IADC COMMITTEE NEWSLETTER

TOXIC AND HAZARDOUS SUBSTANCES LITIGATION

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IN THIS ISSUE

Michael Fox and Robert Kum of Duane Morris LLP, analyze IARC's recent re-classification of gasoline as a carcinogen.

An Analysis of IARC's Recent Re-Evaluation of Gasoline: More Questions Than Answers Raised

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On January 12, 2024, the International Agency for Research on Cancer ("IARC") announced that they were convening a panel of scientists to re-evaluate the carcinogenicity of automotive gasoline (as well as some oxygenated additives). This panel would meet in Lyon, France, from February 25 to March 4, 2025.

So why were they doing this? In 1988, IARC previously classified gasoline as a Class 2B carcinogen (possibly carcinogenic to humans), meaning there was some evidence that it can cause cancer in humans, but the evidence was far from conclusive. Were there new, groundbreaking studies since then that warranted this updated review? Unfortunately, IARC was not transparent in the reasons behind this review.

Cynics looking at this announcement believed that, as an organization that has become increasingly more political and a haven for plaintiff's experts, IARC had already made up their mind that they were going to conclude that gasoline was going to be bumped up to Class 1 known human carcinogen status. Waiting a year before the formal announcement was just window dressing to give their review an air of credibility. After all, the epidemiological studies concerning gasoline exposure were consistent that exposure to gasoline does not increase the risk of cancer.

Supporters of IARC insisted a thorough review of the scientific evidence would be conducted and any new findings would reflect such a methodology.

On March 21, 2025, the working group of 20 scientists revealed their findings in *The Lancet Oncology*.[1] The cynics' fears were verified. IARC did indeed re-classify gasoline as a Class 1 known human carcinogen, causing specifically acute myelogenous leukemia ("AML") and bladder cancer. This determination contradicts the findings of various governmental agencies including the Environmental Protection Agency.[2]

What was the basis of this conclusion? IARC claims to have examined studies involving both humans and experimental animals, as well as underlying mechanistic evidence. As for human data, IARC notes that it reviewed occupational epidemiology studies involving service station attendants and gasoline distribution workers that assessed gasoline exposure and found them supportive of their conclusions. What is troubling about their purported reliance is that the papers cited in their *Lancet* article do not support such a conclusion. For example, in support of a causal connection between gasoline and

[1] Turner MC, Godderis L, Guénel P, Hopf N, Quintanilla-Vega B, Soares-Lima SC, et al. Carcinogenicity of automotive gasoline and some oxygenated gasoline additives, Lancet Oncol. Published online 21 March 2025.

[2] U.S. EPA. Evaluation of the Carcinogenicity of Unleaded Gasoline. U.S. Environmental Protection Agency, Washington, D.C., EPA/600/6-87/001.

AML, they cite a 2007 mortality study involving thousands of refinery and petroleum workers.[3] The conclusion section however states: "The only findings that showed clear evidence of an occupational cancer hazard were those for mesothelioma in oil refinery workers." Moreover, the underlying data indicated that there was "no clear-cut excess of AML (or the slightly larger grouping of ANLL) … ." Nowhere in this paper do they claim that gasoline or working in or around gasoline causes AML.

How IARC could rely on this to support their conclusion is unknown. It is possible that IARC is not following traditional notions of statistical significance for standard mortality rates ("SMR") or 95% confidence intervals. To do this though would be an abandonment of common scientific principles that are generally used in analyzing such studies.

The lack of a causal connection found in this 2007 paper is consistent with other large-scale studies looking at refinery workers, including those involved in transporting or distribution.[4]

Studies involving service station or mechanics also consistently show that they are not at an increased risk of AML.[5] Therefore, it is unknown what studies upon which IARC is relying for the claims that these studies support their findings.

It is also important to note that they found that there was only "limited" evidence that gasoline causes myelodysplastic syndrome ("MDS"). Such a finding contradicts their position that they found mechanistic data sufficient to show that gasoline causes AML.

There is no dispute that MDS and AML are similar hematological diseases along the same continuum. As a result, treatment options are also similar for MDS and AML patients (e.g., chemotherapy, stem cell transplant, etc.). A normal blast cell count in humans is 5% or less. When a blast cell count is within the 5% to 20% range, this is indicative of MDS. AML is generally defined as a 20% or greater blast cell count in the bone marrow. It is this reason that it is common for MDS to progress to AML, a fact IARC concedes. If gasoline does indeed cause AML, then mechanistically, they should have

[3] Sorahan T. Mortality of UK oil refinery and petroleum distribution workers, 1951–2003. Occup Med 2007; 57: 177–85.

[4] O. Wong, L. Trent, F. Harris, Nested case-control study of leukemia, multiple myeloma, and kidney cancer in a cohort of petroleum workers exposed to gasoline, Occup Environ Med, 56 (1999) 217-221; O. Wong, F. Harris, T.J. Smith, Health effects of gasoline exposure. II. Mortality patterns of distribution workers in the United States, Environ. Health Perspect, 101 Suppl 6 (1993) 63-76.

[5] D.P. Loomis, D.A. Savitz, Occupation and leukemia mortality among men in 16 states: 1985-1987, Am. J. Ind. Med, 19 (1991) 509-521; J.N. Poynter, M. Richardson, M. Roesler, C.K. Blair, B. Hirsch, P. Nguyen, A. Cioc, J.R. Cerhan, E. Warlick, Chemical exposures and risk of acute myeloid leukemia and myelodysplastic syndromes in a population-based study, Int J Cancer, 140 (2017) 23-33; C.F. Robinson, J.T. Walker, M.H. Sweeney, R. Shen, G.M. Calvert, P.K. Schumacher, J. Ju, S. Nowlin, Overview of the National Occupational Mortality Surveillance (NOMS) system: leukemia and acute myocardial infarction risk by industry and occupation in 30 US states 1985-1999, 2003-2004, and 2007, Am. J. Ind. Med, 58 (2015) 123-137.

similar findings for MDS, which they did not find.

The methodology IARC employed to determine gasoline causes bladder also appears flawed, and their apparent dismissal of smoking as a confounding factor (i.e. a variable that may mask an actual association or influence both the supposed cause and effect). Smoking is not just a confounding factor but *the confounding factor* that would influence the results of a bladder cancer analysis.

Cigarette smoking causes approximately 50% of all bladder cancers in the U.S.[6] Persons who smoke are at a fourfold excess risk of developing bladder cancer. Therefore, it can be assumed that cigarette smoke is responsible for at least half of the bladder cancers being examined in any study.

IARC attempts to argue that any "[c]oncerns about confounding by smoking or engine exhausts were allayed because lung cancer incidence or mortality was generally not elevated in the available studies." This reasoning dismissed smoking as a confounding factor because the persons in the studies they relied on did not have both lung and bladder cancer. The incidence of persons in the U.S. that have both lung and bladder cancers are rare, and there are no well-defined statistics as to how many of these persons exist. In addition, it is likely that the studies they relied upon failed to determine if these same populations had other non-cancerous smoking related diseases such COPD, emphysema and coronary heart disease. Thus, if a person had both bladder cancer and COPD (of which the number one cause is smoking), it is likely their bladder disease is smoking related. If you flip the use of this methodology on its head and state that you can only prove that smoking causes bladder cancer if you also determine they did not also have lung cancer, that will of course likely lead to an erroneous conclusion.

While there have been a few nuclear verdicts involving alleged gasoline exposure, the plaintiff's bar has, for the most part, not filed these types of cases in mass quantities. Historically, this was primarily due to the fact that general causation was difficult to prove. However, now armed with IARC's new classification, we are likely to see not only more cases concerning persons who are exposed to gasoline in an occupational setting but potentially consumer-based lawsuits from individuals who simply fueled their vehicles.

The formal monograph will provide more specific detail as to all the studies IARC reviewed in support of its finding. Such monographs are likely not to be published until the later part of 2025 or early part of 2026. Given what information they provided to date in their recent disclosure, we would bet on the cynics who say that any insight into their underlying methodology will raise more questions than answer.

[6] https://www.nih.gov/news-events/nih-research-matters/smoking-bladder-cancer

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