

Annals of Leukemia Research

Review Article

Occupational and Environmental Benzene Exposure and Risk of Adult Acute Myeloid Leukemia

Balazs Farkas^{1*}, Vesela Kolev² and Eleni Stavrou³

¹Department of Clinical Immunology, Semmelweis University, Budapest, Hungary

²Department of Haematology, Medical University of Plovdiv, Plovdiv, Bulgaria

³Department of Bioinformatics, Aristotle University of Thessaloniki, Thessaloniki, Greece

*Address for Correspondence: Balazs Farkas, Department of Clinical Immunology, Semmelweis University, Budapest, Hungary

Received: 11 February 2026; Accepted: 30 April 2026; Published: 05 August 2026

Citation of this article: Farkas, B., Kolev, V., Stavrou, E. (2026) Occupational and Environmental Benzene Exposure and Risk of Adult Acute Myeloid Leukemia. Ann Leuk Res, 7(1): 04-06. doi: 10.5281/zenodo.22247821

Copyright: © 2026 Balazs Farkas, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Summary

That benzene causes acute myeloid leukaemia is settled. What is not settled is the shape of the curve at low exposure, and that shape determines every occupational limit set from it. Applying two different historical exposure reconstructions to the same cohort of workers produces a linear relationship under one and a quadratic one under the other.

Objectives: To review the published evidence on benzene exposure and adult acute myeloid leukaemia: what risk is reported at high and low cumulative exposure, how the dose-response relationship has been modelled, why estimates differ between cohorts, and what follows for exposure limits.

Material and Methods: Narrative review of occupational cohort studies, case-cohort analyses, meta-analyses, risk assessments and agency evaluations indexed in PubMed and the major occupational health and epidemiology journals. Studies were eligible if they reported the association between quantified benzene exposure and myeloid malignancy in a defined adult population. No new patient data were generated and no participants were recruited; every estimate quoted is one published by the original investigators.

Results: In a case-cohort analysis following 110,631 Chinese workers between 1972 and 1999, risk of myelodysplastic syndrome or acute myeloid leukaemia rose with cumulative exposure, with an excess relative risk per 100 parts per million-years of 0.30 (95% CI -0.02 to 1.09, trend $p = 0.08$), corresponding to a rate ratio of 1.30. Pooled analysis gave an odds ratio of 1.94 (95% CI 0.95 to 3.95) for cumulative exposure below 40 parts per million-years. Among petroleum distribution workers, myelodysplastic syndrome risk was elevated above 2.93 against below 0.348 parts per million-years, at an odds ratio of 4.33 (95% CI 1.31 to 14.3), an exposure range more than ten times lower than the pooled analysis low category. Significant dose-response for acute myeloid leukaemia has been reported from cumulative exposures just below 1 part per million-year. In the historically pivotal cohort, one exposure reconstruction predicted a linear dose-response and another a quadratic one, with the departure from linearity borderline at $p = 0.08$.

Conclusion: The causal relationship is not in question and the quantitative basis for regulation is weaker than its use implies. Effects are being detected at exposures well below limits derived from cohorts whose lowest category exceeds them by an order of magnitude.

Abbreviations: AML – Acute Myeloid Leukaemia, MDS – Myelodysplastic Syndrome, AMML – Acute Myelomonocytic Leukaemia, ANLL – Acute Non-Lymphocytic Leukaemia, ppm – Parts Per Million, ERR – Excess Relative Risk, RR – Rate Ratio, OR – Odds Ratio, OEL – Occupational Exposure Limit, TWA – Time-Weighted Average, TLV – Threshold Limit Value, LMS – Linearized Multistage, IARC – International Agency for Research on Cancer.

Keywords: Acute Myeloid Leukaemia; Benzene; Occupational Exposure; Dose-Response; Exposure Limits

Introduction

Benzene is among the most thoroughly studied human carcinogens, classified by the International Agency for Research on Cancer as causing acute myeloid leukaemia in humans [1,2]. The evidence derives principally from occupational epidemiology, and international agreement supports the causal relationship even where debate continues over lymphoid neoplasms [3,4].

Exposure is not confined to industry. Benzene occurs in petrol, exhaust fumes and tobacco smoke, so the general population is exposed at low concentrations continuously [5,6]. Workplace limits have been reduced repeatedly over the past three decades, and the eight-hour

time-weighted average limit stands at 1 part per million in several jurisdictions, with one advisory body lowering its threshold value to 0.02 parts per million [5,7].

Those limits are derived quantitatively from cohort data by extrapolating a dose-response relationship down from the high exposures historically experienced [8,9]. The extrapolation depends on the assumed shape of the curve, and that shape is where the epidemiology becomes contested rather than settled [10,11].

This review sets out what risk is reported at high and low exposure, how the relationship has been modelled, why cohorts disagree, and what follows for the limits derived from them.

Material and Methods

Search strategy: PubMed and the archives of the major occupational health, epidemiology and haematology journals were searched for studies of benzene and myeloid malignancy. Search terms combined benzene with acute myeloid leukaemia, myelodysplastic syndrome, occupational exposure, cumulative exposure, parts per million-years, dose-response and exposure limits. Reference lists of retrieved cohort analyses and agency evaluations were screened by hand.

Eligibility: Studies were included if they reported the association between quantified benzene exposure and myeloid malignancy in a defined adult population, or modelled that relationship for risk assessment. Studies of haematological effects short of malignancy are cited where they bear on the plausibility of low-dose effects and are identified as such.

Data extraction and handling: Cohort, follow-up period, exposure assessment method, exposure metric, lag applied and reported effect estimates were extracted as published. The exposure range covered is stated alongside every estimate, because the central finding of this review is that the word low denotes ranges differing by more than an order of magnitude between literatures. No pooling or re-analysis was performed and no participant-level data were obtained. Figures 1 to 4 and Tables 1 to 3 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

The largest prospective evidence: A multicentre case-cohort study followed 110,631 Chinese workers between 1972 and 1999, estimating exposures by hierarchical modelling of occupational factors calibrated against historical routine measurements [3,12]. For the exposure window of two to under ten years, risk of myelodysplastic syndrome or acute myeloid leukaemia rose with cumulative exposure, giving an excess relative risk per 100 parts per million-years of 0.30 (95% CI -0.02 to 1.09, trend $p = 0.08$) and a corresponding rate ratio of 1.30 (Figure 1) [12].

Cumulative exposure and average intensity were highly correlated at 0.94 in that window, so the two metrics could not be separated and the cumulative results were reported [12]. Across the studies compared, a positive exposure response was observed for all leukaemias or acute myeloid leukaemia, with risks highest at the highest exposures [12].

Results below 400 parts per million-years were generally compatible across studies, given wide confidence intervals reflecting small numbers of leukaemia cases in most cohorts [12]. Above that level, risks in the historically pivotal rubber hydrochloride cohort exceeded those in the Chinese study, probably reflecting higher exposures within that category [12,13].

Earlier analysis of the same Chinese population reported cause-specific mortality and incidence of haematopoietic malignancy across a retrospective cohort design [12], and the original dose-related incidence analysis established the exposure-response that later work refined [20]. Exposure assessment in that cohort has itself been described in detail, which is unusual and valuable [21].

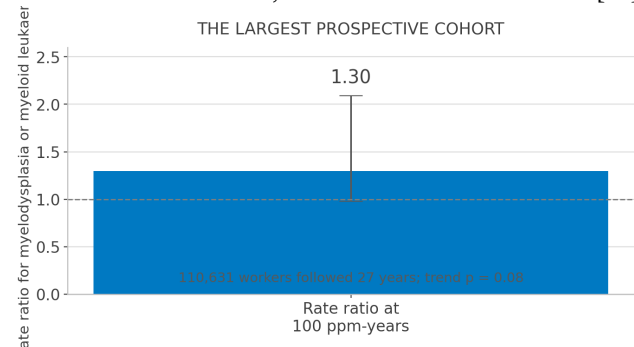


Figure 1: Rate ratio at 100 parts per million-years in a cohort of 110,631 workers [12]

What is reported at lower exposure: A systematic review and meta-analysis synthesising the evidence by cumulative exposure category reported a pooled odds ratio of 1.94 (95% CI 0.95 to 3.95) for exposures below 40 parts per million-years (Figure 2) [14]. The interval crosses unity, so the pooled low-exposure estimate is compatible with no effect as well as with a near doubling.

Studies conducted at genuinely low exposure report larger effects. Among petroleum distribution workers, myelodysplastic syndrome risk was elevated in those exposed above 2.93 parts per million-years compared with below 0.348, at an odds ratio of 4.33 (95% CI 1.31 to 14.3) [15,16]. A significant dose response for acute myeloid leukaemia has been reported from cumulative exposures just below 1 part per million-year [17], and an elevenfold risk from exposures well below that level [16,18].

Effects short of malignancy appear lower still. Total white cell and platelet counts were significantly reduced in workers exposed at 1 part per million compared with unexposed controls [16,19], and increased aneuploidy has been reported in germ cells at the same

concentration [16].

Whether these lower-exposure findings should carry equal weight is contested. They rest on few cases and wide intervals, and the populations differ from the historical cohorts in co-exposures and in smoking prevalence [14,15]. They are nonetheless the only evidence generated at concentrations workers actually encounter today [5,7].

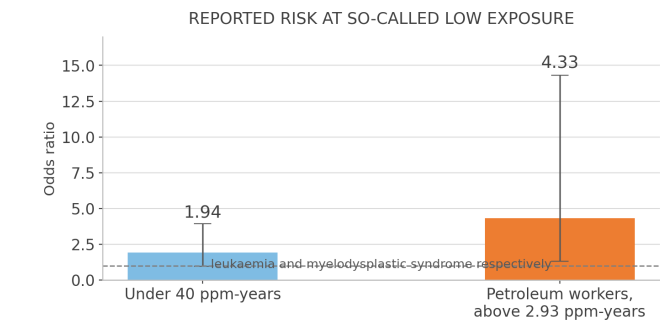


Figure 2: Reported risk at low cumulative exposure across two literatures [14,15]

Study	Population	Exposure range	Principal reported finding
Crump 1994 [10]	Rubber hydrochloride	High	Curve shape depends on exposure estimates used
Hayes et al. 1997 [20]	Chinese workers	Wide	Benzene and the dose-related incidence of leukaemia
Khalade et al. 2010 [14]	Pooled	Under 40 ppm-years	Pooled odds ratio 1.94, interval crossing unity
Schnatter et al. 2013 [15]	Petroleum distribution	Under 3 ppm-years	Myelodysplasia elevated at very low exposure
Stenehjem et al. 2015 [17]	Offshore petroleum	Under 1 ppm-year	Significant dose response below 1 ppm-year
Linnet et al. 2019 [12]	110,631 workers	Wide	Rate ratio 1.30 at 100 ppm-years

Table 1: Principal studies reviewed, with exposure range and the finding as reported by the original investigators

What low means: The comparison above is not like for like, and the mismatch is the point. The pooled analysis defines its lowest category as under 40 parts per million-years [14]. The petroleum worker study divides its entire cohort into tertiles at 0.348 and 2.93 parts per million-years, so its highest exposure group sits more than tenfold below the pooled analysis's lowest (Figure 3) [15].

An effect detected in the second study occurs at exposures that the first would classify as effectively zero. Investigators have noted this

explicitly, observing that their tertiles are much lower than the low cumulative exposure category used in pooled work and than those in the historically pivotal cohorts [15].

This matters directly for regulation. Occupational limits set at around 1 part per million as an eight-hour average [5,7] correspond, over a working lifetime, to cumulative exposures in the range where effects are now being reported rather than the range from which the limits were extrapolated [16,17,18].

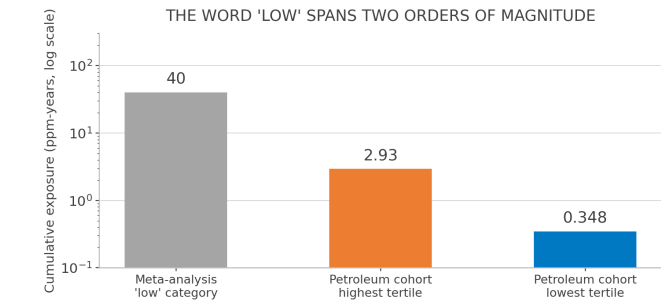


Figure 3: Exposure ranges denoted by the word low in different literatures, logarithmic scale [14,15]

The curve shape is not determined by the data: The historically pivotal cohort showed a significant excess of acute myelocytic or monocytic leukaemia, with 8 to 10 cases observed against 1.61 expected and a strong dose-response trend [10,13]. Quantitative risk estimates proved robust to whether the myelomonocytic endpoint or all leukaemia was modelled, and to which of two exposure reconstructions was used, differing by no more than twofold, provided a linear model was applied [10].

The qualification is decisive. One set of exposure estimates predicted a linear dose response and the other predicted a quadratic one, with the departure from linearity borderline significant at $p = 0.08$ (Figure 4) [10]. Same workers, same leukaemias, different reconstruction of what they breathed decades earlier, different curve.

A linear curve implies risk at any exposure and no safe threshold; a quadratic one implies risk falling away steeply at low doses and a defensible limit. The two lead to different regulatory conclusions from identical outcome data, and the choice between them rests on retrospective exposure estimation rather than on the leukaemias observed [8,10,11].

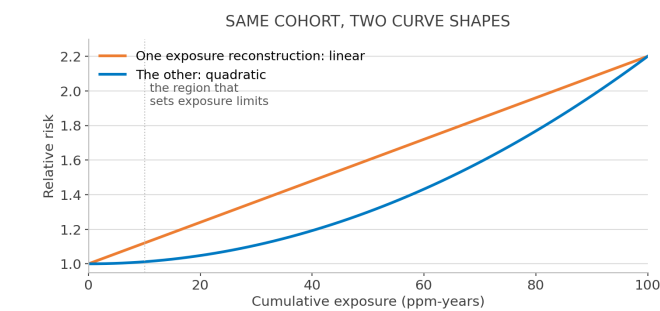


Figure 4: Dose-response shapes derived from the same cohort under two exposure reconstructions [10]

Question	Status	Source
Does benzene cause myeloid leukaemia?	Settled	Agency evaluation [1,2]
Is there a dose-response?	Yes, at high exposure	Multiple cohorts [12,20]
What is the curve shape at low dose?	Contested	Depends on reconstruction [10,11]
Is there a safe threshold?	Not established	Effects reported below 1 ppm-year [17,18]
Do lymphoid neoplasms occur?	Debated	No exposure-response for lymphoma [3]

Table 2: What is settled and what remains contested

How benzene causes the disease: The mechanism supports rather than settles the low-dose question. Benzene metabolites damage haematopoietic progenitors through oxidative injury, topoisomerase inhibition and disruption of chromosome segregation, producing the clastogenic effects characteristic of the exposure [27,28]. Chromosome-wide analysis in exposed workers demonstrated aneuploidy across the genome rather than at isolated loci [29].

Susceptibility varies between individuals. Polymorphism in the gene encoding NAD(P)H quinone oxidoreductase, which detoxifies benzene metabolites, associates with haematological toxicity in exposed workers [30], so a population-average dose-response necessarily averages across people at different risk from the same air concentration.

Where the injury produces a clone that persists, the resulting disease follows the pathway established for secondary myeloid neoplasms generally [31], and would be classified and managed under current criteria for myeloid malignancy [32,33]. Benzene sits alongside prior chemotherapy and radiation among the established environmental causes of an otherwise largely idiopathic disease [34]. **Other cohorts and other endpoints:** Refinery and petrochemical cohorts have been followed for mortality over several decades [25], and hospital-based case-control work has examined personal, lifestyle and environmental risk factors by disease subtype [24].

Analyses of B-lymphocyte malignancy in industrial cohorts address the contested lymphoid question separately [26], where no exposure-response has been demonstrated despite raised counts in exposed workers [3,4].

Evidence type	Strength	Principal weakness
High-exposure cohorts	Consistent dose-response	Exposures no longer encountered [12,13]
Pooled low-exposure estimate	Combines small studies	Interval crosses unity [14]
Contemporary low-exposure studies	Relevant to current limits	Few cases, wide intervals [15,17]
Mechanistic and biomarker work	Effects at 1 ppm demonstrated	Not malignancy endpoints [19,29]

Table 3: Categories of evidence and the limitation of each for setting exposure limits

Discussion

Two things are true simultaneously and are frequently confused. Benzene causes acute myeloid leukaemia, and the evidence for that is as strong as occupational epidemiology produces [1,2,20]. The quantitative relationship used to set exposure limits is considerably less secure than its regulatory application suggests [8,10,11].

The insecurity is specific rather than general. At high cumulative exposure the cohorts broadly agree [12,13]. Below roughly 40 parts per million-years the confidence intervals widen enough to include no effect [14], and the extrapolation into that region is where limits are actually derived.

Why the reconstruction matters more than the outcome data

Historical exposure in these cohorts was not measured contemporaneously for most workers. It has been reconstructed decades later from job histories, process descriptions and sparse industrial hygiene samples [10,12,21]. Two competent reconstructions of the same cohort yielded linear and quadratic dose-responses respectively [10].

The outcome data did not change between those analyses; only the assumption about what workers breathed. That places the weight of a regulatory decision on the least well-measured element of the study, and it is why later work has invested heavily in hierarchical exposure modelling calibrated against routine measurements [12,21].

The evidence at contemporary exposure levels

Modern occupational exposures are far below those in the historical cohorts, so the practical question is what happens in a range those cohorts barely covered [5,15]. Studies conducted at that range report effects: myelodysplasia at an odds ratio of 4.33 above 2.93 parts per million-years [15], dose-response for acute myeloid leukaemia below 1 part per million-year [17], and haematological changes at 1

part per million [16,19].

These findings are individually imprecise, with wide intervals reflecting small case numbers [14,15]. They are nonetheless consistent in direction, and they arise in exactly the range that limits were extrapolated into rather than derived from, which is the situation in which extrapolation is least reliable [8,11].

Study limitations

The limitations belong to the underlying literature. Historical exposure was reconstructed rather than measured for most workers in the formative cohorts, and different reconstructions produce different dose-response shapes from identical outcomes [10,21]. Leukaemia case numbers are small in most cohorts, producing wide confidence intervals that limit what low-exposure categories can establish [12,14]. Diagnostic classification changed across the periods covered, and some cases originally recorded as acute myeloid leukaemia were reclassified as myelodysplasia or myeloproliferative disease on review [15,22]. Co-exposure to other solvents is common in the industries studied and is variably adjusted for [15,20]. And smoking is both a benzene source and a leukaemia risk factor, which confounds occupational estimates in cohorts where it is incompletely recorded [6,14].

Two gaps deserve naming. No study reviewed here resolves the curve shape at low exposure, and the question cannot be settled by enlarging cohorts whose exposures were reconstructed by the disputed methods [10,12]. And no analysis reviewed reports myeloid malignancy risk against biological exposure markers measured prospectively, which would remove the reconstruction problem entirely [16,23].

Conclusion

Benzene causes acute myeloid leukaemia, and cohorts of exposed workers agree at high cumulative exposure, with a rate ratio of 1.30 at 100 parts per million-years in the largest prospective study. Below about 40 parts per million-years the pooled estimate is 1.94 with an interval crossing unity, while studies conducted at genuinely low exposure report myelodysplasia at an odds ratio of 4.33 above 2.93 parts per million-years and dose-response for acute myeloid leukaemia below 1 part per million-year, ranges that pooled work classifies as effectively unexposed. The curve shape at low dose, which determines every limit extrapolated from these data, is not fixed by the outcomes: applying two historical exposure reconstructions to the same cohort produced a linear relationship under one and a quadratic one under the other, with the departure from linearity borderline at $p = 0.08$. The regulatory question turns on the least well-measured element of the evidence.

Acknowledgements

The authors thank the occupational cohorts and their investigators whose published analyses form the substance of this article.

Funding

No dedicated funding from public, commercial or charitable sources supported this review.

Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

References

1. International Agency for Research on Cancer. (2012) Chemical agents and related occupations. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100F. Lyon: IARC.
2. International Agency for Research on Cancer. (2018) Benzene. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 120. Lyon: IARC.
3. Linet, MS., Gilbert, ES., Vermeulen, R., Dores, GM., Yin, SN., Portengen, L., et al. (2019) Benzene exposure response and risk of myeloid neoplasms in Chinese workers: a multicenter case-cohort study. *J Natl Cancer Inst*, 111(5): 465-474.
4. Vlaanderen, J., Lan, Q., Kromhout, H., Rothman, N., Vermeulen, R. (2011) Occupational benzene exposure and the risk of lymphoma subtypes: a meta-analysis of cohort studies incorporating three study quality dimensions. *Environ Health Perspect*, 119(2): 159-167.
5. Capleton, AC., Levy, LS. (2005) An overview of occupational benzene exposures and occupational exposure limits in Europe and North America. *Chem Biol Interact*, 153-154: 43-53.
6. Fiebelkorn, S., Meredith, C. (2018) Estimation of the leukemia risk in human populations exposed to benzene from tobacco smoke using epidemiological data. *Risk Anal*, 38(7): 1490-1501.
7. American Conference of Governmental Industrial Hygienists. (2020) Threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati: ACGIH.
8. Rinsky, RA., Smith, AB., Hornung, R., Filloon, TG., Young, RJ., Okun, AH., Landrigan, PJ. (1987) Benzene and leukemia: an epidemiologic risk assessment. *N Engl J Med*, 316(17): 1044-1050.
9. Paustenbach, DJ., Bass, RD., Price, P. (1993) Benzene toxicity and risk assessment, 1972-1992: implications for future regulation. *Environ Health Perspect*, 101(Suppl 6): 177-200.
10. Crump, KS. (1994) Risk of benzene-induced leukemia: a sensitivity analysis of the pliofilm cohort with additional follow-up and new exposure estimates. *J Toxicol Environ Health*, 42(2): 219-242.

11. Crump, KS., Allen, BC. (1984) Quantitative estimates of the risk of leukemia from occupational exposure to benzene. Washington: Occupational Safety and Health Administration.
12. Linet, MS., Yin, SN., Gilbert, ES., Dores, GM., Hayes, RB., Vermeulen, R., et al. (2015) A retrospective cohort study of cause-specific mortality and incidence of hematopoietic malignancies in Chinese benzene-exposed workers. *Int J Cancer*, 137(9): 2184-2197.
13. Rinsky, RA., Hornung, RW., Silver, SR., Tseng, CY. (2002) Benzene exposure and hematopoietic mortality: a long-term epidemiologic risk assessment. *Am J Ind Med*, 42(6): 474-480.
14. Khalade, A., Jaakkola, MS., Pukkala, E., Jaakkola, JJ. (2010) Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis. *Environ Health*, 9: 31.
15. Schnatter, AR., Glass, DC., Tang, G., Irons, RD., Rushton, L. (2013) Acute myeloid and chronic lymphoid leukaemias and exposure to low-level benzene among petroleum workers. *Br J Cancer*, 109(6): 1783-1789.
16. Zhang, Y., Ding, X., Chen, Y., Wang, J., Yang, L., Wang, H., et al. (2024) Leukemia risk assessment of exposure to low-levels of benzene based on the linearized multistage model. *Front Public Health*, 12: 1383654.
17. Stenehjem, JS., Kjaerheim, K., Bratveit, M., Samuelsen, SO., Barone-Adesi, F., Rothman, N., et al. (2015) Benzene exposure and risk of lymphohaematopoietic cancers in 25 000 offshore oil industry workers. *Br J Cancer*, 112(9): 1603-1612.
18. Talbott, EO., Xu, X., Youk, AO., Rager, JR., Stragand, JA., Malek, AM. (2011) Risk of leukemia as a result of community exposure to gasoline vapors: a follow-up study. *Environ Res*, 111(4): 597-602.
19. Lan, Q., Zhang, L., Li, G., Vermeulen, R., Weinberg, RS., Dosemeci, M., et al. (2004) Hematotoxicity in workers exposed to low levels of benzene. *Science*, 306(5702): 1774-1776.
20. Hayes, RB., Yin, SN., Dosemeci, M., Li, GL., Wacholder, S., Travis, LB., et al. (1997) Benzene and the dose-related incidence of hematologic neoplasms in China. *J Natl Cancer Inst*, 89(14): 1065-1071.
21. Dosemeci, M., Li, GL., Hayes, RB., Yin, SN., Linet, M., Chow, WH., et al. (1994) Cohort study among workers exposed to benzene in China: exposure assessment. *Am J Ind Med*, 26(3): 401-411.
22. Bain, BJ. (1999) The relationship between the myelodysplastic syndromes and the myeloproliferative disorders. *Leuk Lymphoma*, 34(5-6): 443-449.
23. Boogaard, PJ., van Sittert, NJ. (1995) Biological monitoring of exposure to benzene: a comparison between S-phenylmercapturic acid, trans,trans-muconic acid, and phenol. *Occup Environ Med*, 52(9): 611-620.
24. Wong, O., Harris, F., Yiyang, W., Hua, F. (2010) A hospital-based case-control study of acute myeloid leukemia in Shanghai: analysis of personal characteristics, lifestyle and environmental risk factors by subtypes of the WHO classification. *Regul Toxicol Pharmacol*, 55(3): 340-352.
25. Huebner, WW., Wojcik, NC., Rosamilia, K., Jorgensen, G., Milano, CA. (2004) Mortality updates (1970-1997) of two refinery/petrochemical plant cohorts at Baton Rouge, Louisiana, and Baytown, Texas. *J Occup Environ Med*, 46(12): 1229-1245.
26. Bukowski, JA., Huebner, WW., Schnatter, AR., Wojcik, NC. (2003) An analysis of the risk of B-lymphocyte malignancies in industrial cohorts. *J Toxicol Environ Health A*, 66(7): 581-597.
27. Smith, MT. (2010) Advances in understanding benzene health effects and susceptibility. *Annu Rev Public Health*, 31: 133-148.
28. McHale, CM., Zhang, L., Smith, MT. (2012) Current understanding of the mechanism of benzene-induced leukemia in humans: implications for risk assessment. *Carcinogenesis*, 33(2): 240-252.
29. Zhang, L., Lan, Q., Guo, W., Hubbard, AE., Li, G., Rappaport, SM., et al. (2011) Chromosome-wide aneuploidy study (CWAS) in workers exposed to an established leukemogen, benzene. *Carcinogenesis*, 32(4): 605-612.
30. Rothman, N., Smith, MT., Hayes, RB., Traver, RD., Hoener, B., Campleman, S., et al. (1997) Benzene poisoning, a risk factor for hematological malignancy, is associated with the NQO1 609C to T mutation and rapid fractional excretion of chlorzoxazone. *Cancer Res*, 57(14): 2839-2842.
31. Sperling, AS., Gibson, CJ., Ebert, BL. (2017) The genetics of myelodysplastic syndrome: from clonal haematopoiesis to secondary leukaemia. *Nat Rev Cancer*, 17(1): 5-19.
32. Döhner, H., Wei, AH., Appelbaum, FR., Craddock, C., DiNardo, CD., Dombret, H., et al. (2022) Diagnosis and management of AML in adults: 2022 recommendations from an international expert panel on behalf of the ELN. *Blood*, 140(12): 1345-1377.
33. Kantarjian, H., Kadia, T., DiNardo, C., Daver, N., Borthakur, G., Jabbour, E., et al. (2021) Acute myeloid leukemia: current progress and future directions. *Blood Cancer J*, 11: 41.
34. Deschler, B., Lübbert, M. (2006) Acute myeloid leukemia: epidemiology and etiology. *Cancer*, 107(9): 2099-2107.