre in the Lesions of Asbestos Workers,” Tubercle, June 1929, 407. See, Stewart, M. J. and A.C. Haddow, “Demonstration of the Peculiar Bodies of Pulmonary Asbestosis” Journal of Pathology and Bacteriology, 32 (1929), 172. See, for example, the references to “curious” or “peculiar” bodies, Cooke, W. E., and C.F. Hill, “Further Observations on Pulmonary Asbestosis, with Special Reference to Asbestos Dust and the Curious Bodies Found in the Lungs,” Journal of the Royal Microscopical Society, Ser. 3, Volume 50 (1930), 15-19.

¹⁹ Oliver, Thomas, “Pulmonary Asbestosis in its Clinical Aspects,” Journal of Industrial Hygiene, (November 1927), 483-485. (Also printed as “Clinical Aspects of Pulmonary Asbestosis,” in The British Medical Journal, 2, No. 3491, (December 3, 1927), 1026-27. Also reported in JAMA, 89 (1927), 2285.

more attention than it has had.”²⁰ Cooke himself, in an article in the prestigious British Medical Journal noted that “Pulmonary asbestosis has become a question of international interest and importance.”²¹ In 1929, the British Medical Journal noted that asbestos had become such a significant problem in the UK that the government was considering making it a compensable disease.²² Authors suspected that a “wider recognition of this disease” would lead to more cases being identified and that “the mortality statistics [would therefore] probably be swelled.” The latency of this disease also made recognition difficult for it was usually first recognized more than five years following exposure.²³ It was clear that asbestosis was a dread disease for which there was little treatment and no cure. As W. Burton Wood remarked in 1929, “when the diagnosis is established, the prognosis appears to be grave.”²⁴

By 1930 when the first clinical report of an asbestos victim was reported in the United States a huge literature had developed that documented the importance of asbestos as a cause of the pneumoconiosis, asbestosis. Asbestosis was documented by clinicians, by government inspectors and insurance officials, by researchers in university laboratories, by roentologists and pathologists, by editors of leading medical journals in both the United States and the UK and by industry executives themselves through their trade publications. It was even being considered for

²⁰ Editorial, “Pulmonary Asbestosis,” Journal of the American Medical Association, 90 (2), (January 14, 1928), 119-20.

²¹ Cooke, W. E. “Pulmonary Asbestosis,” The British Medical Journal, 2, No. 3534, (September 29, 1928), 585. Numerous case reports appeared in Europe and South Africa. See, for example, appendix for some of these and specifically, Simson, F. W., “Pulmonary Asbestosis in South Africa,” The British Medical Journal, 1 (May 26, 1928), 885-887 for reports as far away as South Africa and Lovisetto, Domenico, “Pulmonary Asbestosis,” Records of the International Conference on Silicosis at Johannesburg, August 1930, No. 13, Geneva, International Labour Office.

²² “Medical Notes in Parliament,” The British Medical Journal, 2 (December 21, 1929), 1182.

²³ Haddow, A.C., “Clinical Aspects of Pulmonary Asbestosis,” The British Medical Journal, 2, No. 3586, (September 28, 1929), 580-581.

²⁴ Wood, W. Burton, “Pulmonary Asbestosis,” Tubercle, 10 (May 1929), 356.

inclusion in workmen's compensation legislation in Britain. The first claim recorded in the United States was in 1927 in Massachusetts.²⁵

The first clinical reports in the United States were by Mills, Lynch and Smith, and Soper in 1930, the latter two in the national publications, JAMA and the American Review of Tuberculosis. In Mills' first paper published in a state medical society journal, Minnesota Medicine, he reflected on the growing attention to this disease in the United States during the 1920s, although, he claimed, he was the first to document a case of what he called "an incurable disease." He opined that because "asbestos is mined or manufactured in many portions of this country," he was reporting the case "in the hope of arousing more widespread interest in the disease and establishing it as an entity with which American physicians surely will have to deal."²⁶ Indeed, JAMA picked up on Mills' report bringing it to the attention of physicians all across the United States, pointing out that "asbestos is mined and manufactured in many parts of this country and that pulmonary asbestosis surely will be encountered."²⁷

The scope of knowledge about the dangers from asbestos was captured in an authoritative epidemiological study of 363 asbestos textile workers, about a quarter of who showed signs and symptoms of fibrosis of the lungs in England. Organized by E.R.A. Merewether, the Medical Inspector of Factories and C.W. Price, the Engineering Inspector of Factories for the British

²⁵ Lanza, A.J., "Asbestosis," JAMA, 106 (February 1, 1936), 368: "While Hoffman called attention to the possible harmfulness of asbestos dust in 1918, it was not until February 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workmen's compensation in Massachusetts."

²⁶ Mills, Ralph G. "Pulmonary Asbestosis: Report of a Case," Minnesota Medicine, 13 (July 1930), 495 - 499. See also, Soper, Willard, "Pulmonary Asbestosis," American Review of Tuberculosis, 22 (1930) 571-584 and Lynch, Kenneth M. And W. Atmar Smith, "Asbestos Bodies in Sputum and Lung," JAMA, 95 (August 30, 1930), 659-661 and Lynch, Kenneth M. And W. Atmar Smith, "Pulmonary Asbestosis II: Including the Report of a Pure Case," American Review of Tuberculosis, 23 (1931), 643 - 660.

government, the report synthesized the huge literature that had developed over the previous decades. Citing the U.S. British, South African, French, Italian and German literature, it laid out the broad dimensions of the problem not just in the textile industry but in a broad range of other industries as well. The dangerous trades they identified included paper manufacturing, tile manufacturing, the production of insulation materials, the use of asbestos in brake and clutch linings, and its use as a sheathing for electrical conductors, cables and wiring. The scope of the problem led them to conclude "that the inhalation of asbestos dust over a period of years results in the development of a serious type of fibrosis of the lungs." They also identified various engineering controls that were necessary for the suppression of dust in industry.²⁸ In his report he notes that among the "preventive measures" should be educating the worker "to a sane appreciation of the risk."²⁹ Merewether also reported on his work in the United States publishing that same year in the Journal of Industrial Hygiene.³⁰

Cross-sectional epidemiological studies were also initiated at the same time by American investigators. Between 1929 and 1931, A.J. Lanza and his colleagues at Metropolitan Life Insurance Company performed a study of Canadian asbestos miners and fabricators in both

²⁷ "Pulmonary Asbestosis," JAMA, 95 (November 8, 1930), 1431.

²⁸ Merewether, E.R.A., and C.W. Price, Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry, London, Her majesty's Stationary Office, 1930. A few years later, Merewether was even more explicit: asbestos was used in "a vast number of articles, ranging from matches to filter pads, from paints to roofing tiles, from high pressure jointing to electrodes, and from brake-linings to insulating materials in great variety. The phenomenal expansion of the motor car industry in the past twenty years has caused a corresponding increase in the demand for asbestos fabric brake linings, asbestos composition clutch rings, and latterly of moulded asbestos brake linings. Many other examples will come to mind." See: Merewether, E. R. A., "A Memorandum on Asbestosis," Tubercle, 15 (November, December, 1933 and January 1934) 69-81; 109-118; 152-159 at 71.

²⁹ Merewether, E.R.A., and C.W. Price, Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry, London, Her majesty's Stationary Office, 1930, p. 17.

³⁰ Merewether, E.R.A., "The Occurrence of Pulmonary Fibrosis and other Pulmonary Affections in Asbestos Workers," Journal of Industrial Hygiene, 12 (May 1930), 198 - 222.

Canada and the U.S. for the asbestos industry which they published in 1935. They found that the longer a worker was exposed to asbestos the greater the chance that his or her X-ray would show that “prolonged exposure to asbestos dust caused a pulmonary fibrosis of a type different from silicosis.”³¹

The widespread acknowledgement of the dangers of asbestos can be gleaned from the fact that in 1930 alone there were reports about asbestos disease by the International Labor Organization, the Monthly Labor Review, the Canadian Public Health Journal, the British Medical Journal, The Lancet as well as in other medical journals and at an international conference (see appendix C).³²

The concern with asbestos disease in the United States corresponded to the growth in the uses of asbestos in a wide variety of industries. Production increased dramatically in the United States in the first three decades of the twentieth century from “96,490 tons in 1910 to 434,938 tons in 1929.”³³ Henry Pancoast and Eugene P. Pendergrass pointed out, in their review of the

³¹ Lanza, A. J., William J. McConnell, and J. William Fehnel, “Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers,” Public Health Reports, 50 (January 4, 1935), 1- 12. See also Page, Robert C. “A Study of the Sputum in Pulmonary Asbestosis,” American Journal of Medical Science, 189 (1935), 44-55. Here the authors point out the short length of time necessary for the appearance of the disease: “The length of exposure to dust has varied within wide limits, but it is clear that gross disease may follow even as short an exposure as 18 months, provided the dust concentration has been sufficiently high.”

³² See for examples: Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Work, Geneva: International Labour Organizations, 1930, pp. 189-191; International Labour Office, Studies and Reports, Series F (Industrial Hygiene), No. 13, “Silicosis: Records of the International Conference Held at Johannesburg, 13-27 August 1930,” Geneva, 1930, p.27; “Pulmonary Asbestosis,” Monthly Labor Review, July 1930, pp. 74- 76; Pedley, Frank G., “Asbestosis,” Canadian Public Health Journal, 21 (1930), 576-577; “Pulmonary Asbestosis,” Asbestos: A Monthly Market Journal Devoted to the Interests of the Asbestos and Magnesia Industries, 11 (March 1930), 20; “Pulmonary Asbestosis,” The Lancet, April 19, 1930, pp. 870-871.

³³ Egbert, Dan S., “Pulmonary Asbestosis,” American Review of Tuberculosis, 31 (1935), 25-34 at p.25. See also, for uses: “Memorandum on the Industrial Diseases of Silicosis and Asbestosis,” London, His Majesty’s Stationary Office, 1932: The processes and industries included among others: “crushing, opening of crude asbestos, blowing, mixing and carding of it”; “making insulating mattresses and slabs or sections composed wholly or partly of

field in 1931, the long latency and high incidence that characterized this chronic disease: "As the number of years at work grows for a worker, they show fibrosis. For those working 20 years and over 80.9% showed fibrosis." They also pointed out the tragic consequences of prolonged exposure without respiratory protection: "Prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection...."³⁴ The growing documentation of asbestosis in the early 1930s led observers to remark on the difficulty of keeping up with the literature. One observer in the Lancet remarked on this phenomenon: "During the past two or three years observations on the asbestosis body have been published with such frequency that it is very difficult to keep track of them all."³⁵ The growing attention to the pneumoconioses in general and the documentation of asbestosis in the medical literature led industry to sponsor research at the prestigious Saranac Laboratory in upstate New York. Leroy Gardner and Donald Cummings began their toxicological, inhalation and animal studies of asbestosis in January, 1928 and because "so much interest has recently been manifested in the subject of asbestosis, a preliminary report [was] submitted" in 1930. They concluded, "The physical and chemical properties of asbestos are so unlike those of any other dust previously studied that, when inhaled,

asbestos;" "the sawing, grinding and turning in the dry state of articles composed wholly or partly of asbestos such as motor car brake and clutch linings, electric conductors and packings and jointings."

³⁴ Pancoast, Henry K. And Eugene P. Pendergrass, "A Review of Pneumoconiosis: Further Roentgenological and Pathological Studies," American Journal of Roentgenology and Radium Therapy, 26 (October 1931), 556-614 at pp. 585, 588, 592-593.

³⁵ Gloyne, S. Roodhouse, "The Asbestosis Body," The Lancet, 2 (1932), 1351. See also, Simson, F.W., and A. Sutherland Strachan, "Asbestosis Bodies in the Sputum: A Study of Specimens from Fifty Workers in an Asbestos Mill," The Journal of Pathology and Bacteriology, 34 (1931), 1-4. In this survey "asbestos bodies... were present [in the sputum] in 48 out of 50 workers examined." Editorial, "German Work on Pulmonary Asbestosis," The Lancet, 2 (July 9, 1932), 93: "The statistical tables appear to indicate that moderately severe asbestosis takes some five years to develop, while none of the workers examined who had had ten years or more exposure was free from the signs of the disease."

particles of this substance provoke an unusual type of reaction in the lung.”³⁶ During this period research increased on the biological mechanisms by which asbestos created fibrosis in the lungs (see appendix C). Also, during this period recognition that the lungs were not the only organ affected by asbestos began to accrue. Medical literature showed that organs far from the site of primary contact ended up diseased by asbestos: “In asbestosis the lung is not the only organ invaded by asbestosis bodies. As we have shown, the spleen and the lymph nodes may harbor large asbestosis bodies.”³⁷ Industrial engineering journals recognized the potential hazards of dust and asbestos specifically pointing out that “Asbestos and every operation in which it is used” is “seriously harmful.”³⁸ These uses were expanding. The asbestos industry in Pennsylvania, for example, “consists mainly of several fabricating plants engaged in the making of asbestos cloth, brake lining, insulating tape, asbestos rope and wick, and other miscellaneous products.” It was also used in textiles, weaving, gasket making as well as insulation in steam pipes, asbestos cements, shingles, lumber, molded fittings, and household appliances.³⁹ In England, by 1932, asbestosis was included as a compensable disease⁴⁰ as it was in Wisconsin for a worker whose “occupation was that of cleaning and restoring the asbestos on pipes.”⁴¹

³⁶ Gardner, Leroy U. And Donald E. Cummings, “Studies on Experimental Pneumonokoniosis. VI. Inhalation of Asbestos Dust: Its Effect upon Primary Tuberculosis Infection,” Journal of Industrial Hygiene, 13 (February 1931), 65 - 81. Conclusion – JIH, 13 (March 1931), 97 - 114 at pp. 65 and 103.

³⁷ Stewart, Harold L. and Carl J. Bucher, “Asbestosis: Report of Two Cases,” Archives of Pathology, 12 (1931), 909-916 at p. 916.

³⁸ Willson, Frederick, “The Very Least an Employer Should Know About Dust and Fume Diseases,” Safety Engineering 62 (November 1931), 317-318.

³⁹ William S. Fulton, “Asbestosis,” Pennsylvania, Bureau of Industrial Standards, Department of Labor and Industry, 1935.

⁴⁰ “Memorandum on the Industrial Diseases of Silicosis and Asbestosis,” London, His Majesty’s Stationary Office, 1932.

⁴¹ “Effects of Dusts Upon the Respiratory System,” Industrial Commission of Wisconsin, Proceedings of a Conference, Chicago, Nov. 16-17, 1932, p. 180.

By 1932 while there was uncertainty as to the hazards of many mineral and organic dusts, silica and asbestos were singled out as the two about which there was no question. For example, Advisory Committee on Employment of Minors in Hazardous Occupations issued a report in Monthly Labor Review, the U.S. Department of Labor's official publication, "that Minors under the age of 18 [should] be excluded [from] occupations involving exposure to free silica dust, asbestos dust, or other dusts in injurious quantities."⁴² In addition, an article in Industrial Medicine cited one author who noted that "aside from silicosis and asbestosis, exact knowledge is not possessed on other dust diseases of the lungs."⁴³ Alice Hamilton, among the leading figures in occupational medicine, also highlighted the singular importance of the diseases silicosis and asbestosis in her summary of the activities of the Industrial Hygiene Section of the APHA: "Problems presented by industrial dusts are probably the most absorbing and important, and these particularly revolve around silicosis and asbestosis."⁴⁴

The medical, engineering, and social dimensions of asbestosis were well recognized by the early 1930s. C.O. Sappington captured the multidimensional nature of the problem thusly: "Asbestosis bodies are demonstrable in the sputum, in the lung tissue, and more recently, have been found in the lymph nodes and the spleen. The treatment of asbestosis apparently has been as unsuccessful as that of silicosis. It is believed that removal from exposure does not impede the progress of the disease, when once it is fully established. Prevention depends upon the same

⁴² "Child Labor: Report of Advisory Committee on Employment of Minors in Hazardous Occupations, Monthly Labor Review, 35 (1932), 1319.

⁴³ "Silicosis and other Dust Diseases," Industrial Medicine, 1 (November 1932), p. 95.

⁴⁴ "Review of Present Status," Industrial Medicine, 1 (November 1932), 108

procedures as in silicosis, with the added recommendation that mechanical devices be substituted for dusty hand work and that wet methods be used instead of dry.”⁴⁵

Industry, insurance, and government agencies were aware of this problem. The “Konicide Club” (Killer Dust Club) was composed of leading industrial hygienists and researchers concerned with the growing crisis over silicosis and pneumoconiosis in general. While “the liability crisis”⁴⁶ around silicosis spurred its development, asbestosis also was a concern. At its 1933 meeting Leroy Gardner of Saranac Laboratory “spoke briefly on ‘What is Known About Asbestosis,’ an early contribution to a subject that looms so large today in the occupational health field.”⁴⁷ By 1935 industry formed the Air Hygiene (later Industrial Hygiene Foundation). At its founding meeting of some 200 representatives of the “dusty trades” formed committees, one of which was to look specifically at asbestos, cork and non-metallic dusts.⁴⁸ A representative of the Johns Manville Corporation, Vandiver Brown, was one of seven people nominated for the Temporary Organization Committee. In his report about the meeting to another asbestos company, Brown noted that “only two forms of dust, namely, free silica and asbestos, are definitely known to produce disabling fibrosis of the lung.”⁴⁹

⁴⁵ Sappington, C.O., “Silicosis and Other Dust Diseases,” Industrial Medicine, 1 (December 1932), 158-68

⁴⁶ Beyer, David S., “The Mechanical Control of occupational Diseases,” National Safety News, August 1933. 21-22, 34: “an analysis of approximately \$300,000 in losses of one insurance company doing a general workmen’s compensation and liability business, for recent industrial disease claims, showing 54 per cent for cases involving pneumoconiosis (including silicosis, asbestosis, etc.)”

⁴⁷ Hatch, Theodore F. and Eugene P. Pendergrass, “The Konicide Club (1932-1940),” Journal of Occupational Medicine, 19 (May 1977), 352.

⁴⁸ Symposium on Dust Problems held at Pittsburgh, PA, January 15, 1935. “A Brief Outline of the Events leading up to and occurring at the ‘Symposium on Dust Problems’ held in Pittsburgh, Pa., January 15, 1935.

⁴⁹ Brown to M.F. Judd, Secretary Raybestos-Manhattan, Inc, Bridgeport, Ct, January 22, 1935.

By the mid-1930s, asbestos was used in a wide variety of products, exposing workers, bystanders, and others to its ill-effects. One author noted, “the asbestos industry has had a remarkably rapid expansion and by reason of the widespread and varied uses of this products which include ‘matches, filter pads, paints, roofing, high pressure jointing, electrodes, brake linings, clutch rings, and insulating material in a great variety.’” The industry “occupies an important, increasing, and permanent place in our economic establishment.”⁵⁰ With this developing knowledge of the dangers of asbestos came attempts to define means of protecting the workforce. In the mid-to-late 1930s authors suggested levels of exposure to asbestos that could be “thresholds” of danger, above which harm was likely to occur. Dreesen and his colleagues, in a report for the U.S. Public Health Service in 1938, suggested that workers not be exposed to more than 5 million particles of asbestos per cubic foot (mppcf). He argued that workers breathing in asbestos dust resulted in the “initiation of fine, interstitial, pulmonary fibrosis” whose symptoms were dyspnea, variable cough “which sometimes raises blood streaked sputum and loss of weight.”⁵¹ Despite the quantification of this exposure limit Warren Cook, an industrial hygienist for Zurich General Accident and Liability Insurance Company observed that the “toxic limit for asbestos is five million particles of dust per cubic foot of air.” But he pointed out that “this is a very small concentration, so small in fact that the condition may look good even to a critical eye and still present an exposure greater than this low limit.” Thus, if asbestos dust in the air was

Merewether, E. R. A., “A Memorandum on Asbestosis,” Tubercle, 15 (November, December, 1933 and January 1934) 69-81; 109-118; 152-159.

⁵⁰ McPheeters, S.B., “A Survey of a Group of Employees Exposed to Asbestos Dust,” Journal of Industrial Hygiene and Toxicology, 18 (April 1936), 229 -239 at p. 229.

⁵¹ Dreesen, W.C., et al., “A Study of Asbestosis in the Asbestos Textile Industry,” Public Health Bulletin No. 241, August 1938. See also, Sayers, R.R., and W.C. Dreesen, “Asbestosis,” American Journal of Public Health, 29

visible, it was clearly above the threshold.⁵² The American Conference of Governmental Industrial Hygienists adopted this 5 mppcf threshold.⁵³

Researchers had always showed interest in the relationship of asbestosis to other lung diseases such as silicosis and tuberculosis. But, by the early 1930s, it was established that asbestosis alone was a serious disease that slowly killed its victims. In the mid-1930s troubling observations that some asbestos victims appeared on autopsy to also suffer from cancer of the lung arose. In 1934, a British study of one hundred workers exposed to asbestos for between six months and 15 years, fifty three of whom (thirty-four women and nineteen men) had asbestosis, revealed that two of the asbestos victims also had carcinoma of the lung and one had “a few deposits of growth in the pleura.”⁵⁴ The issue of the relationship between cancer and asbestos exposure was “still an open question” which would occupy researchers for much of the next decade.⁵⁵ In 1935, researchers in the United States began to look seriously at the relationship. Kenneth Lynch and W. Atmar Smith reported in the American Journal of Cancer on a weaver in an asbestos factory who died from “pulmonary asbestosis with associated carcinoma of the lung.” The authors considered that it was “worthy of record in the interest of both diseases, as well as their possible relationship.”⁵⁶ Anthony Lanza of Metropolitan Life reviewed the insurance company’s files and observed that of six asbestosis victims confirmed on autopsy, one had

(March 1939), 205-214; National Safety Council, “Industrial Dust,” Health Practices Pamphlet No. 4, 1939.

⁵² Warren A. Cook, “The Occupational Disease Hazard,” Industrial Medicine, 11(April, 1942), p.194.

⁵³ “Threshold Limit Values for 1955,” adopted by ACGIH Public Health Reports, 71(February, 1956), 201-203.

⁵⁴ Wood, W. Burton, and S. Roodhouse Gloyne, “Pulmonary Asbestosis: A Review of One Hundred Cases,” The Lancet, 2 (December 22, 1934), 1383-85.

⁵⁵ Gloyne, S. Roodhouse, “Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis,” Tubercle, 17 (October 1935), 5-10.

carcinoma.⁵⁷ That same year, 1936, Dan Egbert and Arthur Geiger specifically addressed the possible relationship of cancer and asbestosis in an article in the American Review of Tuberculosis. They believed that their report on a Hungarian immigrant weaver in an asbestos factory was “especially interesting since it is apparently the first to describe primary carcinoma of the lung as an associated condition.” They argued “that the irritating effects of the inhaled asbestos particles may in this case have been a significant factor concerned in the development of the primary lung cancer seems sufficiently plausible to be worthy of consideration.”⁵⁸ Gloyne, who had published the first piece linking asbestos and cancer in 1935, followed up this article with another case of “an oat cell carcinoma” a year later.⁵⁹ In the late 1930s, reports began to cite this early literature, bringing attention to a possible relationship.⁶⁰ In 1938 the issue was of enough concern that A.J. Vorwald, who would become the director of Laboratories at Saranac Laboratories, conducted a study of “Postmortem examination of 3739 individuals exposed to dust, as compiled from the literature and from our own series” which “revealed 30 individuals with pulmonary carcinoma, or an incidence of 0.8 per cent.” He concluded at the time that “this incidence [was] lower than that reported in routine autopsy examinations of the general populations” leading him to conclude that “Inhaled dusts, therefore, except those containing recognized carcinogenic substances such as radium and tar, cannot in general be considered as

⁵⁶ Lynch, Kenneth M. and W. Atmar Smith, “Pulmonary Asbestosis III: Carcinoma of Lung in Asbesto-Silicosis,” American Journal of Cancer, 24 (1935), 56-64.

⁵⁷ Lanza, A.J., “Asbestosis,” JAMA, 106 (February 1, 1936), 368-69.

⁵⁸ Egbert, Dan S. and Arthur J. Geiger, “Pulmonary Asbestosis and Carcinoma,” American Review of Tuberculosis, 34 (1936), 143 - 150.

⁵⁹ Gloyne, S. Roodhouse, “A Case of Oat Cell Carcinoma of the Lung Occurring in Asbestosis,” Tubercle, 18 (December 1936), 100-101.

⁶⁰ Anderson, C.S. and J. Henry Dible, “Silicosis and Carcinoma of the Lung,” Journal of Hygiene, 38 (1938), 185-

etiological factors in the development of primary pulmonary carcinoma.”⁶¹ JAMA reported on Vorwald’s study and results.⁶² The International Labor Organization (ILO) was more cautious in its interpretation of this limited evidence. It noted that exposure to asbestos was shown to result in “four main complications” -- bronchitis, pneumonia, TB, emphysema – but also to two others “which are less common” including carcinoma. In 1938 “the number of cases ... described [was] small and it [was] not yet established that there is any aetiological connection between the two diseases,” yet there was “sufficient evidence to warrant careful observation in the future.”⁶³ The ILO warned that “a practical maxim of the greatest value is that every translation of fiberised asbestos in the factory produces dust which, if not controlled, is dangerous.”⁶⁴ J.V. Sparks picked up on Gloyne’s research and published in the British Journal of Radiology. This was, in turn, reported on in the American Journal of Industrial Hygiene and Toxicology in 1939 where industrial physicians and hygienists learned that among the chief complications of pulmonary asbestosis was carcinoma.⁶⁵

Between 1938 and 1942 there were at least six additional articles reported in the American and British literature about the relationship of asbestos and cancer. Some of these were reports or abstracts of German-language studies, including a 1938 article by Nordmann who reported on the

204.

⁶¹ Vorwald, Arthur J. And John W. Karr, “Pneumoconiosis and Pulmonary Carcinoma,” American Journal of Pathology, 14 (January 1938), 49-58. He also exposed 3,338 animals to various dusts and found a lower-than-expected carcinoma rate.

⁶² “Pneumoconiosis and Pulmonary Carcinoma,” JAMA, 110 (June 18, 1938), 2068.

⁶³ International Labour Office, *Occupation and Health: Encyclopedia of Hygiene, Pathology and Social welfare*, ILO, 1938, pp.9-10.

⁶⁴ International Labour Office, *Occupation and Health: Encyclopedia of Hygiene, Pathology and Social welfare*, ILO, 1938, p.14.

⁶⁵ Sparks, J.V. “Asbestosis,” British Journal of Radiology, 11 (June 1938), 371 - 377. Reported in Journal of Industrial Hygiene and Toxicology, 21 (1939), 54.

high number of cancers revealed on autopsy of workers who had asbestosis.⁶⁶ American researchers Lynch and Smith cited Nordmann and others as providing further documentation of the link between asbestos, asbestosis and cancer. They reported on another case but acknowledged that the evidence-base was too small to establish a causal relationship. By 1939 there were a total of eight documented cases linking asbestos to lung cancer.⁶⁷ By 1942 the number of reported cases had increased to ten and that inhalation of asbestos caused “irreparable damage” even in workers employed for long periods of time where “exposure to asbestos dust was relatively low.”⁶⁸

Wilhelm Hueper, the eminent student of industrial disease, published the classic, comprehensive study of Occupational Tumors and Allied Diseases in 1942, noting the growing number of case reports in England, Germany and the United States. In this massive tome Hueper lays out the basis for connecting lung cancer deaths to exposure to asbestos at the workplace. First, he noted that the “incidence of lung cancer in asbestosis of the lung ... is definitely excessive.” Second, “the young age of an appreciable portion of the cases ... and the relationship existing between the time of exposure to asbestos dust and the manifestation of the neoplasms” was “suggestive” of such a relationship.⁶⁹ The following year Hueper reported that “Asbestosis

⁶⁶ Abstract (by L. Teleky) of article by Nordmann, “Abstract of the Literature of Industrial Hygiene,” Supplementary to Journal of Industrial Hygiene and Toxicology, 20 (1938), 184 of M. Nordmann, “Occupational Cancer in Asbestos Workers,” Ztschr. F. Krebsforsch., 47 (1938), 288-302.

⁶⁷ Lynch, Kenneth M. And W. Atmar Smith, “Pulmonary Asbestosis: A Report of Bronchial Carcinoma and Epithelial Metaplasia,” American Journal of Cancer, 36 (1939), 567-574. Others as well were still unsure of the relationship because of small sample size. See: Macklin, Madge Thurlow and Charles C. Macklin, “Does Chronic Irritation Cause Primary Carcinoma of the Human Lung,” Archives of Pathology, 30 (1940), 924 - 955.

⁶⁸ Holleb, Herbert B and Alfred Angrist, “Bronchiogenic Carcinoma in Association with Pulmonary Asbestosis,” American Journal of Pathology, 18 (1942), 123-131 at p. 129.

⁶⁹ Hueper, W.C., Occupational Tumors and Allied Diseases, Springfield IL: Charles C Thomas, 1942, p.404. See also, Davis, Edwin, “Chemical Carcinogenesis, Drugs, Dyes, Remedies and Cosmetics with Particular Reference to

cancer of the lung is the most recent newcomer among the occupational cancers of this organ” in an article published in The Bulletin of the American Society of the Control of Cancer. He noted that “asbestosis cancer of the lung has for us a special hygienic and sociologic significance” since “the asbestos industry is most extensively developed in this country.”⁷⁰

During World War II the medical literature on both sides of the Atlantic continued to follow each other’s reports of cancer and asbestos exposure. Specifically, articles in the American literature, including the New England Journal of Medicine, the Journal of Industrial Hygiene and Toxicology, and the Industrial Hygiene Foundation’s Industrial Hygiene Digest, cited German studies that continued to document the link.⁷¹ The latter two abstracted an article by H.W. Wedler which noted that the “German and foreign literature” clearly documented the relationship: “These figures [that twenty percent of autopsied asbestosis victims had lung cancer] clearly show that lung cancer is the most common complication encountered in cases of asbestosis, with the exception of agonal pneumonia and cardiac insufficiency. Even tuberculosis, whose rate of incidence is generally far higher than that of cancer, is considerably lower than for lung cancer in these cases. This constitutes conclusive evidence of the close correlation between asbestosis and lung cancer.”⁷² Also, during the war the official publications of the major medical and public

Bladder Tumors,” The Journal of Urology, 49 (1943), 14 - 27.

⁷⁰ Hueper, W.C., “Cancer in Its Relation to Occupation and Environment,” American Society of the Control of Cancer, Bulletin, 23 (1943), 63-69.

⁷¹ Tabershaw, Irving R. And Manfred Bowditch, “Industrial Hygiene in 1944,” the New England Journal of Medicine, 231 (November 23, 1944), 706 - 710. cites Homburger and Welz. “Abstract of the Literature of Industrial Hygiene,” Supplementary to Journal of Industrial Hygiene and Toxicology, 26 (November 1944), 183; Abstract in Industrial Hygiene Foundation, Industrial Hygiene Digest, 9 (January 1945), 19 – Same as previous Abstract.

⁷² H. W. Wedler, “Lung Cancer in Asbestosis Patients,” Dtsch. Arch. Klin. Med. , 191 (1943), 189 - 209. (In German but cited several times in subsequent literature.)

health societies in the United States, the American Medical Association and the American Public Health Association, as well as the International Labour Organization, reported on the suspected link between asbestos and cancer.⁷³

Shortly after the war ended Wilhelm Hueper published articles in JAMA and the journal, Occupational Medicine about this relationship. In the latter Hueper was especially vivid in his description of what occupational cancers, including those caused by asbestos, did to American workers: "The continued occurrence of occupational cancers,... represents a challenge not only to the intelligence but also to the social conscience of human society, because industrial cancers may be compared with a biologic bomb having a delayed time fuse which may be placed in the body of the victim without his knowledge and realization and which may display its deadly effect many years later when the conditions connected with its introduction are often forgotten."⁷⁴ The responsibility of employers and sellers alike to warn anyone who came in contact with hazardous materials, including dusts, was recognized by the leading trade association for the chemical industry in 1945: "The education of employees regarding chemical hazards is, and must remain, the direct responsibility of their employers. However, such hazards are not confined to employees alone, and information concerning them should, so far as practical, reach every person using, transporting, or storing chemicals. The most practical means for the seller to disseminate this

⁷³ See: "Environmental Cancer," [Editorial] JAMA, 140 (November 25, 1944), 836, Levin, Morton L. "The Epidemiology of Cancer," American Journal of Public Health, 34 (June 1944), 611 -620 and International Labour Office, "Occupation and Health; Encyclopedia of Hygiene, Pathology and Social Welfare," Special Supplement: Industrial Health in Wartime, Montreal, p.9.1944,

⁷⁴ Hueper, W.C., "Significance of Industrial Cancer in the Problem of Cancer," Occupational Medicine, 2 (1946), at p.200 and Hueper, W.C., "Industrial Management and Occupational Cancer," JAMA, 131 (June 29, 1946), 738-741. See also, Perry, Kenneth, M.A., "Diseases of the Lung Resulting from Occupational Dusts Other Than Silica," Thorax, 2 (1947), 91 -120, at p.116: "Incidence of carcinoma of lung is abnormally high in those exposed to arsenic

information appears to be by labels affixed to containers of hazardous chemicals, bearing appropriate precautionary statements and instructions stated as simply and briefly as circumstances permit.”⁷⁵

The already documented cases of cancer of the lung and asbestosis spurred others to investigate this further. Following the war many more such cases were reported. In 1949, the publication of the “Annual Report of the Chief Inspector of Factories for the Year 1947” quantified the relationship between asbestosis and lung cancer, noting that between 1924 and 1946 13.2 percent of a sample of 235 autopsies of asbestosis victims also had lung cancer. This was ten times higher, the report noted, than the lung cancer rate among silicosis victims confirming that asbestos was indeed a cause of lung cancer. The average length of exposure for those victims with lung cancer was 16.5 years.⁷⁶ Studies in the United States also noted the high incidence of asbestos-related cancer.⁷⁷

While there was some dissent,⁷⁸ by 1949, leading American publications accepted this data and wrote that asbestos caused lung cancer. An editorial in JAMA, “Asbestosis and Cancer

dust and to asbestos.”

⁷⁵ Manufacturing Chemists Association, Manual L-1 “A Guide for the Preparation of Warning Labels for Hazardous Chemicals,” Adopted 1945; Revised April 1946, Part 1, p.1.

⁷⁶ “Annual Report of the Chief Inspector of Factories for the Year 1947,” January 1949, at p.70. See Merewether’s report on this study in Merewether, E. R. A., “The Pneumokonioses: Developments, Doubts, Difficulties,” Canadian Medical Association Journal, 62 (February 1950), 169-173. The importance of these finding were noted at the time: See: Mayers, May R. “Industrial Cancer of the Lungs,” Compensation Medicine, 4 (March-May 1952), 11 -18: P. 14: “The relation of asbestos to lung cancer, long suspected, has received new confirmation from the 1949 Annual Report of the Chief Medical Inspector of Factories in England.” See also, Kennaway, E. L. and N. M. Kennaway, “A Further Study of the Incidence of Cancer of the Lung and Larynx,” British Journal of Cancer, 1 (1947), 260-298 for evidence of the growing epidemiological study of this relationship.

⁷⁷ Lynch, Kenneth M. And W. M. Cannon, “Asbestosis: VI Analysis of Forty Necropsied Cases,” Diseases of the Chest, 14 (January-February, 1948), 874-885.

⁷⁸ See, for example, Smith, William E., “Survey of Some Current British and European Studies of Occupational Tumor Problems,” Archives of Industrial Hygiene, 5 (1952), 242 – 263 and the controversy found in “Cancer of the

of the Lung,” remarked on the sea-change that had overtaken medical thinking. “Until recently the coexistence of asbestosis and cancer of the lung was considered by many investigators a coincidence.... A causal relation between asbestosis and cancer of the lung is supported by the following observations: the incidence rate of cancer of the lung in this group is excessive, since the normal death rate from cancer of the lung among adults examined at necropsy at present is about 1 per cent of all necropsies.” This was particularly important “Since some 20,000 workers are employed in the asbestos-producing industries of this country and Canada and many additional thousands in various asbestos-consuming industries, increased attention to this probable occupational hazard of cancer of the lung by the medical profession is desirable.”⁷⁹

Among these “asbestos-consuming industries” were “the manufacture of fire-fighting suits, safety curtains, and boiler mattresses. Asbestos enters into the composition of lagging material for steam pipes, jointing for steam pipes, panelling [sic] of rooms, tiles, the lining of chemical pans, the coating of bulkheads of ships and marine piers and perhaps most significantly into the brake linings and clutch rings of motor cars.”⁸⁰ An article in Scientific American also listed asbestos as a human carcinogen.⁸¹ The number of articles documenting this relationship cascaded in the early 1950s (see appendix C).

The studies in the early 1950s by Hill and Doll Sir Richard Doll, confirmed for virtually all serious scientists the relationship between cancer and asbestos. In his article, “Mortality from

Lung: An Evaluation of the Problem,” Proceedings of the Scientific Session, Annual Meeting, American Cancer Society, November 3-4, 1953, New York: American Cancer Society, 1956.

⁷⁹ “Asbestosis and Cancer of the Lung,” JAMA, 140 (August 13, 1949), 1219-20.

⁸⁰ Wyers, H., “Asbestosis,” Postgraduate Medical Journal, December 1949, 631 – 638 at p.632.

⁸¹ Conklin, Groff, “Cancer and Environment,” Scientific American, 180 (January 1949), 11-15. See also, Doig, A.T., “Other Lung Diseases Due to Dust,” Postgraduate Medical Journal, December 1949, 639 – 649 and Smith, Lawrence

Lung Cancer in Asbestos Workers,” published in the British Journal of Industrial Medicine, he looked at the autopsy reports of 113 men who worked in asbestos factories and found “an increased mortality from lung cancer, other respiratory diseases and cardiovascular diseases, in association with asbestosis.” “From the data [which showed 11 lung cancer deaths when only 0.8 were expected] it can be concluded that lung cancer was a specific industrial hazard of certain asbestos workers.” He went on to say “that the average risk among men employed for 20 or more years has been of the order of 10 times that experienced by the general population. The risk has become progressively less as the duration of employment under the old dusty conditions has decreased.”⁸² Doll’s work was cited by the prestigious British Medical Journal in an editorial on lung cancer and occupation.⁸³ That same year Wilhelm Hueper, the chief of the Environmental Cancer Section of the National Cancer Institute in the United States published an editorial in the American Journal of Clinical Pathology, in which he concluded that “the epidemiologic and pathologic evidence supporting a causal relationship between asbestosis and cancer of the lung ... is quite substantial” and that “the available evidence is adequate for recognizing asbestosis cancer of the lung for medicolegal reasons as an occupational disease.”⁸⁴ By the end of the 1950s asbestos was generally cited as a cause of lung cancer in American medical textbooks.⁸⁵ This was

W. , “Pneumoconiosis and Lung Cancer,” Compensation Medicine, 2 (November, 1949), 3 - 10.

⁸² Doll, Richard, “Mortality from Lung Cancer in Asbestos Workers,” British Journal of Industrial Medicine, 12 (1955), 81-86 at p.86. See also his earlier study, Doll, Richard, “Bronchial Carcinoma: Incidence and Aetiology,” British Medical Journal, 2 (September 5, 1953), 521 - 527.

⁸³ Editorial, “Cancer of the Lung in Industry,” British Medical Journal, 2 (September 24, 1955), 780-781.

⁸⁴ Hueper, W.C., “Editorial: Silicosis, Asbestosis, and Cancer of the Lung,” American Journal of Clinical Pathology, 25 (December 1955), 1388-1389.

⁸⁵ DuBois, Kenneth P. and E. N. K. Geiling, Textbook of Toxicology, (New York: Oxford University Press, 1959), p.110; Cecil, Russell L. and Robert F. Loeb, eds., A Textbook of Medicine, 10th ed., vol. 2, Philadelphia: W.B. Saunders Co., 1959)p.993; Eckardt, R.E., Industrial Carcinogens, New York: Grune & Stratton, 1959, p.4.

confirmed by much evidence including prospective cohort studies of workers in a number of different trades. It was found that “asbestos workers had the second highest observed lung cancers ... of the 10 occupations.”⁸⁶

Although the risk of asbestos disease in bystanders was known, this issue, including the risk of both asbestosis and cancer, increased during the 1960s. In 1962, Milton Scheinbaum reported in the American Industrial Hygiene Association Journal that there were serious threat from airborne asbestos particles to “anyone working within fifty feet” of someone applying asbestos cement plaster with a pneumatic tool.⁸⁷ By the mid-1960s Wilhelm Hueper warned that “asbestosis and asbestos cancer hazards related to an inhalatory exposure to asbestos exist not only for asbestos workers proper ... but also for the large number of individuals who may sustain such contacts on an incidental basis. Such persons may be employed permanently or temporarily in or near operations where asbestos and asbestos products are produced or handled and where they inhale...”⁸⁸

The scope of the asbestos problem expanded dramatically in the post World War II era as the number of potential victims increased with the growing number of uses of the material. At a major conference on asbestos at the New York Academy of Sciences in 1964, Wilhelm Hueper stated that world-wide production of asbestos increased from 500 tons in 1880 to 330,000 tons in 1925 and quadrupled from 446,000 metric tons in 1938 just before the War to over 2,000,000 in

⁸⁶ Dunn, John E., George Linden and Lester Breslow, “Lung Cancer Mortality Experience of Men in Certain Occupations in California,” American Journal of Public Health, 50 (October 1960), 1475 – 1487 at 1478.

⁸⁷ Scheinbaum, Milton, “Some Health Hazards Associated with the Building Trades,” American Industrial Hygiene Association Journal, 23(Sept-October, 1962),353-358.

⁸⁸ Hueper, W.C., “Occupational and Nonoccupational Exposures to Asbestos,” Annals of the New York Academy of Sciences, 132 (1965), 184 – 195 at p.187.

1958, two decades later. By 1964, production was over 3 million tons. This was especially important in the United States as it was “the principal consuming country.”⁸⁹ At the same conference the keynote speaker, J.C. Gilson, the British epidemiologist, told his fellow scientists that “Asbestos can truly be called the 20th century mineral. Its output has increased over a 1000-fold in 60 years, compared with a mere 50 fold for oil – an industry often regarded as the symbol of industrial growth.”⁹⁰ By 1964 observers noted the wide range of uses for asbestos and the “multiplicity of circumstances under which asbestosis can be encountered.” In a report of the Section on Nature and Prevalence Committee on Occupational Diseases of the Chest of the American College of Chest Physicians it was noted that there were – “3,000 specific conditions in which asbestos serves uniquely as a protectant against excessive heat, cold and corrosion.”⁹¹ Major uses of asbestos included textiles, cement products, friction materials and gaskets, paper, floor tile, paints, roof, caulks, plastics. But G.W.H. Schepers “stressed that the medical and health problem originating through exposure to asbestos dust may come about equally through minor uses,” as well.⁹² Another author noted the increasing dangers of asbestos produced for use in wide variety of products, including blankets, clothing, threats, rope, tape, brake linings, wall paper, wallboard, shingles, floor covering, plastics, insulation for pipes, boilers, wire, heating pads. Like

⁸⁹ Hueper, W.C., “Occupational and Nonoccupational Exposures to Asbestos,” Annals of the New York Academy of Sciences, 132 (1965), 184 – 195 at p.186 and Wagner, J.C., J.C. Gilson, G. Berry, V. Timbrell, “Epidemiology of Asbestos Cancers,” British Medical Bulletin, 27(1971), 71-76.

⁹⁰ Gilson, J.C., “Man and Asbestos,” Annals of the New York Academy of Sciences, 132 (1965), 9 – 11.

⁹¹ “Asbestosis,” Report of the Section on Nature and Prevalence Committee on Occupational Diseases of the chest, American College of Chest Physicians,” Diseases of the Chest, 45 (January 1964), 107 – 111 at p. 107.

⁹² Hendry, N.W., “The Geology, Occurrences, and Major Uses of Asbestos,” Annals of the New York Academy of Sciences, 132 (1965), 12 – 22 and Schepers at p.21. See also, “Complications of Asbestosis,” British Medical Journal, 1 (April 30, 1960), 1345 - 1353. P. 1350 Comment by Dr. Hugh-Jones: “Asbestosis is a pneumoconiosis arising from the inhalation of asbestos dust during the manufacture of asbestos goods, such as asbestos sheeting, fireproof clothing, brake linings, lagging for boilers and pipes, etc.”

Schepers and others before him, H.B. Eisenstadt remarked that “due to the rapid expansion of its use, an ever-increasing number of craftsmen and laborers are exposed to the mineral.” He even cited a case of an auto mechanic who acquired asbestosis “while undercoating vehicles.”⁹³

The first U.S. case reports of cancers other than those of the lung was recorded in 1947 when Tracy Mallory, Benjamin Castleman and Edith Parris reported in the New England Journal of Medicine “case 33111” from the Massachusetts General Hospital. “Case 33111” was a 37 year old worker from Sweden whose work was cutting asbestos insulating board. He was diagnosed with mesothelioma of pleura and pericardium. The physicians noted the unusual character of this case for “a number of papers have been written to the effect that there is no such tumor as mesothelioma of the pleura, that the cells lining the pleura do not form tumors and that these tumors really arise from a small focus in the lung.” They had “held a similar opinion for a long time.” But the case forced them to reevaluate their belief. “This is perhaps the first case in which we believed that there was actually such a tumor.”⁹⁴

J.C. Wagner, C. A. Sleggs, and Paul Marchand documented the relationship between asbestos exposure and mesothelioma in a 1960 article published in the British Journal of Industrial Hygiene where they detailed “33 histologically proven cases” of mesothelioma, which they described as being “regarded as an uncommon tumour” during the previous four years. They

⁹³ Eisenstadt, H.B., “Plural Asbestosis,” American Practitioner, 13 (September 1962), 573 -578.

⁹⁴ Mallory, Tracy B., Benjamin Castleman, Edith E. Parris, “Case Records of the Massachusetts General Hospital: Case 33111,” New England Journal of Medicine, 236 (March 13, 1947), 407 - 412. See also Wyers, H., “Asbestosis,” Postgraduate Medical Journal, December 1949, 631 – 638; Owen, T.K., “Carcinoma and Asbestosis of the Lung: Report of a Case,” British Journal of Cancer, 5 (1951), 382-3; Hochberg, Lew A., “Endothelioma (Mesothelioma) of the Pleura,” American Review of Tuberculosis, 63 (1951), 150 – 175 for early reports of mesothelioma related to asbestos exposure.

reviewed the existing literature⁹⁵ and concluded, "Our findings suggest that mesothelioma occurs 20 to 40 years or more after exposure to dust."⁹⁶ As early as 1962 researchers noted that "mesothelioma is now accepted by the majority of pathologists as an entity and in recent years diffuse or malignant mesothelioma has been shown to be associated with pulmonary asbestosis in Canada, Holland, South Africa, and Germany." It was noted that not only workers were at risk but those "who lived in areas where asbestos was mined or treated" were also at risk. He also warned that there were "increasing number of people manufacturing, handling, or using these products." He worried that the "widespread... use of asbestos today" has resulted in "mesothelioma of the pleura and peritoneum in people who have occupations in which association with asbestos is not suspected."⁹⁷ Evidence continued to mount regarding the risk to bystanders and consumers of products using asbestos fiber. In their summary of their findings, Dermot Hourihane argued, "the association of these tumours [mesotheliomas] with asbestos bodies in lung tissue is confirmed." He pointed out that "many of the cases gave no history of industrial exposure, and it is possible

⁹⁵ Wagner, J.C., C. A. Sleggs, and Paul Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," British Journal of Industrial Medicine, 17 (1960), p. 267: "In 1924 Robertson denied the existence of primary malignant tumours of the pleura and considered them to be secondary in origin. Since then, on the one hand, Willis (1948, 1953) and Smart and Hinson (1957) have supported Robertson's views, while on the other hand, primary neoplasms of this nature have been described by many authors in recent years. These include Tobiassen (1955) in Sweden, Belloni and Bovo (1957) in Italy, Godwin (1957) in the United States, and McCaughey (1958) in Britain. Evidence of the mesothelial origin of these tumours can be found in the tissue culture experiments of Stout and Murray (1942) and Sano, Weiss, and Gault (1950). They also noted Doll's study which included one case of mesothelioma.

⁹⁶ Wagner, J.C., C. A. Sleggs, and Paul Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," British Journal of Industrial Medicine, 17 (1960), 260 - 271. A year later they reported on eight more cases in Sleggs, C.A. and J. C. Wagner, "Diffuse Pleural Mesotheliomas in South Africa," South African Medical Journal, 35 (January 14, 1961), 28 -34. See also, "Complications of Asbestosis," British Medical Journal, 1 (April 30, 1960), 1345 - 1353 and Eisenstadt, H.H. and F.W. Wilson, "Primary Malignant mesothelioma of the Pleura," The Lancet, (November 1960), 511 - 514 and Hueper, W.C. "Environmental and Occupational Cancer Hazards," Symposium on Chemical Carcinogenesis, Clinical Pharmacology and Therapeutics, 3 (1962), 776 - 813.

⁹⁷ Thomson, J.G., "Mesothelioma of Pleura or Peritoneum and Limited Basal Asbestosis," South African Medical

that temporary or relatively trivial exposure may have occurred.” The severity of the disease was grave as “once symptoms from the tumours have arisen, the prognosis appears to be measured in months rather than years.”⁹⁸ In the United States, Irving Selikoff built on the British and South African literature with an extensive epidemiological study of insulation workers in the building trades who had “relatively light, intermittent, exposure to asbestos.” He found that of 632 workers who “entered the trade before 1943 and were traced through 1962, forty-five died of cancer of the lung or pleura, whereas only 6.6 such deaths were expected. Three of the pleural tumors were mesotheliomas.” Remarking on the mesothelioma findings, Selikoff wrote “the incidence of more than 1% of deaths from pleural mesothelioma is strikingly high for a tumor which is generally considered to be extremely rare.” This augured poorly for other building trades occupations: “insulation workers undoubtedly share exposure with their workmates in other trades; intimate contact with asbestos is possible for electricians, plumbers, sheet-metal workers, steamfitters, laborers, carpenters, boiler makers, and foremen.”⁹⁹ Selikoff also expressed concern that mesothelioma specifically was more than an occupational hazard and was one which “will be found to be a community problem” as well.¹⁰⁰ Others worried about the growing dangers of asbestos exposure. A British report noted that “(1) carcinoma of the lung, and (2) diffuse

Journal, September 8, 1962), 759 - 760.

⁹⁸ Hourihane, Dermot O’B., “The Pathology of Mesothelioma and an Analysis of Their Association with Asbestos Exposure,” Thorax, 19 (1964), 268 - 278.

⁹⁹ Selikoff, Irving J., Jacob Churg and E. Cuyler Hammond, “Asbestos Exposure and Neoplasia,” JAMA, 188 (April 6, 1964), 22 - 26. See also, Selikoff, I.J., J. Churg, and E.C. Hammond, “The Occurrence of Asbestosis Among Insulation Workers in the United States,” Annals of the New York Academy of Science, 132 (1965), 139 - 155.

mesothelioma of the pleura and peritoneum,” were clearly recognized and that “there is some suggestion of an association also with gastro-intestinal carcinoma, and possibly ovarian tumours.” Because of the long latency of the disease, “further cases of these associated tumours are expected to occur for many years to come, even if dust exposures are now greatly reduced.”¹⁰¹

Wilhelm Hueper summarized the state of knowledge and the depressing implications of the information available in a paper presented at the New York Academy of Sciences in 1964:

“Numerous reports by many investigators from various countries establish the fact that an occupational or nonoccupational inhalation of dust of different chemical and physical types of asbestos results in the development of asbestosis of the lungs which, depending upon the type of exposure may be designated as an occupational, neighborhood or household pneumoconiosis. Since 1935 an increasing amount of epidemiologic, clinical and pathologic evidence, moreover, incriminates this health hazard as one of the environmental sources of cancers of the lung, and more recently also of mesotheliomas of the pleura and peritoneum, although some commercially interested parties and their medical guardians and protectors still prefer for their own reasons and motives, to deny

¹⁰⁰ Selikoff, Irving J, Jacob Churg, and E. Cuyler Hammond, “Relation Between Exposure to Asbestos and Mesothelioma,” New England Journal of Medicine, 272 (March 18, 1965), 560 - 565.

¹⁰¹ “Report and Recommendations of the Working Group on Asbestos and Cancer,” British Journal of Industrial Medicine, 22 (1965), 165 -171. See also, Selikoff, Irving J, Jacob Churg, and E. Cuyler Hammond, “Relation Between Exposure to Asbestos and Mesothelioma,” New England Journal of Medicine, 272 (March 18, 1965), 560 – 565: “It appears that mesothelioma must be added to the neoplastic risks of asbestos inhalation , and joins lung cancer (53 of 307 deaths) and probably cancer of the stomach and colon (34 of 307 deaths) as a significant complication of such industrial exposure in the United States.” See also, Hardy, Harriet, “Asbestos Related Disease,” American Journal of Medical Sciences, 250 (October 1965), 381 - 389.

the existence of these dangerous and usually fatal sequelae of a respiratory contact with asbestos dust. The obvious fallacy of such allegations is well demonstrated by the remarkable and a progressive rise in the number of cases of asbestosis and cancers of the lung, pleura, and peritoneum reported from various countries during the past two decades.”¹⁰²

For the next four decade researchers would build on these basic observations. By the 1970s the federal government had incorporated the expanding literature on asbestos, asbestosis and cancer into the regulations adopted by the newly-formed federal Occupational Safety and Health Administration. In 1972, shortly after its formation, the National Institute of Occupational Safety and Health issued a “Criteria Document for a Recommended Standard ... Occupational Exposure to Asbestos” that warned that “The recommended standard [was] designed primarily to prevent asbestosis,” and that “for other diseases associated with asbestos, there is insufficient information to establish a standard to prevent such diseases including asbestos-induced neoplasms by any all-inclusive limit other than one of zero.”¹⁰³ That same year, OSHA adopted the NIOSH recommendation for a “standard of 8 hour TWA airborne concentrations of asbestos dust not exceeding five fibers longer than five micrometers per millimeter.” OSHA “concluded that there should be one minimum standard to asbestos applicable to all workplaces exposed to any kind, or

¹⁰² Hueper, W.C., “Occupational and Nonoccupational Exposures to Asbestos,” Annals of the New York Academy of Sciences, 132 (1965), 184 - 195.

¹⁰³ “Criteria for a Recommended Standard ... Occupational Exposure to Asbestos,” U.S. Department of Health, Education and Welfare, PHS, CDC, National Institute for Occupational Safety and Health, 1972.

mixture of kinds, of asbestos.”¹⁰⁴ NIOSH revised its recommendation in 1976, summarizing the consensus that had evolved through the scientific, epidemiological and medical studies of the dangers from asbestos to workers, by-standers and the population at large:

“Available studies provide conclusive evidence that exposure to asbestos fibers causes cancer and asbestosis in man. Lung cancers and asbestosis have occurred following exposure to chrysotile, crocidolite, amosite, and anthophyllite.

Mesotheliomas, lung and gastrointestinal cancers have been shown to be excessive in occupationally exposed persons, while mesotheliomas have developed also in individuals living in the neighborhood of asbestos factories and near crocidolite deposits, and in persons living with asbestos workers....¹⁰⁵

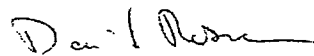
Animal studies confirmed the observation that “all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas after intrapleural or IP injection.”¹⁰⁶ The available data indicated that asbestos had no safe level where workers or others would be protected from cancers. “There are data that show that the lower the exposure, the lower the risk of developing cancer,” NIOSH suggested.

¹⁰⁴ Federal Register, title 29 – Labor, Chapter XVII – Occupational Safety and Health Administration, Department of Labor, Part 1910 – Occupational Safety and Health Standards, “Standard for Exposure to Asbestos Dust,” Federal Register, 37 (June 7, 1972), 11318 – 11322.

¹⁰⁵ “Revised Recommended Asbestos Standard,” U.S. Department of Health, Education and Welfare, PHS, CDC, National Institute for Occupational Safety and Health, Dec.1976, p.92.

¹⁰⁶ “Revised Recommended Asbestos Standard,” U.S. Department of Health, Education and Welfare, PHS, CDC, National Institute for Occupational Safety and Health, Dec.1976, p.92.

But “excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a ‘safe’ level of asbestos exposure.” The standard that was recommended, 100,000 fibers $>5\mu\text{m}$ in length/ m^3 , was intended to “protect against the noncarcinogenic effects of asbestos,” and also to “materially reduce the risk of asbestos-induced cancer.” But, NIOSH explained that “only a ban can assure protection against effects of asbestos....”¹⁰⁷



David Rosner, PhD
July 3, 2014

¹⁰⁷ “Revised Recommended Asbestos Standard,” U.S. Department of Health, Education and Welfare, PHS, CDC, National Institute for Occupational Safety and Health, Dec. 1976, p.93.

AFFIDAVIT

STATE OF Florida
COUNTY Howard

§
§

AFFIDAVIT OF RALPH DONSON –
SWORN INFORMATION FORM PURSUANT TO FL STAT. 774.205(3)(a-d)

Upon first being duly sworn on oath, I depose and state as follows:

The facts stated below are true and correct to the best of my knowledge.

- (a) My name is Ralph Donson; my current address is 7301 Fourwind Ln., Spring Hill, FL, 34606; Date of birth is 10/16/1952; Marital status is "married".
- (b) No secondary exposure claim is herein alleged.
- (c) The specific location of my exposure was in the Navy at A-School in Great Lakes, IL, aboard the USS Wasp, USS Davis, and at Charleston, SC navy shipyard. I was also exposed at Factory Mutual plant in Florham Park, NJ.
- (d) I was exposed to all Defendants' asbestos-containing products at the sites listed in (c) from about 1970-76 and 1980-82. I allege that I was continually exposed to these Defendants' asbestos-containing products throughout this period.
- (e) I was exposed to all Defendants' asbestos-containing products while working at all the sites as a Boiler Tender.
- (f) I have asbestosis.
- (g) Please see the attached Modh Report to Court.

FURTHER AFFIANT SAYETH NAUGHT

Ralph Donson
RALPH DONSON

HAVING PRODUCED A DRIVER'S LICENSE AS IDENTIFICATION
SUBSCRIBED AND SWORN TO BEFORE ME on this 27 day of September, 2016.

Amy Lauren Zoons

Notary Public, In and For the State of FL

My commission expires: 7/5/2020

Amy Lauren Zoons
Printed Name of Notary



AFFIDAVIT

STATE OF Florida §
COUNTY Hillsborough §

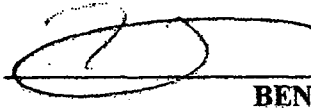
AFFIDAVIT OF COUNSEL IN RE: COLLATERAL SOURCE
PURSUANT TO FLA. STAT. 774.207

Upon first being duly sworn on oath, I depose and state as follows:

1. My name is Ben Vinson, Esq., and I have personal knowledge of the facts stated in this Affidavit and the facts stated are true and correct to the best of my knowledge.
2. The collateral source payments we expect to receive are:

KACC	\$1,698
Quigley	\$180
G-I	\$614
AC&S	\$434
CE	\$1,392
Yarway	\$500
Congoleum	\$450
EPI	\$4,000
Bondex	\$550
Johns-Manville	\$1,438
CEL	2,240
HW	\$854
HAL	\$854
FLX	\$203.00
FB	\$1,248
OC	2220
B&W	\$1,190
AWI	3,535
T&N	\$810.00
WRG	2210
USG	\$2,341
A.P. Green	\$374

FURTHER AFFIANT SAYETH NAUGHT


BEN VINSON

TO ME PERSONALLY KNOWN

SUBSCRIBED AND SWORN TO BEFORE ME on this 15 day of Sept., 2016.



Amy Lauren Zoons
Notary Public, In and For the State of FL

My commission expires: 7/5/2020

Amy Lauren Zoons
Printed Name of Notary

IN THE CIRCUIT COURT OF THE THIRTEENTH JUDICIAL CIRCUIT
IN AND FOR HILLSBOROUGH COUNTY, FLORIDA
CIVIL DIVISION

RALPH DONSON,

Plaintiff,

v.

Case No: New Case

AIR AND LIQUID SYSTEMS, INC.

Division: Z

As Successor-in-Interest to Buffalo Pumps, Inc.,

Et al.

Defendants.

REPORT OF DR. ASHOK MODH

I am a medical doctor licensed to practice medicine in the state of Florida. I am board certified in the specialties of Pulmonary Medicine, Internal Medicine, and Critical Care Medicine. I am also a Fellow of the College of Chest Physicians.

I examined Mr. Ralph Donson on 7/8/16. Attached hereto as "Exhibit A" and made a part hereof is a true and accurate copy of the report I created concerning my examination of Mr. Donson on 7/8/16. As part of my examination of Mr. Donson, I obtained an occupational history from him wherein he explained to me how he was exposed to asbestos during his employment. I also obtained a history of his military service. I asked him about his military service, if any, to determine whether there might have been asbestos exposure then. I also discussed with him his potential exposure to other pulmonary irritants and toxins including the nature, duration, and level of the exposures including asbestos.

Also as part of my examination, I obtained a detailed medical and smoking history including his past and present significant medical conditions and their most likely causes. This included obtaining an explanation of his respiratory symptoms from him wherein he explained the respiratory symptoms he was experiencing. I also obtained an explanation of his past pulmonary history because some diseases can mimic asbestosis. Further, I questioned him about what medications he was taking at the time of the examination. I asked this because this information sometimes gives insight as to why a patient may not be experiencing pulmonary symptoms. Finally, I obtained a smoking history from him wherein he explained his past use of cigarettes and other tobacco products to me. Even though cigarette smoking can cause chest x-ray changes similar to those caused by asbestos, it is extremely rare. Further, the fibrogenic potency of asbestos is much greater than cigarette smoke.

I performed a pulmonary examination on Mr. Donson on 7/8/16 and the results of the pulmonary examination are listed in my report attached as Exhibit A. I listened to Mr. Donson's breath sounds, his heart, observed his general appearance, and examined Mr. Donson's hands for clubbing of the fingers. The absence of abnormal breath sounds does not rule out a diagnosis of asbestosis. It means only that the disease has not advanced to the point of causing abnormal breath sounds. I also reviewed a 6/23/16 B-Reader interpretation of a 3/30/15 chest x-ray taken of Mr. Donson ("Exhibit B"). It shows that Dr. Vuskovich, who is a NIOSH-certified B-Reader and board-certified in Occupational Medicine, interpreted the film as a Quality 1 film (of good diagnostic quality) that depicts the presence of small opacities (type s in predominance) consistent with pneumoconiosis with a profusion of 1/1.

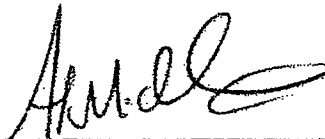
Based on the information I obtained from Mr. Donson on 7/8/16, my examination of Mr. Donson on 7/8/16, and Dr. Vuskovich's B reader interpretation of his chest x-ray, I diagnosed Mr. Donson with bilateral pulmonary asbestosis. This is a condition for which exposure to asbestos is a substantial contributing factor. In making this diagnosis, I followed the guidelines set forth in *The Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos*, Am. J. Respir. Crit. Care Med. 2004; 170(6):691. This article is an article published by the American Thoracic Society and is generally accepted in pulmonary and occupational medicine for establishing the criteria for the diagnosis of non-malignant asbestos disease.

The process of considering all of the information in the various histories given by Mr. Donson, consideration of the latency period of at least ten years between Mr. Donson's reported asbestos exposures and the date of my examination, consideration of information obtained from the examination of Mr. Donson, consideration of the interpretation of Mr. Donson's chest x-ray, and application of principles of occupational medicine, including the principles set forth in *The Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos*, Am. J. Respir. Crit. Care Med. 2004; 170(6):691 was the method I used to reach the diagnosis of asbestosis. In applying this methodology, I considered other causes for the abnormalities seen on Mr. Donson's chest x-ray. Mr. Donson's occupational exposures and the latency period corroborated the changes I saw on his chest x-ray. My diagnosis of asbestosis was based on a reasonable degree of medical certainty.

It is not a necessary criterion for the diagnosis of asbestosis to have quantification of the asbestos to which a worker was exposed. See, *The Diagnosis and Initial*

Management of Nonmalignant Diseases Related to Asbestos, Am. J. Respir. Crit. Care Med. 2004; 170(6):691. As a practical matter, a physician rarely has quantitative data on a patient's asbestos exposure. Further, it is not the usual course of an examining physician to take steps to personally investigate the veracity the history given to the examiner by the patient. Such an investigation would be impracticable. Further, because the exposures occurred many years ago (asbestosis is a latent disease) the workplace conditions have changed.

As to the cause of Mr. Donson's dyspnea, I reviewed Mr. Donson's 6/1/16 pulmonary function test ("Exhibit C"), which showed FVC at 63% of predicted, and FEV1/FVC at 73%, which is greater than the predicted normal lower limit. His TLC also was below the predicted lower limit of normal at 75%. He had no response to bronchodilators. Thus Asbestosis, rather than COPD, is a substantial contributing factor to Mr. Donson's pulmonary impairment. Further, based on my review of his medical records and pulmonary history Mr. Donson's medical findings and impairment are not more probably the result of some other cause than his asbestos exposure. Finally, Mr. Donson has a permanent respiratory impairment rating of at least Class 2 (Moderate) as defined by and evaluated pursuant to the AMA Guides to the Evaluation of Permanent Impairment.



Ashok Modh, M.D., F.C.C.P.

Tampa Bay Pulmonary Associates, P.A.

Ashok K. Modh, M.D., F.C.C.P.

Naishadh K. Mandaliya, M.D., F.C.C.P.

Jerges J. Cardona, M.D.

Nirav B. Patel, M.D.

PULMONARY EVALUATION

PATIENT: Ralph Tom Donson

MRN: 92489

DATE: 07/08/16

PAGE 1 of 3

REFERENCE: Mr. Ben Vinson

Dear Mr. Vinson:

I had an opportunity to do a physical examination on Mr. Ralph Tom Donson today.

HISTORY OF PRESENT ILLNESS: This is a very nice 63-year-old gentleman, who was referred to me by his attorney. Today, I concluded physical examination and personal interview with Mr. Donson with proper standards of care determining the presence of asbestos related lung disease/injury according to ATS Standards. During the examination, I personally discussed with him the history of asbestos exposure as well as potential irritants and toxins. I have also discussed the use of tobacco products, pulmonary symptoms and general health history. Following are the results of my interview with Mr. Donson's symptoms.

This is a very nice 63-year-old gentleman, who gets short of breath very easily. According to him, walking from the car with a briefcase in his hand by the time he gets into the house he gets short of breath. He cannot finish a sentence at times. He does have a cough, which is mostly dry cough. He does not have any fever or chills. He does have significant amount of shortness of breath and chest discomfort. His exercise capacity has gone down significantly over last few years. Also, the patient loses his voice and has hoarseness of voice intermittently.

In the morning, his voice starts normal, but by the afternoon he loses his voice and has significant hoarseness of voice.

2810 West Waters Avenue
Tampa, FL 33614
Phone (813) 935-5501
Fax (813) 933-8784
E-mail: tbpa.lungs@verizon.net

PATIENT: Ralph Tom Donson

PAGE 2 of 3

PAST MEDICAL HISTORY: History of asthma, COPD, asbestos related lung disease, hypertension, insomnia, and low energy level.

MEDICATIONS: At this time, the patient is on Advair, alprazolam, atenolol, bupropion, lisinopril, lovastatin, ranitidine, Spiriva, and zolpidem.

OCCUPATIONAL EXPOSURE HISTORY: The patient worked in engine room for US Navy from 1970 to 1992. He was out for one year from 1972 to 1973 and went back working for Navy. The patient used to operate the boiler and repair the boiler. He used to cut out the asbestos pipes and replace them and cover them with asbestos.

According to him, the pipes were very thin walled and they had to take the asbestos off to test them and then put the asbestos back on. According to him, in mid 90s they recognized the problems and some of the asbestos was covered with pink color and they were told that the pink stuff they do not have to worry about, white stuff they have to worry about, but he continued to work with them until 1992.

SMOKING HISTORY: The patient has a history of smoking approximately 1½ pack a day for about 15-20 years. He has not smoked for last 25 years or so.

MILITARY HISTORY: As mentioned earlier, the patient worked in military from 1970 to 1992 taking one year off in 1973.

PHYSICAL EXAMINATION: The patient is alert, awake, and oriented. O2 saturation is 98%. Blood pressure 138/88 and heart rate 80. The patient is 6'2" and weighs 248 pounds. Does have slight hoarseness of voice. HEENT looks normal. Neck shows no jugular venous distension. No thyromegaly. No cervical lymphadenopathy. Heart, S1, S2 normal. Lungs show rales at both the lung bases present. There are some expiratory wheezes present also. Abdomen is soft and nontender. No distension. Extremities show no edema or clubbing. Neurologically, the patient is stable.

I have looked at the B-reader report on him from March 30, 2015, which showed that the patient does have some small densities in both the lungs which demonstrate bilateral pulmonary interstitial fibrosis. Also has pleural plaques in both the lungs suggestive of asbestos related lung disease.

ASSESSMENT:

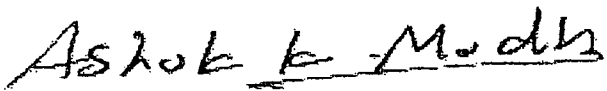
1. Considering the patient's asbestos exposure history, considering the x-ray findings and physical findings, Mr. Donson does have asbestosis. He has bilateral pulmonary interstitial fibrosis.
2. Tobacco abuse history. The patient has a history of tobacco abuse approximately 1½ pack a day for about 15-20 years suggestive of possibility of underlying chronic lung disease also.
3. The patient does have hoarseness of voice. Intrapulmonary pathology needs to be ruled out. I have discussed that with the patient and told him to consult his private physician and see if the CAT scan of chest will be indicated.

PATIENT: Ralph Tom Donson

PAGE 3 of 3

DISCUSSION: I have informed Mr. Donson of findings. I have informed him that asbestosis has no treatment and there is no cure for the disease. In addition, I have informed him that the disease process may progress with time without any additional exposure. He should avoid additional exposure to all pulmonary toxins and irritants. I have further informed him that he is at an increased risk of developing certain type of malignancies because of the asbestos exposure and asbestosis. He should consult a physician for annual physical examination. He should consult a physician for annual physical examination. He should seek immediate medical attention if the symptoms get worse.

Sincerely,

A handwritten signature in black ink, appearing to read "Ashok K. Modh". The signature is written in a cursive, somewhat stylized font.

Ashok Modh, M.D., F.C.C.P.

cc: Mr. Ben Vinson

AM/dp

N 0708-002.01109 2016 70001306.doc

1544192534

DATE OF RADIOGRAPH

MONTH	DAY	YEAR
03	30	2015

DOB: 10/16/52

WORKER'S Social Security Number:

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ROENTGENOGRAPHIC INTERPRETATION

X-ray No. : _____

TYPE OF READING

☐ A	☒ B	☐ P
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Note: Please record your interpretation of a single film by placing an "x" in the appropriate boxes on this form.

Donson, Ralph T.

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3D. DIFFUSE PLEURAL THICKENING (mark site, calcification, extent, and width) Chest wall: <table border="1"><tr><td>O</td><td>R</td><td>E</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr></table> In profile: <table border="1"><tr><td>O</td><td>R</td><td>E</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr></table> Face: <table border="1"><tr><td>O</td><td>R</td><td>E</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr></table>		O	R	E	O	X	X	O	X	X	O	X	X	O	R	E	O	X	X	O	X	X	O	X	X	O	R	E	O	X	X	O	X	X	O	X	X	Extent (chest wall, constant for in profile and face only): Up to 1/4 of lateral chest wall = 1 1/4 to 1/2 of lateral chest wall = 2 > 1/2 of lateral chest wall = 3 Width (in profile only): 3 to 5 mm = a 5 to 10 mm = b > 10 mm = c <table border="1"><tr><td>O</td><td>R</td><td>E</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr><tr><td>O</td><td>X</td><td>X</td></tr></table>		O	R	E	O	X	X	O	X	X	O	X	X																								
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4A. ANY OTHER ABNORMALITIES?		YES ☒ Complete Sections 4B, 4C, 4D, 4E NO ☐ Proceed to Section 5																																																																									
4B. OTHER SYMBOLS (OBLIGATORY) <table border="1"> <tr> <td>a</td><td>b</td><td>c</td><td>d</td><td>e</td><td>f</td><td>g</td><td>h</td><td>i</td><td>j</td><td>k</td><td>l</td><td>m</td><td>n</td><td>o</td><td>p</td><td>q</td><td>r</td><td>s</td><td>t</td><td>u</td><td>v</td><td>w</td><td>x</td><td>y</td><td>z</td> </tr> </table>		a	b	c	d	e	f	g	h	i	j	k	l	m	n	o	p	q	r	s	t	u	v	w	x	y	z	If other diseases or significant abnormalities, findings must be recorded on reverse (section 4C/D) Date Physician or Worker notified: MONTH DAY YEAR																																															
a	b	c	d	e	f	g	h	i	j	k	l	m	n	o	p	q	r	s	t	u	v	w	x	y	z																																																		
4E. Should worker see personal physician because of findings in section 4? YES ☐ NO ☒		Proceed to Section 5																																																																									

FILM READER'S INITIALS

DATE OF READING

MAV

06

23

2016

OccOptions, Inc.

P.O. Box 5069

Gainesville, FL 32627

Matthew A. Vuskovich, M.D., M.S.P.H.

4961192530

IC: MARK ALL BOXES THAT APPLY: (Use of this list is intended to reduce handwritten comments and is optional)

Abnormalities of the Diaphragm

- ☐ Eventration
- ☐ Hiatal hernia

Airway Disorders

- ☐ Bronchovascular markings, heavy or increased
- ☐ Hyperinflation

Bony Abnormalities

- ☐ Bony chest cage abnormality
- ☐ Fracture, healed (non-rib)
- ☐ Fracture, not healed (non-rib)
- ☐ Scoliosis
- ☐ Vertebral column abnormality

Lung Parenchymal Abnormalities

- ☐ Azygos lobe
- ☐ Density, lung
- ☐ Infiltrate
- ☐ Nodule, nodular lesion

Miscellaneous Abnormalities

- ☐ Foreign body
- ☐ Post-surgical changes/sternal wire
- ☐ Cyst

Vascular Disorders

- ☐ Aorta, anomaly of
- ☐ Vascular abnormality

4D. OTHER COMMENTS

Public reporting burden of this collection of information is estimated to average 3 minutes per response, including time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. An agency may not conduct or sponsor, and a person is not required to respond to a collection of information unless it displays a currently valid OMB control number. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to CDC, Project Clearance Officer, 1600 Clifton Road, MS E-11, Atlanta, GA 30333, ATTN: PRA (09020-0020). Do not send the completed form to this address.

Pulmonary & Sleep AssociatesJay P. Kumar, M.D., F.C.C.P.
10075 Cortez Boulevard, Brooksville, FL 34613

Phone: 353-597-6600

Fax: 352-597-6601

Name: Donson, Ralph

ID: 153464994

Gender: Male

Date: 06/01/16

Age: 63 Race: Caucasian

Temp: 28 PBar: 757

Height(in): 74 Weight(lb): 245

Ref MD: Pulsipher

Any Info: COPD/50 pack-yrs

Technician: J. Garofano, CPFT

Spirometry (BTPS)		PRED	PRE-RX		POST-RX		% Chg
			BEST	%PRED	BEST	%PRED	
FVC	Liters	5.21	3.28	63	2.97	57	-9
FEV1	Liters	4.15	2.40	58	2.40	58	-0
FEV1/FVC	%	78	73		81		
FEF25-75%	L/sec	4.08	1.69	41	2.27	56	34
FEF50%	L/sec	5.01	2.28	45	2.82	56	24
PEF	L/sec	9.47	6.94	73	7.39	78	6
MVV	L/min	150					

Lung Volumes (BTPS)

TLC	Liters	7.57	5.66	75
RV	Liters	2.70	2.38	88
RV/TLC	%	38	42	
FRC PL	Liters	3.89	2.82	72
VC	Liters	5.21	3.28	63

Diffusion

DLCO	mL/mmHg/min	28.5	19.4	68
DL Adj	mL/mmHg/min	28.5	19.4	68
DLCO/VA	mL/mHg/min/L	3.79	3.83	101
DLVA Adj	mL/mHg/min/L		3.83	
VA	Liters		5.06	

Resistance

	Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
Raw cmH2O/L/sec	1.13	0.52	46			
Gaw L/sec/cmH2O	0.933	1.919	206			
sRaw cmH2O/L/s/L	4.40	1.84	42			
sGaw L/s/cmH2O/L	0.228	0.544	239			

Interpretation:

Flow is compatible with mild restrictive ventilatory pattern with no bronchodilator response

Jay P. Kumar, M.D., F.C.C.P.

3L Syringe Calibration was performed prior to Spirometry Testing

Spirometry data is ACCEPTABLE and REPRODUCIBLE

2 puffs of Albuterol MDI given for Post-Tx study