LitigationWatch: BENZENE

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TRACKING CASE NEWS AND DEVELOPMENTS IN BENZENE AND RELATED LITIGATION

IN THIS ISSUE:

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Benzene: A Litigation Perspective

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Author Bios on Page 5

Introduction

The National Toxicology Program, U.S. Department of Health and Human Services, *Report on Carcinogens*, Tenth Edition, "Carcinogen Profiles 2002," identifies benzene as a known human carcinogen.[1] Benzene is a clear, colorless liquid used in numerous industrial processes. It is also ubiquitous in the environment, and human exposure to low levels of benzene is unavoidable. Non-occupational exposure occurs primarily through tobacco smoke, automobile service station emissions, car exhaust, industrial emissions, glues, paints, furniture wax and detergents. Workplace exposures occur in industries ranging from oil, shipping, automobile repair, shoe manufacturing and many more. Human exposure occurs via inhalation and dermal contact. Both case reports and epidemiologic studies support the association of benzene exposure with leukemia in humans and it is estimated that as many as three million workers may have been exposed to benzene.[2] Personal injury lawsuits relating to workplace and environmental exposures are on the rise. This article discusses the state of the benzene litigation, benzene disease, and governmental regulations.

Benzene State of the Art

Regulation of Benzene in the Workplace
The American Conference of Government Industrial Hygienists (ACGIH) first set a Threshold Limit Value (TLV) for benzene exposure in 1946 at 100 parts per million. In 1947, the ACGIH lowered the TLV to 50 parts per million; in 1948 to 35 parts per million; in 1957 to 25 parts per million.[3] TLVs are guidelines issued by a private professional association rather than a government agency. Many states adopted the ACGIH-TLV as their law relating to exposure limits.

The Occupational Safety & Health Administration's (OSHA) first regulation relating to benzene was adopted in 1971. The permissible exposure limit at that time was 10 parts per million based on an eight-hour time-weighted average.[4] When Infante, et. al., published their first cohort study relating to the incidence of leukemia in exposed workers in 1977, OSHA attempted to issue an emergency temporary standard of 1 part per million as an eight-hour time-weighted average.[5] That standard was challenged by the American Petroleum Institute, and the Fifth Circuit Court of Appeals found the emergency regulation invalid.[6] In 1978, OSHA attempted to issue a permanent standard of 1 part per million based on an eight-hour time-weighted average. Again, the American Petroleum Institute challenged the standard, and again the Fifth Circuit Court of Appeals struck down the

Continued on Page 4

Benzene: A Litigation Perspective

Continued from Page 1

regulation.[7] The United States Supreme Court granted certiorari and took the appeal. Justice Stevens, writing for the majority, in *Industrial Union Department v. American Petroleum Institute*, stated: "OSHA's rationale for lowering the permissible exposure limit from 10 ppm to 1 ppm was based, not on any finding that leukemia has ever been caused by an exposure to 10 ppm of Benzene and that it will not be caused by exposure to 1 ppm, but rather on a series of assumptions indicating that some leukemia might result from exposure to 10 ppm and that the number of cases might be reduced by lowering the exposure level to 1 ppm."[8] The Court held that the Secretary of Labor exceeded the power outlined in the Occupational Safety and Health Act by failing to first establish that a more stringent standard was necessary to ensure workplace safety.

OSHA's current standard for benzene (1987) establishes a permissible exposure limit of 1 part per million, eight-hour time-weighted average and a short-term exposure limit of 5 parts per million over any fifteen minute period.[9] OSHA's substance safety data sheet identifies the long term effect of benzene exposure as follows: "Repeated or prolonged exposure to benzene, even at relatively low concentrations, may result in various blood disorders, ranging from anemia to leukemia, an irreversible, fatal disease. Many blood disorders associated with benzene exposure may occur without symptoms."[10] The National Institute of Occupational Safety & Health (NIOSH) recommends an exposure limit of 0.1 parts per million on an eight-hour time-weighted average and a short term limit of 1 part per million on a fifteen-minute exposure.[11]

The Benzene Literature

The first case report identifying leukemia in a benzene-exposed worker appeared in an article by Delore and Borgomano in 1928. Isolated case reports reporting leukemia can be found after 1928.[12] The first epidemiological study linking benzene exposure to leukemia was published in the mid-1970s by McMichael et. al., a study of tens of thousands of rubber workers which associated leukemia with benzene exposure.[13]

Infante, et. al. published their first cohort study of benzene-exposed workers in 1977. That study examined workers exposed to benzene from Pliofilm (a type of food wrap). The authors concluded that workers exposed to benzene at that plant were five to ten times more likely to develop leukemia.[14]

Nevertheless, skepticism still existed as to whether benzene could indeed cause leukemia. The existing studies were criticized as improper or inconclusive. "Benzene and Leukemia," published in the *Lancet* in 1977 criticized Infante, et. al., for improperly combining cohorts.[15] The 1981 version of "Patty's Industrial Hygiene and Toxicology" criticized the McMichael study and stated: "The isolation of a human leukemia virus by Gallagher and Gallo puts a new complexion on the presumptive etiologic agent in so-called benzene leukemia. The 60 cases tabulated between 1931 and 1960 by Browning, the bulk of which were one or two cases per plant, and the relatively few cases of leukemia found in McMichael's studies of tens of thousands of rubber workers, create a pattern consistent with an hypothesis that benzene, rather than being a carcinogen per se, triggers increased activity of the leukemia virus, present in only an occasional worker."[16]

The scientific community that recognized a link between benzene exposure and leukemia began studies to determining what level, if any, constituted a safe level of exposure at which leukemia would not occur. Grilli et. al., in "Possible Implications from Results of Animal Studies in Human Risk Estimations for Benzene: Nonlinear-Dose Response Relationship Due to Saturation of Metabolism" in 1987, examined the data regarding human exposure and exposure to small rodents to determine the incidence of leukemia at different levels of exposure. The study attempted to determine if a linear dose-response relationship was supported by the incidence of leukemia at the 100 parts per million exposure limit versus the 1 part per million exposure limit. Grille, et. al., found that the incidence of leukemia at the 10 parts per million was actually two to three times higher than that which would have been expected if a linear dose response relationship existed.[17]

Austin, et. al., in "Benzene and Leukemia: A Review of the Literature and a Risk Assessment," (1988), stated: "The assessments that we consider acceptable suggest that, among 1,000 men exposed to benzene at 10 ppm for a working lifetime of 30 years, there would occur about 50 excess deaths due to leukemia in addition to the baseline expectation of seven deaths. However, this estimate is speculative and whether or not enough confidence can be placed in it to justify a lower occupational benzene standard remains a decision for policy makers."[18]

In 1989, Brett, et. al., published an article in the *Journal of Environmental Health Perspectives* entitled, "Review and Update of Leukemia Risks Potentially Associated with Occupational Exposure to Benzene." The article criticized the 1977 Infante, et. al. study as improperly characterizing the exposure histories of the various cohort groups and, instead, advocated an approach that analyzed individual exposure histories. Applying this individual assessment approach, Brett et. al. re-evaluated seven previous studies of Plioform workers and concluded that only 0.5 excess leukemia deaths resulted per 1,000 workers exposed for 45 years at a level of 1 part per million of benzene, while 7.9 excess deaths resulted per 1,000 workers exposed at a level of 10 parts per million for 45 years.[19] The Brett, et. al., study, thus, seemed to support an argument for a "low-dose" benzene defense as it relates to development of leukemia.

Also in 1989, Lance Wallace published "Major Sources of Benzene Exposure." He analyzed air samples of more than 700 subjects from various urban settings and concluded that benzene exposure occurs largely due to personal and day-to-day activities as opposed to occupational activities. He concluded that exposures from automobiles, first- and second-hand cigarette smoke and consumer products accounted for the largest percentages of benzene exposure, while occupational benzene exposure constituted no more than a few percent. He suggests that the non-occupational exposures are important issues to explore in Benzene litigation.[20]

Wallace's results are supported by the U.S. Department of Health and Human Services, the Public Health Services' National Toxicology Program's 2002 Tenth Edition *Report on Carcinogens*. Passive smoking is identified as the largest source of Benzene exposure in the general population, although interestingly, cigarette smoke is listed far below automobiles in terms of the amount of benzene emissions.[21]

Steffen, et. al., examined the relationship between benzene-exposed children and childhood leukemia in 2004. The study observed both children directly exposed to benzene based on residential proximity to vehicular benzene emission and secondarily exposed via mothers exposed to benzene occupationally during pregnancy. The study concluded that there was a relationship between benzene exposure and childhood leukemia in the directly-exposed group, though the relationship was less remarkable in the secondarily-exposed group.The study conceded that further research was needed to determine the relationship between benzene exposure and childhood leukemia. However, this study could be significant in evaluating latency issues in benzene litigation.[22]

The Benzene Case

The literature suggests that the health effects caused by benzene exposure

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depend upon the level of exposure. Acute inhalation has been associated with convulsions, dizziness, drowsiness and unconsciousness in humans. Chronic benzene exposure has been associated with irreversible injury, including leukemia, to blood forming organs.[23] The literature identifies other specific diseases that have been attributed to benzene exposure including: acute myeloid leukemia (AML), acute lymphocytic leukemia (ALL), non-Hodgkin's lymphoma (NHL), multiple myeloma (MM) and myelodysplasia.[24] One must examine the literature carefully to determine what, if any, threshold exists between exposure to Benzene and any of these serious conditions.

In non-acute myeloid leukemia cases, a successful defense focuses on the scientific literature to determine if studies show a statistically significant relationship between benzene exposure and a particular disease. For example, recent articles suggest that there is no association between benzene and non-Hodgkins lymphoma and multiple myeloma.[25] If the lawsuit involves acute myeloid leukemia, the defense should focus on low dose since the scientific literature has consistently reported data that indicates that acute myeloid leukemia is related only to cumulative exposure greater than 40 ppm/ years.

In defending the benzene case, one must consider the source of the alleged benzene exposure. If the exposure involves a benzene-containing product or trace benzene exposure, a successful defense may revolve around the fact that the exposure is far below proscribed OSHA regulations. Obviously, for the plaintiff to prevail on a claim, he or she will need to establish that benzene exposure occurred at a sufficient level for a sufficient period of time to cause the identified health effect.[26] In low level benzene cases, the causation element frequently is difficult to prove. Plaintiffs might be barred from asserting a failure to warn case, as both OSHA and ANSI standards require warnings for benzene only if the benzene content is greater than 0.1 percent.[27]

Recent Litigation

Benzene litigation has been clustered in California and Texas, but new filings are being reported all over the country. Historically, cases have originated out of the petroleum industry, but more recent cases include alleged exposure in the rubber, paint and adhesives industries.

Many of the cases rise or fall on the adequacy of the plaintiff's experts. A significant number of plaintiff's experts are unable to withstand *Daubert/Havner/Frye* challenges and cases are dismissed on summary judgment. For example, in *Frias v. Atlantic Richfield Company*, 104 S.W. 3d 925 (Tex. App. – Houston, 2003), the trial court entered summary judgment in favor of defendants on general and specific causation grounds from which the plaintiffs appealed. The appellate court affirmed, finding that studies cited by the plaintiff's expert did not meet the 95 percent confidence level and did not establish an association between the studies and the plaintiff to prove general causation. The Court further found insufficient evidence of the frequency and duration of plaintiff's exposure to benzene while working at the refinery.[28]

The trial court in *Austin v. Kerr-McGee Refining Corp.,* 25 S.W.3d 280 (Tex. App. – Texarkana 2000) struck the plaintiff's experts' medical causation evidence. There, the plaintiff contracted chronic myelogenous leukemia (CML) which he claimed was caused by his use of mineral spirits to clean pipes on oil rigs. The appellate court affirmed, finding that the medical literature cited by plaintiff's expert failed to show a sufficient association between benzene and CML and the degree or level of benzene to which plaintiff was actually exposed. The court also found that the plaintiff failed to sufficiently exclude other possible causes.[29]

One defense sometimes asserted by product manufacturer defendants is that of the learned intermediary or bulk supplier. The manufacturer contends that it discharges its duty to warn by providing information to a third person upon whom it can reasonably rely to communicate the warning to the ultimate end user of the product.[30]

A particularly noteworthy benzene decision is *Mobil Oil Corp. vs. Ellender*, 968 S.W. 2d 917 (Tex. 1998), wherein Mobil's appeal of a punitive damages award of $6,000,000 was denied. The decedent was an independent contractor millwright at Mobil's Beaumont, Texas refinery and chemical plant from 1963 to 1977, who claimed exposure to benzene while maintaining equipment and product lines. Plaintiff alleged that, as a result of this exposure, the decedent contracted and died from acute myelogenous leukemia (AML). Plaintiff sued Mobil for failure to warn of exposure to and the risks associated with benzene and failure to protect the decedent. The jury awarded $622,888.97 in compensatory damages and $6,000,000 in punitive damages.

On appeal, the Texas Supreme Court determined that there was legally sufficient evidence to support the jury's finding of gross negligence by Mobil and thus the Court upheld the punitive damages award. In addition to expert testimony, the court cited Mobil's' membership in associations that publicized the dangers of benzene exposure from the early 1900's (namely the National Safety Council and the American Petroleum Industry), the fact that other similarly situated oil companies had practices and procedures to address benzene exposure risks, the fact that Mobil's medical directors were well aware of benzene-associated risks, and that Mobil's industrial hygiene practices included monitoring and providing protective equipment for benzene exposure to direct employees but not to contractors, as grounds for affirming the verdict.[31]

In *Watts, et al. v. Radiator Specialty, et al.,* No. 2002-364 (Miss. Cir. Ct., Smith Cty. 2004), a jury returned a $2,000,000 verdict in a case filed by a plaintiff who con-

tracted non-Hodgkin's lymphoma. Plaintiff was employed as a mechanic and regularly used the benzene-containing product Liquid Wrench. The product manufacturer/distributor was found 40 percent liable, the supplier of benzene-containing ingredients was held 45 percent liable and the former employer 15 percent liable.

Defendants filed several post-trial motions, including one asserting that plaintiff's expert witnesses failed to satisfy admissibility standards. Defendants claimed that Plaintiff's experts did not quantify the level of exposure to benzene that was necessary to cause non-Hodgkins lymphoma and that there is no literature to support conclusions that Liquid Wrench has been associated with this specific disease.

Oral arguments on these issues were held on Jan. 27, 2006, and in May 2006, the presiding judge, Robert G. Evans, filed his decision on the motion under seal and ordered the parties to mediation. Two weeks later, on May 12, 2006, he released his decision to grant the defendants' motion for judgment notwithstanding the verdict. Judge Evans held that "neither the cohort nor the case control studies relied on by Dr. Barry Levy support his opinion that a causal connection exists between benzene exposure and Non-Hodgkin Lymphoma." As such, he found "Dr. Levy's opinions failed to meet *Daubert* muster," that admission of Dr. Levy's testimony was reversible error, and that without Dr. Levy's testimony there was no proof of causation. As such, the motions for judgment notwithstanding the verdict were granted. *[Editor's note: The plaintiffs have appealed the trial court's decision in Watts. See related story on Page 8.]*

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Footnotes

[1] "Report on Carcinogens, Tenth Edition, Carcinogen Profiles 2002," U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program (2002).
[2] *Id.*
[3] Infante, Peter F. "Benzene: an historical perspective on the American and European occupational setting."
[4] 29 CFR 1910.1028(c) (1971).
[5] 42 Fed. Reg. 22516 (1977).
[6] *Id.* At 27452.
[7] *American Petroleum Institute v. OSHA*, 581 F.2d 493 (1978).
[8] *Industrial Union Dept., AFL-CIO v. American Petroleum Institute, et al.*, 448 U.S. 607, 634 (1980).
[9] 29 CFR 1910.1028(c) (1987).
[10] 29 CFR 1910.1028 Appendix A (1987).
[11] See, "NIOSH Pocket Guide to Chemical Hazards: Benzene."
[12] Delore, P. and Borgomano, C. "Acute leukemia following benzene poisoning." *J. Med. Lyon*, 9, 227-233 (1928).
[13] McMichael, A.J., et al. "Solvent exposure among rubber workers: an epidemiological study." *J. Occup. Med.*, 17, 234-239 (1975).
[14] Infante, Peter F., et al. "Leukemia in Benzene workers." *The Lancet*, 2, 76-78 (1977).
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[16] Clayton, George D. and Clayton, Florence E. "Patty's Industrial Hygiene and Toxicology: Third Revised Edition, " 2891 (1981).
[17] Grilli, Sandro, et al. "Possible implications from results of animal studies in human risk estimation for benzene – non linear dose-response relationship due to saturation of metabolism." *J. Cancer Res. Clin. Oncol.*, 113:349-358 (1987).
[18] Austin, H., et al. "Benzene and leukemia: A review of the literature and a risk assessment." *Amer. J. Epid.*, 127 (1988).
[19] Brett, Susan M., et al. "Review and Update of Leukemia Risk Potentially Associated with Occupational Exposure to Benzene." *Envir. Health Perspectives*, 82, 267-281 (1989).
[20] Wallace, Lance A. "Major Sources of Benzene Exposure." *Envir. Health Perspectives*, 82, 165-169 (1989).
[21] "Report on Carcinogens, Tenth Edition, Carcinogen Profiles 2002," U.S. Department of Health and Human Services, Pubic Health Service, National Toxicology Program (2002).
[22] Steffen, C., et al. "Acute childhood leukemia and environmental exposure to potential sources of benzene and other hydrocarbons; a case-control study." *Occup. Envir. Med.*, 61, 773-778 (2004).
[23] "Toxicological Profile for Benzene" (Draft for Public Comment), Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Public Health Service, U.S. Department of Health and Human Services, (2005).
[24] See *id*; see, *e.g.*, Rinsky, "Benzene and Leukemia", *New England Journal of Medicine*, (April 23, 1987); Aksoy, M., et al. "Clinical observations showing the role of some factors in the etiology of multiple myeloma." *Acta Haematol*, 71:116 (1984); Shu, X., et al. "Parental Occupational Exposure to Hydrocarbons and Risk of Acute Lymphocytic Leukemia in Offspring" *Cancer Epidemiology Biomarkers & Prevention*, Vol. 8, 783-791 (September 1999); Smith, M. et al., "Hydroquinone, a benzene metabolite, increases the level of aneusomy of chromosomes 7 and 8 in human CD34-positive blood progenitor cells", *Carcinogenisis*, Vol. 21, No. 8 pp. 1485-1490 (2000).
[25] Glass, "Leukemia Risk Associated with Low-level Benzene Exposure", *Epidemiology*, Vol. 14, No. 5, (September, 2003).
[26] *Marsch v. Exxon Mobil Corp.*, No. 4:03-CV-1646 CAS, WL 2246006 at *10-11 (E.D.Mo. 2005).
[27] See, *Wright v. Mobil Oil Corp.*, No. 09-93-154 CV, WL 672594 at 1 (Tex. App. 1994); *Laico v. Chevron U.S.A., Inc.*, 123 Cal. App. 4th 649, 655, 20 Cal. Rptr. 3d 307 (6th Dist. 2004).
[28] *Frias v. Atlantic Richfield Company*, 104 S.W. 3d 925 (2003) at 930.
[29] See, also, *Daniels v. Lyondell-Citgo Refining Co. Ltd.*, 99 S.W.3d 722 (2003) (affirming summary judgment for the refinery on the basis that benzene was not shown to be cause of lung cancer); *Knight, et al. v. Kirby Inland Marine, et al.*, No. 01-223 (N.D. Miss.) (granting summary judgment for lack of credible medical evidence that benzene exposure causes bladder cancer or Hodgkin's lymphoma); *Parker v. Mobil Oil Corp., et al.*, No. 2005-02454 (N.Y. Sup. Ct., App. Div., 2nd Jud. Dept.) (appellate court reversed pervious denial of summary judgment, ruling that plaintiff's expert testimony was inadmissible because it did not quantify the amount of alleged exposure to benzene.)
[30] See, *Wood v. Phillips Petroleum Co.,* 119 S.W.3d 870 (Tex. App. – Houston, 2003) and *Curtis v. M&S Petroleum Inc.,* 174 F.3d 661 (5th Cir. (Miss.) 1999) (adopting that rationale).
[31] *Mobil Oil Corp. vs. Ellender*, 968 S.W. 2d 917 (Tex. 1998) at 922-924.