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1,1,1-TRICHLOROETHANE AND FOUR OTHER INDUSTRIAL CHEMICALS

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This publication represents the views and expert opinions of an IARC Working Group on the Identification of Carcinogenic Hazards to Humans, which met remotely, 7–22 October 2021

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International Agency for Research on Cancer



Table S1.5 Exposure assessment review and critique for mechanistic studies in humans exposed to 1,1,1-trichloroethane

Reference and mechanistic end- point	What was the study design? (n subjects)	What methods were used for the exposure assessment? (incl. data source, environmental and biological measurements etc.)	Was the exposure defined well, and what was the definition?	Was exposure assessment qualitative, semiquantitative or quantitative?	Were sampling and collection protocols for chemical measurements appropriate?	What routes of exposure were assessed?	How was the intensity of exposure assessed?	How was the duration of exposure assessed?	Was cumulative exposure assessed?	Was exposure assessed before outcome being ascertained?	What was the timing of exposure relative to the outcome?	Was there known exposure to any other carcinogens?	Could the 'unexposed' group have included exposed?	Summary of methods used to assess exposure	Critique of the quality of the exposure assessment
Muttray et al. (1999) Inflammation (KC6) – proinflammatory cytokines	Exposure chamber in a crossover design (12)	Controlled exposure to > 99% 1,1,1- trichloroethane occurred in an $18m^3$ chamber at 2 time points, 1 week apart (200 ppm and 20 ppm) [1110 mg/m ³ and 111 mg/m ³] measured by a Miran infrared analyser	Yes, well defined	Quantitative	Yes	Inhalation	Measured by infrared analyser	Each session was 4 hours of exposure at 2 time points	No	Yes	Exposure occurred 20 min before biological measurements	Not in chamber. No other information provided	There were no unexposed; lowest exposure was 20 ppm [111 mg/m ³]	Exposure was generated in a controlled chamber for 4 hours each of 2 time points	No history was identified for earlier exposures (including confounding exposures). Methods are appropriate for an exposure chamber study. The inclusion of a 0 ppm exposed group would have been helpful for interpretation
Lemasters et al. (1999) Genotoxicity (KC2) – sister- chromatid exchanges and micronuclei Hill Air Force base solvent exposure study	Pilot study + a prospective exposure- response study in exposed (n = 50) and unexposed workers (n = 8)	Exposure was measured in air and in exhaled breath for correlation with genotoxic changes	Subjects had 3, full 8-hour shift measurements on consecutive days and had breath samples at the end of the 3 days. Results for 1,1,1- trichloroethane were summed with methyl ethyl ketone, xylenes, toluene and methylene chloride to a 'total solvents' exposure due to relatively low individual exposure levels	Quantitative	Unclear. "Though methylene chloride, xylenes, and 1,1,1- trichloroethane were not analysed using a NIOSH method, the same method as for blood toluene and methyl ethyl ketone was used"	Inhalation	Measured in air and breath samples in pilot study, breath only in genotoxic study	Duration was 30 weeks	No	Yes. Workers recruited could not have worked with chemicals 12 months before enrolment	Genotoxic end- points were assessed before beginning work (exposure) and at 15 and 30 week intervals	Yes, other solvent exposures were also measured in this study (toluene, xylenes, methyl ethyl ketone, methylene chloride) and 1,1,1- trichloroethane results were combined as 'solvent exposure'	Unlikely, as they did clerical work	Repeated measures of air exposure and exhaled breath levels as a marker of exposure. Pilot study collected hygiene air measurement data to demonstrate correlation with biological measures, so air concentrations are not otherwise described in second study	While extensive air monitoring data were collected, results are presented as aggregate 'solvent' values by breath and industrial hygiene measurement methods. No history was identified as being taken for earlier exposures (including confounding exposures)
JEM, job-exposure	matrix; KC, key	characteristic.													

References

Lemasters GK, Lockey JE, Olsen DM, Selevan SG, Tabor MW, Livingston GK, et al. (1999). Comparison of internal dose measures of solvents in breath, blood and urine and genotoxic changes in aircraft maintenance personnel. Drug Chem Toxicol. 22(1):181-200. https://doi.org/10.3109/01480549909029731 PMID:10189578 Muttray A, Klimek L, Faas M, Schäfer D, Mann W, Konietzko J (1999). The exposure of healthy volunteers to 200 ppm 1,1,1-trichloroethane increases the concentration of proinflammatory cytokines in nasal secretions. Int Arch Occup Environ Health. 72(7):485-8.

https://doi.org/10.1007/s004200050403 PMID:10541915

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