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Mr. Joseph D. Lonardo
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1909 K Street NW, Suite 900
Washington, DC 20006-1152

August 23, 2010

RE: Hord v. Goodyear Tire & Rubber Company

Dear Mr. Lonardo,

I am a licensed physician specializing in occupational medicine. I am board certified in both occupational medicine and internal medicine. I am qualified as a specialist in the fields of occupational medicine and internal medicine and in the field of epidemiology, especially as it relates to the study of diseases related to exposures to chemical agents. I am Emeritus Professor of Occupational Medicine and Epidemiology at the University of Michigan School of Public Health, and I also hold an appointment as Emeritus Associate Professor in the Department of Emergency Medicine, University of Michigan School of Medicine. I am engaged full-time in research in occupational epidemiology, the training and education of physicians and graduate students in occupational health, and in the clinical practice of occupational medicine.

I received my undergraduate degree with high honors in chemical engineering from Tufts University, Medford, Massachusetts in 1972. I received my M.D. degree from Tufts University School of Medicine, Boston, in 1976. From 1976-77, I served as an intern in internal medicine at Georgetown University Hospital in Washington, D.C. and from 1977-78, I served as a fellow in medical ambulatory care at that same institution. From 1978-80, I served as a resident in occupational medicine at the Harvard School of Public Health, Boston, and received the Master of Public Health degree in 1979 and the Master of Science in Physiology degree in 1980 from that institution. From 1980-81, I served as senior resident in internal medicine at University Hospital, Boston.

Upon completion of my training in 1981, I joined the faculty of the University of Southern California School of Medicine, where I was engaged in research in occupational cancer epidemiology and in the practice of occupational medicine until 1988, when I joined the faculty of the University of Michigan. While I have been on the faculty of the University of Michigan, I have served as Director of Occupational Medicine (1988-94), head of the Occupational Health Program (1992-95), Director of the Center for Occupational Health and Safety Engineering (1990-95), Director of the Occupational & Environmental Epidemiology program (2001-2007),

and Founding Director of the Risk Science Center (2003-present). My research has focused for the past 25 years on the long term health effects of chemicals on humans and I have published over 200 research articles, book chapters, and abstracts related to this area. A copy of my curriculum vitae is attached.

I have served on the Editorial Board of the Journal of Occupational Medicine and currently serve as a reviewer of scientific papers for a number of other journals, including Environmental Health Perspectives, Journal of the National Cancer Institute, Cancer Causes and Control, Cancer Epidemiology Biomarkers and Prevention, American Journal of Epidemiology, and Cancer Research. I was appointed to membership on the Safety and Occupational Health Study Section of the National Institutes of Health (NIH) in 1992 and served on that Study Section until 1996, having been appointed by Secretary of HHS Donna Shalala as Chair for the 1995-96 year. In that position I served as a peer-reviewer of research proposals for the NIH. I am a Fellow of the American College of Occupational and Environmental Medicine (FACOEM) and a Fellow the American College of Preventive Medicine (FACPM). I am currently a member of a number of professional organizations, including the Society for Epidemiologic Research, the International Epidemiological Association, the Society for Risk Analysis, and the American College of Occupational and Environmental Medicine. I received the Excellence in Research Award from the University of Michigan School of Public Health in 2006 and the Research Excellence Award from the Risk Science Center at the University of Michigan School of Public Health in 2007.

Materials Reviewed

I have reviewed the materials that your office forwarded to me including:

- Plaintiff's Pre-Trial Statement
- Reports by
 - Stephen E. Petty, P.E., C.I.H., dated 3/9/2009
 - Peter F. Infante, D.D.S., Dr.P.H., F.A.C.E., dated 3/31/2010
 - Dennis D. Weisenburger, M.D., dated 3/24/2010
 - Richard K. Shadduck, M.D., dated 1/13/2010
 - Pamela Williams, Sc.D., dated 8/17/2010
- Medical Records from
 - Arthur B. Dalton, M.D.
 - Clinical Hematology & Oncology
 - Employment File
 - Pathology Report
 - Physicians Urology
 - Summa Health – Akron City Hospital
 - Waleed F. Nemer, M.D.
- Depositions of
 - Dennis D. Weisenburger, M.D., dated 8/3/2010
 - Richard Shadduck, M.D., dated 7/21/2010

In addition, I have reviewed the scientific literature that is relevant to the issues of causation of non-Hodgkin lymphoma and, specifically, follicular large cell lymphoma. A list of the relevant scientific literature upon which my opinions are based, is attached.

Past Medical History

Mr. Anthony T. Hord was born on 7/18/1931 in Akron, Ohio. He was a past smoker but quit approximately 30 years prior to his death. He also drank alcohol, allegedly quitting after he was medically advised to in July 2004. The various descriptions of Mr. Hord's alcohol intake in the medical records include daily intake of beer, four days a week of liquor and beer, three days a week of 6-8 shots of liquor and 6-8 beers, and daily intake of three glasses of vodka. Mr. Hord's medical history was significant for prostate cancer (1998) with prostatectomy, laparoscopic cholecystectomy, increased liver function tests with nodular hepatomegaly, and hypertension. There was no reported history of lymphohematopoietic disease in Mr. Hord's family.

Occupational History

Mr. Hord started working at Goodyear Tire & Rubber Co. in Akron, Ohio as a utility worker on 7/19/1951 and retired as a grounds crewman on 10/24/1992. According to Dr. Williams' report, Mr. Hord worked as a tire builder for less than 3 months in 1956, a position from which he was subsequently disqualified. He worked in the hose manufacturing department for approximately 20 years in 1958-1979. For the remainder of his employment, he worked in a variety of positions including utility worker, janitor, laborer, stockman, line inspector, truck and general service worker, reroll liner worker, rotocure worker, and grounds crew. He was not in the plant for approximately 6 years between 1951 and 1992 due to furloughs, layoffs, and injuries.

The only positions in which Mr. Hord may have handled or used solvents or cements directly were

- a) Tire builder in Plant 1 (Dept 152A), 9/19/1956 to 12/9/1956
- b) Hose building on the #1 machine or Spiraling Machine (Dept 273A), 9/5/1959 to 9/14/1959 and 8/8/1961 to 11/20/1961.
- c) Processing of irrigation hose (Dept 273D), 11/25/1978 to 5/18/1979

Rubber gloves were always furnished by Goodyear for handling and use of solvents and cements during these operations. Workers were instructed by Goodyear not to use solvents to clean their skin. According to Dr. Williams' report, applying the available benzene air monitoring data to the specific jobs held by Mr. Hord results in a cumulative inhalation exposure to benzene of 0.3 ppm-years. Insofar as there is no reliable evidence that Mr. Hord had appreciable skin contact with solvents it is unlikely that he had any appreciable dermal exposure to benzene.

I am aware that Mr. Petty estimated Mr. Hord's cumulative benzene exposure at Goodyear to be 124.47 ppm-years with a likely range of 109-139 ppm-ys. Of the 124.47 ppm-years, 99.58 ppm-years were calculated to be by dermal exposure and 24.89 ppm-years by inhalation. It is my understanding that there are substantial discrepancies between Mr. Petty's assumptions that underlie his calculations and the written records regarding Mr. Hord's employment, the use and constituents of various solvents used at Goodyear, and the testimony of Goodyear employees.

Lymphohematopoietic Disease Course

Mr. Hord presented to medical attention in June 2004 after noticing darkened pigmentation on his chest and a left parotid lump. He was referred to Dr. Arthur B. Dalton for evaluation of the mass, which Mr. Hord claimed had been present for the past two to three weeks. He also stated that he had lost approximately 50 pounds because of a poor appetite. Dr. Dalton discovered a 3.0 x 3.5 cm “somewhat rubbery, firm soft tissue mass, left preauricular region” on examination. The mass was considered consistent with a parotid neoplasm. A fine needle aspiration was performed and a CT scan of the neck, abdomen and pelvis was ordered. The consultation note also stated that hepatosplenomegaly was evident with a palpable spleen tip and a liver edge, extending 10 to 12 cm below the costal margin.

The fine needle aspiration biopsy was consistent with a reactive lymph node and the CT scan of the neck revealed a 3.3 x 2.7 x 2.2 cm prominent left parotid gland mass with surrounding lymphadenopathy. A prominent right peri-submandibular gland lymph node, 2.4 cm in size, was noted. CT scans of the abdomen and pelvis revealed extensive retroperitoneal and intraperitoneal lymphadenopathy, including a 4.4 cm left inguinal node.

On 7/20/2004, Mr. Hord underwent a left inguinal lymph node biopsy which revealed an “enlarged lymph node extensively involved by high grade, non-Hodgkin lymphoma, follicular large cell type (follicular center cell lymphoma, grade III).” Immunohistochemical studies were performed and CD20, CD10, BCL2, and BCL6 were strongly expressed. CD5 and CD45 RO were not expressed which, according to the pathology report, highlighted relatively numerous accompanying reactive T lymphocytes. These findings confirmed a B-cell follicular center cell immunophenotype.

Mr. Hord was referred to oncology for staging and treatment. Their assessment was that he had follicular large cell (follicular center 3) B-cell, non-Hodgkin lymphoma, stage IV with diffuse adenopathy, borderline splenomegaly and a parotid mass. Between August and December 2004, Mr. Hord completed six cycles of RCHOP chemotherapy with a clinical and radiographic complete response.

Mr. Hord was stable until October 2006, when he developed new ascites and severe lower extremity edema. His liver function tests had been chronically high and he had had nodular hepatomegaly, but a biopsy of the liver (taken 1/05) was read as normal. An abdominal CT scan, taken on 10/5/06, noted pleural-based plaques along the chest wall regions in the lower portion of each hemithorax; a massive amount of ascites throughout the abdomen and pelvis; a nodular liver shrunk in the region of the right lobe and the medial segment of the left lobe, compatible with cirrhosis; and several small lymph nodes in the para-aortic region. A CT scan of the pelvis revealed massive amounts of free fluid in the pelvis and extensive subcutaneous edema throughout the lower abdominal wall and gluteal regions.

On 1/23/07, Mr. Hord presented to his primary physician complaining of increased abdominal distension and shortness of breath. The distension was believed to be because of massive ascites and Mr. Hord was transported to the emergency room for CAT scan and immediate paracentesis. He was admitted for management of multiple medical problems beginning with cirrhosis and portal hypertension with encephalopathy and ascites, in addition to worsening kidney function and exacerbation of congestive heart failure. Mr. Hord deteriorated quickly and passed away on 1/27/2007. His death certificate listed his cause of death as acute respiratory failure due to congestive heart failure due to cirrhosis due to renal failure. Non-

Hodgkin's lymphoma was listed as an other significant condition contributing to death but not resulting in the underlying cause.

My Opinions

1. Mr. Anthony Hord was diagnosed with non-Hodgkin lymphoma, follicular large cell type, in July 2004.
2. It is alleged that Mr. Hord was exposed to benzene and benzene-containing solvents while working at the Goodyear Tire & Rubber Co. facility in Akron, Ohio, in various capacities, including tire builder and hose fabricator. Based on his employment records, his potential solvent exposure was limited to four time periods: September-December 1956, September 1959, August-November 1961, and November 1978 –May 1979. His cumulative exposure to benzene has been estimated by Dr. Williams at 0.3 ppm-years.
3. There have been dozens of epidemiologic studies that have looked for evidence that humans exposed to benzene, petroleum products, paints, glues, adhesives, and other products that contain petroleum distillation fractions are at increased risk of non-Hodgkin's lymphoma (NHL) (Andjelkovic 1976, Australian Institute of Petroleum 2007, Balarajan 1983, Band 2004, Bernard 1984, Blair 1992, Blair 1993, Blair 1998, Bloemen 2004, Blot 1977, Brown 2002, Brownson 1988, Cano 2001, Cartwright 1988, Chen 1999, Cocco 2010, Collingwood 1996, Collins 2003, Colt 2007, Consonni 1999, Costantini 2001, Costantini 2003, Delzell 1992, Divine 1999, Divine 2000, Dryver 2004, Fabbro-Peray 2001, Franceschi 1989, Fritschi 1996, Fritschi 2005, Fu 1996, Garland 1988, Gérin 1998, Girard 1970, Glass 2003, Gray 2001, Greene 1979, Greenland 1994, Hall 1991, Hardell 1981, Hardell 1994, Hayes 1997, Huebner 2004, Hunter 1993, Hunting 1995, Kaplan 1986, Kato 2005, Kauppinen 2003, Kirkeleit 2008, La Vecchia 1989, Lagorio 1994, Leon 1994, Lewis 2000, Lewis 2003, Linos 1991, Lynge 1995, Lynge 1997, Mallin 1986, Mao 2000, Massoudi 1997, McMichael 1974, Mester 2006, Miligi 2006, Miller 1989, Nilsson 1998, Olsson 1988, Orsi 2007, Ott 1989, Parodi 2003, Patel 2004, Persson 1999, Pukkala 1998, Raaschou-Nielsen 2001, Rafnsson 2001, Rego 2002, Richardson 2008, Rinsky 1988, Rinsky 2002, Sans 1995, Satin 1996, Satin 2002, Scherr 1992, Schnatter 1996, Schottenfeld 1981, Schumacher 1988, Schwartz 1987, Seidler 2007, Semenciw 1993, Siemiatycki 1987, Sorahan 2005, Sorahan 2007, Spirtas 1991, Steenland 1999, Tatham 1997, Terstegge 1995, Tsai 2003, Tsai 2007, Vianna 1979, Vineis 2007, Wang 2009, Waxweiler 1983, Wilcosky 1984, Wong 1987, Wong 1999, Wong 2000, Wong 2001, Wong 2010, Xu 2003, Zoloth 1986). Benzene, petroleum products, paints, glues, adhesives, and other products that contain petroleum distillation fractions are not known to cause NHL of any type or to cause follicular large cell lymphoma specifically. It is not generally accepted in the scientific community that benzene, petroleum products, paints, glues, adhesives, and other products that contain petroleum distillation fractions are causes of NHL or of follicular large cell lymphoma.
4. There are numerous review articles and meta analyses that have summarized the evidence regarding NHL and benzene exposure (Alexander 2010, Weed, 2010, Steinmaus 2008, Kane 2010, Smith 2007, Wong 2005). The most methodically rigorous of these studies (Alexander 2010, Weed, 2010, Kane 2010) did not find an association between NHL and benzene exposure when all the peer-reviewed studies were considered.

5. In contrast to the methodologically rigorous studies (Alexander 2010, Weed, 2010, Kane 2010), the Smith 2007 review is based on a biased selection of studies, and within those studies, a biased selection of the positive results. Moreover, Smith 2007 ignores results that are contrary to his opinions. This flawed methodology results in a biased conclusion that there is an association between benzene exposure and NHL.
 - a. For example, Smith relies on the Kato, 2005 article which reports an association between NHL and exposure to paint thinner or turpentine. However, Smith first ignores that the Kato article also reported no association between paints/varnishes and NHL (Odds Ratio = 0.79, 95% CI 0.40-1.58) and then ignores peer reviewed literature that finds no association whatsoever between non-Hodgkin lymphoma and work as a painter or work exposed to paint thinner (Bethwaite 1990, Chiazze 1980, Lundberg 1998).
 - b. Although Smith 2007 cites the Fabbro-Perray 2001 study as showing a positive association between benzene exposure and NHL in his table 1, he ignores that Fabbro-Perray also reported no association between paints and NHL (OR = 0.8, 95% CI 0.5-1.3). It is inexplicable how he could cite the positive association in Kato 2005 and ignore the negative association in Fabbro-Perray 2001 for the same exposure. This shows evidence of bias.
 - c. Similarly, Smith cites the Hardell 1981 finding that high grade exposure to trichloroethylene, perchloroethylene, styrene, and benzene combined is associated with a relative risk (RR) of 4.5 (95% CI 1.9-11.4) for NHL. Smith neglects to mention that in fact only one of the NHL cases had exposure to benzene. So, the Hardell study has little to do with benzene and it reports no findings about benzene by itself. This type of biased picking and choosing, as conducted in the Smith 2007 paper, is not a reliable scientific method. Smith's conclusion that 40 of 43 case control studies showed an elevation of NHL risk has no meaning because Smith simply chose positive studies and ignored negative studies.
6. There are six studies that have examined the association between follicular NHL and benzene exposure specifically. The results of these studies are presented below.

Disease	Study	Source	Cases	Controls	OR	95% CI
Follicular NHL	Wong 2010	Table 9 (Benzene ever v never)	7	2	7.00	1.45-33.70
	Wong 2010	Table 9 (Benzene highest exposure groups)	1	1	2.00	0.13-31.98
	Cocco 2010	Table 4 (High exposure to benzene)	4	52	1.0	*
	Wang 2009	Table 4 (benzene)	29	284	1.2	0.8-1.7

Disease	Study	Source	Cases	Controls	OR	95% CI
	<i>Seidler 2007</i>	Table 4 (benzene > 130 ppm- yrs)	2	12	1.3	0.3-6.4
	<i>Miligi 2006</i>	Table 4 (benzene)	2	35	*	*
	<i>Blair 1993 and Blair 1992</i>	Table 6 (benzene, higher intensity)	5	18	1.9	0.7-5.3

Only one of these studies shows a statistically significant association (Wong 2010), but shows no evidence of dose-response. In the Wong study, the measure of association in the highest exposure group was lower than in the less exposed groups, thus arguing against dose-response. The other five studies show either no association or weak, non-significant associations. The largest study of all (Wang 2009), which had more cases than all the other studies combined, showed only a weak association (Odds Ratio = 1.2) that was not statistically significant.

7. There are seven studies that have examined the association between follicular NHL and organic solvent exposure. The results of these studies are presented below.

Disease	Study	Source	Cases	Controls	OR	95% CI
Follicular NHL	<i>Cocco 2010</i>	Table 3 (All solvents)	111	1009	1.3	1.0-1.7
	<i>Wang 2009</i>	Table 4 (organic solvents)	59	128	1.3	0.8-2.0
	<i>Miligi 2006</i>	Table 4 (aromatic hydrocarbons)	9	35	1.1	0.5-2.5
	<i>Rego 2002</i>	Table 2 (organic solvents)	4	55	2.30	0.54-9.26
	<i>Tatham 1997</i>	Table 3 (chemical solvents)	114	154	1.00	0.79-1.40
	<i>Hardell 1994</i>	Table 4 (organic solvents)	7	*	6.0	0.9-41
	<i>Blair 1993 and Blair 1992</i>	Table 6 (other solvents, higher intensity)	6	38	1.1	0.4-2.7

The largest study of all (Tatham 1997), showed no association whatsoever (Odds Ratio = 1.0). The next largest study, by Cocco (2010), showed a weak association (Odds ratio = 1.3) between all solvents and risk of follicular NHL, that was barely statistically significant. The other five studies combined had less data than Tatham alone, and showed either no association or non-significant associations. There was no evidence of dose response in any of the studies, and in Blair, the highest exposure group had a lower

Odds Ratio than the less exposed group. In summary, there was neither a consistent association between follicular lymphoma and solvent exposure, nor dose response.

8. There is no exposure level to benzene, petroleum products, paints, glues, adhesives, and other products that contain petroleum distillation fractions that is known to place people at increased risk of NHL, or of follicular large cell lymphoma.
9. Insofar as benzene, petroleum products, paints, glues, adhesives, and other products that contain petroleum distillation fractions are not known to place people at increased risk of either NHL or follicular large cell lymphoma, there is no scientific basis for a conclusion that Mr. Hord's alleged exposure to benzene or benzene-containing solvents played any role whatsoever in the causation of his follicular large cell lymphoma.

In summary, it is my opinion to a reasonable degree of medical certainty that the alleged exposures to benzene and benzene-containing solvents that Mr. Anthony Hord encountered while working at Goodyear Tire & Rubber Co.'s Akron, OH facility did not cause, contribute to, or in any way lead to his follicular large cell lymphoma.



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References Relied Upon by David Garabrant, MD, MPH

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