Karen Hudon
4900 SE Hanson Circle
Stuart, FL 34997

May 15, 2019

To: Florida Department of Environmental Protection

Subject: Comments - Triennial Review – May 15, 2019

Please consider my comments in response to this Triennial Review.

I feel that the citizens of Florida did not have adequate notice of this Triennial Review. In order to effectively receive public input, perhaps notice should be published in major newspapers throughout Florida well prior to this workshop.

Many people are concerned about the condition of our waterways, the environment and how these conditions adversely affect public health, wildlife and sea life. The safety of fish consumption, is being questioned because of aquatic deterioration. Cancer clusters have been found in various places. People want to talk about these problems and find solutions and many people would like to weigh in during this Triennial Review, but most people didn't know it would take place. People need time to plan to attend these workshops and should not have to travel unreasonable distances to attend. I also feel that more workshops should be planned throughout Florida – not just two physical workshops and one teleconference.

Exposure to dangerous, carcinogenic chemicals is causing increased rates of cancer and many other illnesses. Chemical contamination must be greatly reduced. I urge the Department of Environmental Protection to greatly reduce chemical allowances into our environment and thus protect all forms of life. Many of the chemicals being regulated in Florida should be outright prohibited. Attached are a few articles about adverse health effects from some of those chemicals. On behalf of Dr. J. William Louda, I have attached his comments along with a short separate comment he wrote about aluminum and its effects on fish. Additionally, in my archives, I found an item Dr. Louda wrote, which I feel should be included herein. Dr. Louda is extremely knowledgeable and experienced. The following is his January, 2017, communication to the members of the Florida Environmental Preservation and Conservation Subcommittee.

"Dear Members of the Environmental Preservation and Conservation Committee;

As a Research Professor active in the study of water quality and teacher of two courses in Environmental Chemistry (CHM-3080, CHS-6611) at FAU, I was appalled by the roll back of protections from toxic substances this past year.

Please work to increase these protections. Many is not most of the toxic materials are bio accumulative. That is as they have a high "octanol/water partition ratio" they preferentially dissolve into and remain with fat cells (adipose tissue) in each level of the food chain, ending with us. At each stage in the food chain, the rule of thumb states that a ten-fold increase in concentration occurs. Thus, long term exposure to very low levels of toxins can

and does result in increasing concentrations in the human body. The limits of chlorinated hydrocarbons, and many other toxics, in our waters should be zero!

Not only do we need to protect man but wildlife as well. The ancient American Indians had a saying-" *We do not inherit the land from our ancestors--we borrow it from our children*"! Think about that and protect us and the environment. Further, the solution to pollution is NOT dilution.

I thank you for your time and consideration.

Sincerely,

Dr. J. William Louda,
Research Professor
Environmental Biogeochemistry Group
Department of Chemistry and Biochemistry
and The Environmental Sciences Program
Florida Atlantic University
777 Glades Road
Boca Raton, Florida 33431 U.S.A.
(561) 297-3309
(561) 297-2759 FAX
blouda@fau.edu"

Thank you for your consideration of my comments and those of Dr. Louda. Please let me know if you have any questions.

Sincerely,

Karen Hudon
Phone: 772-266-8896
kmhudon@comcast.net

Attachments:

American Cancer Society – Benzene and Cancer Risk

Minnesota Department of Agriculture – Ecological Effects of Ammonia

Notice EPA "Aquatic Life Ambient Water Quality Criteria for Aluminum in Freshwater – December 21, 2019

National Toxicology Program, Dept. of Health and Human Services – Trichloroethylene

Statement of Dr. J. William Louda comments for 2019 Triennial Review with short statement regarding aluminum forming aluminum hydroxides

**From the American Cancer Society** Cancer Helpline 800.227.2345

**https://www.cancer.org/cancer/cancer-causes/benzene.html**

# Benzene and Cancer Risk

## What is benzene?

Benzene is a colorless, flammable liquid with a sweet odor. It evaporates quickly when exposed to air. Benzene is formed from natural processes, such as volcanoes and forest fires, but most exposure to benzene results from human activities.

Benzene is among the 20 most widely used chemicals in the United States. It is used mainly as a starting material in making other chemicals, including plastics, lubricants, rubbers, dyes, detergents, drugs, and pesticides. In the past it was also commonly used as an industrial solvent (a substance that can dissolve or extract other substances) and as a gasoline additive, but these uses have been greatly reduced in recent decades.

Benzene is also a natural part of crude oil and gasoline (and therefore motor vehicle exhaust), as well as cigarette smoke.

## How are people exposed to benzene?

The main way people are exposed is by breathing in air containing benzene. Benzene can also be absorbed through the skin during contact with a source such as gasoline, but because liquid benzene evaporates quickly, this is less common.

People can be exposed to benzene:

- At work
- In the general environment
- Through the use of some consumer products

The highest exposures have typically been in the workplace, although these have decreased greatly over the last several decades due to federal and state regulations. Some other exposures have also gone down over time, such as the amount of benzene allowed in gasoline.

### Workplace exposures

Workers in industries that make or use benzene may be exposed to this chemical. These include the rubber industry, oil refineries, chemical plants, shoe manufacturers, and gasoline-related industries. Benzene is also used to make some types of lubricants, dyes, detergents, drugs, and pesticides. Other people who may be exposed to benzene at work include steel workers, printers, lab technicians, gas station employees, and firefighters. Federal regulations limit exposure to benzene in the workplace (see below).

### Community exposures

People can be exposed to benzene in the environment from gasoline fumes, automobile exhaust, emissions from some factories, and waste water from certain industries. Benzene is commonly found in air in both urban and rural areas, but the levels are usually very low. Exposures can be higher for people in enclosed spaces with unventilated fumes from gasoline, glues, solvents, paints, and art supplies. Areas of heavy traffic, gas stations, and areas near industrial sources may also have higher air levels.

Cigarette smoking and secondhand smoke are important sources of exposure to benzene. Cigarette smoke accounts for about half of the exposure to benzene in the United States. Benzene levels in rooms containing tobacco smoke can be many times higher than normal.

People can also be exposed to benzene in contaminated drinking water and some foods (although the levels are usually very low).

## Does benzene cause cancer?

Benzene is known to cause cancer, based on evidence from studies in both people and lab animals. The link between benzene and cancer has largely focused on leukemia and other cancers of blood cells.

## What do studies show?

Researchers use 2 main types of studies to try to determine if a substance causes cancer.

- **Studies in people:** One type of study looks at cancer rates in different groups of people. Such a study might compare the cancer rate in a group exposed to a substance to the cancer rate in a group not exposed to it, or compare it to the cancer rate in the general population. But sometimes it can be hard to know what the results of these studies mean, because many other factors might affect the results.
- **Lab studies:** In studies done in the lab, animals are exposed to a substance (often in very large doses) to see if it causes tumors or other health problems. Researchers might also expose normal human cells in a lab dish to the substance to see if it causes the types of

The **International Agency for Research on Cancer (IARC)** is part of the World Health Organization (WHO). One of its goals is to identify causes of cancer. IARC classifies benzene as "carcinogenic to humans," based on sufficient evidence that benzene causes acute myeloid leukemia (AML). IARC also notes that benzene exposure has been linked with acute lymphocytic leukemia (ALL), chronic lymphocytic leukemia (CLL), multiple myeloma, and non-Hodgkin lymphoma.

The **National Toxicology Program (NTP)** is formed from parts of several different US government agencies, including the National Institutes of Health (NIH), the Centers for Disease Control and Prevention (CDC), and the Food and Drug Administration (FDA). The NTP has classified benzene as "known to be a human carcinogen."

The US **Environmental Protection Agency (EPA)** maintains the Integrated Risk Information System (IRIS), an electronic database that contains information on human health effects from exposure to various substances in the environment. The EPA classifies benzene as a known human carcinogen.

(For more information on the classification systems used by these agencies, see *Known and Probable Human Carcinogens*.)

## Does benzene cause any other health problems?

Benzene is a potentially dangerous chemical. High levels of exposure can cause both short-term and long-term health effects.

### Short-term effects

Breathing in high doses of benzene can affect the nervous system, which can lead to drowsiness, dizziness, headaches, tremors, confusion, and/or unconsciousness. Consuming foods or fluids contaminated with high levels of benzene can cause vomiting, stomach irritation, dizziness, sleepiness, convulsions, and rapid heart rate. In extreme cases, inhaling or swallowing very high levels of benzene can be deadly.

Exposure to benzene liquid or vapor can irritate the skin, eyes, and throat. Skin exposure to benzene can result in redness and blisters.

### Long-term effects

Long-term exposure to benzene mainly harms the bone marrow, the soft, inner parts of bones where new blood cells are made. This can result in:

changes that are seen in cancer cells. It's not always clear if the results from these types of studies will apply to humans, but lab studies are a good way to find out if a substance might possibly cause cancer.

Often neither type of study provides conclusive evidence on its own, so researchers usually look at both human and lab-based studies when trying to figure out if something causes cancer.

### Studies in people

Rates of leukemia, particularly acute myeloid leukemia (AML), have been found to be higher in studies of workers exposed to high levels of benzene, such as those in the chemical, shoemaking, and oil refining industries.

Some studies have also suggested links to childhood leukemia (particularly AML) as well as acute lymphocytic leukemia (ALL), chronic lymphocytic leukemia (CLL), and other blood-related cancers (such as multiple myeloma and non-Hodgkin lymphoma) in adults. However, the evidence is not as strong for these cancers.

There is much less evidence linking benzene to any other type of cancer.

### Studies done in the lab

When inhaled or swallowed, benzene has been found to cause different types of tumors in lab animals such as rats and mice. These results support the finding of an excess risk of leukemia in humans. However, most studies in humans have not found an increased risk of cancers other than leukemia among people with higher exposures.

Benzene has been shown to cause chromosome changes in bone marrow cells in the lab. (The bone marrow is where new blood cells are made.) Such changes are commonly found in human leukemia cells.

## What expert agencies say

Several national and international agencies study substances in the environment to determine if they can cause cancer. (A substance that causes cancer or helps cancer grow is called a *carcinogen*.) The American Cancer Society looks to these organizations to evaluate the risks based on evidence from laboratory, animal, and human research studies.

Based on animal and human evidence, several expert agencies have evaluated the cancer-causing potential of benzene.

When possible, limiting the time you spend near idling car engines can help lower your exposure to exhaust fumes, which contain benzene (as well as other potentially harmful chemicals).

Use common sense around any chemicals that might contain benzene. Limit or avoid exposure to fumes from solvents, paints, and art supplies, especially in unventilated spaces.

If you are exposed at your workplace, talk to your employer about limiting your exposure through process changes (such as replacing the benzene with another solvent or enclosing the benzene source) or by using personal protective equipment. If needed, the Occupational Safety & Health Administration (OSHA) can provide more information or make an inspection.

### What should I do if I've been exposed to benzene?

For short-term exposure to high levels of benzene, the Centers for Disease Control and Prevention (CDC) recommends getting away from the source of benzene, removing any clothing that may have benzene on it, washing exposed areas with soap and water, and getting medical care as soon as possible.

If you think you may have been exposed to benzene over a long period of time, speak to a doctor. Benzene can be measured in the blood or breath, and breakdown products of benzene can be measured in the urine. These tests can only detect recent exposures to benzene. They cannot predict possible health effects.





The American Cancer Society medical and editorial content team

Our team is made up of doctors and oncology certified nurses with deep knowledge of cancer care as well as journalists, editors, and translators with extensive experience in medical writing.



Last Medical Review: January 5, 2016 Last Revised: January 5, 2016

- Anemia (a low red blood cell count), which can cause a person to feel weak and tired.
- A low white blood cell count, which can lower the body's ability to fight infections and might even be life-threatening.
- A low blood platelet count, which can lead to excess bruising and bleeding.

There is also some evidence that long-term exposure to benzene might harm reproductive organs. Some women who have breathed in high levels of benzene for many months have had irregular menstrual periods and ovary shrinkage, but it is not known for sure if benzene caused these effects. It is not known if benzene exposure affects the fetus in pregnant women or fertility in men.

### Are benzene levels regulated?

Several government agencies regulate benzene levels and exposures.

The Occupational Safety & Health Administration (OSHA) is the federal agency responsible for health and safety regulations in most workplaces. OSHA limits exposure to benzene in the air in most workplaces to 1 ppm (part per million) during an average workday and a maximum of 5 ppm over any 15-minute period. When working at potentially higher exposure levels, OSHA requires employers to provide personal protective equipment such as respirators.

The EPA limits the percentage of benzene allowed in gasoline to an average of 0.62% by volume (with a maximum of 1.3%).

The EPA limits concentrations of benzene in drinking water to 5 ppb (parts per billion). Some states may have lower limits. Likewise, the US Food and Drug Administration (FDA) sets a limit of 5 ppb in bottled water.

The Consumer Product Safety Commission (CPSC) considers any product containing 5% or more by weight of benzene to be hazardous, requiring special labeling.

### Can I limit my exposure to benzene?

If you are concerned about benzene, there are several ways you can limit your exposure.

Stay away from cigarette smoke. If you are a smoker, try to quit. Cigarette smoke is a major source of benzene exposure.

Try to limit gasoline fumes by pumping gas carefully and using gas stations with vapor recovery systems that capture the fumes. Avoid skin contact with gasoline.

https://www.mda.state.mn.us/ecological-effects-ammonia

DEPARTMENT OF AGRICULTURE

# ECOLOGICAL EFFECTS OF AMMONIA

Even at extremely low concentrations aquatic life will be harmed by ammonia. Ammonia occurs naturally in the environment. A small amount of ammonia is generated when lightning strikes and reaches earth in rainfall. But most ammonia is produced by bacteria in water and soil as an end product of plant and animal waste decomposition. It is found in relatively low nontoxic concentrations in soil, air, and water and provides a source of nitrogen for plants.

In soils and water ammonia will go through many complex biochemical transformations. These transformations constitute what is commonly known as the nitrogen cycle. For a more in-depth discussion of the nitrogen cycle see related links.

**Ammonia in Water**

Water reacts with ammonia to form ammonium and hydroxide ions. Ammonia is often referred to as "unionized ammonia". Ammonia is toxic to aquatic organisms but ammonium is non-toxic. There exists an equilibrium in water between the toxic ammonia and the non-toxic ammonium. The equation shifts back and forth depending upon existing or introduced environmental changes

$$NH_3\,(aq) + H_2O\,(l) \rightleftharpoons NH_3 \cdot H_2O\,(aq) \rightleftharpoons NH_4^+\,(aq) + OH^-\,(aq)$$

(ammonia in water) (ammonia + water) (ammonia in water)

The dynamic equilibrium between NH3 and NH4 + is affected by water temperature and pH (acidity). At a pH of six the ratio of ammonia to ammonium is 1 to 3000 but decreases to 1 to 30 when the pH rises to eight (becomes less acidic). Warm water will contain more toxic ammonia then cooler water. When sampling water for ammonia analysis both the temperature and the pH of the surface water body must be measured at the same time the water samples are collected. (See Ammonia, pH & Temperature Calculator (http://www.hbuehrer.ch/Rechner/Ammonia.html))

If ammonia is directly spilled into surface water or if water used by a fire department to depress an ammonia vapor cloud is allowed to reach surface water, aquatic life can be harmed. Even at a concentration of 0.02 mg/L (48 hour LC50) unionized ammonia is lethal to some sensitive freshwater fish. That equates to about ½ a cup of unionized ammonia in one million gallons of water. Ammonia is also highly toxic to freshwater invertebrates having a 48-hour LC50 of 0.66 mg/L for Daphnia magna . Again, water contaminated with fertilizer ammonia should not be allowed to enter any storm drains, rivers, drainage ditches, wetlands or lakes.

**Ammonia in Air and Soil**

After a release of ammonia the vapors will dissipate reacting with the moisture in the air to form ammonium and eventually return to earth in rainfall. Ammonium then quickly binds to the negatively charged soil organic matter and soil clays. Ammonium rarely accumulates in soil because bacteria will rapidly convert the ammonium that is not taken up by plant roots into nitrates (nitrification). Nitrates can also be absorbed by roots or may leach through the soil profile. Since ammonium is soil bound, unless the soil is washed away by rainfall events the contamination will likely stay put horizontally but leach vertically as nitrates through the root zone.

If the fire department suppresses an ammonia vapor cloud with water for a prolonged period of time the applied water may contaminate soil. In this instance the soil may require some method of remediation to prevent adverse environmental affects.

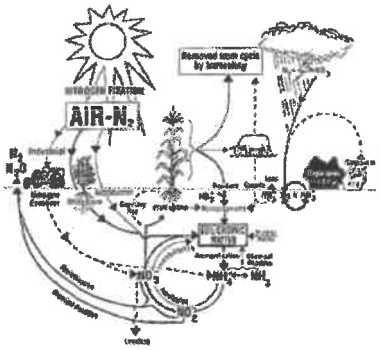
**Ammonia Affecting Plants**

Plants, trees and crops are mostly made up of water. If a large release of ammonia occurs the vapor will likely burn the leaves of nearby downwind vegetation. Ammonia will pull water from the leaves but will not affect the roots so damaged plants will probably fully recover although affected crops may suffer yield loss.

**Ammonia Affecting Livestock**

Be aware that ammonia vapors are toxic to livestock. Dairy, swine and poultry livestock producers operating near to and downwind of a release or potential release must be notified to take appropriate action.

The Nitrogen Cycle in Soil from The Ohio State University Extension Fact Sheet AEX-463-96




## FORMS + RESOURCES

> First Responder Resources (/pesticide-fertilizer/emergency-response-anhydrous-ammonia-releases-spills)

## EXTERNAL LINKS

> The Nitrogen Cycle - Missouri Extension (http://extension.missouri.edu/publications/displayprinterfriendlypub.aspx?p=wq252)

> Ammonia, pH & Temperature Calculator (http://www.hbuehrer.ch/Rechner/Ammonia.html)

> What is LD50 or LC50? (http://www.ccohs.ca/oshanswers/chemicals/ld50.html)

## CONTACT US

Patrick Kelly (/patrick-kelly)
Pesticide & Fertilizer Management
651-201-6387 (tel:651-201-6387)
Patrick.Kelly@state.mn.us (mailto:Patrick.Kelly@state.mn.us)

Report on Aluminum effects on aquatic life:

https://www.federalregister.gov/documents/2018/12/21/2018-27745/aquatic-life-ambient-water-quality-criteria-for-aluminum-in-freshwater

LEGAL STATUS

LEGAL STATUS

# Aquatic Life Ambient Water Quality Criteria for Aluminum in Freshwater

A Notice by the Environmental Protection Agency on 12/21/2018

DOCUMENT DETAILS

**Printed version:**
PDF (https://www.govinfo.gov/content/pkg/FR-2018-12-21/pdf/2018-27745.pdf)

**Publication Date:**
12/21/2018 (/documents/2018/12/21)

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FRL-9988-42-OW

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## AGENCY:

Environmental Protection Agency (EPA).

## ACTION:

Notice of availability.

## SUMMARY:

The Environmental Protection Agency (EPA) is announcing the availability of Aquatic Life Ambient Water Quality Criteria for Aluminum in Freshwater. The EPA first released freshwater criteria for aluminum in 1988 to protect aquatic life from harmful effects of aluminum toxicity. The EPA updated its recommended aluminum criteria to reflect the latest science and to provide users the flexibility to develop criteria based on site-specific water chemistry. The document provides a scientific assessment of ecological effects and is not a regulation. The EPA submitted the draft document for external expert peer review and edited the document considering peer review comments. The EPA subsequently released the draft criteria document for a 90-day public comment period in July 2017. The EPA has considered the public comments and revised the document based on consideration of those comments. The final criteria document provides recommendations for states and authorized tribes to establish water quality standards under the Clean Water Act. The recommendations found in this document supersede the EPA's 1988 national recommended criteria for aluminum in ambient water.

## FOR FURTHER INFORMATION CONTACT:

Diana Eignor, Health and Ecological Criteria Division, Office of Water (Mail Code 4304T), Environmental Protection Agency, 1200 Pennsylvania Avenue NW, Washington, DC 20460; telephone: (202) 566-1143; or email: *eignor.diana@epa.gov (mailto:eignor.diana@epa.gov).*

## SUPPLEMENTARY INFORMATION:

### I. General Information

#### A. How can I get copies of this document and other related information?

*1. Docket.* EPA has established a docket for this action under Docket ID No. EPA-HQ-OW-2017-0260. Publicly available docket materials are available either electronically through *www.regulations.gov (http://www.regulations.gov)* or in hard copy at the Water Docket in the EPA Docket Center, (EPA/DC) EPA West, Room 3334, 1301 Constitution Ave. NW, Washington, DC. The EPA Docket Center Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the Water Docket is (202) 566-2426.

*2. Electronic Access.* You may access this **Federal Register** document ☐ electronically from the Government Printing Office under the **Federal Register** listings FDSys (*http://www.gpo.gov/fdsys/browse/collection.action?collectionCode=FR (http://www.gpo.gov/fdsys/browse/collection.action?collectionCode=FR)*).

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### II. What is aluminum and how does it affect aquatic life?

Aluminum is found in most soils and rocks and is the third most abundant element and the most common metal in the earth's crust. Aluminum can enter the water via natural processes, like weathering of rocks and as a result of human based activities, such as drinking and waste water treatment and mining. Aluminum is considered a non-essential metal because fish and other aquatic life do not need it to function. Elevated levels of aluminum can affect some species' ability to regulate ions and inhibit respiratory functions. Aquatic plants are generally less sensitive than fish and other aquatic life to aluminum.

## III. What are EPA's updated recommended criteria for aluminum in freshwater?

The recommended criteria concentrations for aluminum in freshwater to protect aquatic life depends on a site's water chemistry parameters. Bioavailability is the measure of whether a substance in the environment is available to affect living organisms like fish. The bioavailability of aluminum is dependent on specific water chemistry parameters. The more bioavailable the aluminum is, the more likely it is to cause a toxic effect. The water chemistry parameters that have the greatest impact on aluminum's bioavailability are pH, dissolved organic carbon (DOC) and total hardness.

The final 2018 recommended national criteria are based upon Multiple Linear Regression (MLR) models for fish and invertebrate species that use pH, DOC, and total hardness to quantify the effects of these water chemistry parameters on the bioavailability and associated toxicity of aluminum to aquatic organisms. The MLR models are used to normalize the available toxicity data to reflect the effects of the water chemistry (pH, hardness, DOC) on the toxicity of aluminum to tested species. These normalized toxicity test data are then used in a criteria calculator to generate criteria for specific water chemistry conditions, yielding the water chemistry specific acute and chronic criteria concentrations. This flexible approach is based on the latest science and allows users to develop site-specific aluminum criteria for freshwaters that appropriately reflect important water chemistry parameters. The recommended acute criteria (known as the criteria maximum concentration or CMC) duration is a one-hour average and the recommended chronic criteria (criteria chronic concentration or CCC) duration is a four-day average. The EPA recommends that the CMC and CCC not be exceeded more than once every three years.

These final 2018 recommended national aluminum criteria are expressed as total recoverable metal concentrations. The use of total recoverable aluminum includes monomeric (both organic and inorganic) forms, polymeric and colloidal forms, as well as particulate forms and aluminum sorbed to clays. However, toxicity data comparing toxicity of aluminum using total recoverable aluminum and dissolved aluminum demonstrated that toxic effects increased with increasing concentrations of total recoverable aluminum even though the concentration of dissolved aluminum was relatively constant. If aluminum criteria were based on dissolved concentrations, toxicity would likely be underestimated, as colloidal forms and hydroxide precipitates of the metal that can dissolve under natural conditions and become biologically available would not be measured. The criteria document contains more discussion of the studies that informed the choice to use total recoverable aluminum as the basis for the final 2018 recommended national criteria. The current EPA-approved Clean Water Act Test Methods [1] for aluminum in natural waters and waste waters measure total recoverable aluminum.

The numeric outputs of the 2018 recommended National Aluminum Criteria Calculator will depend on the specific pH, DOC, and total hardness concentrations entered into the models. The model outputs (CMC and CCC) are numeric values that are protective for the set of input conditions. Criteria can be determined in two ways: Use the provided Aluminum Criteria Calculator V.2.0 to enter the pH, DOC, and total hardness conditions at a specific site to calculate the numeric aluminum CMC and CCC corresponding to those local input water-quality conditions, or (2) use the look-up tables provided in the criteria document, developed using the calculator, to find the numeric aluminum CMC and CCC most closely corresponding to the local conditions for pH, DOC, and total hardness. In order to calculate numeric water quality criteria for aluminum that will protect the aquatic life designated uses of a site over the full range of ambient conditions and toxicity, multiple model outputs will need to be considered.

See Table 1 for a comparison of the EPA's 1988 criteria and the updated 2018 criteria for aluminum.

Table 1—Summary of the EPA National Recommended Aquatic Life Criteria for Aluminum

| EPA aquatic life criteria for aluminum | Freshwater acute [a] (1 hour, total recoverable aluminum) | Freshwater Chronic [a] (4-day, total recoverable aluminum) |
|---|---|---|
| 2018 Updated Criteria (Vary as a function of a site's pH, total hardness, and DOC) | 1-4,800 µg/L [b] | 0.63-3,200 µg/L [b]. |
| 1988 Criteria (pH 6.5-9.0, across all total hardness and DOC ranges) | 750 µg/L | 87 µg/L. |

[a] Values are recommended not to be exceeded more than once every three years on average.

[b] Values will be different under differing water chemistry conditions.

## IV. What are recommended water quality criteria developed by the EPA?

Section 304(a)(1) of the Clean Water Act directs the EPA to develop and publish and, from time to time, revise criteria for water quality accurately reflecting the latest scientific knowledge. Water quality criteria developed under section 304(a) are based solely on data and scientific judgments on the relationship between pollutant concentrations and environmental and human health effects. Section 304(a) criteria do not reflect consideration of economic impacts or the technological feasibility of meeting pollutant concentrations in ambient water.

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Section 304(a) criteria provide guidance to states and authorized tribes in adopting water quality standards that ultimately provide a basis for controlling discharges of pollutants. Under the Clean Water Act and its implementing regulations, states and authorized tribes are to adopt water quality criteria to protect designated uses (*e.g.*, aquatic life, recreational use). The EPA water quality criteria recommendations are not regulations. Thus, the EPA recommended criteria do not constitute legally binding requirements. States and authorized tribes may adopt other scientifically defensible water quality criteria that differ from these recommendations. As part of the water quality standards triennial review process defined in section 303(c)(1) of the Clean Water Act, the states and authorized tribes are responsible for maintaining and revising water quality standards. Standards consist of designated uses, water quality criteria to protect those uses, a policy for antidegradation, and may include general policies for application and implementation. Section 303(c)(1) requires states and authorized tribes to review and modify, if appropriate, their water quality standards at least once every three years. Consistent with the EPA regulations at 40 CFR 131.11 (/select-citation/2018/12/21/40-CFR-131.11)(a), protective criteria must be based on a sound scientific rationale and contain sufficient parameters or constituents to protect the designated uses. Criteria may be expressed in either narrative or numeric form. States and authorized tribes have four options when adopting water quality criteria for which EPA has published section 304(a) criteria. They may: (1) Establish numerical values based on recommended section 304(a) criteria; (2) Adopt section 304(a) criteria modified to reflect site-specific conditions; (3) Adopt criteria derived using other scientifically defensible methods; or (4) Establish narrative criteria where numeric criteria cannot be established or to supplement numeric criteria (40 CFR 131.11 (/select-citation/2018/12/21/40-CFR-131.11)(b)).

Dated: December 14, 2018.

Anna J. Wildeman,

Acting Assistant Administrator, Office of Water.

## Footnotes

1. *40 CFR part 136.3 (/select-citation/2018/12/21/40-CFR-136.3) and Appendix C*
Back to Citation

[FR Doc. 2018-27745 (/a/2018-27745) Filed 12-20-18; 8:45 am]

BILLING CODE 6560-50-P

National Toxicology Program

Headquartered at the
National Institute of Environmental
Health Sciences NIH-HHS

# Trichloroethylene

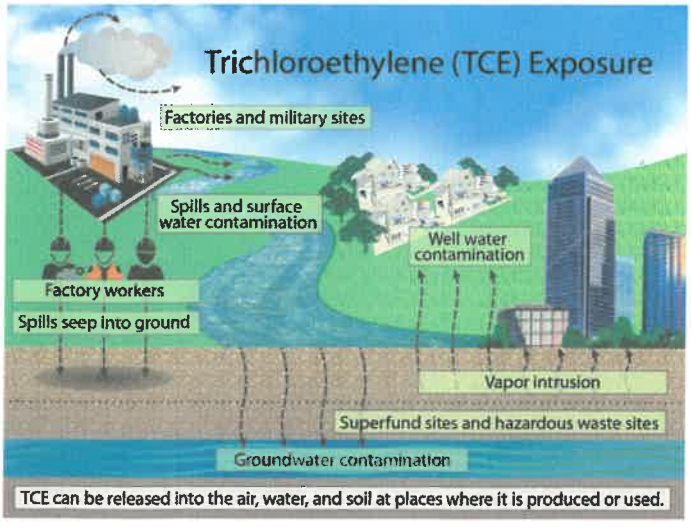
## What is trichloroethylene and how is it used?

Trichloroethylene, also known as TCE, is a colorless, volatile liquid that is produced in large volumes for commercial use.

It is primarily used in two ways — to make hydrofluorocarbon chemicals, especially refrigerants, and as a solvent to remove grease from metal parts, although this use is declining. Additionally, it is used for spot removal in dry cleaning operations and is found in some aerosol cleaning products that consumers may purchase for home and auto maintenance.

## How are people exposed to trichloroethylene?

There are many ways people can be exposed to TCE. It can be released into the air, water, and soil at places where it is produced or used. It breaks down slowly, so it remains in the environment for long periods of time. TCE can move readily through soil and make its way into underground drinking water sources. Because of its widespread use as a metal degreasing agent to maintain military equipment, it has been found in the groundwater at many military sites. Notably, TCE and other chemicals have been found in the water supply wells at the military base in Camp Lejeune, North Carolina. The wells were contaminated by leaking underground storage tanks, industrial area spills, and waste disposal sites.




## Key Points


- *Upgraded to known to be a human carcinogen*
- *Industrial solvent used primarily to make hydrofluorocarbon chemicals*
- *Elevated risk of kidney cancer in exposed people*

14th Report on Carcinogens

The general population can be exposed to TCE by inhaling the chemical in indoor and outdoor air, drinking contaminated water, or eating foods that may have been washed or processed with TCE-contaminated water. People who live or work in buildings near sites that make or dispose of TCE, such as Superfund sites, may experience higher levels of indoor TCE due to vapor intrusion. Vapor intrusion is when volatile chemicals, such as TCE, migrate into buildings from the soil.

Workers involved in commercial degreasing activities are mainly exposed by inhaling vapors or by dermal, or skin, exposure to the vapors or liquid.

PO Box 12233 • Research Triangle Park, NC 27709
Phone: 919-541-3345 • http://ntp.niehs.nih.gov

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https://ntp.niehs.nih.gov/ntp/roc/content/profiles/trichloroethylene.pdf

Report follows

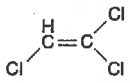
# Trichloroethylene

## CAS No. 79-01-6

Known to be a human carcinogen

First listed in the *Ninth Report on Carcinogens* (2000)

Also known as 1,1,2-trichloroethene or TCE



## Carcinogenicity

Trichloroethylene is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans. This conclusion is based on epidemiological studies showing that it causes kidney cancer in humans, together with supporting evidence from toxicological, toxicokinetic, and mechanistic studies demonstrating the biological plausibility of its carcinogenicity in humans. Epidemiological studies also provide limited evidence that trichloroethylene causes non-Hodgkin lymphoma (NHL) in humans. Supporting evidence is provided by studies in experimental animals demonstrating that trichloroethylene causes cancer at several tissue sites, including some of the same sites as seen in humans. The data available from studies in humans are inadequate to evaluate the relationship between trichloroethylene exposure and liver cancer. Trichloroethylene was first listed in the *Ninth Report on Carcinogens* in 2000 as *reasonably anticipated to be a human carcinogen* based on limited evidence of carcinogenicity from studies in humans, sufficient evidence of carcinogenicity from studies in experimental animals, and information from studies on mechanisms of carcinogenesis.

### *Cancer Studies in Humans*

#### *Kidney Cancer*

Epidemiological studies have demonstrated that trichloroethylene exposure causes kidney cancer, based on the following evidence: (1) consistent findings of increased risk across studies with different study designs, in different geographical areas, and in different occupational settings, (2) evidence of increasing cancer risk with increasing level or duration of exposure (i.e., an exposure-response relationship), and (3) meta-analyses (combined statistical analyses of the data from several different studies) showing statistically significant increased cancer risk across studies.

Numerous studies, primarily among workers, have evaluated the association between trichloroethylene exposure and kidney cancer, including twelve cohort studies or nested case-control studies (case-control studies conducted within specific cohort studies) and seven case-control studies (NTP 2015). Three of these studies—a cohort study of aerospace workers (Zhao *et al.* 2005), a French case-control study of screw-cutting workers (Charbotel *et al.* 2006, 2009), and a case-control study of occupational exposure to trichloroethylene in central and eastern Europe (Moore *et al.* 2010)—were considered to be the most informative, because they had good exposure assessments, detailed analysis of exposure-response relationships, or presumed high levels of exposure. The most convincing evidence that exposure to trichloroethylene causes kidney cancer comes from these three studies and two additional studies, a Nordic cohort of blue-collar workers in companies using trichloroethylene (Raaschou-Nielsen *et al.* 2003) and a case-control study in an area in Germany where workers in several factories were exposed to high levels of trichloroethylene (Brüning *et al.* 2003). All five studies found that workers with the highest exposure to trichloroethylene had a statistically significant increased risk of kidney cancer, and four of the studies found that the risk of kidney cancer increased with increasing duration, intensity, or cumulative level of exposure.

These findings are supported by weaker associations found in several other cohort studies (Morgan *et al.* 1998, Boice *et al.* 2006, Hansen *et al.* 2013, Bove *et al.* 2014, Silver *et al.* 2014) and case-control studies (Dosemeci *et al.* 1999, Pesch *et al.* 2000). Very high risks of kidney cancer were found among German workers exposed to high levels of trichloroethylene (Henschler *et al.* 1995, Vamvakas *et al.* 1998); however, potential methodological biases in these two studies could have distorted the risk estimate.

Two recent robust meta-analyses found statistically significant increased risks of kidney cancer among workers who had ever been exposed to trichloroethylene (overall relative risks: mRR = 1.27, 95% confidence interval [CI] = 1.13 to 1.43, Scott and Jinot 2011; mRR = 1.32, 95% CI = 1.17 to 1.50, Karami *et al.* 2012). One meta-analysis (Scott and Jinot 2011) found a higher overall relative risk for the highest exposure group across studies (mRR = 1.58, 95% CI = 1.28 to 1.96) than for all subjects ever exposed to trichloroethylene, which provides supporting evidence for an exposure-response relationship between the level of trichloroethylene exposure and risk of kidney cancer.

Several studies, including some large studies (Greenland *et al.* 1994, Radican *et al.* 2008, Lipworth *et al.* 2011, Christensen *et al.* 2013, Vlaanderen *et al.* 2013), found little or no evidence for an association between kidney cancer and trichloroethylene exposure or for an exposure-response relationship. However, these studies were limited by imprecise classification of exposure or low sensitivity to detect an association, either because the exposure levels were low or because few workers had high levels of exposure. In general, these limitations would make it harder to detect a true effect and would not likely result in a false positive association (NTP 2015).

It is unlikely that the positive findings across studies could be explained by biases or by potential confounding from smoking or co-exposure to other occupational carcinogens (NTP 2015). Most of the case-control studies found positive associations between trichloroethylene exposure and kidney cancer after controlling for smoking. Furthermore, the cohort studies found little evidence for an association between trichloroethylene exposure and lung cancer, which strongly suggests that smoking did not explain the excess kidney cancer risk. Studies of specific industries found positive associations after considering known occupational co-exposures to other carcinogenic substances in their statistical analysis or study design (Zhao *et al.* 2005, Charbotel *et al.* 2006, 2009). Although other cohort and case-control studies did not provide information about occupational co-exposures, those studies included workers in diverse occupations with differing levels and patterns of co-exposures, and the prevalence of any one specific co-exposure across studies was probably low. Furthermore, positive associations between trichloroethylene exposure and kidney cancer were found across studies with different study designs and in different occupational settings and geographical regions.

#### *Non-Hodgkin Lymphoma*

Epidemiological studies provide limited evidence that trichloroethylene exposure causes NHL, based on positive associations in several studies and evidence for increased risk of NHL across studies combined in two meta-analyses. The evidence across studies is less consistent than for kidney cancer, and alternative explanations for the associations, such as chance or confounding, cannot reasonably be ruled out (NTP 2015). NHL includes a diverse group of cancers arising in cells of the lymphatic system, most commonly the B lymphocytes, a type of white blood cell involved in the immune response.

Studies evaluating the relationship between exposure to trichloroethylene and NHL (including specific histological subtypes of NHL

and other related B-cell lymphomas) included ten cohort or nested case-control studies (Morgan *et al.* 1998, Raaschou-Nielsen *et al.* 2003, Boice *et al.* 2006, Radican *et al.* 2008, Bahr *et al.* 2011, Lipworth *et al.* 2011, Hansen *et al.* 2013, Vlaanderen *et al.* 2013, Bove *et al.* 2014, Silver *et al.* 2014), four case-control studies (Hardell *et al.* 1994, Persson and Fredrikson 1999, Wang *et al.* 2009, Christensen *et al.* 2013, Deng *et al.* 2013), a pooled analysis of four case-control studies by the International Lymphoma Epidemiology Consortium (InterLymph) (Cocco *et al.* 2013), and two recent meta-analyses. The InterLymph pooled analysis (Cocco *et al.* 2013) was considered to be the most informative study, because of its high-quality exposure assessment, large size, and analyses of exposure levels and durations and NHL subtypes (NTP 2015).

The strongest evidence for an association between trichloroethylene exposure and NHL comes from the InterLymph pooled analysis ($P$ = 0.004 for combined analysis of duration, frequency, and intensity of exposure among individuals with the highest probability of exposure) (Cocco *et al.* 2013) and the two meta-analyses (mRR = 1.23, 95% CI = 1.07 to 1.42, Scott and Jinot 2011; mRR = 1.32, 95% CI = 1.14 to 1.54, Karami *et al.* 2013). The risk of NHL increased with increasing level or duration of exposure in the InterLymph pooled analysis, in one of its component studies (Purdue *et al.* 2011), and in another case-control study (Wang *et al.* 2009).

An association between trichloroethylene exposure and NHL is also supported by increased risks of NHL found in several case-control studies (Hardell *et al.* 1994, Wang *et al.* 2009) and cohort studies (Morgan *et al.* 1998, Raaschou-Nielsen *et al.* 2003, Radican *et al.* 2008, Lipworth *et al.* 2011, Hansen *et al.* 2013). However, the evidence from these studies was not considered to be strong, because most of the studies (except for Wang *et al.* 2009) did not observe exposure-response relationships, and the estimated risks were relatively small or not statistically significant (NTP 2015). Nonetheless, these studies collectively contributed to the statistically significant increased risks found in the meta-analyses.

The remaining studies provided little evidence (Persson and Fredrikson 1999, Christensen *et al.* 2013, Bove *et al.* 2014) or no evidence (Bahr *et al.* 2011, Vlaanderen *et al.* 2013, Silver *et al.* 2014) for an association between trichloroethylene exposure and NHL. These studies were considered inadequate for detecting an association with an uncommon cancer like NHL, either because of inadequate methods for determining whether the subjects were exposed to trichloroethylene, or because of limited exposure information, or because few subjects were exposed (NTP 2015). The study of aerospace workers (Boice *et al.* 2006) was not informative, because it included only one trichloroethylene-exposed worker with NHL.

Few individual subtypes of NHL or related B-cell lymphomas have been studied with respect to trichloroethylene exposure. Among these specific types of lymphoma, the strongest evidence for an association with exposure to trichloroethylene is for chronic lymphocytic leukemia and follicular-cell lymphoma (Purdue *et al.* 2011, Cocco *et al.* 2013).

#### *Liver Cancer*

The data available from studies in humans are inadequate to evaluate the relationship between trichloroethylene exposure and liver cancer (NTP 2015). The available database for liver cancer included twelve cohort or nested case-control studies (Greenland *et al.* 1994, Morgan *et al.* 1998, Ritz 1999, Raaschou-Nielsen *et al.* 2003, Boice *et al.* 2006, Radican *et al.* 2008, Bahr *et al.* 2011, Lipworth *et al.* 2011, Hansen *et al.* 2013, Vlaanderen *et al.* 2013, Bove *et al.* 2014, Silver *et al.* 2014) and two meta-analyses (Alexander *et al.* 2007, Scott and Jinot 2011). The only available case-control study (Christensen *et al.* 2013) was not informative, because it included only one trichloroethylene-exposed worker with liver cancer. The epidemiological data suggest that trichloroethylene may be associated with a modest increase in the risk of liver cancer, based primarily on the two meta-analyses. However, the findings are inconsistent across studies, and there was little evidence for exposure-response relationships in the individual studies or the meta-analyses. In addition, the role of chance or confounding by one or more common occupational co-exposures or lifestyle factors could not be completely ruled out.

### *Cancer Studies in Experimental Animals*

Trichloroethylene caused tumors in mice and rats at several different tissue sites by two different routes of exposure. In rats, exposure to trichloroethylene by inhalation or stomach tube caused kidney cancer (tubular adenocarcinoma) and testicular tumors (interstitial-cell tumors) in males (Maltoni *et al.* 1988, NTP 1988, 1990). In mice, exposure to trichloroethylene by inhalation or stomach tube caused benign and malignant liver tumors (hepatocellular adenoma and carcinoma) in both sexes (NCI 1976, Maltoni *et al.* 1988, NTP 1990, IARC 1995), and inhalation exposure also caused lung tumors in both sexes and lymphoma in females (Henschler *et al.* 1980, IARC 1995).

### *Studies on Mechanisms of Carcinogenesis*

Two distinct metabolic pathways for trichloroethylene have been identified that have been found in all mammalian species studied: oxidation by a cytochrome P450 (CYP) enzyme and binding (conjugation) by the compound glutathione (GSH). These two metabolic pathways usually modify substances that enter the body to make them less toxic and more likely to be eliminated by excretion. However, in some cases (as with trichloroethylene), the chemical modifications can instead make the substance more toxic. Although the GSH-conjugation and CYP-oxidation pathways operate in parallel, the oxidative pathway predominates in all species studied (Lash *et al.* 2014). However, genetic differences among individuals or exposure to substances that induce or inhibit the action of CYP can alter the balance between oxidation and GSH conjugation of trichloroethylene, and their effects may be greater at higher trichloroethylene concentrations. Kidney cancer most likely occurs as a result of GSH conjugation of trichloroethylene (EPA 2011, Rusyn *et al.* 2014), whereas liver cancer most likely occurs as a result of the CYP-oxidation pathway (EPA 2011, Rusyn *et al.* 2014). Trichloroethylene metabolites most likely play a role in the development of non-Hodgkin lymphoma; however, less information is available for this type of cancer.

#### *Kidney Cancer*

Toxicokinetic and mechanistic data in both humans and experimental animals provide evidence for a biologically plausible mechanism by which trichloroethylene causes kidney cancer (NTP 2015). The evidence indicates that trichloroethylene causes genotoxicity (such as DNA and chromosomal damage) and cytotoxicity as a result of being metabolized into products that can damage DNA or cells (EPA 2011, Chiu *et al.* 2013, Lash *et al.* 2014). The following key events are likely involved: (1) GSH-conjugated metabolites are either produced in the kidneys or produced elsewhere in the body and transported to the kidneys in the blood, and (2) these metabolites cause mutations, genetic damage, and toxicity in the kidneys (EPA 2011), leading to cancer. Evidence for this proposed mechanism comes from data in both humans and experimental animals. Kidney and liver cells from humans and rodents are able to metabolize trichloroethylene to several GSH-conjugation-derived metabolites, two of which (*N*-acetyl-*S*-dichlorovinyl-L-cysteine and *S*-[2,2-dichlorovinyl]glutathione) have been detected in the urine of humans and experimental animals ex-

posed to trichloroethylene. The importance of the GSH-conjugation pathway in humans is supported by the finding of a significantly increased risk of kidney cancer among trichloroethylene-exposed individuals with a functioning gene that codes for the production of glutathione S-transferase theta 1 (GSTT1) (an enzyme involved in GSH conjugation), but not among individuals who cannot produce GSTT1 (Moore *et al.* 2010).

GSH-conjugated metabolites of trichloroethylene have been shown to cause genetic damage to cultured cells from both humans and experimental animals, most notably kidney cells, to transform cultured rat kidney cells into cancer cells, and to damage DNA and cause micronucleus formation in kidney cells from rats exposed to trichloroethylene. Also potentially contributing to trichloroethylene's carcinogenicity are its toxicity to cells and the associated increase in proliferation of cells in response to cell death (EPA 2011). Studies in humans also provide evidence that trichloroethylene causes kidney toxicity (Brüning *et al.* 1999a,b, Brüning and Bolt 2000, Bolt *et al.* 2004, Vermeulen *et al.* 2012), supporting the relevance of this mechanism in humans.

This proposed mechanism is also consistent with the findings of increased risk of kidney cancer mainly among workers with high exposure to trichloroethylene. Some of the mixed results across studies could be partly explained by differences among the study populations in genetic characteristics or co-exposures to substances that could affect trichloroethylene metabolism and the balance between the GSH-conjugation and CYP-oxidation metabolic pathways (NTP 2015).

### *Non-Hodgkin Lymphoma*

The mechanisms by which trichloroethylene could cause lymphoma are largely unknown. Immune disorders, including suppression of immune function and attacks on the body by its own immune system (autoimmunity), are strongly linked to NHL (Hardell *et al.* 1998, Baecklund *et al.* 2014, Ponce *et al.* 2014). In both types of immune disorder, chronic stimulation of the immune system can activate B cells to produce antibodies against host antigens (autoimmunity) or pathogens (immunosuppression) through a mechanism (somatic hypermutation) in which the B cells undergo complex rearrangements of their DNA to code for these antibodies. Because this process is subject to errors, it is proposed that the increased activation of the B cells can lead to a proliferation of mutated cells, possibly resulting in lymphoma (NTP 2015). Although trichloroethylene or its metabolites can cause both immunosuppression and autoimmunity in humans and animals (EPA 2011), the results from studies in humans and animals that measured indicators of immune function (such as B-cell activation) were not entirely consistent with this hypothesis (Peden-Adams *et al.* 2006, 2008, Keil *et al.* 2009, Lan *et al.* 2010, Hosgood *et al.* 2012, Bassig *et al.* 2013). However, neither the proposed model nor the potential association between lymphoma and trichloroethylene-induced immune effects has been directly tested in either humans or animals.

### *Liver Cancer*

The mechanism by which trichloroethylene causes liver cancer in mice is unknown, but likely is complex, involving key events in several molecular pathways (EPA 2011). Studies in experimental animals provide evidence for several potential modes of action, including genetic damage, oxidative stress, peroxisome proliferation, epigenetic events, and autoimmune hepatitis (EPA 2011, Wang *et al.* 2013). Oxidative metabolites are considered to be more important than GSH-conjugated metabolites in liver carcinogenicity, because trichloroethylene and its oxidative metabolites trichloroacetic acid, dichloroacetic acid, and chloral hydrate have similar toxic effects on the liver. These oxidative metabolites are formed in humans and have been reported to cause genetic damage in several *in vitro* and *in vivo* test systems. Although species differences in sensitivity to the proposed modes of action are likely, no data suggest that trichloroethylene causes liver tumors in mice by mechanisms that are not relevant to humans (NTP 2015).

## Properties

Trichloroethylene is a halogenated alkene that exists at room temperature as a clear, colorless, or blue freely flowing liquid with an ethereal odor. It is slightly soluble in water, soluble in ethanol, acetone, diethyl ether, and chloroform, and miscible in oil. It is relatively stable, but oxidizes slowly when exposed to sunlight in air (HSDB 2014). Upon combustion, trichloroethylene produces irritants and toxic gases, which may include hydrogen chloride. In the presence of moisture and light, it breaks down into hydrochloric acid. Physical and chemical properties of trichloroethylene are listed in the following table.

| Property | Information |
|---|---|
| Molecular weight | 131.4 |
| Specific gravity | 1.4642 at 20°C/4°C |
| Melting point | –84.7°C |
| Boiling point | 87.2°C |
| Log $K_{ow}$ | 2.61 |
| Water solubility | 1.28 g/L at 25°C |
| Vapor pressure | 69 mm Hg at 25°C |
| Vapor density relative to air | 4.53 |

Source: HSDB 2014.

## Use

Trichloroethylene is used as an intermediate in production of hydrofluorocarbon refrigerant (83.6%) and as a degreaser for metal parts (14.7%) (EPA 2014a). The remaining 1.7% is attributed to "other uses," which include use as a spot-removal solvent in the drycleaning industry, as a modifier in polyvinyl chloride polymerization, and in several consumer household aerosol products. Starting in the early 1900s, trichloroethylene was primarily used as a degreaser, to remove grease, wax, or dirt from metal parts before painting, plating, or other processes; however, that use in the United States declined beginning in the 1970s (Bakke *et al.* 2007). Although trichloroethylene was used extensively as a degreaser in the aircraft industry beginning in the 1950s, this use ended in the 1980s. Industries that may currently use trichloroethylene in vapor or cold degreasing operations include fabricated metal products, electrical and electronic equipment, transportation equipment, and miscellaneous manufacturing. Trichloroethylene has also been used as an industrial solvent in the rubber industry; in paints, lacquers, varnishes, adhesives, and paint strippers; and in the production of agricultural chemicals such as fungicides and insecticides (IARC 1995, Bakke *et al.* 2007).

Trichloroethylene has been listed as a major ingredient in several consumer products, such as degreasers intended for use in automotive products, home maintenance, or commercial or institutional use and household aerosol products for arts and crafts uses (HPD 2014, EPA 2014b). However, the only U.S. manufacturer of trichloroethylene-containing spray fixatives used in arts and crafts has voluntarily reformulated the products to replace trichloroethylene with an alternative chemical (EPA 2016). Other consumer products identified as containing trichloroethylene include typewriter correction fluids, paint removers and strippers, adhesives, spot removers, and rug-cleaning fluids (Gist and Burg 1995).

In the past, trichloroethylene was used as a drycleaning agent in a batch process for cleaning large quantities of textiles; as an ex-

traction solvent to remove natural fats and oils from plant materials, to manufacture flavoring extracts from spices and hops, and to remove caffeine from coffee; as an anesthetic and analgesic in obstetrics and for minor surgical procedures; and in cosmetics and drug products. However, its use essentially ended by the 1950s for batch drycleaning (although uses for spot cleaning have continued) and by the 1970s for the other food, drug, cosmetic, and surgical uses (IARC 1995, Bakke *et al.* 2007).

## Production

Trichloroethylene is a high-production-volume chemical commercially produced by 21 companies worldwide, including two in the United States (SRI 2011). The two U.S. producers of trichloroethylene were reported to have a total capacity of 330 million pounds in 2002 (ICIS 2005). In 2014, trichloroethylene was available from 101 suppliers worldwide, including 37 U.S. suppliers (ChemSources 2014). Recent volumes of U.S. trichloroethylene production, imports, and exports are listed in the following table.

| Category | Year | Quantity (million lb) |
|---|---|---|
| Production + imports[a] | 2012 | 225 |
| U.S. imports[b] | 2013 | 2.4 |
| U.S. exports[b] | 2013 | 25.5 |

Sources: [a]EPA 2014a. [b]USITC 2014.

U.S. imports of trichloroethylene generally increased from 1989 to 2007, reaching an all-time high of 27.2 million kilograms (60 million pounds) in 2007, but imports decreased steadily to less than 5% of that level for 2010 to 2013 (USITC 2014). Between 1989 and 2012, U.S. exports of trichloroethylene ranged from a low of 16.6 million kilograms (36.7 million pounds) in 2005 to a high of 48.7 million kilograms (107.4 million pounds) in 1992, showing no consistent trends over that period.

Stabilizers, in the form of antioxidants or acid receptors (such as phenolic, olefinic, pyrrolic, or oxiranic derivatives and aliphatic amines), are usually added to commercial trichloroethylene in concentrations that normally range from 20 to 600 mg/kg but may be as high as 5,000 mg/kg. Which stabilizers are used depends on patent ownership and technical specifications (IPCS 1985).

Trichloroethylene is reported to occur naturally in some algae in temperate to tropical climates and in one red macroalga (IARC 1995).

## Exposure

A significant number of people living in the United States are or have been exposed to trichloroethylene because of its widespread presence from past and current uses. In addition to occupational exposure of workers in industries using trichloroethylene, the general population can be exposed to trichloroethylene in ambient air, drinking-water supplies, certain consumer products, and contaminated foods (ATSDR 1997, 2013). Exposure has been documented by measurement of trichloroethylene blood levels in the general population and by direct measurement of trichloroethylene in groundwater, drinking water, and ambient air in workplace and non-workplace environments. However, recent measurements of trichloroethylene blood levels in the general population suggest an overall decrease in exposure. Several additional lines of evidence support this trend, including changes in major uses of trichloroethylene that suggest decreases in both the numbers of exposed workers and occupational exposure levels, particularly the decreased use of trichloroethylene for solvent degreasing in large commercial and industrial settings; recent decreases in total imports of trichloroethylene; and declining environmental releases of trichloroethylene. Nevertheless, exposure to workers and also to the general population from current use, past use, or disposal of trichloroethylene continues, as evidenced by measurements in the environment.

### *Occupational Exposure*

Workers are exposed to trichloroethylene primarily by breathing vapors and through skin contact with vapors or liquid. According to the U.S. Environmental Protection Agency's (EPA's) Office of Chemical Safety and Pollution Prevention (EPA 2014b), an estimated 300,000 workers are exposed in drycleaning facilities that use trichloroethylene to remove spots from garments before or after cleaning them in drycleaning machines. An estimated 30,000 additional workers are potentially exposed to trichloroethylene at small commercial degreasing operations. EPA considered production of hydrofluorocarbon refrigerant and solvent degreasing in large commercial and industrial settings to have low potential for human exposure to trichloroethylene because of the use of closed-loop process systems and regulatory monitoring and controls (EPA 2014b).

Exposure in occupational settings such as solvent degreasing in large commercial or industrial facilities has decreased over time as a result of regulatory monitoring and controls, but high levels of trichloroethylene in workplace air have still been reported in recent years, particularly in the metals and rubber industries. Based on 1,229 trichloroethylene measurements across industries from the 1930s to 1990s, an average exposure concentration of 38.2 ppm was calculated, and the highest levels were reported for vapor degreasing (44.6 ppm) (Bakke *et al.* 2007). In statistical modeling of trichloroethylene exposures over time, the highest values were found in the 1950s to 1970s (Hein *et al.* 2010). The Occupational Safety and Health Administration (OSHA) Chemical Exposure Health Database (which includes results for 3,600 air samples from 1984 to 2011) reported that from 2000 to 2010, 92 samples exceeded the OSHA permissible exposure limit of 100 ppm, and two exceeded the National Institute for Occupational Safety and Health "immediately dangerous to life or health" level of 1,000 ppm (OSHA 2013).

### *Exposure of the General Population*

The general population is exposed to trichloroethylene primarily by consuming contaminated drinking water and inhaling contaminated indoor air. Water and air become contaminated by releases of trichloroethylene from active industries or from hazardous waste sites. Exposure from consumer products and food also is possible. Results from the third National Health and Nutrition Examination Survey (NHANES), conducted from 1988 to 1994 (in which 677 whole-blood samples were tested for trichloroethylene), suggested that approximately 10% of the U.S. population had detectable levels of trichloroethylene in their blood (limit of detection = 0.01 ng/mL) (Wu and Schaum 2000). However, the NHANES survey data for 2001 to 2002 (922 samples), 2003 to 2004 (1,228 samples), 2005 to 2006 (3,178 samples), and 2007 to 2008 (2,952 samples) detected trichloroethylene in the blood of less than 5% of people in all age groups, genders, and races or ethnicities studied in the surveys (CDC 2009a,b, 2011, 2016).

### *Environmental Releases*

According to EPA's Toxics Release Inventory (TRI) database, environmental releases of trichloroethylene from 211 U.S. facilities in 2011 totaled 2.3 million pounds (TRI 2014). Based on historical TRI data, environmental releases of trichloroethylene have declined by more than 95% since 1988, when over 57 million pounds were released. Trichloroethylene is a common groundwater and drinking-water contaminant (Gist and Burg 1995, IARC 1995, ATSDR 1997, 2013, Heneghan 2000, Wu and Schaum 2000). Trichloroethylene tends to

be persistent in groundwater, because its rate of biodegration is relatively slow, with a half-life of months to years (Howard *et al.* 1991). Industrial wastewater is a source of trichloroethylene released into surface-water systems. Trichloroethylene background levels in 1995 were 0.001 ppb (µg/L) in the Gulf of Mexico, 0.007 ppb in the northeastern Atlantic Ocean, and 0.0008 to 0.039 ppb in rainwater and snow (Gist and Burg 1995). In EPA's Contract Laboratory Program Statistical Database, trichloroethylene was reported in about 3% of surface-water samples and 19% of groundwater samples (IARC 1995). Based on its past widespread use for industrial and maintenance processes (e.g., as a metal degreasing agent) at U.S. military installations, trichloroethylene is also a common groundwater contaminant at military sites (NRC 2006, 2009). For example, the U.S. Marine Corps discovered in 1982 that the water supply from a water-treatment plant at Camp Lejeune, North Carolina, contained trichloroethylene, as a result of contamination of supply wells from leaking underground storage tanks, industrial area spills, and waste-disposal sites (ATSDR 2016). Exposure to contaminated groundwater can also potentially occur near National Priorities List (Superfund) sites, and trichloroethylene has been found in at least 1,045 of the 1,699 sites identified by EPA (ATSDR 2015).

#### *Trichloroethylene in Tap Water*

Based on a trichloroethylene concentration of 3.0 µg/L in drinking water (the median concentration in a large California water survey) and daily water consumption of 2 L, average daily trichloroethylene exposure through drinking water was estimated as 6 µg (Wu and Schaum 2000), which is consistent with the Agency for Toxic Substances and Disease Registry's estimate of 2 to 20 µg for daily exposure of the general population (ATSDR 1997). EPA has set a maximum contaminant level for trichloroethylene of 0.005 mg/L (5 µg/L) for drinking water (see Regulations).

Trichloroethylene volatilizes readily from contaminated tap water, and inhalation exposure to volatilized trichloroethylene may equal or exceed the exposure from ingestion of contaminated drinking water. One study estimated that inhalation exposure from a 10-minute shower in trichloroethylene-contaminated water would equal the exposure expected from drinking the contaminated water (McKone and Knezovich 1991), and another study (Weisel and Jo 1996) determined that approximately equal amounts of trichloroethylene entered the body via inhalation, absorption through the skin, and ingestion during typical daily activities where contaminated tap water was used for drinking and bathing (including showering). However, a modeling study of trichloroethylene exposure of workers showering with trichloroethylene-contaminated water at a metal degreasing facility (Franco *et al.* 2007) estimated that dermal exposure contributed more than inhalation exposure to carcinogenic risk.

#### *Trichloroethylene in Indoor and Outdoor Air*

In studies of the concentration of trichloroethylene in air conducted since the 1980s, trichloroethylene levels are generally lower in recent samples, consistent with the overall decrease in releases to the air and in blood levels in the general population. According to monitoring data from EPA's Air Quality System, trichloroethylene levels in ambient air remained fairly constant from 1999 to 2006, with a mean level of approximately 0.3 µg/m$^3$ (0.000056 ppm); however, the data were not from a statistically based survey and may not be nationally representative (EPA 2011). As part of the Minnesota Children's Pesticide Exposure Study, personal, indoor-air, and outdoor-air trichloroethylene concentrations were measured from May to September 1997 in 284 households with children. The median values for indoor, outdoor, and personal sampling were all between 0.5 and 1 µg/m$^3$ (0.00009 and 0.0002 ppm) (Adgate *et al.* 2004). Trichloroethylene concentrations in ambient air were also measured during EPA's large-scale Total Exposure Assessment Methodology studies conducted in Maryland, New Jersey, and California from 1981 through 1987 (Wallace *et al.* 1996). Median personal trichloroethylene exposure concentrations measured with personal air monitors carried by 750 individuals for 24 hours ranged from 0.3 to 3.0 µg/m$^3$ (0.00006 to 0.0006 ppm).

Migration of volatile chemicals from the subsurface into overlying buildings (vapor intrusion) likely makes an important contribution to indoor air levels of trichloroethylene in offices or residences located near soil or groundwater with high contamination levels (EPA 2011). Contamination of residential indoor air could also result from release of trichloroethylene directly from consumer products into the indoor environment (McHugh *et al.* 2011); however, some previously marketed consumer products have been reformulated to replace trichloroethylene (as discussed under Use, above). Environmental occurrences of trichloroethylene have been reported in locations near sites of past use or disposal (e.g., Superfund sites). Elevated levels of trichloroethylene in indoor air at Superfund sites were reported for office buildings in Mountain View, California (Rust and Drange 2013), and homes in Asheville, North Carolina (Morrison 2014). Trichloroethylene concentrations were as high as 110 µg/m$^3$ in office buildings at the Mountain View site when the heating, ventilation, and air conditioning system was not operating (Welt and Bice 2013) and 14 µg/m$^3$ in the basement of a house at the Asheville site.

#### *Trichloroethylene in Consumer Products or Food*

Trichloroethylene has been a major ingredient in several consumer products. For example, it constituted 80% to 100% of three aerosol spray fixative products for arts and crafts uses and other products intended for use as cleaners or degreasers in automobile or home maintenance (EPA 2014b, HPD 2014). However, an EPA risk assessment (EPA 2014b) was not able to estimate the numbers of consumers or bystanders exposed to trichloroethylene from arts and crafts spray products or degreasers.

The U.S. Food and Drug Administration (FDA) Total Diet Study identified 72 food items containing trichloroethylene, including fruits, beverages, and many foods prepared with oils and fats. The highest mean concentration (0.012 ppm) was found in samples of raw avocado (FDA 2006). Other studies also have found trichloroethylene in a variety of foods, with the highest levels in meats and margarine. Although trichloroethylene has not been used as a solvent for extraction of natural fats and oils, spices, hops, or caffeine (from coffee) since the FDA imposed limitations on these uses in 1977, foods can still be contaminated with trichloroethylene through the use of contaminated water in food processing or the use of food-processing equipment cleaned with trichloroethylene (ATSDR 1997).

## Regulations

***Coast Guard, Department of Homeland Security***

Minimum requirements have been established for safe transport of trichloroethylene on ships and barges.

***Department of Transportation (DOT)***

Trichloroethylene is considered a hazardous material, and special requirements have been set for marking, labeling, and transporting this material.

***Environmental Protection Agency (EPA)***

*Clean Air Act*

*National Ambient Air Quality Standards:* Controlled as a volatile organic compound under state regulations implementing the standards for ozone.

*National Emission Standards for Hazardous Air Pollutants:* Listed as a hazardous air pollutant.

*National Volatile Organic Compound Emission Standards for Aerosol Coatings:* Trichloroethylene is included in product-weighted reactivity (PWR) calculations of aerosol coating products (trichloroethylene reactivity factor = 0.60 g $O_3$/g VOC).
*New Source Performance Standards:* Manufacture of trichloroethylene is subject to certain provisions for the control of volatile organic compound emissions.
*Urban Air Toxics Strategy:* Identified as one of 33 hazardous air pollutants that present the greatest threat to public health in urban areas.

### *Clean Water Act*

Designated a hazardous substance.
*Effluent Guidelines:* Listed as a toxic pollutant.
*Water Quality Criteria:* Based on fish or shellfish and water consumption = 2.5 µg/L; based on fish or shellfish consumption only = 30 µg/L.

### *Comprehensive Environmental Response, Compensation, and Liability Act*

Reportable quantity (RQ) = 100 lb.

### *Emergency Planning and Community Right-To-Know Act*

*Toxics Release Inventory:* Listed substance subject to reporting requirements.

### *Resource Conservation and Recovery Act*

*Characteristic Hazardous Waste:* Toxicity characteristic leaching procedure (TCLP) threshold = 0.5 mg/L.
*Listed Hazardous Waste:* Waste codes for which the listing is based wholly or partly on the presence of trichloroethylene = U228, F001, F002, F024, F025, K018, K019, K020.
Listed as a hazardous constituent of waste.

### *Safe Drinking Water Act*

Maximum contaminant level (MCL) = 0.005 mg/L.

### *Toxic Substances Control Act*

Manufacturers (including importers) or processors of trichloroethylene for use in a consumer product except for use in cleaners and solvent degreasers, film cleaners, hoof polishes, lubricants, mirror edge sealants, and pepper spray are required to notify EPA at least 90 days before commencing, to allow EPA to evaluate the intended use and to regulate prospective manufacturers or processors of trichloroethylene before the use occurs, provided that regulation is warranted under Toxic Substance Control Act section 5(e), 5(f), 6, or 7.

### *Food and Drug Administration (FDA)*

Maximum permissible level in bottled water = 0.005 mg/L.
Trichloroethylene may be used as a solvent in the manufacture of modified hop extract provided the residue does not exceed 150 ppm.
Trichloroethylene may be used as a solvent in the manufacture of specified foods, with maximum residue levels ranging from 10 to 30 ppm.

### *Occupational Safety and Health Administration (OSHA)*

This legally enforceable PEL was adopted from the United States of America Standards Institute (USAI) (later the American National Standards Institute, ANSI) shortly after OSHA was established. The PEL may not reflect the most recent scientific evidence and may not adequately protect worker health.
Permissible exposure limit (PEL) = 100 ppm (8-h TWA).
Acceptable ceiling concentration = 200 ppm.
Acceptable maximum peak above the acceptable ceiling concentration = 300 ppm (5 min in any 2 h).

## Guidelines

### *American Conference of Governmental Industrial Hygienists (ACGIH)*

Threshold limit value – time-weighted average (TLV-TWA) = 10 ppm.
Threshold limit value – short-term exposure limit (TLV-STEL) = 25 ppm.
Biological exposure indices: Trichloroacetic acid in urine (end of shift at end of workweek) = 15 mg/L; trichloroethanol in blood (without hydrolysis; *n*-hexane, methyl *n*-butyl ketone, and trichloroethylene, end of shift at end of workweek) = 0.5 mg/L.

### *Environmental Protection Agency (EPA)*

Integrated Risk Information System (IRIS) oral reference dose (RfD) = 0.0005 mg/kg b.w. per day.
IRIS inhalation reference concentration (RfC) = 0.0004 ppm [0.4 ppb, or 2 µg/m$^3$].
IRIS oral cancer slope factor = $5 \times 10^{-2}$ per mg/kg b.w. per day.
IRIS inhalation unit risk = $2 \times 10^{-2}$ per ppm [$4 \times 10^{-6}$ per µg/m$^3$].
*Regional Screening Levels* (formerly Preliminary Remediation Goals): residential soil = 0.44 mg/kg; industrial soil = 2.0 mg/kg; residential air = 0.21 µg/m$^3$; industrial air = 0.88 µg/m$^3$; tap water = 0.26 µg/L; maximum contaminant level (MCL) = 5.0 µg/L.

### *National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL)*

*Interim acute exposure guideline levels (AEGL):* AEGL-1 (non-disabling) = 77 ppm; AEGL-2 (disabling) = 240 ppm; AEGL-3 (lethal) = 970 ppm (8-h TWAs). These AEGLs are available for use as deemed appropriate on an interim basis by federal and state regulatory agencies and the private sector.

### *National Institute for Occupational Safety and Health (NIOSH)*

Recommended exposure limit (REL) = 25 ppm (10-h TWA).
Ceiling recommended exposure limit = 2 ppm (60-min ceiling) during use as an anesthetic agent.
Immediately dangerous to life and health (IDLH) limit = 1,000 ppm.

## References


Adgate JL, Eberly LE, Stroebel C, Pellizzari ED, Sexton K. 2004. Personal, indoor, and outdoor VOC exposures in a probability sample of children. *J Expo Anal Environ Epidemiol* 14(Suppl 1): S4-S13.

Alexander DD, Kelsh MA, Mink PJ, Mandel JH, Basu R, Weingart M. 2007. A meta-analysis of occupational trichloroethylene exposure and liver cancer. *Int Arch Occup Environ Health* 81(2): 127-143.

ATSDR. 1997. *Toxicological Profile for Trichloroethylene.* Atlanta, GA: Agency for Toxic Substances and Disease Registry. 335 pp.

ATSDR. 2013. *Addendum to the Toxicological Profile for Trichloroethylene.* Atlanta, GA: Agency for Toxic Substances and Disease Registry. 120 pp.

ATSDR. 2015. *ToxFAQs for Trichloroethylene (TCE).* Agency for Toxic Substances and Disease Registry. Last updated: 5/12/15. http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=172&tid=30.

ATSDR. 2016. *Camp Lejeune, North Carolina: Background.* Agency for Toxic Substances and Disease Registry. Last updated 5/5/16. http://www.atsdr.cdc.gov/sites/lejeune/background.html.

Baecklund E, Smedby KE, Sutton LA, Askling J, Rosenquist R. 2014. Lymphoma development in patients with autoimmune and inflammatory disorders ---- what are the driving forces? *Semin Cancer Biol* 24: 61-70.

Bahr DE, Aldrich TE, Seidu D, Brion GM, Tollerud DJ, Muldoon S, *et al.* 2011. Occupational exposure to trichloroethylene and cancer risk for workers at the Paducah Gaseous Diffusion Plant. *Int J Occup Med Environ Health* 24(1): 67-77.

Bakke B, Stewart PA, Waters MA. 2007. Uses of and exposure to trichloroethylene in US industry: A systematic literature review. *J Occup Environ Hyg* 4(5): 375-390.

Bassig BA, Zhang L, Tang X, Vermeulen R, Shen M, Smith MT, *et al.* 2013. Occupational exposure to trichloroethylene and serum concentrations of IL-6, IL-10, and TNF-alpha. *Environ Mol Mutagen* 54(6): 450-454.

Boice JD Jr, Marano DE, Cohen SS, Mumma MT, Blot WJ, Brill AB, Fryzek JP, Henderson BE, McLaughlin JK. 2006. Mortality among Rocketdyne workers who tested rocket engines, 1948-1999. *J Occup Environ Med* 48(10): 1070-1092.

Bolt HM, Lammert M, Selinski S, Brüning T. 2004. Urinary $\alpha_1$-microglobulin excretion as biomarker of renal toxicity in trichloroethylene-exposed persons. *Int Arch Occup Environ Health* 77(3): 186-190.

Bove FJ, Ruckart PZ, Maslia M, Larson TC. 2014. Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: A retrospective cohort study. *Environ Health* 13(1): 13 pp.

Brüning T, Bolt HM. 2000. Renal toxicity and carcinogenicity of trichloroethylene: Key results, mechanisms, and controversies. *Crit Rev Toxicol* 30(3): 253-285.

Brüning T, Mann H, Melzer H, Sundberg AG, Bolt HM. 1999a. Pathological excretion patterns of urinary proteins in renal cell cancer patients exposed to trichloroethylene. *Occup Med (Lond)* 49(5): 299-305.

Brüning T, Sundberg AG, Birner G, Lammert M, Bolt HM, Appelkvist EL, Nilsson R, Dallner G. 1999b. Glutathione transferase alpha as a marker for tubular damage after trichloroethylene exposure. *Arch Toxicol* 73(4-5): 246-254.

Brüning T, Pesch B, Wiesenhütter B, Rabstein S, Lammert M, Baumüller A, Bolt HM. 2003. Renal cell cancer risk and occupational exposure to trichloroethylene: Results of a consecutive case-control study in Arnsberg, Germany. *Am J Ind Med* 43(3): 274-285.

CDC. 2009a. *2001 - 2002 Data Documentation, Codebook, and Frequencies: Volatile Organic Compounds in Blood and Water.* National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention. http://wwwn.cdc.gov/nchs/nhanes/2001-2002/L04VOC_B.htm.

CDC. 2009b. *2003 - 2004 Data Documentation, Codebook, and Frequencies: Volatile Organic Compounds in Blood and Water.* National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention. http://wwwn.cdc.gov/nchs/nhanes/2003-2004/L04VOC_C.htm.

CDC. 2011. *2005 - 2006 Data Documentation, Codebook, and Frequencies: Volatile Organic Compounds in Blood.* National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention. http://wwwn.cdc.gov/nchs/nhanes/2005-2006/VOCWB_D.htm.

CDC. 2016. 2007 - *2008 Data Documentation, Codebook, and Frequencies: Volatile Organic Compounds in Blood.* National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention. https://wwwn.cdc.gov/nchs/nhanes/2007-2008/VOCWB_E.htm.

Charbotel B, Fevotte J, Hours M, Martin JL, Bergeret A. 2006. Case-control study on renal cell cancer and occupational exposure to trichloroethylene. Part II: Epidemiological aspects. *Ann Occup Hyg* 50(8): 777-787.

Charbotel B, Fevotte J, Martin JL, Bergeret A. 2009. Renal cell carcinoma and exposure to trichloroethylene: Are French occupational exposure limits relevant? *Rev Epidemiol Santé Publique* 57(1): 41-47.

ChemSources. 2014. *Chem Sources - Chemical Search.* Chemical Sources International. http://www.chemsources.com/chemonline.html and search on substance name. Last accessed: 6/19/14.

Chiu WA, Jinot J, Scott CS, Makris SL, Cooper GS, Dzubow RC, *et al.* 2013. Human health effects of trichloroethylene: Key findings and scientific issues. *Environ Health Perspect* 121(3): 303-311.

Christensen KY, Vizcaya D, Richardson H, Lavoué J, Aronson K, Siemiatycki J. 2013. Risk of selected cancers due to occupational exposure to chlorinated solvents in a case-control study in Montreal. *J Occup Environ Med* 55(2): 198-208.

Cocco P, Vermeulen R, Flore V, Nonne T, Campagna M, Purdue M, *et al.* 2013. Occupational exposure to trichloroethylene and risk of non-Hodgkin lymphoma and its major subtypes: A pooled InterLymph analysis. *Occup Environ Med* 70: 795-802.

Deng Q, Zheng T, Lan Q, Lan Y, Holford T, Chen Y, *et al.* 2013. Occupational solvent exposure, genetic variation in immune genes, and the risk for non-Hodgkin lymphoma. *Eur J Cancer Prev* 22(1): 77-82.

Dosemeci M, Cocco P, Chow WH. 1999. Gender differences in risk of renal cell carcinoma and occupational exposures to chlorinated aliphatic hydrocarbons. *Am J Ind Med* 36(1): 54-59.

EPA. 2011. *Toxicological Review of Trichloroethylene (CAS No. 79-01-6) in Support of Summary Information on the Integrated Risk Information System (IRIS).* EPA/635/R-09/011F. U.S. Environmental Protection Agency. 1200 pp.

EPA. 2014a. *2012 Chemical Data Reporting.* U.S. Environmental Protection Agency. Last updated: 7/23/14. http://java.epa.gov/oppt_chemical_search and search by CAS number.

EPA. 2014b. *TSCA Work Plan Chemical Risk Assessment. Trichloroethylene: Degreasing, Spot Cleaning and Arts & Crafts Uses.* EPA 740-R1-4002. Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention. 212 pp.

EPA. 2016. EPA takes action to reduce exposure to TCE in art and crafts spray fixatives [news release]. U.S. Environmental Protection Agency. Last updated: 4/18/16. https://www.epa.gov/newsreleases/epa-takes-action-reduce-exposure-tce-art-and-crafts-spray-fixatives.

FDA. 2006. *U.S. Food and Drug Administration Total Diet Study: Market Baskets 1991-3 through 2003-4.* College Park, MD: U.S. Food and Drug Administration. 127 pp.

Franco A, Costoya MA, Roca E. 2007. Estimating risk during showering exposure to VOCs of workers in a metal-degreasing facility. *J Toxicol Environ Health A* 70(7): 627-637.

Gist GL, Burg JR. 1995. Trichloroethylene — a review of the literature from a health-effects perspective. *Toxicol Ind Health* 11(3): 253-307.

Greenland S, Salvan A, Wegman DH, Hallock MF, Smith TJ. 1994. A case-control study of cancer mortality at a transformer-assembly facility. *Int Arch Occup Environ Health* 66(1): 49-54.

Hansen J, Sallmén M, Seldén AI, Anttila A, Pukkala E, Andersson K, *et al.* 2013. Risk of cancer among workers exposed to trichloroethylene: Analysis of three Nordic cohort studies. *J Natl Cancer Inst* 105(12): 869-877.

Hardell L, Eriksson M, Degerman A. 1994. Exposure to phenoxyacetic acids, chlorophenols, or organic solvents in relation to histopathology, stage, and anatomical localization of non-Hodgkin's lymphoma. *Cancer Res* 54(9): 2386-2389.

Hardell L, Lindström G, van Bavel B, Fredrikson M, Liljegren G. 1998. Some aspects of the etiology of non-Hodgkin's lymphoma. *Environ Health Perspect* 106(Suppl 2): 679-681.

Hein MJ, Waters MA, Ruder AM, Stenzel MR, Blair A, Stewart PA. 2010. Statistical modeling of occupational chlorinated solvent exposures for case-control studies using a literature-based database. *Ann Occup Hyg* 54(4): 459-472.

Heneghan AK. 2000. *The Legacy of Woburn, Massachusetts and Trichloroethylene.* Case study for Principles of Environmental Toxicology course, University of Idaho. 23 pp. http://www.webpages.uidaho.edu/etox/resources/case.studies/WOBURN.PDF.

Henschler D, Romen W, Elsasser HM, Reichert D, Eder E, Radwan Z. 1980. Carcinogenicity study of trichloroethylene by longterm inhalation in three animal species. *Arch Toxicol* 43(4): 237-248.

Henschler D, Vamvakas S, Lammert M, Dekant W, Kraus B, Thomas B, Ulm K. 1995. Increased incidence of renal cell tumors in a cohort of cardboard workers exposed to trichloroethene. *Arch Toxicol* 69(5): 291-299.

Hosgood HD 3rd, Zhang L, Tang X, Vermeulen R, Qiu C, Shen M, *et al.* 2012. Decreased numbers of CD4(+) naive and effector memory T cells, and CD8(+) naive T cells, are associated with trichloroethylene exposure. *Front Oncol* 1: 53.

HPD. 2014. *Household Products Database.* National Library of Medicine. http://hpd.nlm.nih.gov/ingredients.htm and search on CAS number. Last accessed: 10/6/14.

HSDB. 2014. *Hazardous Substances Data Bank.* National Library of Medicine. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB and search on CAS number or compound name. Last accessed: 5/14/14.

IARC. 1995. Trichloroethylene. In *Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals.* IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. 63. Lyon, France: International Agency for Research on Cancer. pp. 75-158.

ICIS. 2005. *Chemical Profile - Trichloroethylene.* ICIS Chemical Business. http://www.icis.com/resources/news/2005/12/02/177493/chemical-profile-trichloroethylene.

IPCS. 1985. *Environmental Health Criteria 50, Trichloroethylene.* International Programme on Chemical Safety. http://www.inchem.org/documents/ehc/ehc/ehc50.htm.

Karami S, Lan Q, Rothman N, Stewart PA, Lee KM, Vermeulen R, Moore LE. 2012. Occupational trichloroethylene exposure and kidney cancer risk: A meta-analysis. *Occup Environ Med* 69(12): 858-867.

Karami S, Bassig B, Stewart PA, Lee KM, Rothman N, Moore LE, Lan Q. 2013. Occupational trichloroethylene exposure and risk of lymphatic and haematopoietic cancers: A meta-analysis. *Occup Environ Med* 70(8): 591-599.

Keil DE, Peden-Adams MM, Wallace S, Ruiz P, Gilkeson GS. 2009. Assessment of trichloroethylene (TCE) exposure in murine strains genetically-prone and non-prone to develop autoimmune disease. *J Environ Sci Health A* 44(5): 443-453.

Lan Q, Zhang L, Tang X, Shen M, Smith MT, Qiu C, *et al.* 2010. Occupational exposure to trichloroethylene is associated with a decline in lymphocyte subsets and soluble CD27 and CD30 markers. *Carcinogenesis* 31(9): 1592-1596.

Lash LH, Chiu WA, Guyton KZ, Rusyn I. 2014. Trichloroethylene biotransformation and its role in mutagenicity, carcinogenicity and target organ toxicity. *Mutat Res* 762: 22-36.

Lipworth L, Sonderman JS, Mumma MT, Tarone RE, Marano DE, Boice JD, Jr., McLaughlin JK. 2011. Cancer mortality among aircraft manufacturing workers: An extended follow-up. *J Occup Environ Med* 53(9): 992-1007.

Maltoni C, Lefemine G, Cotti G, Perino G. 1988. Long-term carcinogenicity bioassays on trichloroethylene administered by inhalation to Sprague-Dawley rats and Swiss and B6C3F$_1$ mice. *Ann N Y Acad Sci* 534: 316-342.

McHugh T, Kuder T, Fiorenza S, Gorder K, Dettenmaier E, Philp P. 2011. Application of CSIA to distinguish between vapor intrusion and indoor sources of VOCs. *Environ Sci Technol* 45: 5952-5958.

McKone TE, Knezovich JP. 1991. The transfer of trichloroethylene (TCE) from a shower to indoor air: Experimental measurements and their implications. *J Air Waste Manage Assoc* 41(6): 832-837.

Moore LE, Boffetta P, Karami S, Brennan P, Stewart PS, Hung R, *et al.* 2010. Occupational trichloroethylene exposure and renal carcinoma risk: Evidence of genetic susceptibility by reductive metabolism gene variants. *Cancer Res* 70(16): 6527-6536.

Morgan RW, Kelsh MA, Zhao K, Heringer S. 1998. Mortality of aerospace workers exposed to trichloroethylene. *Epidemiology* 9(4): 424-431.

Morrison C. 2014. EPA to sample air for toxic chemicals near CTS site. *Asheville Citizen-Times* Jun 21. http://www.citizen-times.com/story/news/local/2014/06/21/epa-sample-air-toxic-chemicals-near-cts-site/11226271.

NCI. 1976. *Carcinogenesis Bioassay of Trichloroethylene.* Technical Report Series No. 2. DHEW (NIH) Publication No. 76-802. Bethesda, MD: National Institutes of Health. 225 pp.

NRC. 2006. *Assessing the Human Health Risks of Trichloroethylene: Key Scientific Issues.* National Research Council. Washington, DC: National Academies Press. 448 pp.

NRC. 2009. *Contaminated Water Supplies at Camp Lejeune: Assessing Potential Health Effects.* National Research Council. Washington, DC: National Academies Press. 338 pp.

NTP. 1988. *Toxicology and Carcinogenesis Studies of Trichloroethylene (CAS No. 79-01-6) in Four Strains of Rats (ACI, August, Marshall, Osborne-Mendel) (Gavage Studies).* Technical Report Series no. 273. Research Triangle Park, NC: National Toxicology Program. 303 pp.

NTP. 1990. *Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin) (CAS No. 79-01-6) in F344/N Rats and B6C3F$_1$ Mice (Gavage Studies).* Technical Report Series no. 243. Research Triangle Park, NC: National Toxicology Program. 176 pp.

NTP. 2015. *Report on Carcinogens Monograph on Trichloroethylene.* Research Triangle Park, NC: National Toxicology Program. 254 pp. http://ntp.niehs.nih.gov/ntp/roc/monographs/finaltce_508.pdf.

OSHA. 2013. *Chemical Exposure Health Data.* United States Department of Labor. https://www.osha.gov/opengov/healthsamples.html and search on substance name. Last accessed: 6/11/13.

Peden-Adams MM, Eudaly JG, Heesemann LM, Smythe J, Miller J, Gilkeson GS, Keil DE. 2006. Developmental immunotoxicity of trichloroethylene (TCE): studies in B6C3F$_1$ mice. *J Environ Sci Health A Tox Hazard Subst Environ Eng* 41(3): 249-271.

Peden-Adams MM, Eudaly JG, Lee AM, Miller J, Keil DE, Gilkeson GS. 2008. Lifetime exposure to trichloroethylene (TCE) does not accelerate autoimmune disease in MRL +/+ mice. *J Environ Sci Health A Tox Hazard Subst Environ Eng* 43(12): 1402-1409.

Persson B, Fredrikson M. 1999. Some risk factors for non-Hodgkin's lymphoma. *Int J Occup Med Environ Health* 12(2): 135-142.

Pesch B, Haerting J, Ranft U, Klimpel A, Oelschlagel B, Schill W, *et al.* 2000. Occupational risk factors for renal cell carcinoma: Agent-specific results from a case-control study in Germany. *Int J Epidemiol* 29(6): 1014-1024.

Ponce RA, Gelzleichter T, Haggerty HG, Heidel S, Holdren MS, Lebrec H, Mellon RD, Pallardy M. 2014. Immunomodulation and lymphoma in humans. *J Immunotoxicol* 11(1): 1-12.

Purdue MP, Bakke B, Stewart P, De Roos AJ, Schenk M, Lynch CF, *et al.* 2011. A case-control study of occupational exposure to trichloroethylene and non-Hodgkin lymphoma. *Environ Health Perspect* 119(2): 232-238.

Raaschou-Nielsen O, Hansen J, McLaughlin JK, Kolstad H, Christensen JM, Tarone RE, Olsen JH. 2003. Cancer risk among workers at Danish companies using trichloroethylene: A cohort study. *Am J Epidemiol* 158(12): 1182-1192.

Radican L, Blair A, Stewart P, Wartenberg D. 2008. Mortality of aircraft maintenance workers exposed to trichloroethylene and other hydrocarbons and chemicals: Extended follow-up. *J Occup Environ Med* 50(11): 1306-1319.

Ritz B. 1999. Cancer mortality among workers exposed to chemicals during uranium processing. *J Occup Environ Med* 41(7): 556-566.

Rust S, Drange M. 2013. Google employees face health risks from Superfund site's toxic vapors. Center for Investigative Reporting. http://cironline.org/reports/google-employees-face-health-risks-superfund-sites-toxic-vapors-4291.

Rusyn I, Chiu WA, Lash LH, Kromhout H, Hansen J, Guyton KZ. 2014. Trichloroethylene: Mechanistic, epidemiologic and other supporting evidence of carcinogenic hazard. *Pharmacol Ther* 141(1): 55-68.

Scott CS, Jinot J. 2011. Trichloroethylene and cancer: systematic and quantitative review of epidemiologic evidence for identifying hazards. *Int J Environ Res Public Health* 8(11): 4238-4272.

Silver SR, Pinkerton LE, Fleming DA, Jones JH, Allee S, Luo L, Bertke SJ. 2014. Retrospective cohort study of a microelectronics and business machine facility. *Am J Ind Med* 57(4): 412-424.

SRI. 2011. *Directory of Chemical Producers.* Menlo Park, CA: SRI Consulting. Database edition. Last accessed: 9/21/11.

TRI. 2014. *TRI Explorer Chemical Report.* U.S. Environmental Protection Agency. http://www.epa.gov/triexplorer and select Trichloroethylene. Last accessed: 6/19/14.

USITC. 2014. *USITC Interactive Tariff and Trade Dataweb.* United States International Trade Commission. http://dataweb.usitc.gov/scripts/user_set.asp and search on HTS no. 290322. Last accessed: 6/19/14.

Vamvakas S, Brüning T, Thomasson B, Lammert M, Baumüller A, Bolt HM, *et al.* 1998. Renal cell cancer correlated with occupational exposure to trichloroethene. *J Cancer Res Clin Oncol* 124(7): 374-382.

Vermeulen R, Zhang L, Spierenburg A, Tang X, Bonventre JV, Reiss B, *et al.* 2012. Elevated urinary levels of kidney injury molecule-1 among Chinese factory workers exposed to trichloroethylene. *Carcinogenesis* 33(8): 1538-1541.

Vlaanderen J, Straif K, Pukkala E, Kauppinen T, Kyyrönen P, Martinsen JI, *et al.* 2013. Occupational exposure to trichloroethylene and perchloroethylene and the risk of lymphoma, liver, and kidney cancer in four Nordic countries. *Occup Environ Med* 70(6): 393-401.

Wallace L, Buckley T, Pellizzari E, Gordon S. 1996. Breath measurements as volatile organic compound biomarkers. *Environ Health Perspect* 104(Suppl 5): 861-869.

Wang G, Wang J, Ma H, Ansari GA, Khan MF. 2013. *N*-Acetylcysteine protects against trichloroethene-mediated autoimmunity by attenuating oxidative stress. *Toxicol Appl Pharmacol* 273(1): 189-195.

Wang R, Zhang YW, Lan Q, Holford TR, Leaderer B, Zahm SH, *et al.* 2009. Occupational exposure to solvents and risk of non-Hodgkin lymphoma in Connecticut women. *Am J Epidemiol* 169(2): 176-185.

Weisel CP, Jo WK. 1996. Ingestion, inhalation, and dermal exposures to chloroform and trichloroethene from tap water. *Environ Health Perspect* 104(1): 48-51.

Weit SB, Bice NT. 2013. *Indoor Air Sampling Report* [unpublished report]. Oakland, CA: Geosyntec Consultants. Prepared for the United States Environmental Protection Agency, Region 9, San Francisco, CA. 178 pp.

Wu C, Schaum J. 2000. Exposure assessment of trichloroethylene. *Environ Health Perspect* 108(Suppl 2): 359-363.

Zhao Y, Krishnadasan A, Kennedy N, Morgenstern H, Ritz B. 2005. Estimated effects of solvents and mineral oils on cancer incidence and mortality in a cohort of aerospace workers. *Am J Ind Med* 48(4): 249-258.

Dr. J. William Louda, Research Professor
Environmental Biogeochemistry Group
Department of Chemistry and Biochemistry
and The Environmental Sciences Program
Florida Atlantic University
777 Glades Road
Boca Raton, Florida 33431 U.S.A.
(561) 297-3309
(561) 297-2759 FAX
blouda@fau.edu

**a)- Increasing the limit on ANY toxin is stupid. The ONLY benefactor of increased limits on toxins in OUR waters are the polluters themselves-PERIOD. This obviously includes all EPA listed toxic metals (Cadmium and chromium discussed in the TR).**

**b)- Increasing turbidity restrictions is a good thing but this should be extended into brackish and fresh waters--the ultimate sources, in addition to direct land runoff, to turbidity in the marine environment. Turbidity in estuaries decreases light to SAV as it does in fresh waters also.**

**c)- Need to strongly investigate regulate and prosecute nutrient pollution leading to CHLa > 20 ug/L. Example, in March I measured 63 ug CHLa / L in Taylor Creek by the rim canal. (Chpt. 62-302FAC)**

**d)- 62-303 FA: Why not PROJECT future nutrient and cHLa levels. Not doing so is "GIVING UP" Why not try to understand and be PROACTIVE in prevention??**

**e) Adding WBIDs to Kissimmee River, Taylor Creek and Fisheating Creek is good. BUT should also add Indian Prairie, Harney Pond, Nubbin Slough.**

**In general MUCH MUCH more monitoring along each river / canal is needed and point as well as non-point sources need to be identified and fixed (septic to sewer, ag BMPs and Ag holding ponds on-site with their own STAs. Clean THEIR water before putting it into OURS.**

**(Short statement about aluminum)**

**The thing about aluminum is that it forms aluminum hydroxides which can stick to fish gills and lead to fish kills if concentrated enough. That is why one should not use ALUM to precipitate phosphorus (besides in anoxic condition of the sediments the Pis released anyway.**