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IN THE SUPREME COURT OF APPEALS OF WEST VIRGINIA

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SUPREME COURT OF APPEALS
OF WEST VIRIGINIA

ROBERT H. CASDORPH, JR.

Petitioner,

V.

WVOIC and
WEST VIRGINIA STATE POLICE,

Respondents.

Appeal No. 73940
Claim No.: 2003048198

PETITION FOR APPEAL
from an Order of the
WORKERS' COMPENSATION BOARD OF REVIEW
dated December 5, 2006

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IN THE SUPREME COURT OF APPEALS OF WEST VIRGINIA

ROBERT H. CASDORPH, JR.

Petitioner,

V.

Appeal No. 73940
Claim No.: 2003048198

WVOIC and
WEST VIRGINIA STATE POLICE,

Respondents.

PETITION FOR APPEAL

I.

STANDARD FOR REVIEW

This matter requires the interpretation of a ruling the by West Virginia Workers' Compensation Board of Review which presents a purely legal question subject to a de novo review. *Appalachian Power Co. v. State Tax Dept.,* 195 W.Va. 573, 466 S.E.2d 424 (1995).

II.

TYPE OF PROCEEDING/RULING OF BOARD OF REVIEW

Robert Casdorph filed an occupational disease claim after contracting chronic myelogenous leukemia (hereinafter "CML") from 21 years of significant exposure to benzene and 2-Butoxyethanol. The Decision of the Administrative Law Judge dated August 4, 2005 ruled Mr. Casdorph's claim compensable.[1] However, this Decision was subsequently reversed by the

[1]See Decision of Administrative Law Judge dated August 4, 2005 in Appendix at Exhibit 1.

West Virginia Workers' Compensation Board of Review by Order dated December 20, 2006.[2]

Distinguished experts in the areas of epidemiology, occupational medicine, toxicology, hematology and oncology have all opined that Mr. Casdorph's CML and resulting death was caused from his significant occupational exposure to benzene-containing products and products that contain 2-Butoxyethanol. Even the employers industrial hygiene expert rendered opinions which support this claim. Further, numerous peer-reviewed scientific studies, which form the basis of the current state of the scientific literature, show that exposure to benzene causes CML.

Based upon the weight of the evidence, the Claimant/Appellee asserts that the Decision of the Administrative Law Judge was clearly right and the ruling from the Board of Review was clearly wrong and should be reversed.

III.

STATEMENT OF FACTS

A. Exposure History

Robert H. Casdorph, Jr. was born on March 5, 1954 and was a lifetime non-smoker.[3] In approximately January, 1981, at the age of 26, he went to work for the West Virginia State Police ("WVSP"). Between 1981 and approximately 1982, he primarily worked as a janitor. However, in approxi-

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[2]See Order of West Virginia Workers' Compensation Board of Review dated December 5, 2006 in Appendix at Exhibit 2.

[3]See Deposition Transcript of Robert H. Casdorph, Jr., July 11, 2003, Vol. I at p. 73 in Appendix at Exhibit 3.

mately 1982, he began working as a mechanic and he continued in this capacity until his medical retirement in 2003 at the age of 49.[4]

In his capacity as a mechanic, Mr. Casdorph worked on and replaced brakes and brake rotors, transmissions, replaced air filters, greased bearings, replaced fuel filters, changed tires, replaced batteries, changed oil and oil filters, and many other mechanical type jobs for a fleet of 450 vehicles.[5] He used many petroleum based products to, among other things, degrease and lubricate parts and he had repeated daily dermal contact with and inhalation of gasoline.[6] Specifically, Mr. Casdorph had significant inhalation and dermal exposure while cleaning and reassembling vehicle parts without gloves.[7] Gasoline and degreasers were constantly dripping and spraying on his exposed skin and onto his clothing.

Inhalation and dermal exposures were inevitable given the nature of his job functions. Gasoline and solvents spilled onto the floor of the garage, combined with cleaning of parts, paint fumes and lack of ventilation created an environment for continuous exposure to benzene-containing compounds. Also, Mr. Casdorph frequently ingested gasoline as a result of the practice of having workers, such as Mr. Casdorph, siphon gas from/to vehicle fuel tanks

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[4] Id. at pp. 6, 51.

[5] Id. at pp. 7-9, 76-83.

[6] Id. at pp. 7, 11-19, 21-34, 39.

[7] See Section IV, C, 2, infra at p. 15.

by mouth when performing fuel pump repairs.[8] The removed gasoline was stored in an open trash can in close proximity to his work space.[9] Also, his primary work area was very small with no exhaust fan and was quite "smelly."[10]

As evidence of overexposure to benzene containing products, as set forth infra, Mr. Casdorph testified that he would get dizzy, lightheaded and "a little bit sick" approximately three of five work days when working around gasoline and other products.[11] Further, Mrs. Casdorph testified that Mr. Casdorph frequently mentioned to her that he got dizzy from the fumes at work.[12] She also testified that two or three nights a week, Mr. Casdorph delayed eating when he came home from work because of nausea and dizziness.[13] Moreover, she stated that Mr. Casdorph had a terrible odor that smelled like paint thinner on his clothing when he came home from work.[14]

Perhaps the best evidence of overexposure to benzene-containing products came from E. K. Harper, one of Mr. Casdorph's supervisors. Mr.

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[8]See Deposition Transcript of Robert H. Casdorph, Jr., July 11, 2003, Vol. I, at pp. 9, 91-95 in Appendix at Exhibit 3.

[9]Id. at pp 10-15, 102.

[10]See Deposition Transcript of E. K. Harper, August 28, 2003, at pp. 25-29 in Appendix at Exhibit 4.

[11]See Deposition Transcript of Robert H. Casdorph, Jr., July 11, 2003, Vol. I at pp. 15, 25, 46, 104-105 in Appendix at Exhibit 3.

[12]See Deposition Transcript of Melba Casdorph, June 7, 2004, at pp. 16, 19, 38 in Appendix at Exhibit 5.

[13]Id.

[14]Id.

Harper testified that Mr. Casdorph had the "hardest and dirtiest job there . . . . Bobby had a dirty, nasty job."[15]

B. Medical History

In January, 2002, Mr. Casdorph developed a severe cough. He was evaluated by his family physician, Brad Henry, M.D., who discovered that his white blood count was abnormally high.[16] Dr. Henry referred Mr. Casdorph to James Frame, M.D. who ordered a bone marrow core biopsy. The biopsy showed that Mr. Casdorph had CML in an accelerated phase. He was treated with the medication Gleevec.[17]

Between January, 2002 and June, 2002, Mr. Casdorph was seen numerous times by Dr. Frame. He was evaluated clinically and underwent laboratory studies. On June 12, 2002, at the direction of Dr. Frame, Mr. Casdorph had a bone marrow test. The results showed that Mr. Casdorph was in Gleevec induced remission of CML.[18]

In July, 2002, Mr. Casdorph was evaluated by Dr. Richard K. Shadduck, Director of Western Pennsylvania Cancer Institute.[19] Between June, 2002 and March, 2003, Mr. Casdorph was evaluated numerous times by Dr. Frame and

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[15]See Deposition Transcript of E. K. Harper, August 28, 2003, at pp. 15-16 in Appendix at Exhibit 4.

[16]See Deposition Transcript of Robert H. Casdorph, Jr., July 11, 2003, Vol. I at pp. 42-43 in Appendix at Exhibit 3.

[17]Id.

[18]Id. at pp. 42-43, 47 in Appendix at Exhibit 3.

[19]See Deposition Transcript of Richard Shadduck, M.D., May 4, 2004, at pp. 6-10 in Appendix at Exhibit 6.

Dr. Shadduck. In addition to clinic evaluations, Mr. Casdorph underwent numerous laboratory tests and several additional bone marrow procedures.[20]

In March, 2003, Mr. Casdorph's bone marrow studies showed that his CML had returned. Mr. Casdorph was immediately admitted to Western Pennsylvania Hospital for induction of chemotherapy.[21]

Between March and July, 2003, Mr. Casdorph continued to be evaluated and treated by Dr. Shadduck. He underwent many lab tests, diagnostic studies, pulmonary function tests, and bone marrow biopsies.[22]

In July, 2003, he was admitted to Western Pennsylvania Hospital for a bone marrow transplant by an unrelated donor.[23]

After the bone marrow transplant, Mr. Casdorph continued to be evaluated by Dr. Shadduck between July, 2002 and March, 2004. During this period he underwent numerous tests including several more bone marrow biopsies.[24]

Mr. Casdorph was admitted to Western Pennsylvania Hospital in March, 2004, for nausea and vomiting. Mr. Casdorph never recovered and ultimately died on April 9, 2004 at the age of 50. He died as a result of CML.[25]

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[20] Id.

[21] Id.

[22] Id.

[23] Id.

[24] Id.

[25] Id.

IV.

RULINGS OF BOARD OF REVIEW AND ADMINISTRATIVE LAW JUDGE

Mr. Casdorph appeals the Order of the Board of Review dated December 20, 2006 reversing the ruling of the Administrative Law Judge with regard to compensability.

V.

ASSIGNMENT OF ERROR

The Board of Review erred by reversing the Decision of the Administrative Law Judge with regard to compensability.

VI.

DISCUSSION OF LAW AND ARGUMENT

THE EVIDENCE PRESENTED OVERWHELMINGLY SHOWS THAT THE BOARD OF REVIEW RULING WAS "CLEARLY WRONG" AND SHOULD BE REVERSED

A. Petroleum Based Products Contain Benzene

All petroleum-based products contain benzene, including gasoline and organic solvents. It is well known in the scientific literature that gasoline contains benzene. Further, gasoline manufacturers have included a reference to benzene on their Material Safety Data Sheets for years.

The American Conference of Governmental Industrial Hygienist ("ACGIH-") Documentation of the Threshold Limit Values for "Gasoline" summarizes the literature and the research on the benzene content of gasoline as follows:

> Because benzene is the most significant, potent health hazard found in gasoline, benzene deserves special consideration in establishing a TLV. Elkins, H.B.; Comproni, E.M.; Pagnotto, L.D.: Industrial Benzene Exposure from Petroleum Naphtha. II. Pertinent Physical Properties of Hydrocarbon Mixtures. *Am. Ind. Hyg. Assoc. J.* 24:99-102 (1963). Phillips and Jones reported that the average concentration of benzene in American gasoline was 1% or less and that 2% was seldom exceeded. Phillips, C.F.; Jones, R.K.: Gasoline Vapor Exposure During Bulk Handling Operations. *Am. Ind. Hyg. Assoc. J.* 39:118-128 (1978). One large refiner had a self-imposed limit of 4%. However, analysis of 86 samples of gasoline from American filling stations revealed that 5% contained more than 2.5% benzene, with the highest benzene content being 4.8%. McDermott, J.J.; Killiany, Jr., S.E.: Quest for a Gasoline TLV. *Am. Ind. Hyg. Assoc. J.* 39:110-117 (1978).

ACGIH, TLV Documentation, "Gasoline" at 1 (7th ed. 2001).

Based on Motor Vehicle Manufacturers Association Survey results, Infante reported in 1993 that the benzene volume percentage in gasoline in the United States has averaged between 1.5 and 2.0% over the past decade.[26] Additionally, the Material Safety Data Sheets ("MSDS") and other documents generated by gasoline manufacturers have clearly listed the percentage of benzene in their products.[27]

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[26] *See* Infante, Peter F., State of the Science on the Carcinogenicity of Gasoline with Particular Reference to Cohort Mortality Study Results. *Environ. Health Perspect.* 101 (Suppl. 6)105-109 (1993) in Appendix at Exhibit 7.

[27] Shell Oil Company's (Shell) February 22, 1982 MSDS for unleaded gasoline indicated that the maximum benzene content was 4%. Mobil Oil's February 3, 1982 MSDS for gasoline indicated that it may contain up to 5% benzene. Shell's MSDS dated September 18, 1986 and February 18, 1988 reported a benzene concentration in Shell unleaded gasoline of less than 5%. An internal ARCO Petroleum Products Company memorandum, dated May 25, 1984, placed the range of the benzene content in gasoline between 1.2% and 2.7% depending on the refinery location. Gulf Oil Corporation's October 30, 1985 MSDS indicates that "Good Gulf" lead gasoline may contain 4.9% benzene. The June 5, 1986 MSDS for "Good Gulf" leaded gasoline indicated that 5% is a typical concentration of benzene

B. Benzene Causes Chronic Myelogenous Leukemia

With regard to gasoline, epidemiologic studies, analyses and case reports have shown an association between gasoline exposure and leukemia. The hazards of benzene in gasoline have been recognized since at least 1928.[28] Numerous studies support gasoline's ability to cause leukemia.[29] As

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in its gasoline. Chevron's MSDS for regular unleaded gasoline dated June 5, 1986, states that the "typical composition" of its product contained 5% benzene. Texaco, Inc.'s November 24, 1987 MSDS reports a range of between 1% and 3.99% benzene concentration in its gasoline. On July 1, 1992, CENEX reported in its MSDS on automotive gasoline (regular, unleaded and premium) that its benzene content was "<5" percent. Sinclair Oil reported in 1992 benzene content as "0.6-5.3" percent in its leaded gasoline and "0.9-4.8" percent in its unleaded gasoline. Unocal Corporation's MSDS for reformulated gasoline dated November 29, 1994, indicates that the benzene content of its gasoline could reach 1.3%. Phibro Energy USA, Inc., in 1995 reported on its MSDS that its unleaded gasoline contained "1-5%" benzene.

[28] *See* Aksey, J.M. Aplastic Anemia Due to Benzol Poisoning. *Calif West Med* 29:262-263 (1928) in Appendix at Exhibit 8 (case report of aplastic anemia in U.S. workers exposed to gasoline); *See* Tondel M., Persson B. and Carstensen J. Myelofibrosis and Benzene Exposure. *Occup Med* 45:51-52 (1995) in Appendix at Exhibit 9 (case report of myelofibrosis in a Swedish petrol station attendant); *See* Infante, P.F., Schwartz, E., and Chaill, R. Benzene in Petrol: A Continuing Hazard. *Lancet* ii:814-815 (1990) in Appendix at Exhibit 10 (case report of aplastic anemia in a U.S. roofer who used petrol to clean seams before fitting rubberized roofing material).

[29] *See* Australian Institute of Petroleum Health Surveillance Program, Health Watch, Tenth Report 1998, University of Melbourne, Department of General Practice and Public Health, Melbourne, Australia in Appendix at Exhibit 11; *See* Infante, Peter F., State of the Science on the Carcinogenicity of Gasoline with Particular Reference to Cohort Mortality Study Results. *Environ. Health Perspect.* 101(Suppl. 6):105-109 (1993) in Appendix at Exhibit 7; *See* Jakobson R., Ahlbom A., Bellander T. and Lundberg I., Acute Myeloid Leukemia Among Petrol Station Attendants. *Arch. Environ. Health* 48:255-259 (1993) in Appendix at Exhibit 12; *See* Lindquist, Ragnhild, *et al.*, Acute Leukemia in Professional Drivers Exposed to Gasoline and Diesel, *European J. Haematol.* 47:98-103 (1991) in Appendix at Exhibit 13; *See* Lumley M., Barker H. and Murray J.A., Benzene in Petrol. *Lancet* 336:1318-1319 (1990) in Appendix at Exhibit 14; *See* Naizi G.A. and Fleming A.F., Blood Dyscrasia in Unofficial Vendors of Petrol and Heavy Oil and Motor Mechanics in Nigeria, *Tropical Doctor* 19:55-58 (1989) in Appendix at Exhibit 15; *See* Ruston, L., A 39-Year Follow-up of the U.K. Refinery and Distribution Center Studies: Results for Kidney Cancer and Leukemia. *Environ. Health Perspect.* 101(Suppl. 6):77-84 (1993) in Appendix at Exhibit 16 (higher than expected leukemia rate for distribution workers); *See* Schnatter, A.R., *et al.*, A Retrospective Mortality Study

shown in the Summary Chart in Appendix at Exhibit 19 and as fully explained below, the current state of the scientific literature provides a statistically significant connection between inhalation and dermal exposures to benzene and the development of CML.

A population-based case-control study of 486 men and women was conducted in Shanghai China from 1987 to 1989 to evaluate the association of selected occupational exposures with the risk of leukemia. The authors reported that exposure to benzene was found to be associated with the elevated risk of CML. The odds ratio was 2.5 and statistically significant (CI=1.3–10.2). In essence those individuals with occupational exposure to benzene demonstrated **a two and one-half fold increase in the development of CML.**[30]

In reviewing the reanalysis of the Pliofilm cohort by Wong,[31] Savitz and Andrews calculated a statistically significant **relative risk** for non-AML

Among Canadian Petroleum Marketing and Distribution Workers. *Environ. Health Perspect.* 101(Suppl. 6):85-99 (1993) in Appendix at Exhibit 17(significantly elevated SMR for leukemia in truck drivers with highest exposure); and *See* Schwartz E., Proportionate Mortality Ratio Analysis of Automobile Mechanics and Gasoline Service Station Workers in New Hampshire, *Am. J. Ind. Med.* 12:91-99 (1987) in Appendix at Exhibit 18.

[30] *See* Adegoke, Olufemi, *et al.*, Occupational History and Exposure and the Risk of Adult Leukemia in Shanghai. *AEP.* 13(No. 7): 485-494 (2003) in Appendix at Exhibit 20.

[31] *See* Wong. O., Risk of Acute Myeloid Leukemia and Multiple Myeloma in Workers Exposed to Benzene, *Occup. Environ. Med.* 52:380-384 (1995) in Appendix at Exhibit 21.

leukemia (which would include CML) of 3.0 based on 8 cases with confidence interval of 1.3 to 5.8.[32]

The Chinese cohort study by Dr. Yin and others reported an imprecise 2.5 relative risk for CML from exposure to benzene (CI=0.7 – 16.9), not statistically significant.[33] Although imprecise, this two and one-half excess risk is consistent with other studies finding an association.

Dr. Peter Infante, formerly Director of Standards at the Occupational Safety and Health Administration and an expert in this claim, reanalyzed the specific cell-type of leukemia cases report by Yin and others for the years 1972 – 1981.[34] In his reanalysis, Infante broke down the 30 leukemia cases by cell-type among the benzene-exposed workers in the cohort and compared them with the distribution of leukemia cases identified from the general population of China. In doing so, the cell-type specific standard mortality ratio (SMR) for CML was 4.24 ($p < 0.01$). This four and one-quarter excess risk for CML among benzene-exposed workers was statistically significant.[35]

Also, a case-control study of 1,257 cases of leukemia and 339 cases of aplastic anemia in China for the years between 1986 and 1988 found a

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[32] *See* Savitz, David, Kurtis W. Andrews. Risk of Myelogenous Leukemia and Multiple Myeloma in Workers Exposed to Benzene. *Occup. Enviror. Med.* 53:357 (1996) in Appendix at Exhibit 22.

[33] *See* Yin S-N, *et al.*, A Cohort Study of Cancer Among Benzene-Exposed Workers in China: Overall Results. *Am. J. Ind. Med.* 29:227-235 (1996) in Appendix at Exhibit 23.

[34] *See* Yin, S-N, *et al.*, Occupational Exposure to Benzene in China. *Br. J. Ind. Med.* 44:192-195 (1987) in Appendix at Exhibit 24.

[35] *See* Infante, P.F., Benzene and Leukemia: Cell Types, Latency and Amount of Exposure Associated with Leukemia. *Adv. Occup. Med. Rehab.* 1:107-120 (1995) in Appendix at Exhibit 25.

statistically significant excess of CML for patients exposed to benzene. (OR=1.65).[36]

Martha Linet, along with other researchers from the National Cancer Institute, utilized the Swedish Cancer-Environment Registry (which links cancer incidence and employment information) in an attempt to provide additional information on the cause of leukemia. By reviewing 19 years of data (1961 to 1979), 5,351 leukemia cases were found with 19% CML. When the CML cases were cross-referenced with occupational information, a one and one-half fold excess of CML was revealed for motor mechanics (auto, truck, and other types of engines) who were exposed to gasoline and its additives, oils, grease and solvents. (SIR = 1.5, $p<.05$).[37]

In 1979, the Australian petroleum industry took the initiative to set up a health surveillance program to report on the long-term health of its employees. *Health Watch* consists of a cohort study, in which the long-term health of oil industry employees is followed over time, and a case-control study, which focuses on the relationship of certain blood and bone-marrow cancers to benzene exposure. From time to time a report is published by the universities handling the information and in addition, reports are published in the medical and scientific literature. In 1991, a paper was published reporting the

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[36] *See* Chung-Kuo, Hsueh Ko, Hsueh Yuan Hsueh, Countrywide Analysis of Risk Factors for Leukemia and Aplastic Anemia. Chinese Epidemiologic Study Group of Leukemia and Aplastic Anemia, *Pao Acta Academiae Medicinae Sinicae* 14:185-189 (1992) (Translated from the original Chinese version) in Appendix at Exhibit 26.

[37] *See* Linet, Martha S., Malker, Hans S.R., *et al.*, Leukemia and Occupation in Sweden: A Registry-Based Analysis. *Am. J. Ind. Med.* 14: 319-330 (1988) in Appendix at Exhibit 27.

incidence of cancer in the employees of the Australian petroleum industry from 1981 to 1989. The authors report a **four-fold excess of myeloid leukemia (which includes CML) over the expected case.** This 4.0 standard incidence ratio was statistically significant (CI= 1.6 – 8.2).[38]

In France, Goguel *et al,* in 1967 described 50 instances of leukemia in the Paris region from 1950 to 1965 (6 of which had already been reported). The 44 new cases included 13 chronic myelocytic leukemia, 8 chronic lymphocytic leukemia, and 23 acute leukemia. **Almost 30% of the benzene leukemias were of the CML cell-type.**[39]

Researchers from the National Cancer Institute and the Chinese Academy of Preventive Medicine have concluded:

> Chronic myeloid leukemia [CML] has been linked with benzene exposure in clinical reports, a small case-control study [from France], the cohort of Chinese workers followed-up through 1981 by the Chinese Academy of Preventive Medicine, and the expanded cohort of Chinese workers followed-up through 1987 by the [National Cancer Institute and the Chinese Academy of Preventive Medicine].

Hayes, Richard B., S-N Yin, Mustafa Doseci, and Martha Linet, Benzene and Lymphohematopoietic Malignancies in Humans. *Am. J. Ind. Med.* 40:117-126 (2001) (supporting citations omitted) in Appendix at Exhibit 30.

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[38] *See* Chrisite, D., *et al.,* A Prospective Study in the Australian Petroleum Industry. II. Incidence of Cancer. *Br. J. Ind. Med.* 48:511-514 (1991) in Appendix at Exhibit 28.

[39] *See* Goguel, A., Benzene Leukemia in the Paris Area Between 1950 and 1965. [Benzene Leukemia in the Paris Region From 1950 to 1965"], *Nouv. Rev. Fr. D'Hemat.* 7:465-480 (1967). (Translated from the original French version) in Appendix at Exhibit 29.

Finally, a December, 2004 study published by Dr. Martyn T. Smith and others, found that exposures to benzene at levels below the threshold deemed safe under American law, reduced counts of protective white blood cells and weakened some of the cells that build the blood supply.[40] This was the first study of a large group of workers who were breathing air with very low levels of benzene. After these results were made public, Dr. Nathaniel Rothman, one of the authors and a senior investigator at the National Cancer Institute, stated: "The fact that we see biological effects occurring in an important organ, the blood, raises questions and adds a bit more concern that we understand what's going on." See Revkin, Andrew, C., Study Shows the Chemical Benzene Reduces Cell Count in Workers, The New York Times (December 2, 2004), in Appendix at Exhibit 32. Also, Dr. Robert A. Rinsky, an epidemiologist at Cincinnati Children's Hospital who conducted significant federal studies in the 1970's and 1980's linking high benzene to leukemia, stated that the findings, while subtle, are a clear sign that even these low levels of exposure to benzene pose a risk.[41] Also, Dr. Martyn Smith, an author of the paper and a toxicologist at the University of California, Berkeley, stated there is no safe level of exposure to benzene that science will ever define as harmless.[42]

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[40]Lan, Q., *et al.*, Hematotxicity in Workers Exposed to Low Levels of Benzene, *Science,* Vol. 306 (December, 2004) in Appendix at Exhibit 31.

[41]See Revkin, Andrew, C., Study Shows the Chemical Benzene Reduces Cell Count in Workers, The New York Times (December 2, 2004) in Appendix at Exhibit 32.

[42]Id __.

C. The Evidence Overwhelming Proves That Mr. Casdorph's Exposure to Benzene Caused His Chronic Myelogenous Leukemia

1. Richard Shadduck, M.D.

Dr. Richard Shadduck is an expert in hematology and oncology, was Mr. Casdorph's bone marrow transplant treating physician and was not retained by any party. Dr. Shadduck is the Director of the Western Pennsylvania Cancer Institute in Pittsburgh, Pennsylvania and has treated patients with CML for 35 or 40 years.[43] His curriculum vitae contains 34 pages of published articles, books, book chapters, reviews, invited papers, letters, and abstracts in the areas of hematology and oncology.[44]

After reviewing Mr. Casdorph's medical records, the testimony of Mr. and Mrs. Casdorph, the testimony of Mr. Casdorph's co-workers and supervisors, as well as the testimony and opinions rendered by Stephen E. Petty, P.E., P.I.H., an industrial hygenist, Dr. Shadduck opined, inter alia, that Mr. Casdorph's exceptionally high level of exposure to benzene in the workplace caused his CML and resulting death.[45]

2. Stephen E. Petty, P.E., C.I.H.

Mr. Petty is president of Energy & Environmental Solutions, Inc. He is a Registered Professional Engineer, a Certified Industrial Hygienist, and he

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[43] *See* Deposition Transcript of Richard Shadduck, M.D., May 4, 2004, at pp. 31 in Appendix at Exhibit 6.

[44] *See* Curriculum Vitae of Richard Shadduck, M.D. in Appendix at Exhibit 33.

[45] *See* Deposition Transcript of Richard Shadduck, M.D. at. pp. 53, 64, 76, 87, in Appendix at Exhibit 6.

possesses a Bachelor of Science and a Masters of Science in Chemical Engineering as well as a Masters in Business Administration.[46]

Mr. Petty was retained to evaluate Mr. Casdorph's occupational exposure to aromatic hydrocarbons. Based on his review and knowledge of the industrial hygiene and safety literature, the documents produced in this matter and others by the Employer, his experience in the industrial hygiene and safety fields, and the testimony of the Claimant and his co-workers, he opined that Mr. Casdorph was routinely and on a continuing basis exposed to cancer causing substances including gasoline, solvents, degreasers, and aromatic hydrocarbon products during his employment.

Further, he opined, inter alia, as fully set forth in his report dated October 31, 2003, that:

- Mr. Casdorph suffered chronic exposures to hazardous chemicals, including carcinogens such as benzene.
- Protection of Mr. Casdorph from chemical exposures was inadequate given job functions. Inhalation and dermal exposure was inevitable given the nature of the job functions.
- Background levels from gasoline and solvents spilled onto the floor of the garage, combined with cleaning of parts, paint fumes and lack of ventilation created an environment for continuous exposure to benzene-containing compounds. For example, constant exposure to hazardous chemicals occurred as a result of the practice of having workers such as Mr. Casdorph siphon gas from/to vehicle fuel tanks by mouth and storing the fuel in an open trash can. The work area was reported to be quite "smelly".
- Dermal exposure, for example, from the cleaning and reassembly of vehicle parts, including manifolds, fuel filters and

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[46] *See* Curriculum Vitae of Stephen E. Petty, P.E., C.I.H. in Appendix at Exhibit 34.

fuel tanks were not done with gloves on. Further gasoline and degreaser was constantly dripping onto worker clothes and exposed skin parts. Gloves were not used by workers until after Mr. Casdorph was diagnosed with CML.

- Auto exhaust was exhausted into closed garages daily with no ventilation.

- Gasoline, paint and various solvent fumes were known to be present.

- Work practices were not protective of human health. This was recognized by the company since practices such as closed garages, little or no ventilation and siphoning of gasoline by mouth continued throughout most of Mr. Casdorph's 22-year career. For example, workers had to siphon gasoline from/into fuel tanks by siphoning by mouth. Mr. Casdorph's personnel review in 1992 stated that he was "properly using facilities, equipment, etc.," even though they changed the practice of siphoning. This practice continued until about 2000. The fact the process changed reflects knowledge that it was wrong.

- Based upon the depositions reviewed, workers reported that they smelled fumes but little or nothing was reportedly done to correct the situation. Workers reported odor levels of 7 or 8 on a 1-to-10 scale supporting this contention. Supervisors were reportedly aware of these complaints.

- In addi tion, an effecti ve respi ratory protec tion program appears to have been virtually non-existent during the time interval from 1992 to about 2003.

- Records of training in the Claimant's files suggest no training on hazards despite that recommendation in 1992 that "immediate updates on hazardous waste through in-service school" be completed.

- No respiratory monitoring or protection has apparently ever been completed, even after this date.

- Further, until Mr. Casdorph's illness, no systematic use of PPE of any kind appears to have been used. Now some mechanics were gloves.

See Report of Steven Petty, P.E., C.I.H. dated October 31, 2003 in Appendix at Exhibit 35.

3. Peter Infante, Dr. P.H.

Dr. Infante is a epidemiologist and former Director of Standards at the Occupational Safety and Health Administration ("OSHA").[47] Based upon his review and knowledge of the medical and scientific literature, his review of Mr. Casdorph's medical records, his extensive experience in epidemiology, and the review of the testimony of Mr. and Mrs. Casdorph and Mr. Casdorph's co-workers, Dr. Infante testified that Mr. Casdorph's development of CML was causally related to his work as a mechanic at the West Virginia State Police facility in South Charleston. He opined that his CML was caused from exposure to gasoline, solvents, degreasers, and aromatic hydrocarbon products.[48] Additionally, Dr. Infante testified that Mr. Casdorph had significant exposure to 2-Butoxyethanol that was found in sufficient concentrations (some as much as 10%) in several products used by Mr. Casdorph.[49] 2-Butoxyethanol has been associated with blood abnormalities of exposed workers.[50] Further, 2-Butoxyethanol enhances the toxicity of benzene in workers exposed to 2-Butoxyethanol.[51]

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[47] *See* Curriculum Vitae of Peter F. Infante, Dr. P.H. in Appendix at Exhibit 36.

[48] *See* Deposition Transcript of Peter Infante, Dr. P.H. at pp. 8-9 and 54-56 in Appendix at Exhibit 37.

[49] *Id.* at pp. 61-62, 111-112.

[50] *Id.* at p. 111.

[51] *Id.* at pp. 8-9, 61-62, 111, 118-119.

4. Nachman Brautbar, M.D.

Dr. Brautbar was retained as an expert in occupational medicine and toxicology.[52] After interviewing Mr. Casdorph, reviewing his family, medical and work histories as well as his exposure history and reviewing the testimony of Mr. and Mrs. Casdorph, Mr. Casdorph's co-workers and supervisors, Dr. Brautbar opined (1) that Mr. Casdorph had a particularly high level of exposure to benzene and benzene-containing gasoline, paints and solvents; (2) that benzene, gasoline, paints and solvents which contain benzene have been causally associated with chronic myelogenous leukemia; (3) that there is strong epidemiologic evidence indicating that occupational exposure to benzene, gasoline, paints and solvents is a cause of chronic myelogenous leukemia and (4) as a result of Mr. Casdorph's high level of exposure to benzene in the workplace, that Mr. Casdorph's development of chronic myelogenous leukemia was medically caused by his workplace exposures to benzene.[53]

5. Myron Mehlman, Ph.D.

Dr. Mehlman is an internationally known toxicologist.[54] Based upon his review of the records in this case as well as the testimony of Mr. Casdorph and Mr. Casdorph's co-workers and supervisors, Dr. Mehlman has opined, inter alia, that Mr. Casdorph's chronic myelogenous leukemia was a direct

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[52] *See* Curriculum Vitae of Nachman Brautbar, M.D. in Appendix at Exhibit 38.

[53] *See* Deposition Transcript of Nachman Brautbar, M.D., May 6, 2004, at pp. 43-49, 52, 58-61, 87, 92, 95-101, 111-112 in Appendix at Exhibit 39.

[54] *See* Curriculum Vitae of Myron Mehlman, Ph.D. in Appendix at Exhibit 40.

result of his occupational exposures to gasoline, solvents, degreasers, and aromatic hydrocarbon products while he was employed at the West Virginia State Police.[55]

6. Raymond B. Weiss, M.D.

Dr. Weiss was retained by the employer as an expert in the area of oncology. He opined that exposure to benzene cannot cause chronic myelogenous leukemia.[56] However, Dr. Weiss could not cite a scientific article that concluded that benzene did not cause CML.[57]

His opinion was based on the five (5) articles he cited in his report as well as his own medical knowledge.[58] However, Dr. Weiss either was not aware or did not recall the scientific article authored by Adegoke.[59] Also, he "did not pay much attention to" the scientific study authored by Chung Kuo.[60] Dr. Weiss also did not recall the study by Linet which found a statistically significant standard incident ratio (1.5) for CML in auto mechanics.[61] Ironi-

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[55] *See* Deposition Transcript of Myron Mehlman, Ph.D., May 13, 2004, at pp. 15, 17-18, 36, 44-47,49, 52, 66, 77-78, 92-93, 96-100, 101-103 in Appendix at Exhibit 41.

[56] *See* Deposition Transcript of Raymond B. Weiss, M.D. at p. 37 in Appendix at Exhibit 42.

[57] *Id.* at p. 19.

[58] *Id.* at p. 37.

[59] *Id.* at p. 23. [Adegoake, *et al.* found a 2-1/2 fold increase in CML for workers exposed to benzene.]

[60] *Id.* at p. 24. [Kuo, *et al.* found a statistically significant excess (OR = 1.65) of CML in workers exposed to benzene.]

[61] *Id.* at p. 27.

cally, Dr. Weiss was not familiar with this study or the author who, like Dr. Weiss, did work for the National Cancer Institute.[62] Moreover, Dr. Weiss did not recall the Nisse, *et al.* abstract[63]; the study by Pollan and Gustafson[64]; the study published by Savitz, *et al.*[65]; or the study authored by Yin, *et al.*[66]

7. Francis W. Weir, Ph.D.

Dr. Weir was retained by the employer as an expert in the area of industrial hygiene. Dr. Weir's opinions were actually beneficial to Mr. Casdorph and support causation. He opined that:

- "Based on benzene's health effects on the hematopoietic system, i.e. aplastic anemia and leukemia, the benzene content of gasoline vapor has been recognized as one of the most toxicologically significant constituents of the vapor."[67]
- OSHA has concluded that low level exposure to benzene can cause chromo-somal aberrations.[68]
- Chromosomal changes are deleterious.[69]

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[62] *Id.*

[63] *Id.* at pp. 29-31, [Nisse found that a quantitative exposure to solvents was associated with CML in 110 cases when compared with 440 controls].

[64] *Id.* at 32, [Pollan & Gustafson showed almost a 4-fold increase in CML in organic solvent exposed workers].

[65] *Id.* at p. 33, [Savitz, *et al.* concluded that benzene was likely to cause CML].

[66] *Id.* at pp. 33-34, [Yin, *et al.* reports a relative risk of 2.6 developing CML from exposure to benzene].

[67] *See* Deposition Transcript of Francis W. Weir, Ph.D., May 3, 2004, at p. 11 in Appendix at Exhibit 43.

[68] *Id.* at p. 72.

[69] *Id.* at p. 6.

- When atmospheric testing has not taken place and there is a reasonable likelihood that a hazard exists, a respirator is necessary under OSHA regulations.[70]

- Based on the McDermott article provided by Dr. Weir, a person working around gasoline who, like Mr. Casdorph, develops headaches, nausea or dizziness, would have been exposed to at least 1,000 ppm for 60 minutes.[71]

- Exposure to 1,000 ppm in the air of gasoline vapor exceeds the present OSHA benzene regulation.[72]

- The employer violated OSHA safety regulations by not having a respiratory protection program.[73]

- The employer should have provided gloves to Mr. Casdorph that were impervious to gasoline.[74]

- It was not safe for Mr. Casdorph to syphon gasoline by using his mouth.[75]

- The employer had no training program for hazards in violation of OSHA regulations.[76]

- The employer provided no information to its employees, including Mr. Casdorph, about the hazardous nature of the chemical products they were utilizing, in violation of OSHA regulations.[77]

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[70] *Id.* at p. 45.

[71] *Id.* at pp. 54-55.

[72] *Id.* at p. 55.

[73] *Id.* at pp. 62-63.

[74] *Id.* at pp. 50-51.

[75] *Id.* at pp. 52, 54.

[76] *Id.* at p. 63.

[77] *Id.* at p. 68.

8. Mohammed Ranavaya, M.D.

Dr. Ranavaya was retained by the Workers' Compensation Commission to review this case. He issued a report opining that exposure to benzene does not cause CML. However, he had little basis for his opinion.

Dr. Ranavaya was deposed on June 7, 2004 by counsel for the Claimant. In his deposition, Dr. Ranavaya was totally unprepared, evasive, argumentative and, at times, rude.[78]

By reading Dr. Ranavaya's testimony, it is obvious that he was totally unprepared and could not defend his opinion. Moreover, he had not read the 1995 Infante study;[79] the Adegoke study;[80] the Kuo study;[81] the Goguel study;[82] the Linet study;[83] the Nisse study;[84] Pollan and Gustavsson;[85] or

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[78] *See* Deposition Transcript of Mohammed Ranavaya, M.D., June 7, 2004, at pp. 22, 23, 29-31, 33-34, 38, 42-44, 49, 60 in Appendix at Exhibit 44.

[79] *Id.* at pp. 23-24, 26 [Infante reanalyzed the specific cell type of leukemia cases reported by Yin (*see* fn. 34 supra) and found a 4-1/4 excess risk for CML among benzene-exposed workers.]

[80] *Id.* at pp. 40-41, [Adegoke, *et al.* found a 2-1/2 fold increase in CML for workers exposed to benzene.]

[81] *Id.* at pp. 42-44 [Kuo, *et al.* found a statistically significant excess (OR = 1.65) of CML in workers exposed to benzene.]

[82] *Id.* at p. 51 [Goguel, *et al.* found that 30% of benzene leukemias were CML cell-type.]

[83] *Id.* at p. 52 [Linet, *et al.* found a 1-1/2 found excess of CML for auto and truck mechanics.]

[84] *Id.* at p. 54 [Nisse found a quantitative exposure to solvents was associated with CML in 110 cases when compared with 440 controls.]

[85] *Id.* at p. 54 [Pollan, *et al.*, showed almost a 4-fold increase in CML in organic solvent exposed workers.]

Savitz.[86] Moreover, he opined that certain scientific studies were flawed even though he admitted not reading them.[87] Additionally, this was Dr. Ranavaya's first benzene-related, blood disorder case.[88]

Also, despite the request from Claimant's counsel for a copy of all materials reviewed by Dr. Ranavaya upon which he relied as the basis in forming his opinion, none were ever produced.[89]

D. Mr. Casdorph Has Proven and The Prevailing Scientific Evidence Shows That He Was Exposed to Benzene and 2-Butoxyethanol and That Such Exposure Contributed, To A Significant Degree, To His Chronic Myelogenous Leukemia and Resulting Death

In West Virginia, a disease is deemed to have been incurred in the course of or to have resulted from employment only if:

> it is apparent to the rational mind, upon consideration of all the circumstances (1) that there is a direct causal connection between the disease, (2) that it can be seen to have followed as a natural incident of the work as a result of the exposure occasioned by the nature of the employment, (3) that it can be fairly traced to the employment as the proximate cause, (4) that it does not come from the hazards to which workmen would have been equally exposed outside of the employment, (5) that is incidental to the character of the business and not independent of the relation of employer and employee, (6) that it must appear to have had its origin in a risk connected with the employment and to have flowed from that source as a natural consequence, though it need not have been foreseen or expected before its contraction.

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[86] *Id.* at p. 54 [Savitz, *et al.* concluded that benzene was likely to cause CML.]

[87] *Id.* at pp. 46-47.

[88] *Id.* at p. 75.

[89] *Id.* at p. 77.

W.Va. Code §23-4-1. See also, *Powell v. State Workmen's Compensation Comm'r,* 166 W.Va. 327, 273 S.E.2d 832 (1980).

In *Thacker v. Workers' Comp. Division,* 207 W.Va. 241, 531 S.E.2d 66 (1999), this Court held that "[i]n all types of compensation cases, conflicts in evidence, medical or otherwise are to be construed in favor of the claimant."

In *Javins v. Workers' Compensation Commission,* 173 W.Va. 747, 320 S.E.2d 119 (1984), this Court held:

> In all types of compensation cases, conflicts in evidence, medical or otherwise, are to be construed in favor of the claimant. The time-honored liberality rule is not only a rule of statutory construction, but is also an evidentiary rule. *Workman v. Workmen's Compensation Commissioner,* 160 W.Va. 656, 236 S.E.2d 236, 238 (1977); *Pennington v. State Workmen's Compensation Commissioner,* 154 W.Va. 378, 387, 175 S.E.2d 440, 445 (1970); *Pripich v. State Compensation Commissioner,* 112 W.Va. 540, 543, 166 S.E. 4, 5 (1932). It should be clear that in evidentiary matters the law dictates that the claimant be given the benefit of all reasonable inferences the record will allow; and any conflicts must be resolved in favor of the claimant. *Persiani v. State Workmen's Compensation Commissioner,* 248 S.E.2d 844, 846 (W.Va. 1987); *Myers v. State Workmen's Compensaion Commissioner,* 160 W.Va. 766, 239 S.E.2d 124, 126 (1977); *McGeary v. State Compensation Director,* 148 W.Va. 436, 438-39, 135 S.E.2d 345, 347 (1964).

Javins, 173 W.Va. at 758, 320 S.E.2d at 130.

West Virginia Code § 23-4-1g provides, insofar as herein pertinent:

> (a) For all awards made on or after [July 1, 2003] resolution of any issue raised in administering this chapter shall be based on a weighing of all evidence pertaining to the issue and a finding that a preponderance of the evidence supports the chosen manner of resolution. The process of weighing evidence shall include, but not be limited to, an assessment of the relevance, credibility, materiality and reliability that the

> evidence possesses in the context of the issue presented. . . If, after weighing all of the evidence regarding an issue in which a claimant has an interest, there is a finding that an equal amount of evidentiary weight exists favoring conflicting matters for resolution, the resolution that is most consistent with the claimant's position will be adopted.

W.Va. Code § 23-4-1g.

In this case, Mr. Casdorph, his doctors, the general state of scientific knowledge, and the prevailing case law have shown:

1. That there is a casual connection between Mr. Casdorph's CML and his 21 years of exposure to benzene and 2-Butoxyethanol;

2. That his CML followed as a natural incident of exposure to benzene and 2-Butoxyethanol during and in relation to his employment;

3. That there is no evidence that he was equally exposed to benzene and 2-Butoxyethanol outside his employment;

4. That the use of benzene-containing and 2-Butoxyethanol-containing products was incidental to the character of the business; and

6. That the risk of CML is a national consequence of exposure to benzene and 2-Butoxyethanol.

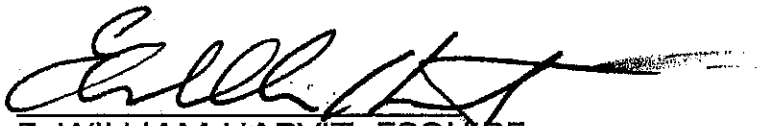
VII.

RELIEF PRAYED FOR

WHEREFORE, Petitioner prays that his Petition for Appeal will be granted, that the Order of the West Virginia Workers' Compensation Board of Review dated December 20, 2006 will be **REVERSED**, that Mr. Casdorph's claim will be held compensable.

Respectfully submitted,

ROBERT H. CASDORPH, JR.,
By Counsel



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CERTIFICATE OF SERVICE

I, E. William Harvit, do hereby certify that I have served the foregoing Petition for Appeal, upon the following named individuals, by United States Mail, postage prepaid, and addressed as follows, this 19th day of January, 2007.

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