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Lancet. 2014 Aug 30;384(9945):755-65. doi: 10.1016/S0140-6736(14)60892-8. Epub 2014 Aug 13.

# Body-mass index and risk of 22 specific cancers: a population-based cohort study of 5·24 million UK adults.

Bhaskaran K[1], Douglas I[2], Forbes H[2], dos-Santos-Silva I[2], Leon DA[2], Smeeth L[3].

**Author information**

## Abstract

**BACKGROUND:** High **body-mass index** (BMI) predisposes to several site-**specific cancers**, but a large-scale systematic and detailed characterisation of patterns of **risk** across all common **cancers** adjusted for potential confounders has not previously been undertaken. We aimed to investigate the links between BMI and the most common site-**specific cancers**.

**METHODS:** With primary care data from individuals in the Clinical Practice Research Datalink with BMI data, we fitted Cox models to investigate associations between BMI and **22** of the most common **cancers**, adjusting for potential confounders. We fitted linear then non-linear (spline) models; investigated effect modification by sex, menopausal status, smoking, and age; and calculated population effects.

**FINDINGS:** 5·24 **million** individuals were included; 166,955 developed **cancers** of interest. BMI was associated with 17 of **22 cancers**, but effects varied substantially by site. Each 5 kg/m(2) increase in BMI was roughly linearly associated with **cancers** of the uterus (hazard ratio [HR] 1·62, 99% CI 1·56-1·69; p<0·0001), gallbladder (1·31, 1·12-1·52; p<0·0001), kidney (1·25, 1·17-1·33; p<0·0001), cervix (1·10, 1·03-1·17; p=0·00035), thyroid (1·09, 1·00-1·19; p=0·0088), and leukaemia (1·09, 1·05-1·13; p≤0·0001). BMI was positively associated with liver (1·19, 1·12-1·27), colon (1·10, 1·07-1·13), ovarian (1·09, 1.04-1.14), and postmenopausal breast **cancers** (1·05, 1·03-1·07) overall (all p<0·0001), but these effects varied by underlying BMI or individual-level characteristics. We estimated inverse associations with prostate and premenopausal breast cancer **risk**, both overall (prostate 0·98, 0·95-1·00; premenopausal breast cancer 0·89, 0·86-0·92) and in never-smokers (prostate 0·96, 0·93-0·99; premenopausal breast cancer 0·89, 0·85-0·94). By contrast, for lung and oral cavity cancer, we observed no association in never smokers (lung 0·99, 0·93-1·05; oral cavity 1·07, 0·91-1·26): inverse associations overall were driven by current smokers and ex-smokers, probably because of residual confounding by smoking amount. Assuming causality, 41% of uterine and 10% or more of gallbladder, kidney, liver, and colon **cancers** could be attributable to excess weight. We estimated that a 1 kg/m(2) population-wide increase in BMI would result in 3790 additional annual **UK** patients developing one of the ten **cancers** positively associated with BMI.

**INTERPRETATION:** BMI is associated with cancer **risk**, with substantial population-level effects. The heterogeneity in the effects suggests that different mechanisms are associated with different cancer sites and different patient subgroups.

**FUNDING:** National Institute for Health Research, Wellcome Trust, and Medical Research Council.



## Comment in

The obesity and cancer link. [Ann Oncol. 2015]
Overweight and obesity are linked to 10 common **cancers** and more than 12,000 **UK** cases. [BMJ. 2014]
[Fat people have common **cancers**]. [MMW Fortschr Med. 2014]
Obesity: a certain and avoidable cause of cancer. [Lancet. 2014]
[Obesity and cancer]. [Soins. 2014]

PMID: 25129328 [PubMed - indexed for MEDLINE] PMCID: PMC4151483 **Free PMC Article**

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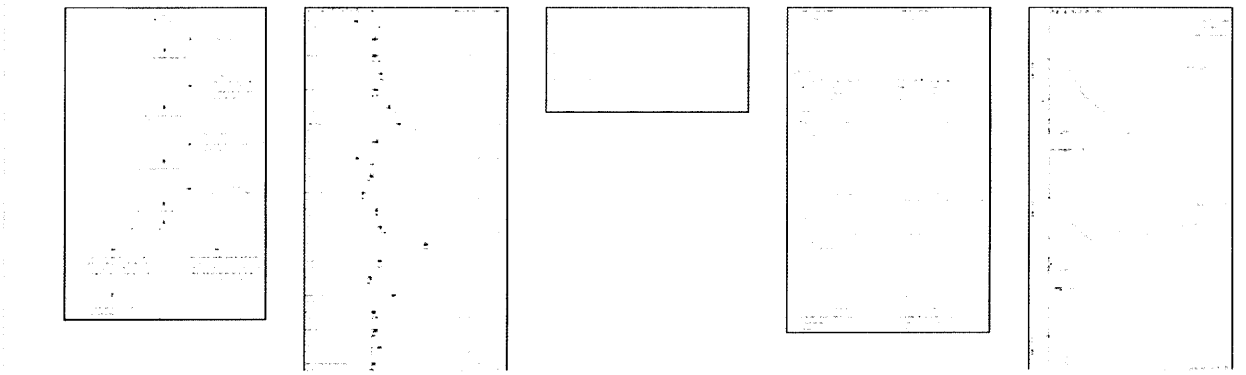

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Ann Oncol. 2013 Mar;24(3):807-16. doi: 10.1093/annonc/mds508. Epub 2012 Oct 26.

# Alcohol drinking and all cancer mortality: a meta-analysis.

Jin M[1], Cai S, Guo J, Zhu Y, Li M, Yu Y, Zhang S, Chen K.

## Author information

## Abstract

**BACKGROUND:** Epidemiological studies have suggested an inconsistent relationship between **alcohol drinking** and risk of all **cancer mortality**. As far as we know, no **meta-analysis** has been conducted to explore this issue.

**PATIENTS AND METHODS:** We carried out a PubMed search to find relevant articles published before April 2012 in English. Categorical and dose-response meta-analyses were conducted to identify the impact of **alcohol drinking** on all **cancer mortality**. Potential sources of heterogeneity were detected by meta-regression and stratification analyses. Sensitivity and cumulative meta-analyses were also carried out.

**RESULTS:** Eighteen independent cohort studies met the inclusion criteria. Compared with non/occasional drinkers, the pooled relative risks (RRs) were 0.91 [95% confidence interval (CI) 0.89-0.94] for light, 1.02 (95% CI 0.99-1.06) for moderate, and 1.31 (95% CI 1.23-1.39) for heavy drinkers. Former drinkers presented a higher risk (RR = 1.32, 95% CI 1.15-1.50) than current drinkers (RR = 1.06, 95% CI 0.98-1.16). There was a J-shaped relationship between all **cancer mortality** and **alcohol** consumption in males but not in females.

**CONCLUSIONS:** This **meta-analysis** confirms the health hazards of heavy **drinking** (≥50 g/day) and benefits of light **drinking** (≤12.5 g/day). Large-sample, well-designed, prospective epidemiological studies, especially on heavy **drinking** among women, should be developed in future.

## Comment in

Re: light **drinking** has positive public health consequences. [Ann Oncol. 2013]
Heavy consumption of **alcohol**: a risk factor for **cancer** deaths? [Natl Med J India. 2013]
Light **drinking** has positive public health consequences. [Ann Oncol. 2013]

PMID: 23104725 [PubMed - indexed for MEDLINE] Free full text

## Publication Types, MeSH Terms

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Cancer Res. 2014 Jun 1;74(11):3076-83. doi: 10.1158/0008-5472.CAN-13-2430.

# Breast cancer risk after occupational solvent exposure: the influence of timing and setting.

Ekenga CC[1], Parks CG[2], D'Aloisio AA[2], DeRoo LA[3], Sandler DP[2].

Author information

## Abstract

Organic solvents are ubiquitous in **occupational** settings where they may contribute to risks for carcinogenesis. However, there is limited information on organic solvents as human **breast** carcinogens. We examined the relationship between **occupational exposure** to solvents and **breast cancer** in a prospective study of 47,661 women with an **occupational** history in the Sister Study cohort. **Occupational solvent exposure** was categorized using self-reported job-specific **solvent** use collected at baseline. Multivariable Cox regression analyses were used to assess **breast cancer risk**, adjusting for established **breast cancer risk** factors. A total of 1,798 women were diagnosed with **breast cancer** during follow-up, including 1,255 invasive cases. Overall the **risk** of invasive **breast cancer** was not associated with lifetime **exposure** to solvents [HR, 1.04; 95% confidence interval (CI), 0.88-1.24]. Parous women who worked with solvents before their first full-term birth had an increased **risk** of estrogen receptor-positive invasive **breast cancer** compared with women who never worked with solvents (HR, 1.39; 95% CI, 1.03-1.86). A significantly elevated **risk** for estrogen receptor-positive invasive **breast cancer** was associated with **solvent exposure** among clinical laboratory technologists and technicians (HR, 2.00; 95% CI, 1.07-3.73). **Occupational exposure** to solvents before first birth, a critical period of **breast** tissue differentiation, may result in increased vulnerability for **breast cancer**. Our findings suggest a need for future studies in this area to focus on **exposure** time windows and **solvent** types in different **occupational** settings.



PMID: 24879566 [PubMed - indexed for MEDLINE] PMCID: PMC4059370 [Available on 2015-06-01]

## Publication Types, MeSH Terms, Substances, Grant Support

## LinkOut - more resources

Abstract



JAMA Intern Med. 2014 Dec;174(12):1922-8. doi: 10.1001/jamainternmed.2014.5219.

# Estimation of cigarette smoking-attributable morbidity in the United States.

Rostron BL[1], Chang CM[1], Pechacek TF[2].

## Author information

## Abstract

**IMPORTANCE:** **Cigarette** smoking has been found to harm nearly every bodily organ and is a leading cause of preventable disease, but current estimates of **smoking-attributable morbidity** by condition for the **United States** are generally unavailable.

**OBJECTIVE:** To estimate the burden of major medical conditions attributable to **cigarette** smoking in the **United States**.

**DESIGN, SETTING, AND PARTICIPANTS:** The disease burden of smoking was estimated using population-attributable risk calculations, taking into account the uncertainty of estimates. Population estimates came from 2009 US Census Bureau data and smoking prevalence, disease prevalence, and disease relative risk estimates came from National Health Interview Survey data for surveyed adults from 2006 through 2012. National Health and Nutrition Examination Survey spirometry data obtained from medical examination of surveyed adults from 2007 through 2010 was used to adjust for underreporting of chronic obstructive pulmonary disease.

**EXPOSURES:** Smoking status was assessed from self-reported National Health Interview Survey data.

**MAIN OUTCOMES AND MEASURES:** The number of adults 35 years and older who had had a major **smoking-attributable** disease by sex and condition and the total number of these conditions were estimated for the **United States** in 2009.

**RESULTS:** Using National Health Interview Survey data, we estimated that 6.9 million (95% CI, 6.5-7.4 million) US adults had had a combined 10.9 million (95% CI, 10.3-11.5 million) self-reported **smoking-attributable** medical conditions. Using chronic obstructive pulmonary disease prevalence estimates obtained from National Health and Nutrition Examination Survey self-reported and spirometry data, we estimated that US adults had had a combined 14.0 million (95% CI, 12.9-15.1 million) **smoking-attributable** conditions in 2009.

**CONCLUSIONS AND RELEVANCE:** We estimate that US adults have had approximately 14 million major medical conditions that were attributable to smoking. This figure is generally conservative owing to the existence of other diseases and medical events that were not included in these estimates. **Cigarette** smoking remains a leading cause of preventable disease in the **United States**, underscoring the need for continuing and vigorous smoking-prevention efforts.

Environ Health. 2014 Feb 19;13(1):10. doi: 10.1186/1476-069X-13-10.

# Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study.

Bove FJ[1], Ruckart PZ, Maslia M, Larson TC.

## Author information

## Abstract

**BACKGROUND:** Two drinking water systems at U.S. Marine Corps Base **Camp Lejeune**, North Carolina were contaminated with solvents during 1950s-1985.

**METHODS:** We conducted a retrospective cohort mortality study of Marine and Naval personnel who began service during 1975-1985 and were stationed at **Camp Lejeune** or **Camp** Pendleton, California during this period. **Camp** Pendleton's drinking water was uncontaminated. Mortality follow-up was 1979-2008. Standardized Mortality Ratios were calculated using U.S. mortality rates as reference. We used survival analysis to compare mortality rates between **Camp Lejeune** (N = 154,932) and **Camp** Pendleton (N = 154,969) cohorts and assess effects of cumulative exposures to contaminants within the **Camp Lejeune** cohort. Models estimated monthly contaminant levels at residences. Confidence intervals (CIs) indicated precision of effect estimates.

**RESULTS:** There were 8,964 and 9,365 deaths respectively, in the **Camp Lejeune** and **Camp** Pendleton cohorts. Compared to **Camp** Pendleton, **Camp Lejeune** had elevated mortality hazard ratios (HRs) for all cancers (HR = 1.10, 95% CI: 1.00, 1.20), kidney cancer (HR = 1.35, 95% CI: 0.84, 2.16), liver cancer (HR = 1.42, 95% CI: 0.92, 2.20), esophageal cancer (HR = 1.43 95% CI: 0.85, 2.38), cervical cancer (HR = 1.33, 95% CI: 0.24, 7.32), Hodgkin lymphoma (HR = 1.47, 95% CI: 0.71, 3.06), and multiple myeloma (HR = 1.68, 95% CI: 0.76, 3.72). Within the **Camp Lejeune** cohort, monotonic categorical cumulative exposure trends were observed for kidney cancer and total contaminants (HR, high cumulative exposure = 1.54, 95% CI: 0.63, 3.75; log10 $\beta$ = 0.06, 95% CI: -0.05, 0.17), Hodgkin lymphoma and trichloroethylene (HR, high cumulative exposure = 1.97, 95% CI: 0.55, 7.03; $\beta$ = 0.00005, 95% CI: -0.00003, 0.00013) and benzene (HR, high cumulative exposure = 1.94, 95% CI: 0.54, 6.95; $\beta$ = 0.00203, 95% CI: -0.00339, 0.00745). Amyotrophic Lateral Sclerosis (ALS) had HR = 2.21 (95% CI: 0.71, 6.86) at high cumulative vinyl chloride exposure but a non-monotonic exposure-response relationship ($\beta$ = 0.0011, 95% CI: 0.0002, 0.0020).

**CONCLUSION:** The study found elevated HRs at **Camp Lejeune** for several causes of death including cancers of the kidney, liver, esophagus, cervix, multiple myeloma, Hodgkin lymphoma and ALS. CIs were wide for most HRs. Because <6% of the cohort had died, long-term follow-up would be necessary to comprehensively assess effects of drinking water exposures at the base.

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Environ Health Perspect. 2014 Apr;122(4):325-34. doi: 10.1289/ehp.1307359. Epub 2014 Feb 14.

# Human health effects of tetrachloroethylene: key findings and scientific issues.

Guyton KZ[1], Hogan KA, Scott CS, Cooper GS, Bale AS, Kopylev L, Barone S, Makris SL, Glenn B, Subramaniam RP, Gwinn MR, Dzubow RC, Chiu WA.

**Author information**

## Abstract

**BACKGROUND:** The U.S. Environmental Protection Agency (EPA) completed a toxicological review of **tetrachloroethylene** (perchloroethylene, PCE) in February 2012 in support of the Integrated Risk Information System (IRIS).

**OBJECTIVES:** We reviewed **key findings** and **scientific issues** regarding the **human health effects** of PCE described in the U.S. EPA's Toxicological Review of **Tetrachloroethylene** (Perchloroethylene).

**METHODS:** The updated assessment of PCE synthesized and characterized a substantial database of epidemiological, experimental animal, and mechanistic studies. **Key scientific issues** were addressed through modeling of PCE toxicokinetics, synthesis of evidence from neurological studies, and analyses of toxicokinetic, mechanistic, and other factors (tumor latency, severity, and background rate) in interpreting experimental animal cancer **findings**. Considerations in evaluating epidemiological studies included the quality (e.g., specificity) of the exposure assessment methods and other essential design features, and the potential for alternative explanations for observed associations (e.g., bias or confounding).

**DISCUSSION:** Toxicokinetic modeling aided in characterizing the complex metabolism and multiple metabolites that contribute to PCE toxicity. The exposure assessment approach-a **key** evaluation factor for epidemiological studies of bladder cancer, non-Hodgkin lymphoma, and multiple myeloma-provided suggestive evidence of carcinogenicity. Bioassay data provided conclusive evidence of carcinogenicity in experimental animals. Neurotoxicity was identified as a sensitive noncancer **health** effect, occurring at low exposures: a conclusion supported by multiple studies. Evidence was integrated from **human**, experimental animal, and mechanistic data sets in assessing adverse **health effects** of PCE.

**CONCLUSIONS:** PCE is likely to be carcinogenic to humans. Neurotoxicity is a sensitive adverse **health** effect of PCE.

Environ Health. 2014 Aug 13;13:68. doi: 10.1186/1476-069X-13-68.

# Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study.

Bove FJ[1], Ruckart PZ, Maslia M, Larson TC.

## Author information

## Abstract

**BACKGROUND:** Two drinking water systems at U.S. Marine Corps Base **Camp Lejeune**, North Carolina were contaminated with solvents during 1950s-1985.

**METHODS:** We conducted a retrospective cohort mortality study of 4,647 civilian, full-time workers employed at **Camp Lejeune** during 1973-1985 and potentially exposed to contaminated drinking water. We selected a comparison cohort of 4,690 **Camp** Pendleton workers employed during 1973-1985 and unexposed to contaminated drinking water. Mortality follow-up period was 1979-2008. Cause-specific standardized mortality ratios utilized U.S. age-, sex-, race-, and calendar period-specific mortality rates as reference. We used survival analysis to compare mortality rates between **Camp Lejeune** and **Camp** Pendleton workers and assess the effects of estimated cumulative contaminant exposures within the **Camp Lejeune** cohort. Ground water contaminant fate/transport and distribution system models provided monthly estimated contaminant levels in drinking water serving workplaces at **Camp Lejeune**. The confidence interval (CI) indicated precision of effect estimates.

**RESULTS:** Compared to **Camp** Pendleton, **Camp Lejeune** workers had mortality hazard ratios (HRs) >1.50 for kidney cancer (HR = 1.92, 95% CI: 0.58, 6.34), leukemias (HR = 1.59, 95% CI: 0.66, 3.84), multiple myeloma (HR = 1.84, 95% CI: 0.45, 7.58), rectal cancer (HR = 1.65, 95% CI: 0.36, 7.44), oral cavity cancers (HR = 1.93, 95% CI: 0.34, 10.81), and Parkinson's disease (HR = 3.13, 95% CI: 0.76, 12.81). Within the **Camp Lejeune** cohort, monotonic exposure-response relationships were observed for leukemia and vinyl chloride and PCE, with mortality HRs at the high exposure category of 1.72 (95% CI: 0.33, 8.83) and 1.82 (95% CI: 0.36, 9.32), respectively. Cumulative exposures were above the median for most deaths from cancers of the kidney, esophagus, rectum, prostate, and Parkinson's disease, but small numbers precluded evaluation of exposure-response relationships.

**CONCLUSION:** The study found elevated HRs in the **Camp Lejeune** cohort for several causes of death including cancers of the kidney, rectum, oral cavity, leukemias, multiple myeloma, and Parkinson's disease. Only 14% of the **Camp Lejeune** cohort died by end of follow-up, producing small numbers of cause-specific deaths and wide CIs. Additional follow-up would be necessary to comprehensively assess drinking water exposure effects at the base.

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J Occup Environ Med. 2013 Feb;55(2):198-208. doi: 10.1097/JOM.0b013e3182728eab.

# Risk of selected cancers due to occupational exposure to chlorinated solvents in a case-control study in Montreal.

Christensen KY[1], Vizcaya D, Richardson H, Lavoué J, Aronson K, Siemiatycki J.

**Author information**

## Abstract

**OBJECTIVE:** To evaluate the association between **exposure** to **chlorinated solvents** and cancer.

**METHODS:** We conducted a **case-control study** of **occupational** exposures and cancer in **Montreal**, Quebec, Canada, including 3730 cancer cases and 533 population controls. **Occupational** exposures were derived using a combination of subject-reported job history and expert assessment. We examined the associations between two chemical families and six **chlorinated solvents** with 11 sites of cancer.

**RESULTS:** The majority of the associations examined were null, although many were based on small numbers. We found two significantly elevated odds ratios (ORs), one between perchloroethylene and prostate cancer (OR = 4.3; 95% CI: 1.4 to 13) and another between trichloroethylene and melanoma (OR = 3.2; 95% CI: 1.0 to 9.9).

**CONCLUSIONS:** There was little evidence of associations between **chlorinated solvents** and cancer. Limited power precludes strong inferences about absence of **risk**. We raise hypotheses about two possible associations: perchloroethylene with prostate cancer and trichloroethylene with melanoma.

PMID: 23147555 [PubMed - indexed for MEDLINE]

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QJM. 2015 Mar;108(3):205-12. doi: 10.1093/qjmed/hcu195. Epub 2014 Sep 9.

# The risk of kidney cancer in patients with kidney stones: a systematic review and meta-analysis.

Cheungpasitporn W[1], Thongprayoon C[2], O'Corragain OA[2], Edmonds PJ[2], Ungprasert P[2], Kittanamongkolchai W[2], Erickson SB[2].

**Author information**

## Abstract

**BACKGROUND:** The objective of this **meta-analysis** was to evaluate the association between a history of **kidney stones** and **kidney cancer**.

**METHODS:** A literature search was performed from inception until June 2014. Studies that reported odds ratios or hazard ratios comparing the **risk** of renal cell carcinoma (RCC) and transitional cell carcinoma (TCC) of the upper urinary tract in **patients** with the history of **kidney stones** versus those without the history of **kidney stones** were included. Pooled **risk** ratios (RRs) and 95% confidence interval (CI) were calculated using a random-effect, generic inverse variance method.

**RESULT:** Seven studies were included in our analysis to assess the association between a history of **kidney stones** and RCC. The pooled RR of RCC in **patients** with **kidney stones** was 1.76 (95% CI, 1.24-2.49). The subgroup analysis found that the history of **kidney stones** was associated with increased RCC **risk** only in males (RR, 1.41 [95% CI, 1.11-1.80]), but not in females (RR, 1.13 [95% CI, 0.86-1.49]). Five studies were selected to assess the association between a history of **kidney stones** and TCC. The pooled RR of TCC in **patients** with **kidney stones** was 2.14 (95% CI, 1.35-3.40).

**CONCLUSION:** Our study demonstrates a significant increased **risk** of RCC and TCC in **patients** with prior **kidney stones**. However, the increased **risk** of RCC was noted only in male **patients**. This finding suggests that a history of **kidney stones** is associated with **kidney cancer** and may impact clinical management and **cancer** surveillance.



PMID: 25208892 [PubMed - in process]

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Abstract

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Environ Health Perspect. 2000 May;108 Suppl 2:161-76.

# Trichloroethylene and cancer: epidemiologic evidence.

Wartenberg D[1], Reyner D, Scott CS.

## Author information

## Abstract

**Trichloroethylene** is an organic chemical that has been used in dry cleaning, for metal degreasing, and as a solvent for oils and resins. It has been shown to cause liver and kidney **cancer** in experimental animals. This article reviews over 80 published papers and letters on the **cancer** epidemiology of people exposed to **trichloroethylene**. Evidence of excess **cancer** incidence among occupational cohorts with the most rigorous exposure assessment is found for kidney **cancer** (relative risk [RR] = 1.7, 95% confidence interval [CI] 1.1-2.7), liver **cancer** (RR = 1.9, 95% CI(1.0-3.4), and non-Hodgkin's lymphoma (RR = 1.5, 95% CI 0.9-2.3) as well as for cervical **cancer**, Hodgkin's disease, and multiple myeloma. However, since few studies isolate **trichloroethylene** exposure, results are likely confounded by exposure to other solvents and other risk factors. Although we believe that solvent exposure causes **cancer** in humans and that **trichloroethylene** likely is one of the active agents, we recommend further study to better specify the specific agents that confer this risk and to estimate the magnitude of that risk.

## Comment in

Errors in TCE analysis. [Environ Health Perspect. 2001]
The a posteriori probability of a kidney **cancer** cluster attributed to **trichloroethylene** exposure. [Environ Health Perspect. 2002]
Carcinogenicity of **trichloroethylene**. [Environ Health Perspect. 2002]
Meta-analyses of TCE carcinogenicity. [Environ Health Perspect. 2000]

PMID: 10807550 [PubMed - indexed for MEDLINE] PMCID: PMC1637753 **Free PMC Article**

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