EXHIBIT 23

Correspondence from Judith Knight February 3, 2023

Affidavit from David O. Carpenter, MD dated February 14, 2023

Curriculum Vitae of David Carpenter, MD

Exposure to and health effects of volatile PCBS document submitted by Judith Knight on behalf of David Carpenter, MD

Jim Wilusz

From: Judith Knight <jknight@judithknight.com>
Sent: Friday, February 3, 2023 10:31 AM
To: Robert Wespiser
Cc: Carisa Vincent; Cristobal Bonifaz; Jim Wilusz; Tim Gray; jopads@msn.com
Subject: Re: 11/19/22 LBOH Adj Hearing-submitting SA as an exhibit

Dear Dr. Wespiser,
I appreciate your response.
Thank you.

Sincerely,

Judith Knight, Esq.

On Fri, Feb 3, 2023 at 9:11 AM Robert Wespiser <rwespiser@gmail.com> wrote:
Ms Knight,
I believe Lee BoH will agree that a clear definition of the monitoring of the UDF is very important. I suggest this should be included in a comprehensive, specific delineation of the manner in which the sediment will be dewatered, loaded, transported from site to dump, transported to the dump from other sites, and moved from site to the "high PCB concentration" facility, and other specific concerns not listed here.
Any effort that HRI can make in this more comprehensive approach will be important.
Thank you,
Bob Wespiser

On Tue, Jan 31, 2023 at 5:18 PM Judith Knight <jknight@judithknight.com> wrote:
Dear Dr. Wespiser,
On behalf of HRI, I am hereby submitting this letter and the Settlement Agreement ("SA") between GE, Town of Lee and other parties as exhibits to become part of the public record in above referenced 11/19/2022 LBOH Adjudicatory Hearing. The SA in pdf form is attached below in its entirety.

While all of the SA is relevant to the LBOH's consideration in this matter, I would like to draw the LBOH's attention to pages 8-12 of the SA, section III entitled: *Disposal of Excavated Material in Rest of River Remedial Action.*

In Paragraphs C & D of section III, the size and dimensions of the UDF (PCB-landfill) and some limited information about the liner(s) that will be used in the UDF are provided. Also set forth is the fact that the placement of the bottom liner to the UDF can be as close as 15 feet above the Town of Lee's water table.

Paragraphs F through H of section III set forth in surprisingly vague terms that GE will be required to "inspect and maintain" the UDF once the PCB contaminated soil is dumped there. However, no timeline is provided as to when, how often, or for how long GE will be required to "maintain, inspect the UDF and conduct groundwater samplings".

Secondly, there is no description as to what actions GE must take to constitute a proper "maintenance and inspection" of the UDF.

Lastly, there is no description of, or even a reference to, what scientifically acceptable standard GE must meet in order to be in compliance with the terms of SA.

It is my legal opinion that the terms in the SA which purportedly require GE to inspect and maintain the UDF are so vague that they are unenforceable.

I understand that the legal issues pertaining to the SA are not in the LBOH's domain. However, the unanswered questions and concerns about whether, in what manner, and for how long GE will be required to "inspect and maintain" the UDF going forward once the UDF is built, are relevant to the issues before the LBOH in this matter. This is especially true given the UDF's close proximity to the Town of Lee's water table, its residential neighborhoods and downtown Main Street.

Thank you for your time and consideration in this matter.

Sincerely,

Judith C. Knight, Esq.
342 Main Street
Great Barrington, MA 01230
Off: 413-528-0505
Cell: 413-329-4665
Jknight@judithknight.com

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Judith Knight, Esq.
Cell: 413-329-4665
Sent from my iPhone

Jim Wilusz

From:	Judith Knight <jknight@judithknight.com>
Sent:	Wednesday, February 15, 2023 7:29 PM
To:	Robert Wespiser; Jim Wilusz; Cristobal Bonifaz
Cc:	Tim Gray
Subject:	Affidavit of David o. Carpenter, MD for submission to 11.19.22 LBOH
Attachments:	AFFIDAVIT OF DAVID O. CARPENTER, MD.pdf; EXHIBIT A to Affidavit of David O. Carpenter, MD.pdf; EXHIBIT B of Affidavit of David O. Carpenter, MD (May 2015 Envir. Health Article).pdf

Dear Dr. Weipiser, Attorney Bonifaz and Mr. Wilusz,

On behalf of HRI, I am submitting herewith pdf copies of Dr. Carpenter's Affidavit with Exhibits A and B to the affidavit for your consideration and to be part of the public record for the 11/19/22 Adjudicatory Hearing before the Lee Board of Health.

Please find the following documents attached below:
1) Affidavit of David O. Carpenter, MD (5 pages);
2) Exhibit A to the Affidavit- Dr. Carpenter's Curriculum Vitae (38 pages); and
3) Exhibit B to the Affidavit- Dr. Carpenter's Published Article entitled "*Exposure to and Health Effects of Volatile PCBs,* May of 2015 (11 pages).

Because of the lengths of Exhibits A and B, I had to attach the exhibits separately.

Please let me know if you have any questions or concerns.

Sincerely,

Judith C. Knight, Esq.
342 Main Street
Great Barrington, MA 01230
Off: 413-528-0505
Cell: 413-329-4665
Jknight@judithknight.com

IN RE THE NOVEMBER 19, 2022
LEE BOARD OF HEALTH
ADJUDICATORY HEARING

Affidavit of David O. Carpenter, M.D.

1. I, David O. Carpenter, M.D., am an expert in the field of Environmental Medicine. One of my subspecialities is the health effects caused by human exposure to PCBs.[1] My Curriculum Vitae is attached herewith as **Exhibit A**.

2. I have written extensively on the serious and long-lasting adverse health effects caused to humans from exposure to unsafe levels of PCBs. These health effects include cancer, reproductive health as well as many other diseases which are listed and discussed in my published article entitled "*Exposure to and Health Effects of Volatile PCBs*" dated May 31, 2015 report attached here as **Exhibit B**.[2]

3. I testified at the November 19, 2022 Lee Board of Health Adjudicary Hearing ("11/19/22 LBOH") via Zoom. However, there was some difficulty with internet connection during my testimony that day. I would like to supplement my expert testimony with this affidavit.

4. I am familiar with General Electric Company's proposed Upland Disposal Facility ("UDF") at Woodland Road Location in Lee, MA.

5. I have reviewed the relevant aspects of the Settlement Agreement between General Electric Company ("GE") and the Town of Lee, et al. which provide specific information about the UDF. The Settlement Agreement ("SA") indicates that the UDF will be located two miles from downtown Main Street in Lee, MA. As to the

[1] Polychlorinated biphenyl
[2] The findings of the adverse health effects of PCBs as described in the article remain current and relevant today.

size of the UDF, the SA states that the UDF will have a capacity of 1.3 million cubic yards with a footprint of 20 acres and an elevation of 1,099 feet above mean sea level. Placing a PCB-contaminated disposal facility of this magnitude in a location so close to the heart of the Town of Lee and residential areas is a dangerous plan. It will threaten the health of the residents who live up to a four-mile radius of the PCB landfill. My research team has numerous publications showing that people who live within a four-mile radius of a PCB-contaminated waste site in New York State are more likely to be hospitalized with a number of different diseases (including diabetes, hypertension, heart disease, asthma, COPD and other diseases) than those who do not. The route of their exposure to PCBs is through the inhalation of vapor-base PCBs coming off the PCB waste site.

6. Landfills, such as the UDF, do not prevent PCBs from passing through the cover of the landfill and into the air. PCBs will escape into the air from even an enclosed and dormant landfill.

7. The SA states that the soil dredged from the Housatonic River with concentration levels of PCBs between 20 to 25 parts per million and lower will be dumped and stored at the UDF. It leaves open the possibility that GE could dump PCB-contaminated soil with concentration levels of PCBs as high as 25 to 49 parts per million at the UDF.[3] Our studies, referenced in Paragraph 5 above, show that for some diseases, especially diabetes and hypertension, it is the lower-chlorinated PCBs congeners that are more volatile and responsible for the elevated risk of disease.

8. While there may be good intentions behind the language in the SA promising to only store the PCB-contaminated soil with the lower-concentration levels of PCBs, this does little to control the risk of dangerous exposure to PCBs to those living within a four-mile radius of the UDF. The important distinction lies between lower *concentration* levels of PCBs verses lower-*chlorinated* PCBs. The soil with the lower-concentration levels of PCBs between 20 and 25 parts per million will still

[3] The Toxic Substance Control Act requires that contaminated soil with concentration level of PCBs at levels of 50 parts per million or more must be transported to a designated facility out of state.

contain PCBs with fewer chlorines. It is the PCBs with lower amounts of chorine that volatize more quickly into the air and increase the risk of exposure. The SA does not reference this important fact nor does it mention any plan to test for of the PCB-contaminated soil stored at the UPD for its chlorine content which is critical to understanding the likelihood of the risk of exposure to airborne PCBs coming from the UDF.

9. I understand that the UDF project includes a thirteen-year plan to transport the PCB-contaminated soil and sediments from various points of removal along the Housatonic River to the UDF. All of the PCB-contaminated soil and sediments dredged from the river will contain some lower-chlorinated PCBs. The constant movement of these lower-chlorinated PCBs and their continual exposure to the air will cause those PCBs to volatized, become airborne and inhaled. Breathing in airborne PCBs is the primary way in which human beings are exposed to dangerous levels of PCB. The thirteen-year plan to transport the PCB-contaminated soil from the Housatonic River to the UDF will logistically involve a minimum of four steps. Each step of the process will cause the lower-chlorinated PCBs in the soil to volatilize again and again. For example, the opportunity for the lower-chlorinated PCBs to volatilize will occur at each of the following points of transport:
    a. Every time the PCB-contaminated soil is dredged from the Housatonic River to the open air;
    b. Every time the PCB-contaminated soil is loaded on to the trucks for transport to the UDF;
    c. Every time the PCB-contaminated soil is transported from the point of removal to the UDF on public roads;
    d. Every time the PCB-contaminated soil is unloaded from the trucks and dumped into the UDF: and
    e. The lower-chlorinated PCBs will volatize even more quickly when wet as the soil and sediments will be wet when its first dredged from the Housatonic River for transport to the UDF. As the soil containing the lower-chlorinated PCBs dries out, those PCBs will rapidly evaporate and become airborne.

10. The SA states that the UDF will have "*a double liner and a leachate collection system with a low permeability cap and vegetation. The liners shall have permeability equal or less than 1 x 10 (minus 7) cm/sec with a minimum thickness of 30 mils and be chemically compatible with PCBs.*" These liners, whether there is one liner or two, will eventually leak. All of the known data on the life of the liners used in toxic

landfills, such as the UDF, support this conclusion. The location of the UDF is 1,000 feet from the Housatonic River as the crow flies and only 15 feet above the Town of Lee's water table (as stated in the SA). Given the location of the UDF, it is more likely than not that when the liners in the UDF do leak, the PCB contaminates stored at the UDF will eventually run back into the Housatonic River and contaminate the ground water and water table for the Town of Lee. This conclusion is supported by Dr. David DeSimone's findings in his report submitted as Exhibit 6 in the online public record for the 11/19/22 LBOH.[4]

11. Given the likelihood of exposure to potentially dangerous levels of airborne PCBs to the residents of Town of Lee living within a four-mile radius of the UDF, if the water table for Town of Lee was also contaminated with PCBs, it would be catastrophic to the long-term health of these residents.

12. Based on the above factual information, it is my expert opinion, to a degree of medical certainty, that the location and size of the UDF, as well as the thirteen-year plan to transport the PCB-contaminated soil from the Housatonic River to the UDF, will cause the residents in the Town of Lee living within a four-mile radius of the UDF an array of serious and long-lasting adverse health effects as referenced in this affidavit and its attachments which are incorporated herein by reference.

[4] Dr. DeSimone states, in relevant part, that the ground underneath the UDF is quite porous and will provide little, if no, retention base for the PCB-contaminates that leak from the UDF. Specifically, he describes the ground underneath the UDF as being "underlain by Stockbridge Foundation carbonate rock which primarily consists of dolomite marble and that the marble naturally contains fractures and joints which allow ground water and any contaminates to flow through" to the ground water below.

Dated: February 14, 2023

SIGNED UNDER PENALTIES OF PERJURY

DAVID O. CARPENTER, M.D.

STEPHEN HOUT
Notary Public, State of New York
Qualified in Albany County
Reg. No 01HO6288676
My Commission Expires Oct. 7, 2025

2/14/2023

David O. Carpenter*

Exposure to and health effects of volatile PCBs

Abstract

Introduction: Polychlorinated biphenyls (PCBs) are persistent, lipophilic contaminants that are known to increase risk of a number of human diseases. Although ingestion of animal fats is a major route of exposure, there is increasing evidence that inhalation of vapor-phase PCBs is also important and may be as or even more important than ingestion under some circumstances.

Methods: The evidence that inhalation of PCBs may cause cancer, heart disease, hypertension, and diabetes is reviewed and presented in this report.

Results: PCBs are known human carcinogens. A husband and wife, occupationally required to 'smell' PCB-containing oils, both developed thyroid cancer, malignant melanoma/severely melanocytic dysplastic nevus (a precursor to malignant melanoma) and the husband, a non-smoker, developed and died of lung cancer. The serum of both had highly elevated concentrations of lower chlorinated, volatile PCB congeners. In other studies, residents living near PCB-containing hazardous waste sites, and thus breathing PCB-contaminated air, have elevated rates of hospitalization for cardiovascular disease, hypertension, diabetes and reduced cognitive performance, whereas other studies in defined populations show that there is an elevated risk of all of these diseases in individuals with elevated serum PCBs.

Conclusions: These results are consistent with the conclusion that inhaled PCBs can increase risk of cancer, cardiovascular disease, hypertension, diabetes and reduce cognitive function.

Keywords: cancer; cardiovascular disease; diabetes; hypertension; PCB exposure; volatile PCBs.

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***Corresponding author: David O. Carpenter,** Institute for Health and the Environment, University at Albany, 5 University Place, A217, Rensselaer, NY 12144, USA, E-mail: dcarpenter@albany.edu

Introduction

Polychlorinated biphenyls (PCBs) were manufactured in many countries from the late 1920s until they were found to be persistent and toxic in the late 1970s, when their manufacture and use was stopped in most developed countries. It is reported, however, that they are still being manufactured in North Korea, and even in the US, many transformers and capacitors that are still being used contain PCBs.

PCBs consist of mixtures of up to 209 individual congeners, which vary depending on how many chlorines are on the biphenyl rings and where they are located on the molecule. Figure 1 shows the PCB molecule and the convention for identifying different congeners based on the location of chlorines. PCBs were manufactured in many countries as commercial mixtures through the chlorination of biphenyl with anhydrous chlorine in the presence of a catalyst, usually iron. The duration of the reaction determined the average degree of chlorination. In the US, almost all PCBs were manufactured by Monsanto, who sold commercial mixtures under the trade name 'Aroclor'. Aroclor 1242 was 42% chlorine by weight, whereas Aroclor 1260 was 60% chlorine. However, all commercial products contained a variety of PCB congeners, with the exception of Aroclor 1271, which was pure PCB 209 that contained chlorine groups at all 10 sites.

Most widely used commercial PCB mixtures are oils, and the greater the degree of chlorination, the more viscous the oil. They had many useful purposes. However, they had major uses in capacitors and light ballasts given because they are relatively nonflammable and nonconductive. They were widely used as hydraulic fluids, as solvents for paints or caulking, in carbonless copy paper, and in other products requiring a lipophilic solvent.

Although all PCB congeners have some common properties, they also have significant differences in physical properties and routes of exposure to humans. In general, PCBs have low water solubility and volatility. However, those congeners containing fewer chlorines are more water soluble and more volatile than those with more chlorines (1, 2). Table 1 (3) shows vapor pressure, water solubility, log octanol/water partition coefficient (log K_{ow}), and approximate evaporation rates as a function of the number of chlorines on the PCB molecule.

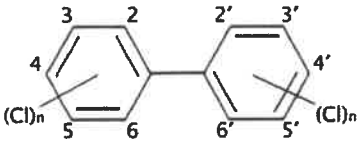

Figure 1: The structure of PCBs. There can be any number of chlorines around the biphenyl ring between one and ten. The convention for labelling the position is shown by the numbers, where the 2 and 6 positions are *ortho*, the 3 and 5 positions are *meta*, and the 4 position is *para*. The prime sign distinguishes in which ring the chlorines are located.

Table 1: Physical characteristics of PCBs by homologue groups at 25°.

PCB homologue group	Vapor pressure, Pa	Water solubility, g/m³	Log octanol/ Water coefficient	Evaporation rate, g/m³/h
Monochloro	1.1	4.0	4.7	0.25
Dichloro	0.24	1.6	5.1	0.065
Tetrachloro	0.012	0.26	5.9	4.2×10^{-3}
Hexachloro	5.8×10^{-4}	0.038	6.7	2.5×10^{-4}
Octachloro	2.8×10^{-5}	5.5×10^{-4}	7.5	1.5×10^{-5}
Decachloro	1.4×10^{-6}	7.6×10^{-4}	8.3	8.5×10^{-7}

Data from Ref (3).

Even commercial mixtures with primarily highly chlorinated congeners contain lower chlorinated congeners at low concentrations. Figure 2 shows the congener pattern of Aroclor 1260 (60% chlorine by weight) and that of PCBs in the vapor phase, resulting from blowing air over the commercial mixture. Clearly, even this highly chlorinated mixture contains lower chlorinated PCBs that volatize. There is also some volatilization of moderately chlorinated congeners, but the overall profile in the vapor phase shifts markedly to the left, indicating that lower chlorinated congeners are more volatile.

PCBs can volatilize from a variety of sources, including commercial mixtures, water, landfills, and commercial products. As lower chlorinated PCBs are more water soluble and more volatile (Table 1) they will selectively dissolve in water and then move from a soluble aqueous phase into the air. PCBs evaporate along with the water (4, 5), and this process is very temperature dependent (6). Volatile loss of PCBs from Lake Superior was calculated to be about 1900 kg per year (7). Outdoor air concentrations of PCBs near New Bedford Harbor, a highly contaminated body of water, ranged from 0.4 to 53 ng/m³ (8); these are significantly higher than those at a comparison site. PCB fluxes to air along the contaminated Hudson River ranged from 0.5 to 13 μg/m²/day (9).

The greater water solubility of lower chlorinated PCBs has implications for drinking water quality. The majority of the higher chlorinated congeners will be bound to particulates in water and then removed by standard drinking water treatments. However, those that are dissolved are more difficult to remove and may be an important route of human exposure, especially if contaminated surface water is used for municipal drinking water.

PCBs will also volatilize from contaminated soils and sediments. As from water, the PCBs volatize with water, and dry sediments lose fewer PCBs to the air as compared with wet sediments or soils (4). PCBs can also volatilize from landfills, depending upon how tightly they are covered (10). Hermanson et al. (11) studied air PCB concentrations near a Monsanto landfill in Anniston, Alabama, the site of a PCB synthesis factory, and compared results to those from a nearby site that had superficial soil PCB contamination. They found less dependence on surface temperature for PCB release to air from the landfill, and suggested that most of the sources of PCBs from the landfill site were materials buried within the landfill.

In addition to the differences in physical properties, congeners have both differences in rates of metabolism in the human body and major differences in mechanisms of action and health effects in humans. PCBs, like most chlorinated compounds, are poorly metabolized and are thus persistent. In general the half-life increases with number of chlorines but other factors like location of the chlorines around the ring also influence rates of metabolism. The half-lives in humans of several individual PCB congeners are shown in Table 2 (12). *Ortho* chlorine substitution usually increases the half-life relative to that of a PCB with the same number of chlorines but with none in the *ortho* position (13).

Many of the volatile mono-, di-, and tri-chloro congeners are metabolized within hours in rats (14). Hu et al. (15) found that labeled PCB 11 (3,3′-dichloro biphenyl) had a half-life of 12 h in male rats. Although human metabolism is generally not as rapid as in rodents, it is sufficiently rapid such that lower chlorinated congeners are rarely found at significant concentrations in human blood. Long half-life makes it convenient to determine the exposure of a person to PCBs in the past, but there is often the assumption that long half-life is indicative of greater health effect. This assumption is not necessary correct. This is because even those congeners that are more rapidly metabolized may have significant toxicity, especially if there is prolonged exposure, as would be the case if they were inhaled on a daily basis.

The major metabolism of PCBs is through cytochrome P450s in the liver and other organs (13). This results in

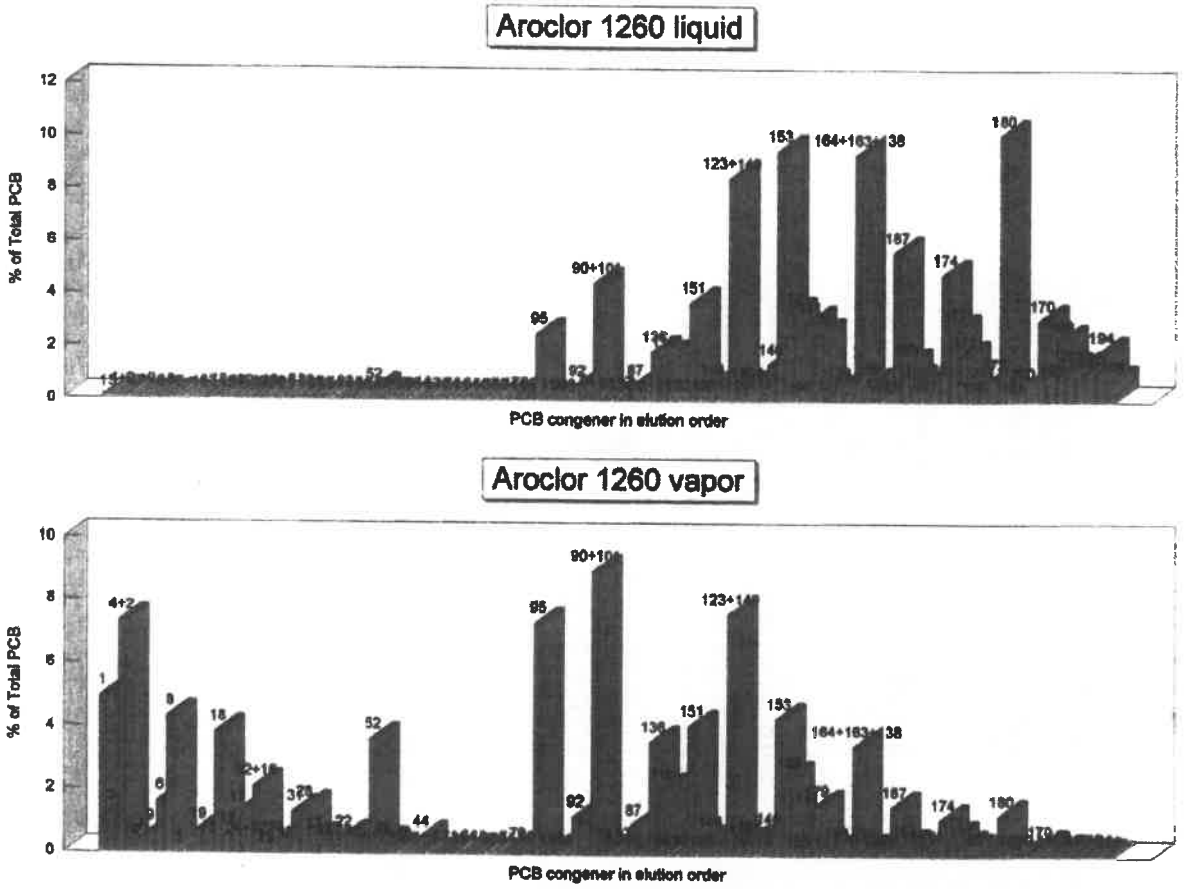

Figure 2: The congener patterns in Aroclor 1260 liquid (top) and the congener pattern seen when passing air over the liquid and collecting and analyzing the vapor-phase PCBs.
Peaks are shown in the order they elute from the column. The numbers above the peaks identify individual congeners or groups of congeners. Those peaks to the left have fewer chlorines.

Table 2: Half-lives of single PCB congeners in the human adult body.

PCB number	PCB structure	Half-life, years
28	2,4,4′ Trichlorobiphenyl	5.5
52	2,2′,5,5′ Tetrachlorobiphenyl	2.6
105	2,3,3′4,4′ Pentachlorobiphenyl	5.2
118	2,3′4,4′,5 Pentachlorobiphenyl	9.3
138	2,2′,3,4,4′,5′ Hexahlorobiphenyl	10.8
153	2,2′,4,4′,5,5′ Hexachlorobiphenyl	14.4
170	2,2′,3,3′,4,4′,5 Heptachlororbiphenyl	15.5
180	2,2′,3,4,4′,5,5′ Heptachlorobiphenyl	11.5

Data from Ref (4).

introduction of oxygen onto the molecule, which then allows for further metabolism by other transferases. Many of the hydroxylated or methyl sulfonated metabolites are somewhat persistent and have biologic activity (16). The position of the chlorines around the PCB molecule influences the rate of metabolism (17). This is why different PCB congeners with the same number of chlorines have different half-lives, as shown in Table 2. In addition, different congeners are targets of different P450s. Many studies have focused on PCB congeners that have dioxin-like activity as well as those that bind to the aryl hydrocarbon receptor, induce P4501A and then induce many different genes (18). Other congeners induce different P450s and many genes, but with a different pattern (19). To make matters even more complex, the profile of genes that are induced may vary from one tissue to another (20). Many of the adverse health effects reported in humans are likely a consequence of different patterns of gene induction.

Despite the more rapid metabolism of lower chlorinated PCBs, evidence for inhalation exposure can be obtained from serum samples. Our group has studied PCB exposure in a Native American population for many years. Many older

adults have a pattern of congeners dominated by a few highly chlorinated and persistent congeners like PCBs 138, 153, 170, and 180. However, we have been able to identify a pattern of lower chlorinated PCBs in the serum of younger Mohawks, which matched closely to the pattern of the PCB profile in air over a contaminated site (21) Figure 3. The pattern could not be observed clearly in older individuals because serum levels increase with age and the PCBs from ingestion obscure those more readily metabolized PCBs.

Herrick et al. (22) measured serum PCB levels in teachers working in a school that had elevated PCBs in indoor air, and found significantly higher concentrations of lower

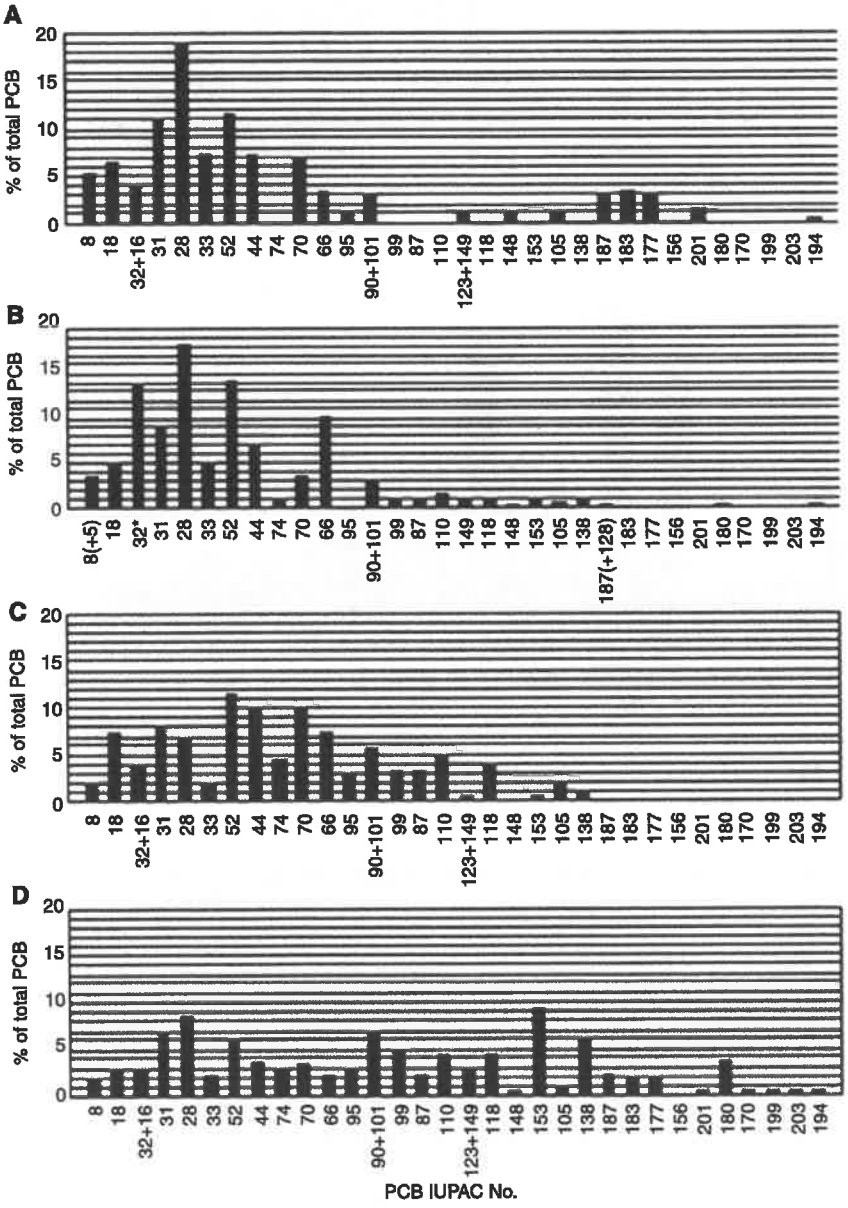

Figure 3: Congener compositions of (A) End-member (EM)-1 as determined by polytopic vector analysis (PVA) of serum PCB congener data for 702 adult Mohawks, (B) air sampled above "Contaminant Cove" at the western boundary of Akwesasne in summer 1993,[17] (C) native commercial A1248 liquid, and (D) serum from the subject with the highest proportion (46.2%) of EM-1. For profiles not generated in the authors' laboratory (i.e., B), the same congener elution order as that in the other samples is presented to facilitate comparisons. Differences in congener coelutions between samples are indicated by brackets; congeners analyzed in the authors' laboratory but not by others are shown in italics. For brevity, CB 138 is listed alone although it coelutes with CBs 163 and 164 for all samples. In addition, CB 32 coelutes with CBs 11, 12, and 13 for the sample shown in (B). *Reprinted from DeCaprio et al.[21] with permission from Elsevier B.V.*

chlorinated congeners (PCBs 6–74) than those found in unexposed teachers. Meyer et al. (23) obtained serum PCB measurements from 134 residents of a flat with high concentrations of PCBs in the indoor air, and compared levels to those of 139 unexposed persons. Levels of 27 congeners, especially lower chlorinated congeners, were found to be four times higher in the serum of the exposed individuals.

The goal of this paper is to review the evidence that the inhalation of PCBs can lead to adverse health effects in humans. The paper will focus on a few specific diseases for which evidence exists to support the conclusion that inhalation is an important route of exposure. The problem is that most scientists who are investigating health effects of PCBs use serum PCB concentration as their exposure assessment measure. Given that most of the more volatile congeners are rapidly metabolized, they are not present in high concentrations in serum samples and, thus, they are usually not considered. However, the typical source of inhaled PCBs is indoor air in homes, schools and offices, places where people spend many hours a day. Under these circumstances, people may be more or less continuously exposed and affected by the lower chlorinated congeners.

Cancer

PCBs have been identified as Group 1, known human carcinogens, by the International Agency for Research on Cancer (24). The specific cancer with the strongest evidence is malignant melanoma. There are, however, many of types of cancer for which strong associations with serum PCB levels have been found (25). However, there is little direct evidence for cancer in humans resulting from inhalation exposure to PCBs.

Until the recent IARC identification of all PCBs being carcinogenic, there was a widespread belief that only dioxin-like PCBs had carcinogenic activity. This is despite clear evidence presented by van der Plas et al. (26). They reported that majority (about 80%) of the tumor-promoting activity of PCBs can be found the in 2–4 *ortho*-substituted congener groups, which have little or no dioxin-like activity. Sandal et al. (27) compared the genotoxic activities of PCB 52 (2,2′,5,5′tetrachloro biphenyl, a non-dioxin-like congener) and PCB 77 (3,3′,4,4′ tetrachlorobiphenyl, a dioxin-like congener) on cultured human lymphocytes. They found that both congeners caused DNA damage as monitored by the comet assay, but that PCB 52 is significantly more potent. Both PCB 9 (2,5 dichlorobiphenyl) (28) and PCB 11 (29) generate reactive oxygen species, known to be a risk factor for cell damage and death. Ludewig et al. (30) found that PCB 3 (4-monochlorobiphenyl) and/or its metabolites increase mutations in rat liver. Tan et al. (31) found that PCBs 8 (2,4 dichlorobiphenyl), 28, 47 (2,2′4,4′-tetrachlorobiphenyl), and 52 are cytotoxic to both neurons and thymocytes, but the dioxin-like congeners PCBs 77, 80 (3,3′,5,5′-tetrachlorobiphenyl) and 81 (3,4,4′,5-tetrachlorobiphenyl) are not. Although not all of these effects are necessarily directly related to cancer, they clearly demonstrate toxicity of lower chlorinated, non-dioxin-like congeners.

Case study

Company X was an analytic services laboratory that provided analysis of fluids from electric transformers. Up until 1977, when their manufacture and new use was outlawed by the US Environmental Protection Agency (EPA) due to their persistence and toxicity, most electric transformers were filled with commercial mixtures of PCBs. However, old transformers that have not been serviced still contain PCBs. Now EPA requires that the fluid from transformers being serviced or discarded be tested to determine whether PCBs are present; if they are, then the EPA requires that the fluid be removed and the transformer cleaned and filled with a non-toxic substitute. All PCB-containing fluids at concentrations <50 ppm are to be treated as hazardous waste, and rules have been established to regulate disposal of oils containing PCBs at concentrations between 2 and 50 ppm.

JM, a relatively dark-skinned Hispanic, was employed by company X between 1994 and 2003 as a laboratory technician. His job was to analyze 100–150 transformer oil samples per day to determine whether they contained PCBs. It was known that 10%–20% of those samples would have PCBs at concentrations ranging from 50 to 499 ppm, and another 10% would have even higher concentrations, some being 100% commercial PCBs. JM was told to smell the fluid to determine whether or not it contained high concentrations of PCBs. PCBs have a subtle but distinctive odor. The reason for smelling the fluids before analyzing them was that running a sample with a high PCB concentration in the gas chromatograph would result in contamination that would then take time to wash out. Thus, if samples with high concentrations could be identified before being run, they could be serially diluted to the point that they would not require extra time to be taken to wash out the gas chromatograph.

JM was born in 1967 and did not smoke nor drink to excess. His medical history was unremarkable except for hypertension, and elevated LDL with a slightly low HDL. On December 14, 2001 he was found to have a greatly

reduced thyroid stimulating hormone (TSH) level, and highly elevated thyroxine (T4) level. On February 28, 2003 he was treated with radioactive ^{131}I, which resulted in a decrease in his TSH level. On March 3, 2003 a large papillary thyroid carcinoma was removed in a subtotal thyroidectomy. The tumor surrounded the vagus nerve and it was difficult to remove. On August 26, 2003 he was found to still have an abnormally elevated uptake of ^{131}I, which was suggestive of recurrent disease. Although he continued to work at company X after his surgery, he was no longer required to analyze for PCBs. In March, 2011 JM had a malignant melanoma removed from his back. In March, 2013 JM was diagnosed with lung cancer, which on biopsy, proved to be a poorly differentiated adenocarcinoma, not a metastasis from the melanoma. JM died later in 2013 with massive hemorrhagic brain metastases.

GM, wife of JM, was born in 1968 and hired by company X in 1996. Her job was to dump oils that were in the GC sampling vials that had been analyzed into 55 gallon drums, separating those with and without high concentrations of PCBs, and ensure that any liquids containing PCBs were not allowed down the drain. She also was required to wash the glassware. She worked in a 50 sq ft room with a hood and waste basin but without windows or air conditioning, and was told to keep the door closed. When the oils were to be dumped, she was told to sniff each sample in order to determine which 55 gallon drum the material should be placed in. If it smelled like PCBs, it would go into one drum, but if not then it should go into the other. The glassware contaminated with PCBs was to be washed with toluene and acetone, followed by soap and water. She was never provided with a lab coat, gloves, or a mask.

GM was also diagnosed with thyroid cancer in May of 2003, after which she stopped working at company X. She had a total thyroidectomy in July, 2003. She completed a course of 100 mC ^{131}I on September, 2003. She had some abnormal uptake of the isotope on August 26, 2003, but there was no evidence of recurrent disease by March, 2004. In 2011, she was diagnosed with a compound melanocytic dysplastic nevus, a highly dangerous mole that is a precursor to melanoma. This was removed. She also had abnormal liver function tests, perhaps a fatty liver, diabetes, and hypertension. She does not drink and does not have hepatitis.

Serum samples were obtained in the late summer and fall of 2005 for measurement of PCBs, and analysis was done by ERGO Forschungsgesellschaft mbH in Hamburg, Germany. The results for six PCB congeners are shown in Table 3.

Table 3: PCB concentrations (μg/kg or ppb wet weight) in serum samples from JM and GM.

PCB congener	JM	GM
28	1.82	3.47
52	1.22	1.60
101	nd	0.33
138	nd	0.22
153	0.17	0.23
180	0.16	0.44
Sum	3.37	6.28

nd, not detected.

There are several remarkable findings in this tragic story. For two persons who are not blood relatives to both develop two relatively rare cancers of the same type (thyroid and melanoma) by chance is extraordinarily unlikely. Malignant melanoma is the cancer for which there is the strongest evidence for causation by PCBs. This is reflected in the recent report from the International Agency for Research on Cancer, which declared PCBs to be Group 1, known human carcinogen, based primarily of occupational studies (24). Although the route of occupational exposure is uncertain in these reports, inhalation is certainly a major component.

Thyroid cancer has been reported in rats exposed to commercial PCB mixtures (32, 33). An elevation in lung cancer has been reported in one occupational cohort after control for other factors (34). Animal studies have shown that exposure of mice to Kanechlor-400 (a Japanese PCB product) resulted in various kinds of lung neoplasms (35). JM was a non-smoker living in an area where radon is not a major problem, and it is likely that his lung cancer was also a consequence of inhaling PCBs.

The pattern of PCB congeners found in the serum sample is striking. In the general population, PCB 153, 138, and 180 are found at much higher concentrations than PCBs 28 and 52. However because PCBs 28 and 52 have fewer chlorines, are much more volatile. In the 2003–04 NHANES, mean concentrations of PCB 28 in adults over 20 was 0.031 and the 95th percentile was 0.067 ppb. For PCB 52, the mean concentration was 0.016 and the 95th percentile was 0.043 ppb. Hence, the concentrations of both congeners are two orders of magnitude higher in both JM and GM. For PCB 153, the levels in both JM and GM are within the background concentrations found among the individuals in the 2003–2004 NHANES (mean, 0.148 ppb, 95th percentile, 0.671 ppb). This pattern of PCBs in serum alone is convincing evidence that the major route of exposure for both JM and GM was inhalation of volatile PCBs.

There is other evidence consistent with the conclusion that lower chlorinated, more volatile PCBs are

carcinogenic. Although those congeners with fewer chlorines are more rapidly metabolized, they generate hydroxylated and other metabolic progeny that exhibit genotoxicity (36) and oxidative stress (29). Maddox et al. (37) demonstrated a non-significant two-fold increase in spontaneous mutations induced by PCB 3 (4 monochloro biphenyl) and 4-OH-PCB 3 in rat lung. Xie et al. (38) showed that PCB 3 is converted to quinones which are very efficient in inducing gene mutations and chromosomal breaks.

Studies using hospitalization diagnoses to assess diseases from inhalation of PCBs

My colleagues and I have performed a series of studies using New York State (NYS) hospitalization data to examine residences near hazardous waste sites containing identified chemicals, particularly PCBs. In NYS, the diseases diagnosed in every patient admitted as an inpatient to a state-regulated hospital (all except federal hospitals like Veterans' Administration and Indian Health Services) must be reported to the Department of Health upon discharge. The data available to us include sex, age, race, method of payment and zip code of residence, as well as up to 15 different disease diagnoses. The data are limited in that we do not know names or street addresses, and do not have any information about personal habits. We do have access to behavioral characteristics by county from the Behavioral Risk Factor Surveillance System (BRFSS), and we have information on median household income and population density by zip code from the US Census. We have matched rates of hospitalization for specific diseases to residence in zip codes that either contain or do not contain a hazardous waste site. The Department of Environmental Conservation lists 814 such sites in NYS and identifies those containing PCBs. Our hypothesis behind these studies is that living near a PCB-contaminated site increases exposure, and that such exposure must be primarily by inhalation. There is no reason to assume that dietary exposure would be different depending upon where you live, and it is unlikely that most people are going to have significant dermal exposure.

There are some important limitations in ecologic studies of this sort, particularly with regards socioeconomic status (SES). Poverty is well known to be an important risk for disease, but we adjust for this the best we can using the BRFSS, which provides some information on personal habits in the locale and census data, from which we can obtain median household income in the zip code. The exposure assessment is also very limited, being only the zip code of residence. We cannot distinguish multiple hospitalizations by one person from those of different individuals. However, despite these limitations, there are some other major strengths. For example, there are 2.5 million hospitalizations each year in NYS, and we have data from 1993 through 2008. We have used results of these studies to generate hypotheses, which we then tested in smaller populations wherein we have better exposure assessment.

Cardiovascular disease

Sergeev and Carpenter (39) examined rates of hospitalization for coronary heart disease and myocardial infarction in NYS residents living in a zip code wherein a PCB hazardous waste site was located, and compared these rates with those living in a zip code without any hazardous waste site after adjustment for age, sex, race, income, and health insurance coverage. They found an odds ratio (OR) of 1.15 (95% confidence interval=1.03–1.29) for coronary heart disease and an OR of 1.20 (1.03–1.39) for myocardial infarction. They then examined a sub-set of the PCB zip codes, that being those along the 200 miles of the contaminated Hudson River. Average income is higher in these zip codes, and BRFSS data show more exercise, less smoking, and greater consumption of fruits and vegetables in these counties than in the rest of NYS. Despite living a healthier life style, the ORs for coronary heart disease and myocardial infarction in these zip codes were 1.36 (1.19–1.56) and OR=1.39 (1.19–1.63), respectively. Thus, living in a zip code containing a PCB hazardous waste site (either a landfill or a contaminated body of water) is associated with an increased risk of coronary heart disease and myocardial infarction, and this is unlikely due to inadequate adjustment for socio-economic status because the elevations in ORs are even higher along the Hudson.

Strokes ('brain attacks') are closely related to myocardial infarctions ('heart attacks'). Shcherbatykh et al. (40) used the same hospitalization records for stroke. They found significant elevations for ischemic stroke for individuals living in PCB-contaminated zip code (OR=1.17, 1.04–1.39) and a slightly greater elevation for individuals living along the Hudson River (OR=1.20, 1.10–1.32) as compared with zip codes without any hazardous waste site.

The above ecologic studies support the hypothesis that exposure to PCBs increases the risk of cardiovascular disease. In order to test this hypothesis, we performed

studies in two PCB-exposed populations where we have measured serum PCB concentrations. We suspect that the route of exposure for those individuals living near PCB hazardous waste sites is inhalation of lower chlorine congeners which are not very persistent. Hence, it is not clear whether the associations seen with measurement of total serum PCBs will give exactly the same results.

Goncharov et al. (41) determined self-reported rates of cardiovascular disease among the Mohawks at Akwesasne, a Native American group living at the US-Canadian border, in relation to measured serum PCBs and serum lipids. They found significantly elevated risk of self-reported cardiovascular disease, but found this to be an indirect effect via an elevation in serum cholesterol and triglycerides. Aminov et al. (42) investigated these same relationships in 575 residents of Anniston, Alabama who live near the Monsanto plant that manufactured PCBs. They also found that increased total serum PCB concentrations was significantly associated with elevated concentrations of total cholesterol and triglycerides, but found no effect on HDL or LDL cholesterol. Thus, there is a clear association between elevation in serum lipids, a major risk factor for cardiovascular disease, and more highly chlorinated PCBs, whereas the ecologic results support the conclusion that the lower chlorinated congeners are also important. At present, the relative importance of lower and higher chlorinated congeners on cardiovascular disease remains to be fully determined. Hennig et al. (43) have demonstrated pro-inflammatory changes induced by PCBs on endothelial cells, which may combine with elevations in serum lipids to increase the risk of cardiovascular disease. Ha et al. (44) have reported that there is a dose-dependent relationship between serum PCB concentrations and cardiovascular disease using data from the National Health and Nutrition Examination Survey (NHANES).

Hypertension

Hypertension is not usually considered to be an environmental disease. However, using the hospitalization data set, Huang et al. (45) reported a significantly elevated OR=1.19 (1.09–1.31) for hospitalization diagnosis of hypertension among individuals living in a zip code with a PCB hazardous waste site. They also found elevated hospitalization for hypertension (OR=1.14; 1.05–1.23) for residents living along the Hudson River.

We have determined the associations between serum PCB levels and blood pressure in 351 residents of Anniston who were not on anti-hypertensive medication. Three measurements of blood pressure were taken in individuals where serum PCBs levels had been measured. We found striking associations between rates of hypertension and serum PCB concentrations (46). After adjustment was age, sex, BMI, serum lipids, smoking and exercise the OR for lowest to highest tertile of PCB concentration was 4.09 (1.3–12.7) for clinical hypertension and 5.28 (1.0–25.8) for both systolic and diastolic hypertension. Even within the normotensive range of blood pressure, there were significant associations with total PCB concentration (47). Serum PCB concentration showed a stronger association than any other factor but age, including BMI, total lipids, sex, race, smoking, and exercise. Associations between serum PCBs and hypertension have also been reported using NHANES data (48, 49).

Diabetes

Kouznetsova et al. (50) analyzed NYS hospitalization data for adult inpatient admissions for diabetes in relation to residence in a zip code containing a PCB-contaminated waste site. Living in a PCB-contaminated zip code was associated with a 23% elevated chance of hospitalization for diabetes as compared with rates for individuals living in a zip code that did not contain a hazardous waste site (OR=1.23; 1.15–1.32), after adjustment for age, race, sex, median household income, and urban/rural residence. Living along the Hudson River was associated with an even greater elevation (OR=1.36; 1.25–1.42). As with the above diseases, the most likely exposure must have come from inhalation.

We have examined rates of physician-diagnosed diabetes in relation to serum PCB concentrations in the Mohawk population at Akwesasne. In a preliminary study, Codru et al. (51) reported that after adjustment for sex, age, BMI and smoking, individuals in the top tertile PCB concentration had a significant 3.9-fold elevated risk of diabetes (95% CI=1.5–10.6). Only two individual congeners were reported, PCBs 74 (2,4,4′,5-tetrachlorobiphenyl) and 153. When adjusted for all other contaminants in addition to the factors listed above, only PCB 74 showed a significant association. We have followed-up on this study (52) with a more complete single congener analysis and with adjustment for all other contaminants but the one under investigation. These results indicate that the only significant association with diabetes is with non- or mono-*ortho* PCB congeners that do not have dioxin-like activity. This is an important observation because these are the lower-chlorinated, volatile congeners. This provides strong support for the hypothesis developed from the hospitalization studies (50), which concluded that

the association between diabetes and living near a PCB-contaminated site is secondary to inhalation of lower chlorinated PCBs.

Discussion and conclusions

These results are consistent with the conclusion that inhalation of PCBs is not only an important route of exposure, but that it can also result in serious disease. PCB exposure is well documented to increase the risk of the diseases reviewed here, namely, cancer, cardiovascular disease, hypertension and diabetes, based on documentation that incidence of these diseases increased with serum concentrations of PCBs. However, the majority of the PCBs found in serum are the more persistent congeners, often with half-lives of a decade or more. These are the congeners found in the higher chlorinated commercial mixtures, and are the ones commonly found in animal fats, which is an important route of exposure to humans. From the point of view of research, the persistence of these higher chlorinated congeners is helpful for establishing associations because a blood sample will provide information about PCB exposure after many years have passed.

This review has focused on only four diseases, chosen because of at least some evidence for elevated risk coming from inhalation exposure. However, these are certainly not the only diseases for which exposure to PCBs is known to increase risk. PCBs are known to cause deficits in learning and memory (53, 54), and there is evidence from animal studies indicating that lowered chlorinated congeners are more neurotoxic than more highly chlorinated congeners (55). Fitzgerald et al. (56) reported decrements of verbal learning and an increase in depressive symptoms in adults living near the contaminated Hudson River, but serum concentrations are not significantly different from those in a comparison population (57). This finding is consistent with inhalation of lower chlorinated, more rapidly metabolized PCBs as the critical factor. PCBs are structurally somewhat similar to T4, and exposure has been shown to suppress thyroid function (58). PCBs also alter sex hormone function, with many congeners and hydroxylated metabolites having estrogenic activity (59). Elevated PCB exposure results in earlier puberty in girls (60) and a reduction in testosterone levels in men and boys (61, 62). PCBs suppress the immune system, leading to increased respiratory infections in children (63, 64) and elevations in cases of asthma (64, 65). PCB exposure to mothers is associated with lower birth weight of infants (66, 67). The relative role of inhalation of lower chlorinated PCBs, to date, has not been studied with regards these diseases and effects.

The PCB congeners that volatilize easily are less highly chlorinated, and most of them are much more rapidly metabolized in the human body. Some, like PCBs 28 and 52, are somewhat more persistent than others, and are frequently found at low concentrations in human serum, although the majority of those congeners with four of fewer chlorines are often not present at detectable concentrations. However, just because they are more rapidly metabolized and do not accumulate does not mean that they do not have adverse health effects. This is particularly the case if the concentrations of these lower chlorinated congeners in air are significant in places where people spend long periods of time (e.g., at home, school, or work). Under these circumstances exposure can be almost continuous, but would not be reflected in high levels of PCBs. Although the specific mechanisms whereby serum PCBs cause neurotoxicity are still uncertain, animal studies have shown that PCB, like lead, are effective in reducing long-term potentiation, an electrophysiologic marker of learning and memory (68).

The most extreme demonstration of the hazards of inhalation of PCBs is the cases of JM and GM, workers occupationally instructed to inhale PCB vapors. Both developed multiple cancers of the same type, and JM died of cancer. Their serum contained elevated concentrations of the lower chlorinated, more volatile PCBs, and only background concentrations of more highly chlorinated congeners that are less volatile.

The ecologic studies showing elevations of cardiovascular disease, hypertension, and diabetes in relation to residences near PCB-contaminated waste sites strongly suggest that inhalation is the route of exposure. However, there are significant limitations to ecologic studies, and they must be viewed as being hypothesis generating. Therefore, we have performed other investigations in defined populations where we have good exposure assessment (albeit with the limitations discussed above for lower chlorinated congeners), as well as access to medical and clinical chemistry information. These studies confirm the hypothesis that PCB exposure is associated with elevated risks of all three diseases. Thus, these studies provide support for the conclusion that inhalation of PCBs is the major cause of the elevated rates of hospitalization.

The implications of these studies are significant for several reasons. First, these results suggest that living near a PCB-contaminated waste site poses risk to health, and by extrapolation this applies also to attending a school with elevated PCBs in the air due to PCB-containing light ballasts or caulk (69–73), working in a contaminated building

(74, 75), working as a fireman around certain house fires (76), and living downwind of sewage sludge drying plants (77). Lower chlorinated PCBs are found in current retail paints, and would be expected to volatilize into room air (78). Urban areas are likely to have more hot spots with higher concentrations than in rural areas, as has been demonstrated in Chicago and Cleveland (79). Thus, many people are being unknowingly exposed to these sources via inhalation. Scientists from the USEPA have recently published a report calling for greater evaluation of health risks from inhaled PCBs (80).

PCBs are dangerous chemicals, but the danger is not restricted to dioxin-like congeners or persistent congeners. These findings reinforce the conclusion that it is imperative to find ways of removing these contaminants from the environment. Furthermore, it is important that risk assessment methodologies no longer rely only on measurement of serum PCB levels and their associations with various diseases, but rather consider air concentrations and the evidence that even low concentrations of PCBs in air constitute an important route of exposure and disease, especially if the exposure is prolonged.

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56. Fitzgerald EF, Belanger EE, Gomez MI, Cayo M, McCaffrey RJ, et al. Polychlorinated biphenyl exposure and neuropsychological status among older residents of upper Hudson River communities. Environ Health Perspect 2008;116:209–15.
57. Fitzgerald EF, Belanger EE, Gomez MI, Wilson LR, Belanger EE, et al. Environmental exposures to polychlorinated biphenyls (PCBs) among older residents of upper Hudson River communities. Environ Res 2007;104:352–60.
58. Schell LM, Gallo MV, Denham M, Ravenscroft J, DeCaprio AP. Relationship of thyroid hormone levels to levels of polychlorinated biphenyls, lead, p,p'-DDE, and other toxicants in Akwesasne Mohawk youth. Environ Health Perspect 2008;116:806–13.
59. DeCastro BR, Korrick SA, Spengler JD, Soto AM. Estrogenic activity of polychlorinated biphenyls present in human tissue and the environment. Environ Sci Technol 2006;40:2819–25.
60. Schell LM, Gallo MV. Relationships of putative endocrine disruptors to human sexual maturation and thyroid activity in youth. Physiol Behav 2010;99:246.
61. Goncharov A, Rej R, Negoita S, Schymura M, Santiago-Rivera A, et al. Lower serum testosterone associated with elevated polychlorinated biphenyl concentrations in Native American men. Environ Health Perspect 2009;117:1454–60.
62. Schell LM, Gallo MV, Deane GD, Nelder KR, DeCaprio AP, et al. Relationships of polychlorinated biphenyls and dichlorodiphenyldichloroethylene (p,p'-DDE) with testosterone levels in adolescent males. Environ Health Perspect 2014;122:304–9.
63. Dallaire F, Dewailly É, Vézina C, Muckle G, Weber JP, et al. Effect of prenatal exposure to polychlorinated biphenyls on incidence of acute respiratory infections in preschool Inuit children. Environ Health Perspect 2006;114:1301–5.
64. Ma J, Kouznetsova M, Lessner L, Carpenter DO. Asthma and infectious respiratory disease in children – correlation to residence near hazardous waste sites. Paediatr Respirat Rev 2007;8:292–8.
65. Hansen S, Strøm M, Olsen SF, Maslova E, Rantakokko P, et al. Maternal concentrations of persistent organochlorine pollutants and the risk of asthma in offspring: results from a prospective cohort with 20 years of follow-up. Environ Health Perspect 2014;122:93–9.
66. Baibergenova A, Kudyakov R, Zdeb M, Carpenter DO. Low birth weight and residential proximity to PCB-contaminated waste sites. Environ Health Perspect 2003;111:1352–7.
67. Govarts E, Nieuwenhuijsen M, Schoeters G, Ballester F, Bloemen K, et al. Birth weight and prenatal exposure to polychlorinated biphenyls (PCBs) and dichlorodiphenyldichloroethylene (DDE): a meta-analysis within 12 European birth cohorts. Environ Health Perspect 2012;120:162–70.
68. Carpenter DO, Hussain RJ, Berger DF, Lombardo JP, Park H-Y. Electrophysiologic and behavioral effects of perinatal and acute exposure of rats to lead and polychlorinated biphenyls. Environ Health Perspect 2002;110:377–86.
69. Gabrio T, Piechotowski I, Wallenhorst T, Klett M, Cott L, et al. PCB-blood levels in teachers, working in PCB-contaminated schools. Chemosphere 2000;40:1055–62.
70. Schwenk M, Gabrio T, Päpke O, Wallenhorst T. Human biomonitoring of polychlorinated biphenyls and polychlorinated dibenzodioxins and dibenzofurans in teachers working in a PCB-contaminated school. Chemosphere 2002;47:229–33.
71. Johansson N, Hanberg A, Wingfors H, Tysklind M. PCB in building sealant is influencing PCB levels in blood of residents. Organohalogen Compounds 2003;63:381–4.
72. Herrick RF, McClean MD, Meeker JD, Baxter LK, Weymouth GA. An unrecognized source of PCB contamination in schools and other buildings. Environ Health Perspect 2004;112:1051–3.
73. Kohler M, Tremp J, Zennegg M, Seiler C, Minder-Kohler S, et al. Joint sealants: an overlooked diffuse source of polychlorinated biphenyls in buildings. Environ Sci Technol 2005;39:1967–73.
74. Wingfors H, Seldén AI, Nilsson C, Haglund P. Identification of markers for PCB exposure in plasma from Swedish construction workers removing old elastic sealants. Am Occup Hyg 2006;50:65–73.
75. Broding HC, Schettgen T, Göen T, Angerer J, Drexler H. Development and verification of a toxicokinetic model of polychlorinated biphenyl elimination in persons working in a contaminated building. Chemosphere 2007;68:1427–34.
76. Ruokojarvi P, Marjaleena M, Ruuskanen J. Toxic chlorinated and polyaromatic hydrocarbons in simulated house fires. Chemosphere 2000;41:825–8.
77. Hsu Y-K, Holsen TM, Hopke PK. Locating and quantifying PCB sources in Chicago: receptor modeling and field sampling. Environ Sci Technol 2003;37:681–90.
78. Hu D, Hornbuckle KC. Inadvertent polychlorinated biphenyls in commercial paint pigments. Environ Sci Technol 2010;44:2822–7.
79. Persoon C, Peters TM, Kumar N, Hornbuckle KC. Spatial distribution of airborne polychlorinated biphenyls in Cleveland, OH and Chicago, IL. Environ Sci Technol 2010;44:2797–802.
80. Lehmann GM, Christensen K, Maddaloni M, Philips LJ. Evaluating health risks from inhaled polychlorinated biphenyls: research needs for addressing uncertainty. Environ Health Perspect 2015;123:109–13.

CURRICULUM VITAE

Name: David O. Carpenter

Home Address: 2749 Old State Road
Schenectady, New York 12303

Positions Held:
Director, Institute for Health and the Environment
University at Albany
Professor, Environmental Health Sciences
School of Public Health, University at Albany
5 University Place, A217, Rensselaer, NY 12144

Honorary Professor
Queensland Children's Medical Research Institute
University of Queensland
Brisbane, Australia

Education:

1959	B.A., Harvard College, Cambridge, MA
1964	M.D., Harvard Medical School, Boston, MA

Positions Held:

9/61-6/62	Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg
7/64-6/65	Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
7/65-2/73	Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
2/73-3/80	Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
3/80-9/85	Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
9/85-1/98	Dean, School of Public Health, University at Albany
9/85-Pres.	Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
9/85-7/98	Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
1/98-1/05	Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
2001-Pres.	Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.
2005-2010	Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New York
2011-Pres.	Honorary Professor, Queensland Children's Medical Research Institute, University of Queensland, Brisbane, Australia

Editor-in-Chief:	Cellular and Molecular Neurobiology, 1981 – 1987
Editor-in Chief:	Reviews on Environmental Health 2012-present
Editor-in-Chief:	Environmental Pollution 2015-2019
Editorial Advisor:	Cellular and Molecular Neurobiology, 1987 – Present
Academic Editor:	Journal of Environmental and Public Health, 2009-2013
Academic Editor:	PLoS ONE 2014-2016
Editorial Boards:	Journal of Public Health Management and Practice, 1995 - 2002 International Journal of Occupational Medicine & Environmental Health 1996 – 2016 Journal of Alzheimer's Disease – Associate Editor,2007-2009 Reviews on Environmental Health; 2008-2012 International Archives of Occupational and Environmental Health; 2009-present. Environmental Health Perspectives, 2010-2017 Global Health Perspective, 2012-present Environment International 2013-present International Journal of Environmental Research and Public Health: 2019-present.

National and International Committees:

1978, 1981	Physiology Study Section (Ad hoc member)
1979-1985	NIH International Fellowship Study Section
1974-1981	Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
1981-1989	Member, USA National Committee for the International Brain Research Organization
1985-1986	Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
1986-1987	Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
1987-1989	Member, Science Advisory Council of the American Paralysis Association
1987-1990	Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
1985-1993	Committee #79, National Council on Radiation Protection and Measurements
1986-1997	Member, Legislative and Education Committees, Association of Schools of Public Health
1989-1994	Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
1994, 1995	Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
1994-1997	Member, Legislative Committee of the Association of Schools of Public Health
1997	Member, Executive Committee of the Association of Schools of Public Health
1997-2000	National Advisory Environmental Health Sciences Council of the National Institutes of Health
1998-2015.	Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
2000-Pres.	Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
2001-2008	United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
2002-2003	Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
2001-Pres.	Member, Board of Directors, Alliance for Public Health and Associates, Inc.
2003-2008	Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
2003-2012	Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
2004-2012	Member, Blue Ocean Institute Curriculum Advisory Board.

2007-2011	Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board, International Joint Commission.
2013	Invited Expert, International Agency for Research on Cancer, Panel for Monograph 107, Carcinogenicity of Polychlorinated Biphenyls.
2013-Pres.	Member, Global Burden of Disease Panel

State and Local Committees:

1980-1987	Executive Secretary, New York State Power Lines Project
1985-1989	Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
1986-1989	Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
1991-1992	Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
1991-1992	Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
1991-1992	Member, State Task Force for the Reform of Middle Level Education in NY State
1992-1993	Member, State Needs Task Force on Health Care and Education
1987-1998	Delegate-at-Large, New York State Public Health Association
1991-1995	Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
1994	Chair, Council of Deans, University at Albany, SUNY
1997-2008.	Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
2000-Pres.	Member, Board of Directors, Healthy Schools Network, Inc.
2000-2003	Member, Medical Advisory Board, Hepatitis C Coalition, New York
2000-2004	Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
2001-2008	Member, Board of Directors, Environmental Advocates of New York
2004-2007	Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
2005-Pres.	Member, Schooling Chefs Curriculum Advisory Board
2005-Pres.	Member, Advisory Board, Healthy Child Healthy World
2005-2008	Member, Board of Directors, Citizens Environmental Coalition
2006-2009	Member, Board of Directors, Marine Environmental Research Institute
2007-2009	Member, New York State Renewable Energy Task Force
2013-2015	Member, Medical Society of the State of New York (MSSNY)
2013-2015	Member, Preventive Medicine and Family Health Committee, MSSNY
2014-Pres.	Member, Board of Directors, Regenerative Research Foundation
2014-Pres.	Member, Board of Directors, International Institute for Health and Education

Honors, Awards and Fellowships:

1959	B.A. awarded magna cum laude. Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, *Ambystoma tigrinum*" (Thesis advisor, Professor George Wald)
	Elected to Phi Beta Kappa and to Sigma Xi
1964	M.D. awarded *cum laude* for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
1964	Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
1970	Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
1971	Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios

	Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months
1982, 1986	Visiting Professor of Physiology, Department of Physiology, Kyushu
1987	University, Fukuoka, Japan, for a period of three months each
1989	Awarded Jacob Javits Neuroscience Investigator Award from the National Institute of Neurological and Communicative Diseases and Stroke
1999	Awarded Homer N. Calver Award from the American Public Health Association for studies in environmental health.
2001	Awarded 2001 Academic Laureate from the University at Albany Foundation.
2010	Awarded the Albion O. Bernstein, M.D. Award in recognition of an outstanding contribution to public health and the prevention of disease though lifelong research of environmental health hazards and for limitless devotion to medical education by the Medical Society of the State of New York.
2011	Awarded the Rodney Wylie Eminent Visiting Fellowship 2011 at the University of Queensland, Brisbane, Australia for a period of four weeks.
2013	Awarded the Annual Kenneth V. Dodgson, M.D., Lectureship at the University of Rochester Department of Occupational and Environmental Medicine Grand Rounds.
2019	Received the Third Age Achievement Award for Education, given by Senior Services of Albany
2020	Awarded the Theo Colborn Career Achievement Award for Research and Advocacy in Environmental Heatlh by the Environmental Health Symposia.

Federal Grants Held: (Principal Investigator Only)

1980-1983	United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs", $76,847 total direct costs.
1982-1988	National Institute of Health, "Mechanisms of Desensitization at Central Synapses", $464,786 total direct costs.
1984-1986	Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs@, $330,504 total direct costs.
1986-1996	National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and Toxicity", 1986-1989 $231,848 total direct costs; 1990-1996 $562,926 total direct costs.
1989-1993	National Institute of Health, "Mechanisms of Lead Neurotoxicity" $373,576 total direct costs
1990-1995	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O. Carpenter, P.I. $5,783,419 total direct costs.
1995-2001	Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. ACentral/Eastern European Environ/Occup Training Program@, D.O. Carpenter, P.I. $657,520 total costs.
1995-2001	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. $12,653,709 total direct costs.
1998-1999	Environmental Protection Agency, AIndoor Air Risk at Akwesasne - Pilot Project@, D.O. Carpenter, P.I. $9,996 total costs.
2000-2002	Association Liaison Office for University Cooperation in Development, ACooperative Program in Environmental Health between the Institute of Public Health at Makerere University, Kampala, Uganda and the School of Public Health, University at Albany, USA@,

D.O. Carpenter, P.I. $96,432 total costs.

2001-2007 Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. AMultidisciplinary Environmental Health Training@, D.O. Carpenter, P.I. $850,000 total costs.

2006-2011 Pakistan-US Science and Technology Cooperative Program (US National Academy of Sciences). "Association of particulate matter with daily morbidity in an urban population," D.O. Carpenter, P.I., $391,104 total costs.

2009-2013 Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2: Environmental contaminants and reproductive health of Akwesasne Mohawk women. $387,825 for year 1. D.O. Carpenter, Co-PI.

2010-2013 Department of the Army, "Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., $636,958 total costs.

2010-2013 Higher Education for Development of the United States Agency for International Development, "Drinking Water Supply, Sanitation, and Hygiene Promotion : Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda". D. O. Carpenter, P.I., $299,736 total costs.

2011-2016 National Institute of Environmental Health Sciences (1RO1ES019620), "Protecting the health of future generations: Assessing and preventing exposures." PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, $471,521 for the period 8/08/11-4/30/12, $2,354,871 for the period 2011-2016.

2017-2022 National Institute of Environmental Health Sciences (2RO1ES19620-06A1), "Protecting the Health of Future Generations: Assessing and Preventing Exposures to Endocrine0Disrupting Flame Retardant Chemicals & PCBs in Two Alaska Native Arctic communities on St. Lawrence Island." PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-PIs. $554,464 for the period 2018.

2020-2025 National Institute of Environmental Health Sciences (RO1 ES032392) "Restoring Northeast Cape for the Health and Well-Being of the Yupik Communities of St. Lawrence Island, Alaska." F von Hippel, C Buck, DO Carpenter, PK Miller, Co-PIs. 11/01/2021-10.31.2025. Total Award Amount (including indirect costs): $2, 985,224.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson's Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, The Public Radio Health Show (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001.

Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Member of the Ethics Board, Town of Guilderland, 2013 – 2018

Albany Mayor's Advisory Committee on Air Pollution in the South End, 2016-present.

Board member and treasurer: Health Schools Network, 200-present.

Board member: Regenerative Research Foundation; 2010-present

Board member: National Toxic Encephalopathy Foundation, 2019 – present.

Board member: RADIX Ecological Sustainability Center, 2018-present.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.
6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibitibility of motoneurons of different sizes. J. Neurophysiol., 28:599-620, 1965.
7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. J. Neurophysiol., 28:560-580, 1965.
8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J. Pharmacol., 148:380-385, 1965.
9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. J. Neurophysiol., 28:958-965, 1965.
10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. Arch. Ital. Biol., 104:73-85, 1966.
11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. J. Neurophysiol., 29:353-368, 1966.
12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in Aplysia neurons. J. Gen. Physiol., 50:1469-1484, 1967.
13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv. Pharmacol., 6A:351-364, 1968.
14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na^+ pump to membrane potential in Aplysia neurons. J. Gen. Physiol., 52:1-21, 1968.
15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch. Neurol., 19:591-597, 1968.
16. Carpenter, D.O. Membrane potential produced directly by the Na^+ pump in Aplysia neurons. Comp. Biochem. Physiol., 35:371-385, 1970.
17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of Aplysia neurons upon Na^+ and Ca^{++}. J. Cell. Physiol., 75:121-127, 1970.

18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepin-ephrine in the presence of tetrodotoxin. J. Pharmacol. Exp. Therap., 73:416-421, 1970.

19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. Science, 169:597-598, 1970.

20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of Aplysia neurons and squid axon as determined by a new technique. Intl. J. Neurosci., 2:35-48, 1971.

21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in Aplysia nervous and non-nervous tissues. Int. J. Neurosci., 2:49-56, 1971.

22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of Aplysia neuron somata. Science, 176:1329-1331, 1972.

23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. Science, 179:1336-1338, 1973.

24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. J. Physiol. (Lond.), 229:471-493, 1973.

25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. Aplysia acetylcholine receptors: Blockage by and binding of α-bungarotoxin. Brain Res., 72:225-240, 1974.

26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. Brain Res., 73:156-160, 1974.

27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in Aplysia suggests neurotransmitter function. Science, 185:364-365, 1974.

28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of Aplysia neurons. J. Cell. Physiol., 84:463-472, 1974.

29. Brownstein, M.J., Saavedra, J.M., Axelrod, J., Zeman, G.H. and Carpenter, D.O. Coexistence of several putative neurotransmitters in single identified neurons of Aplysia. Proc. Natl. Acad. Sci. (USA), 71:4662-4665, 1975.

30. Carpenter, D.O. and Gaubatz, G.L. Octopamine receptors on Aplysia neurons mediate hyperpolarization by increasing membrane conductance. Nature, 252:483-485, 1974.

31. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Afferent nerve fiber activity responding to temperature changes of the scrotal skin of the rat. J. Neurobiol., 38:601-612, 1975.

32. Carpenter, D.O. and Gaubatz, G.L. H_1 and H_2 histamine receptors on Aplysia neurons. Nature, 254:343-344, 1975.

33. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Resistivity of axoplasm. II. Internal restivity of giant axons of squid and Myxicola. J. Gen. Physiol., 66:139-148, 1975.

34. Zeman, G.H. and Carpenter, D.O. Asymmetric distribution of aspartate in ganglia and single neurons of Aplysia. Comp. Biochem. Physiol., 52C:23-26, 1975.

35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents in vitro. Pflugers Arch., 359:349-356, 1975.

36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on Aplysia neurons. Nature, 258:751-754, 1975.

37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on Aplysia neurons. Science, 192:806-809, 1976.

38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. Biophys. J., 16:991-1001, 1976.

39. Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α-bungarotoxin with Aplysia acetylcholine receptors. Mol. Pharmacol., 12:999-1006, 1976.

40. Saavedra, J.M., Ribas, J., Swann, J. and Carpenter, D.O. Phenylethanolamine: A new putative neurotransmitter in Aplysia. Science, 195:1004-1006, 1977.

41. Carpenter, D.O., Swann, J.W. and Yarowsky, P.J. Effect of curare on responses to different putative neurotransmitters in Aplysia neurons. J. Neurobiol., 8:119-132, 1977.

42. Yarowsky, P.J. and Carpenter, D.O. GABA mediated excitatory responses on Aplysia neurons. Life Sci., 20:1441-1448, 1977.

43. Willis, J.A., Myers, P.R. and Carpenter, D.O. An ionophoretic module which controls electroosmosis. J. Electrophysiol. Tech., 6:34-41, 1977.

44. Yarowsky, P.J. and Carpenter, D.O. Receptors for gamma-aminobutyric acid (GABA) on Aplysia neurons. Brain Res., 144:75-94, 1978.

45. Carpenter, D.O., Gaubatz, G., Willis, J.A. and Severance, R. Effects of irradiation of Aplysia pacemaker neurons with 20 MeV electrons. Rad. Res., 76:32-47, 1978.

46. Yarowsky, P.J. and Carpenter, D.O. A comparison of similar ionic responses to gamma-aminobutyric acid and acetylcholine. J. Neurophysiol., 41:531-541, 1978.

47. Blum, B., Auker, C.R. and Carpenter, D.O. A head holder and stereotaxic device for the rattlesnake. Brain Res. Bull., 3:271-274, 1978.

48. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Dopamine-induced muscle contractions and modulation of neuromuscular transmission in Aplysia. Brain Res., 157:167-172, 1978.

49. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Evidence for identified dopamine motor neurons to the gill of Aplysia. Neurosci. Lett., 10:275-280, 1978.

50. Kebabian, P.R., Kebabian, J.W. and Carpenter, D.O. Regulation of cyclic AMP in heart and gill of Aplysia by the putative neurotransmitters, dopamine and serotonin. Life Sci., 24:1757-1764, 1979.

51. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Res. Bull., 4:149-152, 1979.

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