Exhibit: CX 46

EXPERT REPORT

In the Matter of Nicor Gas

Docket No. TSCA-HQ-2015-5017

REPORT ON PUBLIC HEALTH IMPLICATIONS FROM COMMUNITY EXPOSURE TO POLYCHLORINATED BIPHEYNLS (PCBs)

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October 6, 2016

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Introduction

At the request of the U.S. Environmental Protection Agency (EPA), in the Matter of Nicor Gas, Docket No. TSCA-HQ-2015-04017, I have prepared this report in which I discuss the public health implications from residential exposures to polychlorinated biphenyls (PCBs). Since the primary material reviewed consists of documents from ATSDR, I provide background information on the Agency and the ATSDR Toxicological Profiles and public health assessment process. This information was mainly gathered from the ATSDR website (www.atsdr.cdc.gov) and from my knowledge of the Agency and its policies and procedures from having worked as a medical officer for ATSDR.

The issues in the case are related to PCBs found in community settings (which includes homes, churches, schools, and other buildings) from liquid condensate found in natural gas pipelines. The exposure pathway for humans from PCBs in natural gas pipelines is primarily from inhalation, with secondary exposure pathway concerns from dermal contact and incidental ingestion. While information on the adverse effects of PCB exposure to animals is available, this report centers on human health effects; animal studies are discussed only as they pertain to human health.

Agency background

Overview and mission

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency of the U.S. Department of Health and Human Services. ATSDR was established by the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) legislation in 1980 to assist in evaluating public health impacts involving hazardous waste sites and to help prevent or reduce further exposures. ATSDR's responsibilities in environmental public heath were expanded by the Superfund Amendments and Reauthorization Act in 1986.

ATSDR is directed by congressional mandate to perform specific functions concerning the effect on public health of hazardous substances in the environment. These functions include preparation of public health assessments of waste sites, health surveillance and registries, responses to emergency releases of hazardous substances, applied research in support of public health assessments, information development and dissemination, and public and health professional education and training concerning hazardous substances. ATSDR also receives requests to investigate public health concerns from environmental hazardous releases from other federal, state, and local agencies and from community groups or individual citizens. ATSDR does not have a regulatory role at hazardous waste sites, but it makes public health recommendations to the EPA and other government agencies concerning hazardous waste sites and releases.

The mission of ATSDR/NCEH is to protect "people's health from environmental hazards." ATSDR serves the public by applying the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances.

Organizational structure

ATSDR headquarters are in Atlanta, Georgia. ATSDR is organized into two divisions: the Division of Community Health Investigations (DCHI) and the Division of Toxicology and Human Health Sciences (DTHHS).

DCHI has staff in each of the ten EPA regional offices, Washington, DC, and Anchorage, Alaska and Atlanta Headquarters. The responsibilities of DCHI include the work of regional offices which establish working relationships with the federal and state agencies within the region and work closely with community groups and citizens concerning sites and hazardous chemical public health issues. DCHI staff prepare or assist in the development of public health assessment products by reviewing site specific environmental data, evaluating exposures, making recommendations to prevent harmful exposures, conducting exposure investigations, and providing public health education.

DTHHS is based in Atlanta. Among the responsibilities of DTHHS are the coordination of activities associated with the development of Toxicological Profiles and the establishment of the minimal risk levels for hazardous substances. DTHHS also provides technical expertise and site-specific support for hazardous material emergency events and chemical specific consultations.

Toxicological Profiles

Toxicological Profile overview and process

ATSDR publishes Toxicological Profiles for hazardous substances commonly found at National Priorities List (NPL) and other hazardous waste sites. There are about 300 Toxicological Profiles available. The documents summarize and interpret key epidemiologic, health, and toxicological information available about the specific substances.

The selection of the compound evaluated is based on the frequency found at NPL sites, its toxicity, and the potential for human exposure. Each year there is a ranking process where either new chemicals or an update of a chemical are nominated for review. The number of new studies that have been published on a chemical and budgetary constraints both influence the number of substances evaluated or updated in a given year.

After development, the Toxicological Profile on a given substance goes through ATSDR internal review for consistency and accuracy. The Minimal Risk Level Workgroup reviews the document and makes recommendations for the minimal risk level (MRL) for the specific substance. Three to four external expert peer reviewers also review the draft document. After agency clearance the document is released as a Draft for Public Comment.

After the comment period, as appropriate, public comments are incorporated into the document and the Toxicological Profile is released as a final document. If there are a large number of substantive comments, a second group of external peer reviewers may be asked to review the revised document before the final release. For a few substances (for example the PCBs toxicological profile), an external panel of experts is assembled to review the document and the public comments prior to the final release as well. The Toxicological Profiles are reviewed and revised periodically. If there had been a substantial

number of new studies or information, the Toxicological Profile on a substance may be updated or an addendum may be published.

Structure of the Toxicological Profile

The ATSDR Toxicological Profiles follow a similar format. The first chapter is the public health statement, which is provided in question and answer format and written for the general public to understand. Toxicological Profiles include a summation chapter about relevance to public health. Other chapters include chemical and physical information, production and use, potential for human exposure, analytical methods, and regulations and advisories.

The bulk of the document revolves around the chapter on health effects. This chapter systematically goes through the peer reviewed literature about each type of health effect associated with the substance by route of exposure and length of exposure. Health effects include death, systemic effects, immunological effects, neurological effects, reproductive effects, developmental effects, genotoxic effects, and cancer. The primary routes of exposure addressed are inhalation, oral, and dermal exposures. Acute (14 day exposures or less), intermediate (15 to 364 day), and chronic (365 days or longer) effects are reported. These sections on health effects include human studies, including case reports and occupational or epidemiologic studies, as well as animal research studies that may be relevant to human exposures. Sections within this chapter also include toxicokinetics, mechanisms of action, sensitive populations, and biomarkers of exposure.

Appendix A includes the minimal risk levels (MRL) and worksheets. An MRL is a health based guidance level for non-cancer health endpoints. The health endpoints selected are generally for subtle changes in the biological systems considered to be of relevance to humans, not for a serious health effect (such as irreparable damage to the liver or kidneys). Uncertainty factors are applied to account for the lack of precise toxicological information on sensitive populations or the use of an animal study. Below the established MRL, there is no appreciable risk of an adverse non-cancer health effect from daily exposure over the specified time period for the given route of exposure even in sensitive populations. The MRL is a screening level used as part of the initial public health assessment and not a health effect level, so environmental media concentrations of a compound that could result in a dose above the MRL would merit further evaluation, but does not mean that an adverse health effect would be present. The MRL may be revised when new information becomes available. MRLs are published on line at: http://www.atsdr.cdc.gov/mrls/index.asp.

Although MRLs do not address cancer as a health endpoint, ATSDR does establish a media specific cancer comparison value for many of the chemicals which are classified by the EPA as known, probable, or possible human carcinogens. For the most part, DCHI develops these cancer comparison values. ATSDR's Cancer Risk Evaluation Guide (CREG) comparison value uses EPA's cancer slope factor for the chemical, but is calculated using a lifetime of 70 years. The CREG represents a one in one million increased risk of cancer based on a 78-year exposure to the chemical at that concentration. CREGs are not published in the Toxicological Profile.

Polychlorinated Biphenyls (PCBs) Toxicological Profile

The *Toxicological Profile for Polychlorinated Biphenyls (Update)* was released as a final version by ATSDR in November 2000 [ATSDR 2000]. This Toxicological Profile follows the standard format described above and replaced the previous 1997 final version. The hard copy version of the 2000 document includes the word 'update' in the title on the blue cover.

The Draft for Public Comment version of the updated Toxicological Profile for PCBs was released in December 1998. An expert panel for the PCB Toxicological Profile convened in September 1999 to review the draft document and the public comments received on the draft document. The summary report from that meeting was prepared in April 2000. The summary report appears in Appendix E of the final version. A four person external peer-review group also reviewed the final document prior to publication. An addendum to the final version was released in April 2011 (see below).

Appendix A of the PCB Toxicological Profile provides MRLs for both an intermediate (14-364 day) and chronic (365 days or longer) duration oral exposure. The intermediate oral MRL of 0.03 µg PCB/kg body weight/day was derived from a monkey study using a PCB congener mixture and a neurological endpoint. The chronic oral MRL of 0.02 µg PCB/kg body weight/day was also derived from a monkey study but used PCB Aroclor 1254 oral exposure and evaluated immunological responses. An uncertainty factor of 300 was used in the derivation of both MRLs to account for the use of a lowest observed adverse effect level (LOAEL), a non-human study, and human variability. No MRLs are derived for acute oral exposures or for inhalation exposures of any duration.

An ATSDR internal peer reviewed *Addendum to the Toxicological Profile for Polychlorinated Biphenyls* was developed by DTHHS and released by ATSDR in April 2011 [ATSDR 2011]. The Addendum was not released for public comment or submitted for independent external peer review prior to on-line publication. The purpose of the Addendum was to provide a supplement of the scientific data that had been published in open peer reviewed literature since the release of the profile in 2000. The Addendum primarily updated scientific literature pertaining to studies on human or animal health effects. The Addendum did not propose any revisions to the MRLs for PCBs.

PCBs found in mixtures are discussed in ATSDR's Interaction Profile, "*Persistent Chemicals Found in Breast Milk (Chlorinated Dibenzo-p-Dioxins, Hexachlorobenzene, p,p'-DDE, Methylmercury, and Polychlorinated Biphenyls)*" [ATSDR 2004]. The final version of this document was released in May 2004 and is available on the ATSDR website. For mixtures involving chlorinated dibenzo-p-dioxins, chlorinated dibenzofurans and PCBs, ATSDR recommended using a toxicity equivalent factor approach because of the similar molecular site for interactions for some PCBs. The general recommendation for these persistent chemicals was to treat the mixture as having additive toxicity for multiple chemicals.

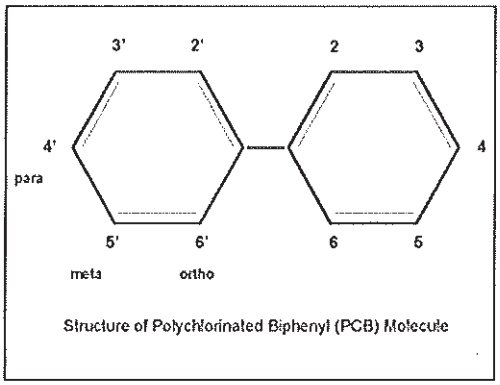
PCBs summary of health effects

The primary sources of information relied on to prepare this section summarizing health effects of PCBs were ATSDR's *Toxicological Profile for Polychlorinated Biphenyls (Update)* [ATSDR 2000], *Addendum to the Toxicological Profile for Polychlorinated Biphenyls* [ATSDR 2011], and *Case studies in Environmental Medicine—Polychlorinated Biphenyls (PCBs) Toxicity* [ATSDR 2014].

Evaluation of PCBs is complicated since they are found as a mixture of congeners that have individual toxicities and have interactions with each other. Assessment of some studies of PCBs are also complicated because of contributions to toxicity from structurally similar compounds, such as chlorinated dibenzofurans, that may also be present. In general, the higher chlorinated PCB congeners are more resistant to metabolism and accumulate in tissues to a greater degree.

Overview

Polychlorinated biphenyls (PCBs) refer to a group of man-made chemicals with a similar structure consisting of two bonded benzene (phenyl) rings surrounded by one to ten chlorine atoms (see figure below). There are 209 possible PCB congeners or structurally related molecules. With the exception of some research studies that isolate specific congeners for evaluation, numerous congeners are generally found together. Some commercial mixtures are called Aroclors and are designated by the percent of chlorine in the mixture. The commercial mixtures might consist of about 100 different congeners. PCBs were produced commercially in the United States from 1929 until 1977.



Source: www.epa.gov

PCB molecules are chemically and thermally stable which made them useful for many commercial applications. These properties also tended to make them very persistent in the environment. They are very soluble in oil and insoluble in water. In general, environmental persistence of the PCB congeners increases with the increasing number of chlorines on the molecule. High heating of PCBs can produce chlorinated dibenzofurans, which are generally a more hazardous by-product. PCBs enter the atmosphere from volatilization from soil, water surfaces, and PCB containing material where they can remain as a vapor or sorb to particles.

Toxicokinetics

Since PCBs are lipophilic, they are readily absorbed into the human body. For the general population, ingestion of contaminated food or inhalation of contaminated air are routes of exposure. Dietary intake, especially from fish which have bioaccumulated PCBs, is a major contributor of exposure. In the gastrointestinal tract, the PCB congeners enter the body by passive diffusion. While the absorption rate by inhalation has not been quantified, qualitative studies of humans exposed to airborne PCBs demonstrate absorption. The dermal route is time dependent and in most non-occupational scenarios does not appreciably contribute to the body burden.

Once absorbed in the body, PCBs have a biphasic distribution, with initial transport in the blood to the liver and muscle and later to the adipose tissue as the primary storage depot. In the blood, PCBs are carried mainly on lipoproteins. Mobilization from adipose tissue can increase during pregnancy, lactation, or in periods with rapid weight loss. PCBs can cross the placenta and enter the fetus.

The majority of PCBs undergo some biotransformation before being excreted in bile or urine. The liver is the primary site of metabolism. The first step is oxidation by the cytochrome P-450 system to form a hydroxylated PCB. The hydroxylated PCBs may be further metabolized by hydroxylation or conjugation reactions. In general, the rate of metabolism decreases with the increasing degree of chlorination. The biologic half-life of the PCB congener can range from about half a year for a congener with three chlorines to nine years for PCB-209 which contains ten chlorines.

Mechanism of action

The mechanism of action of PCBs is complex because PCBs are found in mixtures which may have competing effects. Synergy has also been found with some combinations of PCB congeners. Some PCBs also share similar modes of action to other persistent chemicals such as dioxins and chlorinated pesticides. There are several proposed mechanisms of action of PCB congeners in the human body but they are often grouped into two major categories, aryl hydrocarbon (Ah) receptor dependent (dioxin-like) and independent mechanisms.

Co-planar PCB congeners (congeners without chlorine or with a single chlorine in an ortho position, which allows the rings to exist in the same plane) have a dioxin-like mechanism of action and bind with varying affinity to the Ah-receptor inside the nucleus. The Ah-receptor binding results in health effects through changes in gene expression. Animal studies show induction of some hepatic enzymes, body weight wasting, thymic atrophy, and porphyria.

The non-co-planar PCB congeners are associated with Ah-receptor independent mechanisms. These congeners induce phenobarbital-type hepatic enzymes. Animal studies have shown that there are both neurological and neurodevelopmental effects involving brain dopamine levels and calcium homeostasis that are related to the Ah-receptor independent mechanisms.

Some PCB congener health effects are attributed to both the Ah-receptor independent and dependent mechanisms. These include liver hypertrophy, immune suppression, cancer, reproductive dysfunction, and thyroid hormone disruption. Some lower chlorinated non-co-planar PCBs are estrogen-like in their mechanism of action. These short lived PCBs interact with estrogen receptors and can potentiate estradiol activity.

Non-cancer health effects

There are human health effects related to exposures to PCBs from inhalation, ingestion, and dermal contact. This section summarizes the primary health effects associated with chronic exposures rather than higher concentrations of acute exposures. The non-cancer health effects briefly discussed included developmental, reproductive, immunological, and endocrine effects.

Developmental effects

Developmental effects are the target of public health interventions associated with pre- and post-natal PCB exposure. Fetuses, infants, and children are considered sensitive populations to PCB exposures. Exposures in early life coincide with critical periods of brain development. PCBs cross the placental barrier and maternal PCB body burdens are mobilized during pregnancy and transported to the developing fetus. PCBs are stored in breast milk. Epidemiological studies in children suggest that health effects from *in utero* exposures may be more significant than from breast feeding. Fetuses and young infants generally lack developed liver detoxification mechanisms for PCBs.

PCBs are not known to cause structural birth defects, but influence neurobehavioral and developmental deficits. Multiple studies comparing infants of mothers who ate moderate to high amounts of Great Lakes or other PCB-contaminated fish before or during pregnancy to those who did not eat fish have found neurobehavioral deficits such as abnormally weak reflexes, less responsiveness to stimuli, and greater motor immaturity. Memory and IQ score deficits have also been associated with prenatal exposure to PCBs. Follow-up studies and studies in children exposed to PCBs *in utero* have generally found that neurobehavioral deficits persist and children were more likely to have difficulty paying attention and to lag in verbal and other cognitive abilities.

Developmental effects from *in utero* PCB exposure found in human epidemiological studies include decreased birth weight, head circumference, and gestational age. Occupational studies of women exposed to PCBs in manufacturing jobs have found a decrease in birth weight and gestational age of their infants. Some human studies have not found this association. The range of results may reflect confounding contributions to these metrics, including the presence of other contaminants and differences in PCB doses, congener compositions, and the critical windows of exposure.

Non-human primate studies have supported the developmental and neurobehavioral findings from human studies. A study on post-natal oral exposure to a PCB congener mixture in monkeys from birth to 20 weeks of age that looked at neurobehavioral endpoints was the basis for the derivation of ATSDR's MRL for intermediate oral exposure to PCBs [ATSDR 2000]. The congener mixture used in this study was considered comparable to that found in human breast milk. Learning response decrements were noted in the monkeys with PCB exposure.

Reproductive effects

Human studies are variable in respect to the association of PCBs with reproductive effects in adults. For example, an epidemiological study found modest association in Lake Michigan anglers with risk for conception failure in men, but not in women. Another study determined that decreased menstrual cycle length was associated with the number of fish meals. A cohort of girls exposed to PCBs in their diet suggested a decreased time to menarche. A study on time to menopause found no significant difference with PCB exposure. Non-human primate studies have demonstrated alterations in menstrual cycles, decreased fertility, and increased number of abortions in females exposed to PCBs. Differences in findings may in part be attributable to PCBs inducing both agonistic and antagonistic estrogenic responses depending upon the congener composition.

Immunologic effects

The immune system was considered one of the more sensitive systems in the derivation of ATSDR's MRL for chronic oral exposure to PCBs. An ingestion study on immunological effects in Rhesus monkeys is the basis for the MRL [ATSDR 2000]. The study found significantly reduced IgM and IgG antibody levels in the animals that had received higher daily doses of PCBs when the animals were challenged at 23 months with sheep red blood cells. A similar trend was found at a challenge with sheep red blood cells at 55 months. Studies of shorter duration on non-human primates support the role of PCB on immune function, with decreased antibody responses, increased infections, and histopathological changes in the thymus and spleen being reported.

Both studies on newborns exposed during pregnancy and experimental monkey studies have found a decrease in thymus size with PCB exposure. A study that looked at antibody response to childhood vaccinations found a decreased response with pre-natal exposure to PCBs. In another study, infant susceptibility to infections was found to be positively correlated to maternal serum PCB levels of mothers who consumed Great Lakes and Sheboygan River fish.

Endocrine effects

In addition to the endocrine related health effects reported in the reproductive effects section, studies have found impacts on other endocrine functions. Reductions in serum thyroid hormone levels in workers and in newborns exposed to PCBs during pregnancy have been reported. The latter finding in newborns is of interest because of the role of the thyroid in normal development of the brain. Studies on non-human primates and other experimental animals also support the role of PCB toxicity on thyroid hormones. Some studies on fisherman cohorts and Great Lakes fish consumers support the association of type 2 diabetes mellitus and exposure to PCBs.

Cancer

In 2013, the International Agency for Research on Cancer (IARC) classified PCBs as Group 1, carcinogenic to humans (http://monographs.iarc.fr/ENG/Monographs/vol107/mono107.pdf). This category is generally used by IARC for a chemical where there is either sufficient evidence of carcinogenicity in humans, or there is sufficient evidence in experimental animals and strong evidence in humans that the chemical acts through a relevant mechanism to cause cancer [IARC 2006]. IARC considered more than 70 independent epidemiological studies that demonstrated evidence of PCB carcinogenicity in humans. The Group 1 classification was based on the association between exposure to PCBs and increased risk of melanoma.

The Department of Health and Human Services National Toxicology Program determined that PCBs are reasonably anticipated to be a human carcinogen. Similarly, the U.S. EPA has classified PCBs as a probable human carcinogen.

Some occupational cancer mortality studies have reported increased mortality rates for various cancers including liver cancer, intestinal cancer, brain cancer, thyroid cancer, and non-Hodgkin lymphoma. Other worker cohorts have not found an increased mortality risk for these cancers. Methodological limitations such as worker exposure classification and confounding risk factors may account for inconsistencies between studies. A more recent population-based case control study found exposure to more highly

chlorinated PCBs was associated with increased risk of non-Hodgkin lymphoma. Animal studies have conclusively shown the association of PCBs and the induction of liver tumors.

The potential for PCBs to cause cancer increases with the degree of chlorination of the mixture or congener. Mixtures with greater than 50% chlorination have been found to have the strongest associations with cancer. The derivation for the ATSDR CREG for ingestion exposures for PCBs uses EPA's oral upper-bound cancer slope factor $(2\ mg/kg/day)^{-1}$, which is applied to scenarios that involve high-persistence or dioxin-like PCB exposures and dietary exposures. ATSDR's CREG for inhalation exposure of 0.010 microgram per cubic meter ($\mu g/m^3$), is based on EPA's inhalation unit risk.

Public health assessment (PHA) process

ATSDR has developed an approach to determining public health implications from exposure to environmental contaminants. ATSDR's *Public Health Assessment Guidance Manual* (updated) [ATSDR 2005] describes the methodologies that should be considered by health assessors in their evaluations of sites. As part of the PHA evaluation, environmental and health data are reviewed and community concerns are addressed. ATSDR does not typically collect its own environmental data, but reviews information from EPA or other governmental agencies. If a public health hazard is determined to be present at the site, recommendations are made for reducing or eliminating the exposure.

The two primary components of the health assessment process is the environmental evaluation and the health effects evaluation.

As part of the environmental evaluation, exposure pathways are examined. An exposure pathway is the link between environmental releases and local populations; a person must be exposed to chemical contaminants in the environment before an adverse health effect is possible. An exposure pathway consists of five parts that must all be present to be considered a completed exposure pathway. If one or more of the parts are unknown, it may be considered a potentially completed exposure pathway. Public health interventions are targeted at eliminating at least one step in the exposure pathway to make the pathway incomplete. If there is no completed or potentially completed exposure pathway, than no public health hazard will exist.

The five parts of the exposure pathway are:

- Contaminant source;
- Environmental fate and transport—the release to environmental media or transfer between media;
- Exposure point or area—the location(s) where people might come into contact with a contaminated medium;
- Route of exposure—the route through which the chemical enters the body; and
- Potentially exposed populations.

A completed exposure pathway and contact with a chemical contaminant in and by itself does not necessarily result in adverse health effects. A chemical's ability to affect a person's health is affected by a number of other factors, including the concentration, the duration and frequency of exposure, and the chemical's toxicity. Other factors include a person's history of past exposure to chemicals, current health status, age and sex, or genetic predisposition.

Generally, for non-cancer health effects, a threshold dose must be reached before an adverse health outcome is experienced. The development of cancer is assumed to be a non-threshold health effect. Exposure to the chemical carcinogen increases the risk of developing cancer in proportion to the exposure dose.

After determining the existence or potential of a completed exposure pathway, evaluation is made by comparing the dose an individual may receive to a health screening value for both non-cancer and cancer health effects. For evaluating exposures to the public, ATSDR derives media-specific comparison values from minimal risk levels and EPA reference doses. The comparison values are designed to be protective of the most sensitive populations, including children and pregnant women. These media-specific comparison values are used to identify pollutants that may potentially be of health concern and that required more in-depth evaluation.

Public health implications from exposure to PCBs from natural gas pipelines

Using the ATSDR approach to evaluate public health implications from exposure to environmental contaminants, several completed exposure pathways can be identified. There are many sources of PCBs in the environment. PCBs are present in condensate liquid in natural gas pipelines and can affect residential and commercial appliances such as gas meters, stoves, furnaces, boilers, and water heaters. PCB-contaminated liquid from these natural gas pipelines can seep onto surfaces. PCBs can be released to indoor air by volatilization, where they can remain as a vapor or attach to particles. Transfer of PCBs can occur from airborne deposition to surfaces. Given the location of the appliances and the type of building, the air and surfaces within a home, school, church, or other buildings can be the exposure point for residents, students, teachers, and parishioners. PCBs can enter the body through breathing contaminated air, contact with contaminated surfaces, and incidental ingestion from hand to mouth behavior after contact with surfaces.

Of the exposure routes presented, inhalation and incidental ingestion are the most likely routes of exposure. Because of the time dependency of absorption from dermal exposure, this route may not contribute as appreciably to the body burden. For non-cancer health effects, children, infants, and fetuses are the most sensitive populations for exposure. In the exposure scenarios for residential settings, these groups would be present. As mentioned previously, ATSDR's MRLs for ingestion are based on neurodevelopmental and immunologic effects to young non-human primates. Children exhibit more hand to mouth behaviors than adults. Metabolism and excretion of PCBs are also not fully functional in early life stages, increasing the impact to this population. Because of the slow metabolism, especially of more highly chlorinated PCBs, exposure to PCBs will increase the body burden of PCBs.

The body burden of PCBs will also be increased by other exposure sources. Because of historical uses, residents can be exposed to PCBs from dietary sources and from other airborne building sources such as caulk, paint, and light ballasts. Because of the varying biological and environmental half-lives of PCB congeners, in real-life settings, it would be difficult to attribute body burden to specific sources. Regardless, from epidemiologic studies in the literature, generally, cohorts with higher body burdens or

exposure to PCBs will exhibit any given health effect from PCBs more frequently than populations with lower body burdens or exposure.

For in-utero exposures and exposures to children, the neurodevelopmental and neurobehavioral health effects are understood in terms of populations of exposed individuals. Cognitive impairments are more consistently seen in groups of children with higher maternal contributions or early life exposures to PCBs. More overt clinical health effects to adults and children, such as chloracne and elevated liver enzymes, are only apparent in acute or chronically higher level exposures.

PCBs have been classified by IARC as known human carcinogens and exposures to PCBs increase an individual's risk of developing cancer. Since PCBs are readily absorbed into the body by ingestion and inhalation, similar to non-cancer health effects, these routes of exposure would contribute more appreciably to any increased cancer risk. Increased cancer risk has been associated with higher body burdens of PCBs in people non-occupationally exposed.

There are no specific medical treatments to reduce the body burden of PCBs. Accumulation of PCBs will occur with continued exposure. Minimizing or avoiding exposures to PCBs by removing sources, housekeeping, and good hygiene practices are methods to decrease exposure from airborne or airborne deposition of PCBs.

Conclusions

Completed exposure pathways to PCBs in residential and community settings exist, including those due to the presence of PCBs in natural gas pipelines. There are no known health benefits from PCBs. Efforts to reduce exposure to PCBs would be in the interest of protecting the public's health. Because of the association between increased body burden of PCBs and non-cancer health effects and increased cancer risk, exposure to PCBs is of public health concern.

Works considered

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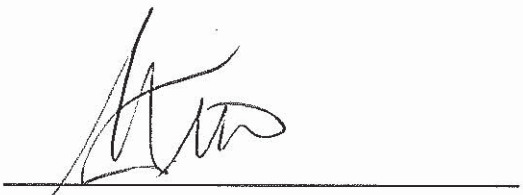
Statement regarding compensation and prior expert testimony

I am receiving no compensation for the preparation of this report or for any testimony I may provide in this matter other than my salary provided by ATSDR since I am serving as an expert as part of my official federal government duties.

I have provided an expert report and have served as an expert witness in United States and Wisconsin v. NCR Corp., et al., Case no. 10-C-0910 (E.D. Wis.).

Curriculum Vitae (attached)



Michelle Watters, MD, PhD, MPH
October 6, 2016