Benzene-contaminated toluene and acute myeloid leukemia: a case series and review of literature

Trevor Peckham¹, Melvyn Kopstein², Jason Klein¹ and James Dahlgren¹

Abstract
We report seven cases of acute myeloid leukemia (AML) with occupational exposure to a toluene-based hydrocarbon solvent. The cases were employed at a facility, which manufactured rubber belts and hoses, between 1950 and 2005 for periods ranging from 21 to 37 total years. Detailed histories were obtained for three workers who were diagnosed with AML within a 3-year period (2003–2005). Death certificates, medical records, and accounts by workers were reviewed. Benzene, a known cause of AML, is typically a contaminant of toluene. Benzene contamination in toluene and other widely used solvents and the potential for concurrent benzene exposure during usage of these solvents in occupational settings are discussed.

Keywords
Benzene, acute myeloid leukemia (AML), toluene, occupational exposure, hydrocarbon solvent

Introduction
Benzene, a well known cause of myelodysplastic syndrome (MDS) and acute myeloid leukemia (AML) (Baan et al., 2009; IARC, 1987; Smith, 2010), is present as a contaminant in many common petrochemicals, including toluene, mineral spirits, and naphtha (Kopstein, 2011). Recent studies indicate that use of hydrocarbon solvents with benzene content of <0.1% can create breathing zone benzene air concentrations that surpass occupational limits (Fedoruk et al., 2003; Kopstein, 2006) (Table 1). Similarly, occupational exposure via dermal absorption to mixtures containing <0.1% benzene may significantly increase cancer risk (Brenner et al., 1998; Kalnas and Teitelbaum, 2000; Petty et al., 2011).

We present seven cases of AML in workers with occupational exposure to a toluene-based hydrocarbon solvent in a factory that manufactured rubber belts and hoses. Case-specific information on a subset of former employees was retrieved from death certificates, medical records, and accounts from workers. The AML cases were employed at the rubber plant between 1950 and 2005 for periods ranging from 21 to 37 total years. Detailed occupational and medical histories were obtained for three of the cases who developed the disease between 2003 and 2005. The toluene-based solvent was used for cleaning and removing misprints on manufactured rubber items and for cleaning ink from printing presses and is the only solvent identified by former workers. No information regarding other solvents being used at the facility or data regarding the benzene content of the toluene solvent were available for this assessment. Employees specify that personal protective equipment (i.e. respirators, impervious clothing, or gloves) were rarely, if ever, utilized when working with the toluene solvent. The solvent was applied to a rag or brush and used without gloves, creating both inhalation and dermal exposures. There was no local exhaust ventilation or other engineering controls present in the workspace.

¹James Dahlgren Medical, Santa Monica, CA, USA
²Potomac, MD, USA

Corresponding author:
James Dahlgren, James Dahlgren Medical, 1158 26th Street, 118, Santa Monica, CA 90403, USA.
Email: dahlgren@envirotoxicology.com
### Case presentation

Case 1, a white male, worked at the facility from 1968 until 1994. In early 2003, he was diagnosed with pan-cytopenia. In December 2003 at the age of 72 years, a bone marrow biopsy showed AML with multilineage dysplasia. He had preexisting MDS. Cytogenetic analysis at this time revealed the presence of an abnormal hypodiploid clone characterized by loss of chromosomes X, 3, 5, 6, and 18, additional material of unknown origin on 4q, 10p, 12q, 13p, and 21p, an inverted duplication of 7p, a derivative chromosome resulting from deletions of both the long and short arms of chromosome 16, and a marker chromosome. In February 2004, a second cytogenetic analysis revealed the clonal expansion of an abnormal hypodiploid cell. The abnormal clone was the loss of chromosome 5, additional material of unknown origin on the short arm of chromosome 6, and the long arm of X chromosome. Nine cells were normal male. These findings are consistent with a clinical diagnosis of MDS or AML. He completed six cycles chemotherapy with idarubicin and ara-C. In July 2004, he relapsed and died from leukemia and congestive heart failure. He smoked cigarettes until 1988, but total pack-years could not be determined. His work history was provided by a coworker with a similar job. Case 1 transferred the toluene solvent into one-half gallon containers with spring-loaded tops and provided fresh solvent to six work stations as often as once per shift. He spent about 50% of his day sorting and cleaning the belts, which involved using the toluene solvent on a rag to wipe incorrectly printed or dirty belts. He used no personal protective equipment.

Case 2, a white female, worked at the facility from 1965 until 1995. She presented with thrombocytopenia, anemia, and leukocytosis and was diagnosed with AML in November 2005 at the age of 58 years. Molecular cytogenetic studies were negative for abnormal fusion of clones of the *PML* gene on 15q to the RARA gene on 17q. Specifically, there was no evidence of the t(15;17)(q22;q11.2) translocation in this specimen. Additional studies were also negative for the rearrangement of the mixed lineage leukemia region at 11q23. The bone marrow and peripheral blood showed >20% blasts. The percentage of myeloid lines that had >50% dysplasia was not specified, and classification using World Health Organization criteria was not possible. Case 2 was classified as AML-M4 under the French–American–British (FAB) Cooperative Group criteria due to having dysplastic monocytes. Chemotherapy with idarubicin and ara-C and eventually cytarabine was given inducing a remission. She relapsed May 2006 and died. She was a nonsmoker. She described her work activities at the plant shortly before her death. Similar to Case 1, she also worked in the prepack department. Her main task was printing and packing the rubber belts. She obtained containers of the toluene solvent from Case 1, who was the service man assigned to her workstation. She would dip brushes or rags into the solvent containers to clean the belts. When the belt printer malfunctioned, it had to be cleaned using large amounts of the solvent, a high exposure task. She rarely wore gloves, and the solvent made her fingertips raw, causing open sores on her hands.

Case 3, a white male, worked at the facility between 1984 and 2005. He was noted to have leukocytosis and malaise in May 2003. He continued working with a diagnosis of myeloproliferative disorder (MPD), subtype not otherwise specified. Some of his hematologists opined that he had MDS based on white blood cell count, which was >12,000, and <5% of monocytosis and blasts in the bone marrow. His treatment included hydroxyurea. His MPD/MDS transformed to AML with multilineage dysplasia in June 2005, at the age of 53 years. The only cytogenetic

### Table 1. Occupational exposure limits for benzene concentration in air.

<table>
<thead>
<tr>
<th>Agency</th>
<th>Description</th>
<th>Level (ppm)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>OSHA</td>
<td>Permissible exposure limit 8-hour TWA</td>
<td>1.0</td>
<td>OSHA, 1987</td>
</tr>
<tr>
<td></td>
<td>STEL</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>ACGIH</td>
<td>Threshold limit value TWA</td>
<td>0.5</td>
<td>ACGIH, 2006</td>
</tr>
<tr>
<td></td>
<td>STEL</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>NIOSH</td>
<td>Recommended exposure limit (10-hour TWA)</td>
<td>0.1</td>
<td>NIOSH, 1986, 2005</td>
</tr>
<tr>
<td></td>
<td>STEL</td>
<td>1.0</td>
<td></td>
</tr>
</tbody>
</table>

OSHA: Occupational Safety and Health Administration; ACGIH: American Conference of Governmental Industrial Hygienists; NIOSH: National Institute for Occupational Safety and Health; TWA: time weighted average; STEL: short time exposure limit; ppm: parts per million.
studies performed on this patient showed a normal male karyotype. Fluorescence in situ hybridization cytogenetic studies, as well as a PCR study, were negative for 9;22 translocation. No abnormal clones were detected at this band level. The patient started induction chemotherapy with idarubicin and received a bone marrow transplant in December 2005. He died in July 2006 from his leukemia while in hospice care. This man had a 5-pack/year smoking history, which ended before 1985. Case 3 worked many years in departments that built the rubber belts. Coworkers reported that this process involved using the toluene solvent in a similar manner as Cases 1 and 2, that is, cleaning misprinted or dirty belts. A fellow employee described the use of this solvent two or three times a day from small squirt bottle. Case 3 was responsible for filling his bottle from a container by pumping by hand.

Four other employees from this facility with probable exposure to the toluene-based solvent presented with AML diagnosis. Death certificates of these additional cases were reviewed, but other information is limited (Table 2). These cases were revealed during the course of discovery for civil litigation involving the three index cases. Two of the four have confirmed history of building or packaging belts, similar to index cases. These additional cases are presented due to the similar time frame of employment. No follow-up has been performed to our knowledge, and there may be other former employees with parallel diagnosis and exposure histories. All but one AML case were employed at the facility beginning in the 1950s or 1960s. Evidence was also obtained for 14 additional workers from this plant with other lymphohematopoietic cancers. Medical records, death certificates, and other information were collected from family and coworkers for the group of all 14 workers, which included reports of diagnoses of chronic lymphocytic leukemia (4 workers), non-Hodgkin’s lymphoma (8 workers), and multiple myeloma (2 workers). Only cases of AML confirmed by death records are reported herein.

### Discussion

Data collected on a subset of past employees from this rubber belt plant indicate seven AML deaths between 1994 and 2006. Lancaster County, Nebraska economic census data indicate that the plastics and rubber manufacturing industries (North American Industry Classification System code 326) employed between 1000 and 2499 workers in 2002 (US Census Bureau, 2002). This range also includes 500 and 999 employees from plastics manufacturing facilities, while the presented cases worked at the only facility in the county classified as rubber products manufacturing. At least seven deaths in a 12-year span at a facility of less than 2000 workers likely represent an elevation in AML mortality. The county population in 2000 was 250,291 (US Census Bureau, 2000). The US national age-adjusted mortality from AML in men and women of all races above the age of 50 years in 2005 is 8.57 per 100,000 population (NCI, 2011). It is notable that four cases expired between 2002 and 2006, two of which had worked in the same department. The Nebraska Cancer Registry reports 89 total leukemia deaths in the county during this period, and that AML accounted for approximately 27% of all leukemia cases in the state from 2003 to 2007 (Nebraska Cancer Registry, 2010). Using this percentage, the presented AML deaths from this facility would hypothetically represent 17% (4 of 24) of all AML mortality in the county between 2002 and 2006.

Cigarette smoking may have contributed to an increased risk of AML from benzene exposure within tobacco smoke in Cases 1 and 3; however, each of

### Table 2. AML in employees at rubber belt and hose plant.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Year of birth</th>
<th>Year of diagnosis</th>
<th>Year of death</th>
<th>Age at death</th>
<th>Years of employment</th>
<th>Employment length</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>F</td>
<td>AML</td>
<td>1922</td>
<td>–</td>
<td>2001</td>
<td>79</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

AML: acute myeloid leukemia; MDS: myelodysplastic syndrome; MPD: myeloproliferative disorder; M: male; F: female.
these cases stopped smoking over 15 years before their AML diagnoses. The cancer risk associated with cigarettes has been reported to diminish over time (Richardson et al., 2008). Smoking data was not available for the four auxiliary cases. Newly diagnosed AML patients have a median age of 65 years (Deschler and Lubbert, 2006). Cases 2 and 3 were under the age of 60 years at time of death, while the average age of the seven AML deaths was 66.4 years. There is no indication of other leukemia risk factors in this group, such as exposure to radiation, formaldehyde, or prior chemotherapy. We propose that decades of occupational exposure to benzene present in the toluene-based solvent used at the facility likely caused or contributed to the development of AML. Inference of benzene contamination in the toluene solvent is supported by the physicochemical properties of toluene, reports of usage of toluene and other hydrocarbons leading to benzene air concentrations above occupational limits, reports of benzene-related disease from toluene exposure, and toluene material safety data sheet (MSDS) reporting up to 5% benzene as recent as 2008 (Table 3).

The International Agency for Research on Cancer (IARC) classifies toluene as a group 3 carcinogen, indicating that the data are insufficient to implicate carcinogenicity to humans (IARC, 1989). Usage of organic solvents such as toluene in occupational settings often lead to overlapping exposures, as they contain multiple aromatic and aliphatic hydrocarbons as impurities. Reports from the 1940s describe toluene-exposed workers exhibiting typical benzene-related hematological effects (Greenburg et al., 1942; Wilson, 1943), which are now attributed to the presence of benzene as a contaminant (ATSDR, 2000). A recent study specifically investigated overlapping exposures to organic solvents and concluded that benzene present in aromatic solvent mixtures was likely to be responsible for the observed elevations in leukemia and lymphoma (Cocco et al., 2010). As early as 1977, a significant increase in total lymphohematopoietic and leukemia deaths was seen in a group of rubber workers exposed only to benzene (Infante et al., 1977; Rinsky et al., 1981). Subsequent research has confirmed the ability of benzene to cause malignant and nonmalignant hematopoietic disorders and suggests that increased risk occurs at levels markedly lower than first reported (Hayes et al., 1997, 2001). In particular, Glass et al. (2003) found, in a case–control study of the Health Watch cohort, that the risk of leukemia was increased at all cumulative exposures above 2 parts per million (ppm)/years with no evidence of a threshold cumulative exposure below which there was no risk (Glass et al., 2003). Prior to reliable epidemiological data, benzene had already been deemed as a leukemogen and potent hematotoxin due to an ample collection of case report series (Aksoy et al., 1974; Vigliani and Forni, 1976). We have included available chromosomal studies in the three index AML cases. There is currently an interest in collecting this data to guide therapy and estimate prognostic differences. A chromosomal pattern or fingerprint that is unique for benzene has been suggested (Gillis et al., 2007); however, available data do not support this claim (Smith, 2008).

### Benzene as a contaminant of toluene and other petrochemicals

American Society for Testing and Materials International (ASTM) has published specifications for the properties of nitration and industrial grades of toluene (ASTM, 1984, 2002). The maximum boiling range establishes the specified purity of both the grades of toluene, and the temperature at which a toluene/benzene mixture starts to boil corresponds to the equilibrium molar concentration of benzene at that temperature. Toluene’s boiling point is 110.6°C. For the nitration grade toluene, the boiling range must be ≤1°C, and for the industrial grade toluene, the boiling range is

### Table 3. Benzene content of toluene in MSDS issued after 2000.

<table>
<thead>
<tr>
<th>Product</th>
<th>Benzene content (ppm) by weight</th>
<th>Benzene content (% by weight)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Commercial grade toluene</td>
<td>20,000 (up to 50,000)</td>
<td>2–5%</td>
<td>Equistar Chemicals LLP, 2008a</td>
</tr>
<tr>
<td>Nitration grade toluene</td>
<td>20,000 (up to 50,000)</td>
<td>2–5%</td>
<td>Equistar Chemicals LLP, 2008b</td>
</tr>
<tr>
<td>Nitration toluene</td>
<td>Up to 10,000</td>
<td>Up to 1%</td>
<td>ExxonMobil Chemical, 2002</td>
</tr>
<tr>
<td>Nitration toluene</td>
<td>Up to 10,000</td>
<td>Up to 1%</td>
<td>ExxonMobil Chemical, 2003</td>
</tr>
<tr>
<td>Commercial grade toluene</td>
<td>Up to 25,000</td>
<td>Up to 2.5%</td>
<td>Chevron Phillips Chemical Company, 2003</td>
</tr>
</tbody>
</table>

MSDS: material safety data sheet; ppm: parts per million.
expanded to 2°C. Using a recirculation type vapor–liquid equilibrium apparatus at a temperature of 107.04°C, benzene comprised 8.1%, or 81,000 ppm, of the benzene/toluene mixture (Kesselman et al., 1968). Similar analysis reveal that toluene solvent meeting ASTM’s boiling point range specification for nitration and industrial grades may have between 22,900 and 44,800 ppm benzene on a volume basis, or approximately 2–4.5% (ASTM, 1984, 2002; Kopstein, 2011). Toluene not meeting ASTM specifications may have a wider boiling point range and much more benzene. Analytical difficulties in measuring lower levels of benzene in mixtures has been noted by Sheehan et al. (2010), Kopstein (2006, 2008), and OSHA (1980). MSDS issued after 2000 shows benzene content of toluene as high as 5% and comport well with the physical chemistry properties (Table 3).

Published scientific literature (Aksoy et al., 1987; Angerer, 1979; Baenfer, 1961; Browning, 1965; Kasahara et al., 1987; Mehlman, 2004; Novaes and Gruenzner, 1981) and other peer reviewed sources (ACGIH, 1991; EPA, 1994; NIOSH, 1973; WHO, 1985) discuss benzene contamination in petrochemical products, which is as high as 25% in toluene. Occupational health textbooks have warned that toluene may contain significant amounts of benzene (Levy, 1995; McCunney, 1994), and it has been recommended that workers exposed to toluene undergo hematological examinations due to benzene contamination (Sittig, 1985; Zenz, 1975). Conversely, publications emanating from members of the American Chemistry Council Hydrocarbon Solvents Panel—which is comprised entirely of CITGO Petroleum, ExxonMobil Chemical Company, Flint Hill Resources LP, Sasol N.A., and Shell Chemicals LP—have argued that benzene concentration in hydrocarbon solvents has been minimal subsequent to an unspecified date in the late 1970s, citing unpublished data (Caldwell et al., 2000; Jaques, 2006; McKee et al., 2007).

**Toluene and exposure to benzene**

Several reports document use of toluene products in occupational settings contributing to significant air benzene concentrations—even if benzene is absent from labeling or MSDS. In a German case report, a woman using an adhesive with a reported 27% toluene content developed aplastic anemia. Subsequent air measurements found the presence of 11 ppm toluene and 8 ppm benzene, although the latter was not included in the product’s label (Lachnit and Reimer, 1959). Analysis of a paint factory in Iran focused on the efficacy of exhaust ventilation systems to reduce the workers’ exposure to toluene and xylene. The authors reported benzene air concentrations averaging 31.98 ppm in the breathing zones of workers. Benzene was not reported as a component of the paint. The benzene air concentration was lowered to 4.5 ppm after the HVAC systems were activated, still many times higher than the US occupational exposure limit. These authors concluded that benzene air level monitoring was needed for solvents likely or possibly contaminated with benzene, even if benzene is not listed on the MSDS (Jafari et al., 2009).

A case series similar to the present report portrayed two workers diagnosed with chronic myelogenous leukemia and thrombocytopenia after exposure to toluene during employment in the printing trade. In addition to toluene, the workers reported using methyl ether ketone (MEK), which is not known to be carcinogenic. Exposures of workers were profiled using information provided in interviews and a review of literature and relevant reports. The author attributes the development of these diseases to benzene exposure arising from the contaminated toluene, and stressed the importance of the retrospective exposure profiles based on worker interviews and workplace descriptions in the absence of air monitoring data or access to the workplace (Kudla, 1997).

**Hydrocarbon solvents and dermal exposure to benzene**

American Conference of Governmental Industrial Hygienists (ACGIH) has issued a ‘skin notation’ to its recommendations on benzene exposure, indicating that dermal absorption of benzene can contribute significantly to the body burden in humans and air sampling alone is insufficient to accurately quantitate exposure (ACGIH, 2006). Repeated exposure to toluene solvents induces irritation and may injure the skin with cracks, lesions, and open sores and allow benzene to cross the skin barrier and enter the bloodstream (ATSDR, 2007; Blank and McAuliffe, 1985). Occupational Safety and Health Administration (OSHA) noted that ‘appreciable quantities of benzene could be absorbed in the case of injured skin. Moreover, absorption of benzene by the skin may be significantly accelerated when benzene is present as a mixture or as a contaminant in solvents known to be readily absorbed such as toluene and xylene’ (OSHA, 1977).
Brenner et al. report the case of a man who developed aplastic anemia after using mixture of acetone, MEK, and toluene during the cleaning and coating of pipes over a 30-year-period. The authors estimated that benzene was present in varying concentrations (0.3-4.5% from 1963 to 1978, 0.03-0.3% from 1978 to 1983, and 0.003-0.03% from 1983 to 1993). The mixture was consistently splashed on the workers' hands and forearms and occasionally other body parts. Dermal dose of benzene was estimated as equivalent to an inhalation exposure of 170 ppm/year (Brenner et al., 1998). Kalnas and Teitelbaum (2000) estimated that a worker who exposes his hands and lower forearms to cleaning solvent containing 0.1% benzene for 2 minutes every 15 minutes throughout the work day will absorb through the skin an amount of benzene equivalent to breathing air with 0.7 ppm benzene or a cumulative exposure of 28 ppm/year in a hypothetical 40-year period. This report also mentions several additional case reports of hematological effects after dermal exposures to liquids containing 0.1% or less benzene (Kalnas and Teitelbaum, 2000). A recent study using a more comprehensive approach obtained similar estimates as Kalnas and Teitelbaum (Petty et al., 2011).

Eight Finnish mechanics were at risk of benzene exposure while performing fuel system repairs and were subjected to air and blood sampling. The authors attributed >65% of total benzene exposure to the dermal route after blood benzene levels were higher than could be expected according to the corresponding time weighted averaged air measurements (Laitinen et al., 1994). Similarly, 60% of total benzene dose was credited to dermal absorption in a series of experiments monitoring showers in a home with benzene-contaminated water (Lindstrom et al., 1994).

**Hydrocarbon solvents and inhalation exposure to benzene**

A database for occupational exposure to hydrocarbon solvents was reported by Caldwell et al. (2000). The authors tabulated data on more than 700 published personal breathing zone exposures to benzene between 1978 and 1997, averaging 13.75 ppm (Caldwell et al., 2000). The authors characterize their findings as a ‘reality check,’ and report that many workplaces did not have proper engineering controls. This is highly relevant since the OSHA Benzene Standard stipulates that a combination of engineering controls and work practices are needed to reduce the 8-hour time weighted average work exposure to below 1 ppm (OSHA, 1987).

Fedoruk et al. investigated the benzene content and subsequent air concentrations associated with the usage of a mineral spirits-based degreaser formulated with severely hydrotreated mineral spirits. The assessment was designed to simulate parts by washing with severely hydrotreated mineral spirits in a degreaser station using a wet brush and sprayers. The simulated work environment did not have any engineering controls. The experiment measured a benzene air concentration of 0.55 ppm when testing a mixture containing 58 ppm benzene (0.0058%) (Fedoruk et al., 2003). Kopstein utilized Fedoruk’s experimental exposure data, employing thermodynamics and mass transfer principles to estimate exposures to benzene arising from products containing higher concentrations of benzene in the mineral spirit mixture (Table 4). Kopstein’s results apply to working conditions similar to the Fedoruk study (Kopstein, 2006). Both Fedoruk et al. and Kopstein conclude that products containing <0.1% benzene can potentially produce air benzene levels that exceed occupational exposure limits. Regular mineral spirits contains far more benzene than the severely hydrotreated mineral spirits used in the Fedoruk study (Hunting et al., 1995; IPCS, 1996; Kopstein, 2011). Using a near field/far field model, Nicas et al. (2006) reported benzene exposures comparable with those measured by the Fedoruk simulation.

Three studies sponsored by industry have found or modeled benzene levels to be much lower than measurements cited above. Sheehan et al. modeled benzene inhalation and dermal exposures arising from the use of solvents containing very-low concentrations of benzene, finding breathing zone benzene concentrations below detection limits (Sheehan et al.,

<table>
<thead>
<tr>
<th>Mean benzene content of petroleum-derived solvent (ppm, v/v)</th>
<th>Percentage benzene by volume</th>
<th>1 hour TWA benzene concentration (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>55.2</td>
<td>0.005%</td>
<td>0.5</td>
</tr>
<tr>
<td>110.4</td>
<td>0.011%</td>
<td>1.0</td>
</tr>
<tr>
<td>552</td>
<td>0.055%</td>
<td>5.0</td>
</tr>
<tr>
<td>1000</td>
<td>0.1%</td>
<td>9.1</td>
</tr>
<tr>
<td>5000</td>
<td>0.5%</td>
<td>45.3</td>
</tr>
</tbody>
</table>

Adapted from Taylor and Francis (Kopstein, 2006).
TWA: time weighted average; ppm: parts per million.
2010). Another modeling study estimated air benzene values using values from non peer reviewed literature for model inputs and predicted air levels would rarely exceed ACGIH or OSHA air levels (Williams et al., 2008). The third modeling study minimized exposures by creating a novel pattern of usage (e.g. complete evaporation or paper-thin solvent layers) assuring low-measured values (Paustenbach et al., 2010).

Conclusions
Case reports have been vital to the understanding of benzene toxicity, as well as other chemicals, and remain so today. Based on available information, the similar, long-term exposure to a toluene-based solvent experienced by the three primary AML cases and toluene’s propensity to contain benzene as an impurity suggest benzene as a probable causative factor for their development of this disease. Although the benzene concentration in widely used solvent mixtures varies, there is a reason to believe that even those containing amounts <0.1% have the potential to generate exposures capable of causing AML among industrial workers. Past research has focused on inhalation as the primary route of benzene exposure; however, dermal absorption is a critical route of exposure for certain occupations.

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Conflict of interest
JD and MK are sometimes retained as experts in civil cases regarding exposure to benzene.

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