



TOWN OF LEE
32 Main Street, Lee, MA 01238
www.lee.ma.us

Select Board

January 2, 2024

His Excellency Joseph Biden, President of the United States
The Honorable Edward Markey, U.S. Senate
The Honorable Elizabeth Warren, U.S. Senate
The Honorable Richard Neal, U.S. House of Representatives
Her Excellency Maura Healey, Governor of Massachusetts
The Honorable Andrea Campbell, Attorney General of Massachusetts
The Honorable Paul Mark, State Senator
The Honorable Smitty Pignatelli, State Representative, 3rd Berkshire
EPA Region 1 Administrator David Cash
EPA Administrator Michael S. Regan
H. Lawrence Culp, Jr., President of General Electric

RE: Housatonic Rest of River – General Electric/EPA PCB Cleanup

The Lee Select Board recently obtained several General Electric (GE) and Monsanto documents that are cause for great concern related to the GE/EPA Housatonic Rest of River Cleanup remedy. Namely, the attached documents show that GE signed an agreement releasing Monsanto of liability for PCB contamination. To our knowledge, this was not disclosed to the EPA or the First Circuit Court of Appeals. In addition, the EPA failed to adequately investigate this relationship between GE and Monsanto. Also attached, is an internal Monsanto document (cancer index) outlining each employee diagnosed with cancer from PCB's to as far back as 1949 (over 2 decades prior to the release with GE). David Carpenter, the foremost expert on the human health impacts of PCBs, testified to the Lee Board of Health in an Affidavit stating that the current location of the projected PCB dump in Lee would be catastrophic to the residents of Lee. His affidavit is attached.

In 1968 Monsanto learned that PCBs were discharged into rivers, and unlike other chemicals, were not carried by the river currents to the sea. Instead, they became imbedded into the settlements of the rivers.

On February 21, 1970, Monsanto's plasticizer department wrote a letter to all its 661 plasticizers customers warning them about contamination to waterways. Monsanto attached information in this letter stating that chemical and industrial plants were located next to rivers and disposed of chemical waste via river discharges. Monsanto also included information in this letter stating that the Commonwealth of Massachusetts only had regulations concerning river discharges for radioactive chemicals.

In August of 1970, Monsanto halted all production of Aroclors 1254 and 1262 due to overwhelming evidence it had that PCBs were toxic and harmful to humans and the environment. By that date, Monsanto had sold more than 1.4 billion pounds of the Aroclors.

On January 21, 1971, Monsanto entered into a contract with GE to continue selling Aroclors 1254 and 1262 for usage in electrical equipment. Monsanto conveyed to GE under the terms of the contract that PCBs were severely harmful to humans and the environment. GE executed this contract assuming all responsibility for any damages Monsanto might have to pay to others for PCB exposures.


In the opinion of the Town of Lee, the January 21, 1971 contract between Monsanto and GE violates Massachusetts Civil Conspiracy Law. The Town is currently planning to file charges against Monsanto this month, and related claims, including a public nuisance claim.

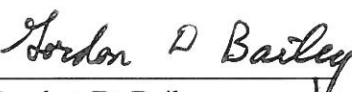
Since the time of the amended EPA permit in 2020, the Town of Lee has continued to express its overall discontent with the cleanup project as a whole. The Town, through numerous elections and public meetings, has objected to almost every aspect of the cleanup including, but not limited to, the toxic waste dump proposed for the Town of Lee, the lack of any alternative technologies for the cleanup remedy, and the potential impact on our infrastructure and human health.

The Town of Lee played no part in the contamination of the river and considers this an environmental injustice that the residents of Lee (with the lowest income of all towns in the river cleanup area) be subject to 13 years of disruption and risk to human health followed by centuries of potential issues from a toxic waste dump in our Town.

Based on the information presented in this letter, the Town of Lee will continue to pursue all avenues for a cleanup remedy where all involved parties are held responsible and all contaminated sediment is removed from Lee and the Berkshires. We look forward to your support in this mission and welcome the opportunity to discuss this situation further. Feel free to contact us through the Town Administrator, Christopher Brittain, at cbrittain@town.lee.ma.us or (413) 409-5976 to discuss this matter further.

Sincerely,


Bob Jones, Chair


Gordon D. Bailey


Sean Regnier

Attachments: Internal GE/Monsanto Documents (Exhibit A), Mickey Friedman 3 Part Series (Exhibit B), Dr. Carpenter Affidavit (Exhibit C).

cc: Berkshire Eagle
Berkshire Edge
Stockbridge Updates
Boston Globe
Springfield Republican
New England Public Radio
New York Times
Washington Post
ABC, CBS, NPR, Fox, CNN

EXHIBIT A

Monsanto

MONSANTO INDUSTRIAL CHEMICALS CO.
800 N. Lindbergh Boulevard
St. Louis, Missouri 63166
Phone: (314) 694-1000

SPECIAL UNDERTAKING BY PURCHASERS OF POLYCHLORINATED BIPHENYLS

Monsanto Company ("Monsanto") manufactures certain polychlorinated biphenyl products ("PCB's") which General Electric Company ("Buyer") desires to purchase. While Buyer desires to purchase PCB's because of certain desirable flame resistant and insulator properties, Buyer acknowledges that it is aware and has been advised by Monsanto that PCB's tend to persist in the environment; that care is required in their handling, possession, use and disposition; that tolerance limits have been or are being established for PCB's in various food products.

Monsanto has therefore adopted certain restrictive policies with respect to its further production, sale and delivery of PCB's, including the receipt of undertakings from its customers as set forth below, and Buyer is willing to agree to such undertakings with respect to sales and/or deliveries of PCB's by Monsanto to Buyer.

Accordingly, Buyer hereby covenants and agrees that, with respect to any and all PCB's sold or delivered by or on behalf of Monsanto to Buyer on or after the date hereof and in consideration of any such sale or delivery, Buyer shall defend, indemnify and hold harmless Monsanto, its present, past and future directors, officers, employes and agents, from and against any and all liabilities, claims, damages, penalties, actions, suits, losses, costs and expenses arising out of or in connection with the receipt, purchase, possession, handling, use, sale or disposition of such PCB's by, through or under Buyer, whether alone or in combination with other substances, including, without implied limitation, any contamination of or adverse effect on humans, marine and wildlife, food, animal feed or the environment by reason of such PCB's.

0041818

a unit of Monsanto Company

EX P-0622
Page 1 of 2

PCB-ARCH0039335

All existing contracts for the sale of PCB's by Monsanto to Buyer are hereby amended to contain the provisions set forth above.

Nothing herein shall create or imply any duty or obligation of Monsanto to sell or deliver any PCB's to Buyer. No conditions, understandings or agreements purporting to modify or vary the terms hereof shall be binding unless hereafter made in writing specifically referring to this agreement and signed by the party to be bound and no modification or variance of the above undertaking shall be effected by the acknowledgment or acceptance of any sale document, purchase order, shipping instruction or other forms containing terms or conditions at variance herewith.

GENERAL ELECTRIC CO.

(Buyer)

BY: Walter A. Schottelbach

TITLE: Vice President and
Corporate Counsel

DATE: January 21, 1972

MONSANTO COMPANY

BY: [Signature]
[Signature]

0041819



SPQR

Service Performance Quality Reliability

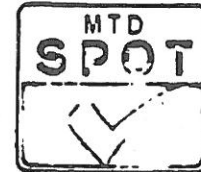


April 18, 1972

SUBJECT: SUPPLY PYRANOL

RECEIVED
AUG 22
EDWARD L DOBBINS

Information Letter - MT-166



To: Holders of SPOT ✓
File this under
Information Letter Tab of Section I

TO: FIELD SALES DISTRICT MANAGERS
FIELD SALES ENGINEERS
PLUS SELECTED PRODUCT DEPARTMENT SALES MANAGERS,
INTERNATIONAL SALES DIVISION, FASO, I&SE &
SERVICE SHOP PERSONNEL

Since early 1970, Monsanto Chemical Company, sole supplier of Pyranol, has advised that this material, under the generic term askarel, contains polychlorinated biphenyls (PCB). Polychlorinated biphenyls are highly stable compounds and are not readily biodegradeable. Therefore, when placed in the environment, they may be considered contaminants and may adversely affect some species of animal and marine life.

At that time all customers were advised through Sales Channels to take every precaution to prevent any entry of this material into the environment through spills, usage, leakage, disposal, vaporization or otherwise.

In the past we have supplied Pyranol to transformer repair shops, and to companies requiring these products for top-up of existing transformers. Pyranol was also available from Monsanto under GE Specification A13B3B without restriction regarding its usage.

These sales have been discontinued by Monsanto as of January 15, 1972, except to those who have entered into special agreement to indemnify Monsanto with respect to this product for use in transformers.

General Electric has agreed to indemnify Monsanto for this use, as have several other major manufacturers. Some manufacturers have chosen not to indemnify Monsanto and have decided to discontinue manufacturing askarel-filled transformers.

The Medium Transformer Products Department will be the only supplier of GE transformer Pyranol effective immediately. This is further defined as follows:

1. All orders for supplying Pyranol for all General Electric Company Pyranol transformers will be placed on the Medium Transformer Products Department, Rome, Georgia.
2. MTPD, Rome, may ship direct to customer or from Monsanto Chemical Company when circumstances warrant such shipment, but orders will always be placed on Rome.

PLAINTIFF'S
EXHIBIT
1100

736592

GENERAL ELECTRIC COMPANY
MEDIUM TRANSFORMER DEPARTMENT, ROME, GEORGIA

GENP 001842

3. Sales by Rome are only for use in askarel-filled transformers including repair and maintenance of any askarel-filled transformer, and for use in any new GE-manufactured transformer.
4. Bulk sales for askarel-filled transformers only will be made to the following customer classes:
 - a) Utilities.
 - b) Industrial and other customers for their own use.
 - c) Service shops for transformer use only.
5. Sales will not be made to:
 - a) External OEM's for their own use.
 - b) Distributors for resale.
 - c) Other external customers for resale, except for an independent Service Shop to fulfill a service contract on their customer's transformer.
6. All bulk orders to MTPD, Rome, from external customers must be acknowledged with the attached statement.

All Sales Departments must not only include this Indemnification Clause but state on the customer's purchase order and the GE requisition that the material is for transformer use only, before the order will be fulfilled by the Medium Transformer Products Department.

Other fluids for transformer use are presently under study which exhibit a higher degree of environmental compatibility. Studies indicate that some of these are feasible for use in transformers. However, until these materials have been proven suitable for transformer use, the above policy will remain in effect.

In the meantime, Handbook Section 5713, pages 1, 2, and 3 have been withdrawn and are being reviewed by the Medium Transformer Products Department, Rome, Georgia.

Please refer all requests for quotations on supplying Pyranol to:

H. J. Pinson
Specialist - Product Service
Medium Transformer Products Department
Rome, Georgia 30161



R. W. FRAHM
MANAGER-MARKETING

:hs
Attach.

736593

GENP 001843

Add to terms and conditions of sale on acknowledgment form which will be sent to the Purchaser in each instance, the following:

This material is sold on the understanding that it is for use in transformers only.

Pyranol[®] contains polychlorinated byphenyls (PCB's) which tend to persist in the environment and, therefore, care is required in its handling, possession, use and disposition. Accordingly, Buyer agrees that it shall defend, indemnify and hold harmless Seller, its directors, officers, employees and agents from and against any and all liability or expense whatsoever arising out of, or in connection with, the possession, handling, use, sale or disposition of such Pyranol[®] purchased by Buyer on this order which relates in any way to contamination of, or adverse effect on, any part of the environment including but not limited to humans, all other animal life, plant life or food by reason of such Pyranol[®].

736594

GENP 001844

DISTRIBUTION:

<u>List</u>	<u>Tabs</u>
1.09	3A, 3B
1.10	1C, 2C, 2E, 3D
1.11B	1A, 1C, 1E, 2D, 2E, 3B, 3E, 4B, 5E
1.12A	2B, 2C, 6A, 7A
1.14	3E, 4A
1.18	1B, 2A, 2C, 2D
1.20	1B, 1D, 9D, 2D, 4B, 3A, 3C, 5B, 5C, 6E, 8D, 8E
1.21	1B, 1C, 2E, 5A, 5C, 6E, 7A
1.22A	8B, 10B, 10C, 1A, 2A, 4A, 5A, 7B, 10E
1.23	1B
1.24	1A
1.25	1A, 1D, 1E, 2A, 2C, 2D
1.28	5A, 5B, 5C, 5D, 6A, 6B, 7A, 7B, 8B, 8D, 9B, 9C

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R. W. Frahm - Rome
R. B. Landwerlen - Rome (100 copies)
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R. A. Branflick - Hickory
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H. A. Brenner - Plainville
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736595

GENP 001845

BCC: J. MASON - JMASO
H. S. BERGEN
E. P. WHEELER - EWHEE
J. D. EARLY - WASHINGTON
W. S. CLARK - WCLAR

February 10, 1971

Mr. Lowell E. Miller, Director
Pesticide Regulation Division
Environmental Protection Agency
South Agricultural Building
Washington, D.C. 20250

Dear Mr. Miller:

I recently noted in United States Department of Agriculture PR Notice 70-25 dated October 29, 1970 that polychlorinated biphenyls must be eliminated from economic poisons. We at Monsanto understand and are in complete agreement with this action as indicated in correspondence Dr. R. E. Kelly, Monsanto's Medical Director, had with Dr. Harry W. Hayes and Dr. C. Cueto, Jr. when the Pesticide Regulation Division was part of the U.S. Department of Agriculture.

I am deeply concerned, however, to note that this elimination has been extended to include the polychlorinated terphenyls. We have thoroughly searched all available literature and communicated with many laboratories, industrial, academic and regulatory, and been unable to find evidence that would indicate the polychlorinated terphenyls are contaminating the environment or that they are responsible for adverse effects on fish or wildlife.

We realize the importance of preventing the contamination of the environment but seriously question whether the polychlorinated terphenyls can be properly classified as contaminants. Does your division have available any information or data which would justify the elimination of these materials from economic poisons?

Sincerely,

W. B. Papageorge
Manager
Environmental Control

/lsc

NEV 012520



711772

BCC: J. MASON - JMASON
H. S. BERGEN
E. P. WHEELER - ETHEE
J. D. EARLY - WASHINGTON
W. S. CLARK - WCLAR

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Sincerely,

W. B. Papageorge
Manager
Environmental Control

/lso



NEV 012520

711772

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CANCER INDEX

Page 1

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
X 4th Quart 1949		Everett - Powerhouse	Reticulum cell sarcoma of the neck.		D-2-10-50
X 4th Quart 1949		New York - Sales	Larynx		D-03-01-58
X 1st Quart 1949		Birmingham - Sales Mgr.	Thyroid		D-June 1950
X 2nd Quart 1949		Gen. Mgr.	Lymphosarcoma of mediastinum & parotid		D-1st Quart 1951
X 3rd Quart 1950		St. Louis - Org. Chem. Div.	Squamous cell carcinoma of esophagus. Explor. operation-July, 1950		D-4th quart 1950
X 1st Quart 1950		St. Louis - Sales	Kidney		
X 3rd Quart 1950		Dayton, Unit I - Guard			
X 3rd Quart 1950		Dayton, Unit V -Accountant	Basal cell carcinoma, rl. post-auricular area		D-03-19-55
X 1947		Queeny Plt -Truck Driver	Multiple squamous cell epitheliomata, face & neck		D-12-28-50
X 2nd Quart 1950		Queeny Plt -Tractor Driver	Adenocarcinoma of sub-maxillary salivary gland		D-9-8-82
X 2nd Quart 1950		Queeny Plt -Operator Phthalic Anhydride	Squamous cell carcinoma of larynx & pharynx in bronchus		D-1st quart 1951 D-3-7-51
X Fall 1947		Queeny Plt -Operator	Bronchogenic carcinoma (with metastases)		D-4-16-48
X 2nd Quart 1950		Queeny Plt -Sulfonamide Dept			
X Fall 1948		Queeny Plt - Supervisory	Rt. temporal astrocytoma		D-06-12-51
X 3rd Quart 1950		Queeny Plt -Chemical Eng. <i>man with the</i>	Ewings Sarcoma - left ilium		
X 2nd Quart 1950		Queeny Plt -Clerk	Myoblastoma -ri. arm.		D-04-04-71
X 3rd Quart 1950		Everett - Mason	Adenocarcinoma of the stomach.		D-06-21-63
X 4th Quart 1950		Everett -Locker Department	Prostate with osteoblastic & nodal metastases		D-12/14/50
X 1950		Queeny Plt-Operator	Lower lip		D-08-21-51
X 3rd Quart 1950		Everett -Locomotive	Carcinoma of lung with brain metastasis.		DEX-P-0013
X 3rd-Quart 1950		Everett -Mason and Yard Sweeper	Obstruction due to carcinoma of rectum		Page 32 of 32 D-12/26/50

CANCER INDEX
Page 2

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
4th Quart 1950		Carondelet	Skin		D-03-21-77
1949		Queeny Plt - Electrician	Basal cell carcinoma of the face just in front of ear.	50	Excised 2/51 with apparent cure
2nd Quart 1951		Everett	Carcinoma of head of pancreas with metastasis	54	D-4/3/51
3rd Quart 1951		Queeny Plt - Mechanic	Bladder. Treated & presumably cured.	43	P-9/23/68
1949		Krummrich Plt	Carcinoma of Colon		D-9/29/51
1949		Everett	Malignant gastric ulcer	39	09-26-57 D-10/21/57
		Springfield - Saflex	Face		10-16-77 D- 10/21/57
		Springfield - Styrene	Face, arm		D-02-25-62
		Springfield - Yard Acetate, Nitrate	Face		
		Springfield - Nitrate	Leukemia		D-07-21-49
		Springfield - Acetates, Nitrates, Resins, Office	Leukemia, Colon		D-06-21-75
		Springfield - Steel wire mill	Face, probable		
		Springfield - elsewhere	Peritoneal metastases, ovaries(?)		D
		Springfield - Payroll	Face		D-11-24-66
		Springfield - Acetate, Nitrate	Tumor of breast (non-malignant)		D-04-14-69
		Springfield	Papillary adenocarcinoma of bladder (recurrent)		
1st Quart 1950		Springfield - Secretary	Carcinoma of prostate	57	D-1/1/52
1st Quart 1950		Mound Lab - Lab Director	Gen. carcinomatosis		EXP-0013
1st Quart 1950		Everett	Carc. of Pancreas & Fall bladder	60	Page 21/6432
1st Quart 1950		Anniston	Main cause of death hepatitis with uremia. Also had cancer of colon & diabetes.	64	D-5/27/52

For Dr. Blaney's memo, 10/24/50, these people had neoplasms identified or known "during the past 3 yrs."

CANCER INDEX

Page 3

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
1952		Queeny Plt - Worker B-5 3/28/51 to 2/13/52 & 3/3/52 to 4/25/52	Prostate	47	D-6-22/52
1952		Shawinigan	Tumor of bowel		D-09-26-54
1952		Nitro	Bladder		D-7-23-85
1952		Springfield	Rectum	67	D-3/31/54
1952		Nitro	Throat (esophagus)	61	D-11/22/53
1952		Dayton (Mound)	Throat - midposition		D-3-21-82
1952		Shawinigan	Colon		D-07-26-54
1952		Main Office	Leukemia	18	D-11/6/52
1952		Springfield	Oat cell carc. of lung involve of pericardium stomach & mediastinum	67	D-10/19/52
March, 1952		Nitro	Bladder		D-08-22-66
1952		Everett	Kidney		D-09-03-76
1952		Nitro	Lungs	62	D-12/15/52
--		Camden	Bladder	72	D-1/5/53
--		Everett	Renal Cell		D-09-03-76
2/6/53		Krummrich Plant	Prostate	61	D-12-28-64
1953		Springfield	Prostate. Terminal stage of pelvic & bone metastases as of 1/27/53	64	D-09-25-53
1953		Queeny	Bile duct, Cirrosis of liver	60	D-5/22/53
1953		Springfield	Right ll. of lung.	65	EX-0013 D-5/20/53 page 30 of 32
1953		Krummrich	Bronchogenic Carcinoma	45	D-7/15/52

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CANCER INDEX

Page 4

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
1953		Queeney Plant	Rectum, metastatic Hydranephrosis	44	9/12/53-D
1953		Everett	(Brain) Glioblastoma multiforme	59	9/27/53-D
10/28/53		Anniston	Lung	48	D-07-03-66
10/29/53		Queeney	Rectum		
1953		Everett	Left lung, Epidermoid Carcinoma	60	10/22/53-D
3/54		Akron - Shipping Clerk	Lung. Lobectomy performed / Employed 33yrs 9/54		D-03-26-57
5/54		Tennessee	Lympho Carcinoma	52	D-5/16/54
8/54		Main Office - Invoice Clerk	Rectum		D-08-31-54
---		Queeney Plt - Salaried	Lung	44	D-8/12/54
9/54		Soda Springs, Idaho	Right kidney		D-11-14-54
12/54		Everett	Stomach	60	D-11/27/54
---		Queeney - wage employe	Lung		D-3/14/55
---		Krummrich Plant	Lung bronchogenic		D-6/27/55
---		Shawinigan	Malignant polyps of colon		D-7/16/55
---		Springfield	Kidney	57	D-7/14/55
---		Krummrich Plant	Stomach	59	D-10/17/55
---		Everett	Rectum	53	D-3/21/55
---		Employed 25 years Night Supervisor - Everett	(metastasis) Lip malignancy	47	EX-P-0013
---		Everett Operator in Everett-Bisulfite Dept.	Adeno carcinoma of rectum	56	Page 26 of 32

0665817

CANCER INDEX

Page 5

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
---		Employed 16 years Everett - Op. in DOP Dept.	Adenocarcinoma of the colon.	54	D-9-17-55
---		Everett	Liver	47	D-11-02-56
Feb. 1957		--	Recticulum Sarcoma Mediastinum	55	D-02-11-57
Feb. 1957		Krummrich - Hourly worker (May be subject to Workmens Nitro Compensation)	Metastasis carcinoma Liver, pancreas & spleen	50	D-02-27-57
---		Addyston - Lead man	Lung	64	P-03/13/57 10-8-57 D-02-11-57
1957		Monsanto, Tenn.	Lung	51	P-11-16-57
March 1958		Mound Laboratory	Malign. of prostate gland	58	D-09-04-62
April 1958		Krummrich Plant	Bronc. cancer carci- noma	52	D-07-04-58
April 1958		Queeney Plt	Bladder (?)		D-04-13-73
April 1958		Queeney Plt	Left lung	49	D-03-30-58
April 1958		Everett Plant	Larynx	66	D-03-17-66
5/19/58		Everett Plant <i>RR on Mont</i> Monsanto, Tennessee	Liver	53	D-12-08-58
5/19/58		Trenton - wage employe	Left lung	58	D-10-19-58
1952		Nitro	Prostate	61	D-8/31/52
5/26/58		Springfield - wage employe	Left lung; exploratory thoracotomy, 3/27/58.	54	D-11-15-57
6/6/58		Everett Plt	Cecum, metastasis		EX P-0013 Page 2 of 528
6/16/58		Springfield, hourly	Carcinoma of pancreas	46	

0665816

CANCER INDEX

Page 6

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
6/24/58		Springfield - salaried	Carcinoma reg. sigmoid	45	D-05-01-61
6/27/58		Texas City - wage employe	Chin	52	D-02-26-70
8/58		Krummrich Plant	Tongue		D-09-25-58
8/58		Krummrich Plant	Bronchogenic sarcoma	52	D-8/28/58
8/58		Barton	Lip		D-08-06-70
9/23/58		Trenton	Lung	59	
9/23/58		Trenton	Lung	38	D-9/13/58
9/30/58		Springfield - wage employe	Pancreas	46	
9/30/58		(Mobay) New Martinsville -salaried	Cecum		D-02-20-63
10/3/58		Queeny Plant - hourly	Pancreas	64	D-11-27-58
10/10/58		(Dept. Mechanic) Queeny Plant - wage employe	Right colon	57	D-9/6/61
10/10/58		Texas City - hourly	Right eye	52	D-02-26-70
10/17/58		Lion Oil - El Dorado wage	Pancreas	54	D-10-29-58
10/58		Shawinigan - Springfield	Left mass racial 1010	46	
11/58		Seattle	Stomach	53	D-11-11-58
12/11/58		Trenton	Bladder	58	D-07-27-65
2/13/59		(Chief Operator) Queeny Plant - hourly	Bladder	54	D-2/25/64
3/6/59		Mon., Tenn. - hourly	Carcinoma of descend- ing colon	57	EX P-0013 Page 27 of 32
3/11/59		St. Louis Office-salaried	Lung	63	D-05-02-61

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
11/12/59		El Dorado, Ark.-Wage	Generalized in lymphatic system of the body	50	D-9/26/59
11/13/59		(Cafeteria - part-time) Queeny Plant-St. L.-Wage	Adenocarcinoma of uterus	45	L - D 9-1-60
11/20/59		Krummrich-Hourly	Carcinoma of Lung	57	D-11/16/59
11/20/59		(Helper-Tech.Melamine) Everett - Hourly	Carcinoma of Bladder	52	D-9-29-60 D-D 9/60
12/8/59		Everett - Salaried	Metaestatic Carcinoma	42	D-02-11-60
12/18/59		Everett - Hourly	Carcinoma of Colon	62	D-03-30-60
12/23/59		Krummrich - Hourly	Carcinoma of Bladder	48	L
12/23/59		Mound Lab - Hourly	Carcinoma of Cervix	40	L
1/22/60		(Mechanic) - Hourly Queeny Plant - Hourly	Carcinoma of Bladder	44	L
1/22/60		(Chemical Operator) Krummrich - Hourly	Basal cell carcinoma of left lateral canthus	34	L
3/11/60		(Chem. Operator) El Dorado - Hourly	Carcinoma of Lung	50	D-01-07-61
3/29/60		(Helper & Laborer) Krummrich-Hourly	Carcinoma of Stomach	53	D-03-06-61
4/15/60		El Dorado Ref.-Hourly (Welder-1st class)	Carcinoma of Pancreas	59	D-07-28-60
6/17/60		(Mechanic) - Hourly Queeny Plant - Hourly	Carcinoma of Colon	55	D-12-17-61
6/24/60		(Chief Operator) Texas City - Hourly	Cancer of the Lung	53	D-02-09-62
7/11/60		Springfield (Office)	Cancer of the breast	43	D-02-21-61
7/18/60		(Foreman in Mfgg.) Monsanto, Tenn.-Salaried	Carcinoma, left vocal cord	57	D-01-31-74
7/18/60		(Chemical Operator) Shawinigan Resins-Wage	Carcinoma of the thyroid	45	EXB-09135-63
7/22/60		(Shift Engr.-Power Dept.) Texas City-Wage	Epidermoid carcinoma of left lung	44	Page 25 of 32 D-01-25-61

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Date Reported	Name	Location & Job	Cancer of:	Age	LIVING DECEASED
3/11/59		St. Louis Office (Sal.)	Stomach	62	D=12/8/59
3/20/59		Spfld. (hourly) Chem. Op.	Metastatic Ca of Hip and pelvis, secondary to Ca. of Hip.	64	D-07-20-59
3/20/59		Texas City (hourly)	Ca. of the Scalp	37	L
6/8/59		Spfld. (hourly)	Basal Cell Ca of Rt. Lower Eyelid	56	L
6/19/59		Queeny (hourly)	Ca. of Lung	54	D-6-22/59
6/26/59		Mound Lab (Sal.)	Endometrial Ca of cervical invading the entire cervical canal	40	D-06-30-60
			Probably the lower uterine segment and anterior lip of cervix		
7/31/59		New York Ofc. (Sal.)	Ca., Rt. Breast	27	L
8/20/59		Dayton (Sal.)	Rt. Lung	63	D-12-18-59
8/21/59		El Dorado Chem. (hourly)	Lower lip	52	L
8/31/59		Anniston (Hourly)	Rectosigmoid	58	D-07-07-63
9/17/59		Krummrich (Hourly)	Metastasis	49	D-9/9/59
10/16/59		Spfld. (Hourly)	Lung	55	10-24-59 D-11-10/59
10/28/59		Krummrich (Hourly)	Lung	60	10-07-59 D-10/59

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CANCER INDEX

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Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
7/29/60		Queeney (Pipefitter)	Urinary Bladder	51	D-07-31-61
8/26/60		Texas C. (Wage) (Operator)	Larynx	62	D-10-07-61
8/26/60		Texas C.-Wage (Operator)	Possible Ca Lungs	61	D-04-26-61
9/28/60		Spfld.-Salaried (Office)	Brain Tumor	30	D-08-17-60
10/13/60		T.C. (Machinist)	Metastatic Adenocarcinoma	31	D-09-17/60
11/11/60		El Dorado - Sal. (Office)	Cervix	38	L
12/5/60		El Dorado - Sal. (Office)	Fallopian Tube	59	D-11-28-60
12/9/60		Queeney - Wage (Chem. Op.)	Bladder	45	D-01-21-70
1/3/61		Shawinigan, Spfld. - Wage	Basal Cell Cancer	37	L
5/8/61		Texas City (Painter-Leadman)	Squamous Cell Carcinoma of left Mandible	54	D-02-26-70
5/23/61		Spfld.-Wage (Production)	Bronchogenic Ca.	59	D-06-05-61
10/24/61		Spfld.-Wage (Production)	Carcinoma of colon	63	D-09-12-62
2/23/62		Krummrich - Wage (Chem. Op.) (Dept. Mechanic)	Multiple myeloma	54	D-02-28-64
3/23/62		Queeney - Wage (PAB)	Carcinoma in situ Bladder	51	D-1-14-83
4/27/62		(Raw Materials Helper) Columbia, Tenn.	Prostate	56	D-07-19-63
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CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
5/13/62		Springfield - W (Production)	Head of pancreas	64	D -10-16-63
9/29/62		Queeny Plant (Chief Op.)	Carcinoma of Right Lung & mastitis		D-9/20/62
9/29/62		Queeny Plant(Dept.Mechanic)	Bronchogenic Cancer		D-8/9/62
10/2/62		Springfield (Production)	Metastatic Carcinoma	56	D-11-23-62
7/15/63		Texas City(Instrument man)	Carcinomatosis	63	08-01-62 D-1962
"		Sharinigan (Clerk-Typist) (Office)	Cancer of Lungs	35	09-01-62 D-1962
"		General Offices - St. Louis	Malignant meanoma of back	57	03-17-62 D-1962
"		Krummrich (Maint.Foreman)	Cancer of Lung	61	04-03-62 D-1962
"		Nitro (Maintenance Man)	Cancer of Lung	53	07-25-62 D-1962
"		General Offices (Office) (Night Superintendent) Texas City	Generalized cancer	45	06-17-62 D-1962
"		General Offices (Sec'y.)	Cancer of Liver	50	06-19-62 D-1962
"		Barton - Sr. Operator (Material Handler) New Martinsville	(Cancer of Liver, Colon, Ovaries,Skull Cirrhosis of Liver	42	06-10-62 D-1962
"		Greeny (Chief Clerk-Purch.) Texas City	Cancer of Pancreas	43	01-07-62 D-1962
"		Carondclet (Utility Man - Production)	Cancer of Lung	50	06-25-62 D-1962
"			Cirrhosis of liver	61	D-1962
"			Cancer of bladder with metastasis to spine	61	04-28-63 D-1963
"		(Plant Guard) New Martinsville	Cancer of Brain	45	04-28-63 D-1963
"				54	EX P-0013 Page 22 of 22 D-1963

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Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
7/15/63	[REDACTED]	Texas City (Operator)	Brain	55	D-12-18-62
7/15/63	[REDACTED]	Queeny (Guard-Prev.Chem.Op.)	Lung	62	D-03-20-63
7/19/63	[REDACTED]	Monsanto, Tenn.	Prostate w/generalized bony metastasis	57	D-07-19-63
9/30/63	[REDACTED]	Mound Lab.-Fireman, Driver	Carcinomatosis Primary unknown at present.	60	D-02-17-64
11/25/63	[REDACTED]	Krummrich (Pipefitter)	Ca. Pancreas	58	D-11/24/63
12/20/63	[REDACTED]	Krummrich (Blacksmith)	Metastatic Ca. of Cervical Gland and Pleural effusion	60	D-01-12-64
12/20/63	[REDACTED]	Texas City (Pipefitter/ Leadman)	Rectum	56	D-12/4/63
1/31/64	[REDACTED]	Springfield (Production)	Rectum	63	D-1/23/64
2/5/64	[REDACTED]	G.O. (Admin)	Brain	42	D-1/20/64
2/5/66	[REDACTED]	Spfld- wage (Production (Chief Maintenance Man) Santa Clara-wage	Lungs	50	D-1/22/64
2/10/64	[REDACTED]		Leiomyosarcoma of the Jejunum with metastasis	59	D-2/1/64
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CANCER INDEX

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Date Reported	Name	Location & Job	Cancer of:	Age	Living Dates
2/18/64		Queeney Plant (Porter)	Prostate	60	D-02-24-67
2/26/64		Spfld., hourly (Production)	Lymphosarcoma	36	D-02-18-65
4/1/64		Nitro-Wage (Gate Attendant)	Abdomen	42	D-3/15/64
4/7/64		El Dorado (Office Worker) Salaried	Abdominal Cavity	56	D-2/11/64
4/9/64		Queeney Plant (Chem. Op.)	Bladder	52	L
5/12/64		Queeney (Packer)	Prostate	56	D-01-29-70
4/9/64		Springfield (Production)	Prostate	64	L
6/5/64		Everett (Op.-Tech.Melamine)	Esophagus]	63	D-05-31-64
7/10/64		Queeney (Chem. Op.)	Lung]	56	D-3/24/65
7/14/64		Everett	Bronchiogenic	60	D-12-04-64
7/17/64		Queeney (Chief Operator)	Pancreas	60	D-10-04-64
8/12/64		(Storeroom Attendant) Krummrich Plant	Rectum	62	D-08-29-71
8/17/64		Krummrich-hourly(Chem.Op.)	Lung	51	D-10-05-64
8/17/64		Lion Oil Distributor	Abdomen	63	D-09-27-64
11/6/64		Spfld.(Materials Clerk)	Bladder	62	D-03-21-73
11/18/64		Spfld.-hourly(stock Handler)	Liver	42	D-11-20-64
11/24/64		(Stock Handler) Spfld.-Shawinigan	Colon Cecum	50	D-7/23/67
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					Page 24 of 32

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CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
12/3/64		(Reduction Op. H-acid) Everett - hourly	Tongue	57	D-11/20/64
12/3/64		(Patent Atty.) St. Louis - G.O.	Right renal cell w/metastasis	43	D-10/1/64
12/22/64		(Chemical Laborer) Krummrich Plant	Lung	49	D-12/16/64
1/13/65		Nitro (Janitor)	Larynx	64	D-01-28-78
1/15/65		(Office Worker) El Dorado - hourly	Lung	59	D-12/31/64
1/22/65		(Rigger) Krummrich - hourly	Widespread cancer- primary site unknown	60	D-1/10/65
2/10/65		Queeney - wage (Chem. Op.)	Pancreas with metastasis to liver and lymph nodes.	54	D-3/31/65
2/17/65		(Office) Springfield - salary	Generalized carcinoma- metastasis due to cancer of breast.	49	D-1/26/65
3/25/65		(Millwright) Queeney Plant - hourly	Stomach	54	D-5/2/65
3/29/65		(Powerhouse Ash Handler) Krummrich Plant -	Lung	52	D-06-27-65
4/29/65		(Finisher) Stonington - hourly	Stomach	55	L
5/3/65		Everett (Head Op.-bisul- fite)	Nose	62	L
"		Queeney - hourly-warehouse- man	Rectum metastasis to liver	54	M. D-8/2/65
"		Krummrich (Chem. Operator)	Larynx	56	L
"		Queeney (Chem. Operator)	Bronchogenic-left lobe	58	(D-9/12/65) EX-P-0013
5/14/65		Everett (l/c Painter)	Lung	45	Page 20 of 32

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Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
5/18/65		MAC Station	right colon	47	D-12/27/65
6/2/65		Krummrich (Chem. Operator)	bronchogenic	56	D-8/10/66
6/9/65		Queeny (Pipefitter)	Hodgkins Discase	43	D-08-2676
			rt. anterior chest wall lymph nodes rt. axilla and left groin.		
6/21/65		Columbia, Tenn. (Elect.)	Lymphosarcoma	40	D-04-08-72
6/23/65		G.O. (Salaried - Admin.) Janitor	Lung	49	D-4/30/65
6/23/65		Port Plastics (wage)	Bronchiogenic with metastasis to brain	60	D-5/26/65
7/28/65		Krummrich (wage) [Painter]	Bronchiogenic w/ cerebral metastasis	58	D-7/10/65
8/17/65		Queeny (salaried) Planner Project Maintenance Dept.	Abdomen	47	D-7/23/65
9/3/65		Queeny Plant (wage) Chemical Operator	Bronchiogenic adeno-carcinoma - rt. upper lung.	56	D-12-22-85
9/28/65		(Finisher) Deep River-Hourly	Breast w/metastases to bones, skin, liver	44	D-9/13/65
9/21/65		Nitro (Pipefitter)	Squamous cell carcinoma - Recurrence	52	L EXP-0013
10/19/65		Krummrich (iron worker helper)	Sarcoma of bone and sclerosis of liver.	56	Page 19 of 32 D-8/26/65

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CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
X 12/15/66		Columbia (hourly) Mechanic	Metastatic ca of abdomen. Primary site unknown.	51	L D-12/22/66
X 12/21/66		Deep River (wage) Mach. Op. B	Malignant lesion 8th vertebra (Widespread carcinomatosis)	49	D-1/13/67
X 12/22/66		Eldorado (hourly) Engineer- (Station pipeline sec.)	Liver (ca widespread)	61	D-2/1/67
X 12/23/66		Deep River (hourly) Maint. Mech. C)	Rectum	58	D-05-29-68
X 1/5/67		Mac Station Supervisor (Machinist)	Anoxia due to infiltration of lung by	39	D-12/5/66
X 1/9/67		Eldorado Chem. Plnt.	Reticulum cell sarcoma	50	D-06-11-67
X 1/19/67		Krummrich (Chemical Operator)	Lungs	57	D-12/13/66
X 2/9/67		Alvin (sal) Analyt. Lab Asst.	Malignant Lymphoma	28	D-1/28/67
X 3/13/67		(Mill Roll Operator) Springfield (hourly)	Looks like Hodgkins)		
X 3/16/67		Columbia ("A" Elect.)	Lung	50	D-2/20/67
X 3/17/67		Texas City (hourly) Operator	Lymphosarcoma w/blood stream involvement	42	D-04-08-72
X 3/17/67		Baxley (Filtered Resins)	Facial skin ca.	56	D-08-30-72
X 3/31/67		Pensacola (sal)	Lung-left upper lobe	60	D-07-21-65-77
X 4/13/67		Greenwood, S. C. (hourly)	Brain tumor, metast.	49	D-2/25/67
X 4/27/67		Krummrich (Day Helper)	(Yarn process operator) Reticulum cell sarcoma	42	D-3/13/67
X 4/27/67		Sharonville (Finisher)	Skin	55	LEX P-0013
			Breast (left)	29	Page 16 of 32 L 5/24/70

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
6/9/66		(Garment Room Supervisor) Krummrich-Sal. Pipefitter	Lung with metastasis	56	D-8/30/66
9/2/66		Queeny Plant (hourly) (Salt Cake Operator)	Stomach	51	L D-4/14/68
9/2/66		Krummrich Plant (hourly)	Lung	54	D-04-29-68
9/6/66		Electrician Columbia (hourly)	Lymphosarcoma	41	D-04-08-72
9/26/66		Queeny (Research Porter)	Metastatic lesion Occipital Lobe	57	D-12-22-85
9/26/66		Gehring Welder	Metastatic ca primary site unknown	36	D-11/27/66
9/30/66		Queeny (hourly)	Brain	45	D-9/11/66
9/30/66		Mound (sal.) Maint. Foreman	Astrocytoma rt. frontal lobe	53	D-9/9/66
10/5/66		Nitro (Production)	Ca bladder	63	D-8/22/66
10/10/66		Gering (hourly) (Boiler Fireman)	Ca bladder	56	D-12-27-75
10/10/66		Springfield (hourly)	Metastatic malignancy	45	D-11/20/66
10/24/66		Springfield (hourly) Engr. (Supervisor-Res. Services) Shawinigan (Spfld) Sal.	Lung	55	D-9/24/66
10/24/66		Pensacola (Sal) (Projects & Mech. Engr.)	Brain	59	D-10/5/66
10/24/66		Fabric Services (Sal.)	Standards Lung and Rectum Metastatic	38	D-10/9/66
10/24/66		Trenton (hourly) Millwright I/C	Ca. primary site un- determined. Pancreas	57	D-9/15/66
12/5/66				43	D-11/26/66
12/5/66					EX P-0013 Page 17 of 32
12/8/66		Columbia (Traffic Switchman)	Prostate	53	D-03-17-75

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Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
4/28/67		Krummrich (Boilermaker)	Anus	57	D-09-22-67
4/28/67		Sharonville (Janitor-Prod.)	Tongue w/metastasis left cervical chain	48	D-02-19-68
5/15/67		Krummrich (Physician)	Abdominal	37	D-3/7/67
5/18/67		Stonington	Prostate (metastatic)	49	D-01-24-74
5/31/67		Springfield (Case maker- Shipping Dept)	Liver	60	L D 11/23/67
6/7/67		Spfld. (General Laborer)	Rt. Testicle	26	L R 8/4/68
6/22/67		Queeney Plant (Chemical Oper.)	Lung	52	D-09-11-68
6/26/67		(Sr. Operator El Dorado Nitric Acid plant)	Nose	50	L
6/30/67		Trenton (Chemical Worker)	Lung	51	D-8/12/67
7/19/67		Pensacola (1st class mechan.)	Metastatic Malignant	29	D 7/9/67
8/8/67		Decatur (sal.) (Sec'y.)	Melanoma Metastatic ca ovaries	51	D-7/17/67
9/1/67		Spfld. (tubes & peritoneum Metastatic ca.	47	D-8-28/67
10/9/67		Krummrich (Office Worker)	Lymphosarcoma	46	D8/31/67
10/16/67		Spfld. (hourly) Press Opr.	Lung	51	D9/24/67
10/16/67		(Loader) ElDorado Refinery (hourly)	Pancreas	62	D 10/1/67
10/19/67		Queeney (Dept. 4 Operator)	Colon	51	L
11/2/67		Queeney (Millwright)	Bladder	48	D-04-13-73
11/9/67		Queeney (Leadman-whse.)	Bladder	62	FD P-0013 Page 15 of 52
11/20/67		Texas City (Sal.Y	Lungs (Maint.Foreman)	52	D-11/2/67

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Date Reported	Name	Location & Job (operator-pelleting plant) El Dorado Plant	Cancer of:	Age	Living Deceased
11/28/67		Neck		53 L	
12/13/67		Krummrich (Bricklayer)	Bladder	55 L	
1/8/68		Bridgeview (Sal.) (Plant Mgr) (Operator)	Pancreas	49	D-10/18/67
1/8/68		Texas City (hourly)	Lymphoma testis	35	D-12/6/67
2/5/68		Queeney	Abdomen-(generalized)	52	L-----
2/5/68		Decatur (Spinning Operator)	Alvcolar Cell Rhabdo-	23	D-1/11/68
2/8/68		Ligonier	myosarcoma Uterine	30	L
2/15/68		Carondelet (hourly) (Plant Guard)	Lung	61	L 2-20-68
4/4/68		Queeney (Electrician)	Left vocal cord	48	L
4/11/68		Ligonier (Factory Inspector)	Bladder	52	L 7-20-78
4/29/68		Nétro (hourly) Santoflex (Operator)	Bronchocarcinoma rt.	54	D-4/15/68
5/2/68		(Electrical Foreman) Pensacola (sal.)	upper lobe Stomach	68	D-4/17/68
5/17/68		(Truck Driver) Krummrich (hourly)	Malignant brain tumor	59	D-4/28/68
5/17/68		(Smiff Helper) Krummrich (hourly)	ca. of lung	62	D-5/7/68
5/24/68		Luling (hourly) Guard	Lungs	55	D-4/30/68
6/8/68		(Maintenance-Lubrication) Deep River (hourly)	Rectum w/metastases	59	D-5/29/68
7/10/68		Krummrich (hourly)	Basal cell Ca.	56	D 10-6-83
7/15/68		T.C. (hourly) (Painter/Lead man)	Left Lung	61	EXP-2013 L 7-20-70 Page 1 of 3

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CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	LIVING DECEASED
2/10/69		Anniston (sal) (Mtls Hndlg supvr)	Pancreas	60	D-1/16/69
2/10/69		Krummrich (Porter-Laborer)	Adenocarcinoma-rectum	54	L 4-5-69
2/19/69		Krummrich (Pipefitter)	Right lung	57	L 10-16-69
3/6/69		El Dorado (Chem. Operator)	Face & Skin	58	L
3/20/69		Delaware River (Chem. Oper.)	Brain	45	L 4-9-70
3/21/69		Nitro (hrly) (head operator)	Pulmonary Embolus	58	D-12/25/68
3/21/69		(Operator)	(due to op. ca of ur- inary bladder operation.		
3/21/69		El Dorado Refinery (hrly)	left lung w/met.	57	D 2/18/69
3/28/69		Kenilworth (sal)(sales corr)	Cirrhosis of liver	48	D-9/11/68
3/28/69		G.O. (enggrg.)	Hypernephroma, met. to peritoneum, liver & tongue.	44	D-3/9/69
3/28/69		Texas City (hrly)(boiler- maker)	rt. lung	63	D-12/8/68
4/8/69		Queeney (Chemical Operator)	Lung	48	D-3/19/69
4/8/69		Columbia (slag foreman)	Lung	59	D-3/14/69
4/8/69		Texas City (operator)	Brain Tumor	49	D-2/14/69
4/15/69		Columbia (Electrician)	Lymphosarcoma with blood stream involvement	44	L 4-8-72
4/28/69		Nitro (hrly)(Gatehouse attendant)	Fight Lung	53	DEX P-9913 Page 12 of 32
4/30/69		Nitro (hrly) (Carpenter)	Right ear	59	L-

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Date Reported	Name	Location & Job	Cancer of:	Age	Lives Deceased
7/29/68		Trenton (hourly)	Gastric ca.	47	L 3-21-69
7/30/68		Texas City (hrly.) (Carpenter)	Papillary Ca. enlarged thyroid gland	49	L
8/20/68		Ligonier (Finisher)	Nose	52	L
8/22/68		Queeny (Cafeteria)	Ovaries	50	D-8-12-68
8/22/68		Springfield (Empl. Serv. Mgr)	Pulmonary Atelectasis	64	D 7/27/68
9/12/68		Textiles N.Y. (Sales Rep.)	Testes	44	D 8/17/68
9/26/68		Kenilworth (hourly) ^{Class A} Mechanic	Lung	54	L 6-11-69
10/18/68		El Dorado Refinery	Anaplastic ca. (truck transport)	35	D-9/23/68
10/21/68		El Dorado Plant (Lead operator)	Mouth	61	D-6-20-80
11/14/68		Queeny (Chem. Operator)	Lung	47	L 3-19-69
11/21/68		Krummrich (hourly) (Tractor Driver)	Lung	58	D-10/23/68
11/22/68		Spfld. (Sal.) (Matl. Handling Engr)	Esophagus	49	D-11/1/68
12/9/68		Krummrich (hrly) (Electrician)	Colon	61	D-11/12/68
1/15/69		G.O. Research	Stomach	49	D-12/15/68
1/22/69		Pensacola (sal) (Managerial)	Malignant Lymphoma	49	D-1/1/69
1/30/69		Springfield (sal) (Business)	Rectum	62	D-12/31/68
2/3/69		Nitro (hrly)	Bronchial ca.	53	L 4-22-69
2/5/69		New York (receptionist)	Brain Tumor -Ca. Colon	52	EX-F-999269 Page 13 of 32

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Date Reported	Name	Location & Job	Cancer of:	Age	Living/Deceased
5/5/69		Yardville (hrly) (finisher)	Colon	42	L
5/7/69		MAC	Lymphosarcoma cell leukemia	43	D-5/3/69
5/19/69		Queeny (hrly) (Chem. Oper.)	Kidney	47	L 2-27-70
6/30/69		Kenilworth (Packer Film Dept)	Multiple Myeloma	59	L
7/9/69		Kenilworth (hrly) (Sr. Mech.)	lung, w/metastasis to brain, liver, and left adrenal.	58	D-6/15/69
7/25/69		Nitro hrly. (Pipefitter)	lung	53	L 2-25-70
7/28/69		(Supt. Cost Greenwood, S. C. analysis)	Carcinomatosis of	55	D-6/13/69
7/29/69		Alvin (Chem. Operator)	Kidney Thyroid	33	
7/31/69		Queeny (Chem. Operator)	nose	62	D-12-21-85
9/2/69		Queeny	Bladder		D-8-17-69
9/10/69		Krummrich (Chem. Operator)	Bladder	61	L 4-20-81
9/22/69		Spfld. (hrly) (Electrician)	Larynx	48	L
9/25/69		Cering (hrly)	Multiple Myeloma	59	D-8/22/69
12/9/69		Akron, (secretary)	Ovarics	49	D-10/26/69
1/12/70		West Port (sal)	Ewings Sarcoma w/Metastases	29	D-11/27/69
1/27/70		Queeny (hrly) Garage Mech.	Bladder	49	DEX #00093
2/26/70		Mound (maintenance foreman)	Colon	56	Page 11 of 32 D-2/5/70

See if there are on mortality on file

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Deaths Exceeded
X 7/26/76	[REDACTED]	JFQ - rigger	ca. bladder	60	D 1-27-85
X 11/9/76	[REDACTED]	Soda Spgs. - Gen'l Acct. Super.	ca; Sigmoid	54	D-9/19/76
X 11/9/76	[REDACTED]	Anniston-Chem. Operator	ca; Bronchogenic	41	D-10/2/76
X 11/18/76	[REDACTED]	Ligonier-Maintenance Laborer	ca: lung	62	D-10/23/76
X 11/24/76	[REDACTED]	Luling - Mfg. Foreman I Ag.	cancer metastatic	56	D-10/22/76
X 11/30/76	[REDACTED]	Deep. River - office worker	generalized carcinoma	47	D-11/30/76
X 3/25/77	[REDACTED]	Westport-Mech./Tool Crib	ca. left ala of nose & left cheek	60	D 7-2-85
					EX P-0013 Page 1 of 32
					0665789

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
2/3/75		Ligonier (finisher)	Breast	42	L
3/21/75		Kearny (central control chemical operator)	Lung	62	D-3/2/75
3/25/75		FRP Co. (chemical operator)	bronchogenic		D
4/21/75		Spfgld. (plant service attendant)	to CA of colon gen. metastases/second	61	D-4/2/75
4/14/75		Alvln-Mech. Maintenance Tech.			L
9/22/75		Sand Mtn. - process tech.	undifferentiated mediastinum w compression lg. vessles	32	D-8/28/75
10/13/75		Trenton - Millright 1/C	carcinomatosis	52	D-9/14/75
12/5/75		Trenton - millright 2/C	ca. throat & tongue	53	L
1/26/76		Spfgld. - stenographer B	ca. rectum	54	D-1/8/76
1/26/76		Houston - secretary	ca: metastatic ovary	63	D-1/1/75
2/20/76		Bloomfield - Engr. Specialist	ca: stomach	54	D-2/2/76
2/3/76		Ligonier - finisher (factory)	carcinomatosis	54	D-1/15/76
4/14/76		G.O. - Gen. Engr. (elec. design)	carcinomatosis-adenaca. of pleura	61	D-3/16/76
3/22/76		Ligonier - Inspector	ca: bladder	60	L
3/25/76		Spfgld. - rigger crane opr. container clerk & wldspread	anaplastic carcinoma	56	D-2/24/76
3/31/76		WGK. - chemical operator	metastatic melanoma	45	D 3-2-84
4/5/75		Bircham Bend - pipefitter	ca: rt. lung met. to rt. scapula	47	L
6/11/76		Akron - Acct. Super. II	ca. Lung	50	Page 2 of 32
6/15/76		Soda Spgs.-mechanic	ca. sigmoid ext. to bowel wall & affecting colon	35	L

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
9/3/74		Columbia Traffic Operator	Prostate	60	L 3-17-75
9/5/74		Texas City (hrly) boiler-maker	Lung	64	D-7/13/74
9/5/74		G.O.	Met. melanoma brain	54	D-8/6/74
9/11/74		Yardville (hrly) Finisher	Met. bronchogenic ca.	55	D-8/28/74
9/19/74		Alvin Maintenance Tech.	Respiratory failure due to met. ca of lung	44	D-9/5/74
9/20/74		Spfld. Still Operator	ca. bile ducts	41	L 12-14-74
9/20/74		G.O.	ca. bladder	56	D-8/30/74
10/22/74		Columbia Tapper Helper	bronchogenic ca.	49	D-9/28/74
10/22/74		Queeny Chem. Opr.	ca. colon w/met.	61	D-9/22/74
11/15/74		Sharonville (finisher)	breast	44	L
11/27/74		Columbia Nodulizing Lead-man	left lung, met. to/pleura	49	L 5-7-75
12/11/74		Shawinigan	sigmoid colon met to/liver	63	D-11/29/74
12/23/74		Decatur Sr. Textile Tech.	stomach w/estens. Met.	60	D-12/2/74
12/23/74		Pensacola Spinnerette Tech.	Met. ca. primary unkn.	43	D-12/18/74
1/7/75		<i>Spfld. L. Boiler</i>	infiltrating adenocarcinoma colon w/o met.	59	D-3-25-79
1/22/75		Springfield hrly	Stomach & liver	59	D-12/29/75
" "		Krummrich hrly (carpenter)	Met. adenocarc. brain	57	D-1/11/75
" "		Columbia -	Lung	46	EXP-0013
2/24/75		Queeny (hrly) millwright - for PAB program 1939-1975	Head. of pancreas w/met. ca. of liver	64	D-2/11/75

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
2/27/74		Yardville (hourly) Machine Chem. Op.	Metastatic Ca. Ca. testicles	22	D-2/2/74
"		Queeny (sal.) Prod. Foreman	Ca. Lung	56	D-2/4/74
"		Chicago (Secretary)	Carcinomatosis/breast, Pneumonia	59	D-5/20/73
"		Avon (hourly) Catalyst Plnt/ Op. B	Ca rectum	58	D-1/23/74
"		Yardville (Finisher)	Bronchogenic ca. w/met & nephritis	55	L 8-28-74
2/26/74		Indian Orchard Process Att.)	Mouth w/met. to neck	48	L 9-25-75
3/21/74		Queeny (Res. Porter)	Esophagal Ca.	60	L 3-21-74
"		Sharonville (Finisher)	Ca. in situ Cervix	40	L
"		St. Peters (Bench Processor)	Ca. in situ	37	L
4/8/74		Lima, Ohio (B Inspector)	Ovary	40	L
4/18/74		Queeny (Engine Operator)	Ca Prostate w/met.	60	D-4/1/74
5/20/74		Nitro (Serv. Bldg. Attend)	Trans. cell ca prostate	49	D-4/29/74
6/17/74		Texas City (Pumper-Gauger)	Bronchogenic carcinoma	61	L 10-4-74
7/1/74		New York (office)	Seminornia -metas.	41	D-5/29/74
7/1/74		Krummrich (Crane Hook-up)	Lung	62	D-6/9/74
7/1		Springfield (Blender Op.)	Ascending Colon	54	L
7/3		Queeny (Chief Operator)	Colon	57	L
8/5/74		Carondelet (Sec. H)	Met. ca to brain, liver, skeleton	46	EX P-0013 Page W/28324
8/12		Columbia (Assistant Supv 60-73 of 61-73)	Ca. Lung w/met.	45	D-7/6/74

0665792

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
4/15/73		Queeny (48-72 Bricklayer (72-73 Shop Clerk (never actually worked for Leonard Construction Co.	Bronchogenic ca.	47	D=3/19/73
5/2/73		Anniston	Distal mid-sigmoid Colon	59	L 10-19-73
6/6/73		Columbia ("B" mechanic)	Met. malignant melano- ma of brain	56	L 8-27-74
7/2/73		Texas City (Pumper Guager)	Met. ca. of rectum	61	D-6/20/73
7/9/73		Trenton (Pipefitter)	Trans. cell ca of bladder.	53	L 12-21-73
7/27/73		Krummrich (hrly) (Leadman) (Injection Molding)	Ca Liver	59	D-6/16/73
8/24/73		Kenilworth (Mach. Opr.)	Breast	47	L
9/5/73		Texas City (Chief Oper.)	Left Lung	58	L 7-6-77
10/1/73		Spfld. (stock cont.-ResiminesProstate)	Ca. Lung (death due to acute myocardial infarct.)	64	D-12/24/73
1/22/74		G.O.			
1/22/74		Trenton (hourly) (Pipefitter)	Ca. Bladder	53	D-12/21/73
1/29 /74		Krummrich (Porter)	Ca. sigmoid colon	59	L 4-22-85
1/29/74		Nitro (sal.) (Utilities Foremga. lung		62	D-1/17/74
1/29/74		Bircham Bend (Mist. (Mist. Opr.)	Reticulum cell ca. left femoral.	29	L
2/27/74		Pensacola Hourly (Mist. Opr.)	Pulmonary ca, wide-spread metastasis to brain bone.	52	EX-106/34

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
3/10/72		Texas City (Chem. Op.)	Lip (lower)	57	L
3/17/72		Pensacola (Electrical Mech.)	Carcinomatoses)Bronchogenic ca.	55	D-3/1/72
3/23/72		Addyston (Office)	Metastatic ca. Brain	50	D-1/13/72
4/20/72		Stonington (Finisher)	Prob. Metastatic Disease, liver. Old ca of breast.	39	L D-6/2/72
5/24/72		Queeny (Mechanic)	Adenocarcinoma lung	63	D-5/13/72
8/23/72		Nitro (Auto Mechanic)	Rectal ca.	49	L 12-8-72
10/3/72		Texas City (Operator)	Metastatic cerebral hepatic carcinoma	62	D-8/30/72
10/30/72		Texas City (Painter)	Adenocarcinoma of prostate metastatic to bone	50	L 1-25-76
11/13/72		El Dorado (Machinist)	left upper lobe-lung	63	L
11/13/72		Ligonier	Abdominal ca	57	D-10/30/72
1/2/73		Greenwood (Plant Security Guard)	Ca of liver-metastatic	61	D-12/2/72
2/73		Springfield (hrly) (Mason)	Ca of lung	56	D-1/8/73
2/73		Bircham Bend (Chem. Opr.)	Ca left lung with metastasis to bones and liver	59	D-1/11/73
2/26/73		Krummrich	Ca of gallbladder	48	D-2/13/73 Page 6 of 32
2/28/73		Krummrich (Yard Laborer)	Ca rt. lung	59	L/25/73

0665794

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living/Deceased
9/9/71		Queeny (Shop Clerk)	Lung	63	D 9-6-71
9/9/71		Queeny (Cafeteria attend.) (Safety & Sec. Inspector El Dorado Plant	Breast	43	L
9/9/71			Prostate gland	62	D-8/1/71
9/20/71		Alvin (I&E Technician)	Basal cell ca rt. cheek	35	L
9/23/71		Bircham Bend (Welder)	Adenocarcinoma	44	L
10/18/71		Krummrich (sal) Lab Analyst	Malignant lymphoma, mixed cell type	47	D-9/28/71
10/21/71		Ligonier (hrly) Finisher	Uterus	53	D-10/20/71
11/1/71		G.O. Off. Mach. Maint.	Metastatic ca. of kidney	51	D-9/17/71
11/23/71		Decatur (hrly)Janitor	Carcinomatosis	61	D-11/5/71
11/23/71		Queeny ?	Prostate	57	L
12/20/71		G.O.	Pancreas (adenocarcinoma)	59	D-11/21/71
1/4/72		Spfld. Lift truck repairman	lower 1/3 esophagus	58	D-10-4-72
1/5/72		Queeny (hrly) Chem. Opr.	urinary bladder (met)	56	D-12/7/71
1/5/72		Ligonier (Maint. Foreman)	Stomach	53	D-12/15/71
1/7/72		Mound (hrly) (Welder)	Carcinomatous from metastatic ca of colon	54	D-11/3/71
1/10/72		Nitro (Yard Laborer)	basal cell ca face & Squamous cell ca of lip	54	D-5-28-72
2/4/72		Cehring (prostate,met. to bone	53	L
2/4/72		Ligonier (Office Worker)	Cervix w/metastasis	42	D-EX 2-0071b
3/1/72		Addyston (Chem. Op)	Colon	35	Page 7 of 32

CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
4/5/71		Krummrich (Machinist)	Lung	52	L 6-5-71
4/13/71		El Dorado	Metastatic malignant melanoma from lesion of back	35	L 10-8-71
5/6/71		ator/spinner Tire Yarn Dept Pensacola (hrly)	Ca of testis with met.	25	D-4/9/71
6/11/71		Texas City (Chief Oper.)	Bronchogenic Ca. w/ Cerebral metastasis	47	L 6-6-71
6/11/71		Krummrich (sal) Office	Bronchogenic	50	D-5/18/71
6/16/71		Krummrich (hrly) (Machinist)	Lung	52	D-6/5/71=
6/18/71		West Port (Shift Mech. C Crew)	Malignant Nevi	38	L
7/1/71		Texas City (Chief Opr.)	Brain tumor, secondary to lung ca.	48	D-6/6/71
7/6/71		Pensacola - Mechanic	Exanguinating hemorrhage due to bronchogenic ca.	57	D-6/12/71
7/20/71		G.O. (Cafeteria)	Metastatic Disease w/ bilateral ureteral obstruction	39	D-7/4/71
7/28/71		Columbia (Bulldozer Oper)	Massive abdominal tumor probably metastatic from old testicle ca	46	L 12-19-74
8/13/71		Ligonier (Inspector)	Transurethral ca	55	EX P-0013 Page 6 of 78

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CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
9/8/70		Everett (Autoclave Opr. Santocel Dept.)	Parkinson's Disease	55 L	9-17-78
9/8/70		Queeny (Whse. Clerk, Shop Laborer)	Rectum	59 L	3-11-78
10/23/70		G.O. (Sec'y)	Breast w/metastasis	54	D-9/18/70
10/15/70		Columbia (Furnace Tapper)	Rt. Upper Lobe	53 L	7-5-72
10/19/70		St. Peters	(Bench Processor Breast Clean & Insp.)	33 D	10/8/70
11/25/70		Texas City (Chief Operator)	Liver	62 L	12-13-70
12/7/70		Queeny (Dept. Mechanic)	Bladder	55 L	
12/10/70		Trenton (Maint. Mechanic)	Hodgkins	33 L	7-9-72
1/26/71		Anniston (Millwright)	Bronchogenic Ca.	59	D-1/5/71
2/5/71		Texas City (Painter)	Respiratory paralysis, secondary to spinal cord metastatic ca. due to ca of rt adrenal	54	D-12/30/70
2/5/71		El Dorado (Field Gauger)	Adenocarcinoma of Pancreas	64	D-1/29/71
2/8/71		Queeny (Coppersmith & Tinner)	Lung	57	5 D 12-10-85
3/10/71		G.O.	Carcinomatosis-primary bronchiogenic ca.	63	D-2/23/71
3/10/71		Everett (Carpenter l/c)	Larvnx	42	L
3/12/71		Caronddet (Chem. Oper.)	Ca. left ear & neck	43 L	EX P-0013
4/1/71		Springfield (Nurse)	Ca. of Ovary	47	Page 9 of 32 D-3/17/71

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CANCER INDEX

Date Reported	Name	Location & Job	Cancer of:	Age	Living Deceased
2/26/70	[REDACTED]	Spfld. (hrly) Pkg. Operator	Sarcoma of lung	50	D-2/12/70
3/2/70	[REDACTED]	Baxley	Lung	61	L 7-21-77
3/5/70	[REDACTED]	Queeny (Chem. Operator)	Bronchiogenic ca.	54	L 11-6-70
3/12/70	[REDACTED]	W. Caldwell (Wiring & Soldering Opr.)	Breast	34	L
3/30/70	[REDACTED]	Spfd. (sal) (Res. Tech.)	Colon	61	D-3/11/70
4/27/70	[REDACTED]	Everett (Sal) (Mfg. Specialist)	Metastatic seminoma to liver and heart	59	D-4/4/70
4/27/70	[REDACTED]	G.O. (sal)	Brain Tumor-lung ca.	53	D-3/31/70
4/30/70	[REDACTED]	Ligonier (Chief Polyflex Inspector)	Cervix	46	L
5/4/70	[REDACTED]	Kenilworth (Benchworker)	Cervix	46	L
6/4/70	[REDACTED]	Trenton (Drum Dryer Operator)	Medullary ca of thyroid	40	L
6/25/70	[REDACTED]	Sharonville (hrly) Finisher	Breast-metastatic	32	D-5/24/70
7/10/70	[REDACTED]	Kenilworth (Benchworker)	Endometrial ca.	41	L
7/13/70	[REDACTED]	Bircham Bend (Shift Supvr.)	Bronchiogenic Ca.	55	D-5/31/70
7/20/70	[REDACTED]	ED	Lymphosarcoma	50	D 7/3/70
7/22/70	[REDACTED]	Ansacola (sal) Maint. Supv. Intermediates and Power	Lymphosarcoma	49	D-7/17/70
8/14/70	[REDACTED]	El Dorado Plant (Chem. Oper.)	Left Kidney	58	L 6-29-72
8/25/70	[REDACTED]	(Mach. Oper.) Sharonville (Blow Molding)	Tetratoma left test.	28	D EX P-0013 8-25-70
9/8/70	[REDACTED]	Queeny hourly	Liver	56	D 8/14/70

EXHIBIT B



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A sign by Woods Pond warning of PCB-contaminated fish. Photo courtesy of the EPA.

IN FOCUS

THE OTHER SIDE: Sue Monsanto, save Lee — a modest proposal in three parts (Part One)

While recent events may preclude legal action against GE, that is not the case when it comes to pursuing claims against the Monsanto Company, the ultimate responsible party for the widespread PCB contamination in the Commonwealth.

BY **MICKEY FRIEDMAN**
POSTED ON **DECEMBER 10, 2023**



I am not an attorney, but for more than 40 years, I have been writing, filming, and organizing about General Electric's (GE's) misuse of polychlorinated biphenyls (PCBs) and how it has affected GE workers, contaminated homes in Pittsfield, a children's playground, a local schoolyard, and, of course, the Housatonic River.

I have learned over these years how we have all been injured, and how our public health and our environment has suffered. All of us in Berkshire County who have lost the use of our Housatonic River, who have breathed in Monsanto's Aroclors, who will have to endure thousands of trucks filled with PCB-contaminated material rumbling through our streets, and especially the citizens of Lee who, for generations to come, will have to live next to a massive PCB dump have suffered an enormous public nuisance.

GE's significant economic, social, and political power, not only in Berkshire County but extending throughout the Commonwealth, coupled with a variety of political pressures on the United States Environmental Protection Agency (EPA), has resulted in a compromised cleanup which will leave large amounts of PCBs in the Housatonic River system and burden the citizens of Lee with the [Upland Disposal Facility \(UDF\)](#). This contamination is—and will be for generations to come—a continuing assault on the public health and the environment of Berkshire County.

I have recently written about all of this for The Berkshire Edge, in case you have time for some research: Read "[Whose Housatonic?](#)"; "[Housatonic Hypocrisy](#)"; and, most recently, "[Massachusetts' Massive PCB Mistake](#)."

Many believe that this imperfect settlement is a done deal. In fact, as part of a series of confidential negotiations, the Massachusetts towns of Lee, Lenox, Stockbridge, Great Barrington, and Sheffield entered into a settlement agreement under which those towns agreed not to appeal the terms of the 2022 CERCLA Order in exchange for a GE payment of \$62 million.

Nonetheless, other parties, including the Housatonic River Initiative (HRI) and HEAL of Connecticut, [unsuccessfully intervened](#) before the EPA's own Environmental Appeals Board (EAB), urging a more comprehensive cleanup and the use of innovative treatment technologies instead of landfilling. EAB, with minor modifications, including the request for a more comprehensive explanation for why EPA wouldn't award a TSCA exemption for a GE on-site landfill, [affirmed the EPA-revised permit](#). The EAB decision prompted confidential negotiations that resulted in the critical change from off-site disposal to mandate the Lee UDF. Meanwhile, two parties went to the United States Court of Appeals for the First Circuit, but the court [dismissed their appeal](#) on July 25, 2023.

Clearly, the ramifications for Lee are highly significant. Even though a majority of Lee residents were/are opposed, the agreement was indeed signed onto by Lee officials, who have since been repudiated by the voters and are no longer serving. Question 1 on the 2022 Lee Town Meeting Warrant read, "Shall the town require the Select Board to rescind the town of Lee's approval of the rest of River Agreement?" 665 residents voted in favor, while 390 opposed. The negotiated settlement precludes any action by local officials and, with the result of the Court of Appeals decision, any action by the courts to challenge this agreement.

I am hoping there might be a new way to address this dilemma and find the money needed to spare the innocent citizens of Lee the massive PCB dump that GE and EPA have foolishly sited near an aquifer in a geologically suspect location.

Sue Monsanto, and save Lee. It is not a coincidence that Lee is the poorest of the Housatonic River communities with the least robust tourist economy—and that the other towns opted to protect themselves and receive financial compensation for agreeing to the dump for Lee that they had previously vigilantly fought against for their own communities.

No lawyer here, but I am hoping that, if successful, this proposed legal action will address the critical public nuisance the UDF would impose and restore some well-deserved environmental justice. And the financial damages can simultaneously be used to pay for the transportation of PCB-contaminated waste out of state to a TSCA-approved landfill, while financing additional remediation of some of the remaining toxic soils and sediments from this and other PCB-contaminated Commonwealth waterways, like New Bedford Harbor. This will reduce threats to public health and the environment, better protect Commonwealth-owned lands that border the Housatonic River, and restore protection for the Housatonic ACEC.

It is important to remember that there were many compelling reasons, including the Commonwealth's repeated and strongly expressed preference in its official comments, for previous EPA decisions that mandated off-site disposal. Here are two persuasive selections from EPA's February 29, 2016 "[Statement of Position in Support of Intended Final Decision](#)":

i. EPA's selection of off-site disposal is supported by the nine permit criteria and the administrative record.

GE claims that EPA concedes that off-site disposal would be no more protective to human health and the environment than on-site disposal. GE SOP at 6. On the contrary, EPA does favor off-site disposal in terms of protectiveness. In addition, and even more significantly, GE treats cost and protectiveness as the sole criteria for decision-making, when they are only two of the nine Permit criteria that EPA evaluated. When viewed in that context, off-site disposal is clearly the best suited disposal option.

One of the Permit factors EPA considered in selecting the remedy is its implementability, including coordination with other agencies, regulatory and zoning restrictions, and availability of suitable facilities. Long-standing and active opposition to on-site disposal threatens the Rest of River remedy with lengthy litigation and community resistance. By proposing off-site disposal, EPA avoids these road-blocks, rendering the entire remedy more likely to be promptly implemented and in that respect more protective of human health and the environment. EPA acted in a manner consistent with the Decree in considering public and governmental objections to on-site disposal because these objections are relevant to the implementability criterion listed in the Permit. In addition, the Decree allows EPA to consider any relevant evidence in the administrative record, including the overwhelming number of public comments opposing on-site disposal. Moreover, the Decree offers multiple public participation opportunities, and these would be meaningless if EPA could not consider the views of the public in remedy selection.

Apart from implementability, EPA also considered the other relevant Permit criteria, including cost. For example, in evaluating long-term reliability and effectiveness, EPA evaluated the suitability of the proposed on-site landfill locations, considering the fact that GE did not establish that the proposed locations were suitable in light of soil permeability, hydrology, and proximity to potential drinking water sources and the Housatonic River. Similarly, EPA recognized that the Woods Pond and Forest Street locations would require the waiver of ARARs designed to protect an ACEC and/or wetlands habitat. EPA further considered the suitability of a pre-existing licensed off-site disposal location in comparison

EPA's February 29, 2016 "Statement of Position in Support of Intended Final Decision." Highlighting added.

The EPA then emphasized its belief that GE was less concerned with environmental science or the suitability of their proposed landfill location than with how much they would have to spend if they were forced to transport the contaminated soils and sediment to an off-site TSCA landfill.

EPA has used its scientific and technical expertise to thoroughly consider GE's technical positions at multiple points in the Rest of River decision-making process. Moreover, EPA has subjected its own analyses to further scrutiny, including review by experienced EPA scientists and engineers nationally, and independent scientific peer review. Furthermore, EPA has afforded GE and the public with an extraordinary degree of participation and input on the Rest of River cleanup decision. Based on that substantive expertise and multiple process opportunities, EPA proposed a remedy that is best suited for the Rest of River.

GE challenges EPA's Intended Final Decision for one reason – to reduce its costs in cleaning up its PCBs. GE attempts to justify its challenge with three main claims: (1) GE allegedly knows better than EPA how to select a remedy in the public interest; (2) GE is allegedly entitled to virtually total certainty and finality in the cleanup, with uncertainties and additional costs all to be borne by the public; (3) EPA allegedly misinterpreted the Decree in requiring restoration of natural resources; and (4) EPA inappropriately applies ARARs. None of these claims are

EPA's February 29, 2016 "Statement of Position in Support of Intended Final Decision." Highlighting added.

In 2016, as well as resounding public support for the Housatonic ACEC, there was large-scale public disapproval of GE's plans to construct another PCB landfill in Berkshire County. GE was previously allowed in 2000 to dump PCB contamination from its cleanup of the first two miles of the Housatonic to two GE landfills, one lined and one unlined, across from the Allendale Elementary School in Pittsfield.

Not surprisingly, as EPA pointed out in 2016 with this latest compromise mandating the Lee UDF, GE will save close to \$200 million in transportation costs. With that one decision, GE accomplished a major task and EPA and the Commonwealth failed to adequately protect the citizens of Lee. GE, one of the most profitable corporations in the world, could easily afford the added transportation costs. After all, the citizens of Lee, along with the citizens of Berkshire County, never profited from the multi-billion dollar manufacture, sales, or use of PCBs, and have only paid a sometimes lethal price for the incompetence and irresponsibility of GE and Monsanto, who together allowed their toxic chemicals to contaminate one of New England's most beautiful rivers.

So, while recent events may preclude legal action against GE, that is not the case when it comes to pursuing claims against the Monsanto Company, the ultimate responsible party for the widespread PCB contamination in the Commonwealth. It is time to see whether the courts will determine that this was criminal negligence.

Money—we discover once again—was always the strongest motivating factor. As internal documents reveal, even as the Monsanto Company—and General Electric and Westinghouse, two of the largest consistent users of Monsanto's PCBs—became aware that the product was toxic, the profit involved in selling PCB-dependent electrical equipment overwhelmed any responsible concern for workers, end-users, local communities, the nation, even the world.

A short bit of history: GE began to manufacture electrical capacitors and transformers at its Pittsfield plant beginning in 1903. PCBs are a group of distinct chemical compounds, none of which occur naturally. While first synthesized in 1881, they were produced commercially by Swann Chemical Company beginning in 1929 in its plant in Anniston, Ala. Monsanto, in its 1969 "[PCB Environmental Abatement Plan](#)," notes, "Monsanto entered the Aroclor market in 1930 by acquiring Swann Electrical Company or known today as our Anniston, Alabama plant. Our first load of Aroclor which incidentally was Aroclor 1254 went out of Anniston, Alabama to GE in 1931."

Monsanto produced PCBs at plants in Sauget, Ill. and Anniston, Ala. until 1978. PCBs were used in capacitors, transformers, hydraulic fluids, lubricants, carbonless copy paper, inks, pesticide extenders, sealants, flame retardants, and building materials like caulk. Monsanto used the trademark "Aroclor" for its PCBs, while GE used the trade name "Pyranol." Monsanto was the sole producer of PCBs in the United States. [See 116 Cong. Record 11695, 91st Congress, (April 14, 1970) ("Insofar as the Monsanto Co., the sole manufacturer of PCB's is concerned ...").]

Both GE and Westinghouse used massive amounts of Monsanto's PCBs as a flame retardant dielectric fluid in transformers and capacitors. It has been estimated that, between 1929 and 1977, about 1.1 billion pounds of PCBs were produced in the United States. And Monsanto's domestic production reached a peak volume of 86 million pounds in 1970. 17.9 percent of Monsanto's sales were of Aroclor 1254. A large amount of Monsanto's Aroclors went into the production of plasticizers.

The EPA details the commercial products that may contain PCBs:

- Transformers and capacitors
- Electrical equipment including voltage regulators, switches, re-closers, bushings, and electromagnets
- Oil used in motors and hydraulic systems
- Old electrical devices or appliances containing PCB capacitors
- Fluorescent light ballasts
- Cable insulation
- Thermal insulation material including fiberglass, felt, foam and cork
- Adhesives and tapes
- Oil-based paint
- Caulking
- Plastics
- Carbonless copy paper
- Floor finish

The Pittsfield Transformer Manufacturing Division of GE manufactured large and medium-sized AC and DC power transformers. Pyranol was used by GE from 1932 until 1977, when they stopped due to the EPA's proposed regulations banning the manufacture of PCBs.

For several decades, GE controlled our understanding of the parameters of our PCB problem, asserting that there was a total of anywhere from 22,000 to 39,400 pounds of PCB that had migrated into the entire Housatonic River system. It was my interviews in "Good Things To Life: GE, PCBs and Our Town" with Edward Bates, the manager of tests at GE Power Transformer, and his assistant, Charles Fessenden, supervisor of calculations, and former Pittsfield Mayor Remo DelGallo that provided a far more accurate portrait of the truly massive extent of the PCB contamination.

From my August 15, 1997 interview with former Pittsfield Mayor Remo DelGallo:

Unfortunately for the City of Pittsfield in many ways the General Electric Company property is located practically in the center of town. And they had many, many drains in the General Electric Company - and they dumped a voluminous amount of oil in those drains. Nobody even knew where those drains went. Some leached into Silver Lake. A good number of them went into the Housatonic River ...

And when I say disposed we're talking about thousands and thousands and thousands of gallons of oil. We're not talking about crank case oil, 150 gallons, we're talking about thousands and thousands of gallons of oil, including that oil tank that they had up on top of what is known as Peck's Bridge, 550,000 gallons of oil. That's how much — it held that capacity and it leaked for years and years and years before 1964 and after 1964. (Emphasis added.)

From my September 12, 1990 interview with Ed Bates:

[P]eople don't realize that Pyranol is twice as heavy as water. You put a gallon of Pyranol in water and it sinks right to the bottom. Within that twelve and a half pounds of Pyranol weighs, seven pounds of every gallon is PCBs. We used to use an average of 20,000 gallons of Pyranol a week. And this is, if you did simple mathematics, this is 140,000 pounds of Pyranol, of PCBs a week that we were handling. And we had a loss rate, spillage, over-filling of about three percent so this says that every week, we would lose between 4,000 and 5,000 pounds of PCBs that would go down the drain and into the river. ... In the meantime about a million and a half pounds of PCBs have been plowed into that river. I imagine a good 30 percent is left. (Emphasis added.)

It wasn't just the liquid Pyranol that was escaping from the factory. Ed Bates explained that there were continuing spills of the oil when filling or putting the transformers through their tests:

Well, Fuller's Earth — it was a substance like sawdust, that if you had a spill of oil or Pyranol, you'd throw it on and take it and shovel it in drums. Then the drums would be unloaded, but I don't know where they were unloaded. Uh it turns out that some of them probably were unloaded up at the Rose Property in Lanesboro from the sound of it, some were unloaded uh where the Allendale School is. Others were unloaded in the big pond in back of Plastics over in Building 59 over there ... every day as part of their cleanup the fellows would put this stuff on the floor and then sweep it up and throw it in the drums and every day they'd be thrown out ... to me there must have been four or five hundred pounds a day thrown out. And if you take that over a period of forty years or thirty years when we were using Pyranol you're talking uh maybe 2,000 pounds a week or a hundred thousand pounds a year or three million pounds over 30 years. It's just mind-boggling if you go into that too ... (Emphasis added.)

This contaminated fill was trucked throughout the county, to the city landfill, and given away to GE employees to use to fill in their front and backyards. MassDEP investigated hundreds of suspected fill properties in the Lakewood area, and 175 residential properties had to be cleaned up and restored.

To get some idea about the scale of GE's miscalculation, here is [an accounting by EPA](#) of the amount of PCB-contaminated soil and sediment remediated so far:

Cleanup is complete at all 20 contaminated areas outside the River ... Approximately 186,000 cubic yards (cy) of soil and sediment have been removed from these 20 cleanup areas ... Cleanup is complete for the Upper ½-Mile Reach. 18,700 cy of contaminated material was removed from river sediment and bank soils ... Cleanup is complete for the 1.5 Mile Reach. 91,700 cy of contaminated material was removed from river sediment and bank soils.

That is a total of 296,400 cubic yards.

As for the anticipated total removal of contaminated soils and sediments from the Rest of River, the [EPA estimates](#):

GE will excavate PCB contamination from 45 acres of floodplain and 300 acres of river sediment, resulting in removal of over one-million cubic yards of PCB-contaminated material. Most of the sediment and floodplain cleanup will happen within the first 11 miles of the Rest of River in the City of Pittsfield and the towns of Lee and Lenox.

When asked if he knew if the PCBs he was working with were dangerous, Ed Bates answered:

No ... Uh the U.S. government ... uh, neither of us blame the GE for that. Any fluid or anything that you used in those days, you'd run a two-week test on it and if you didn't break out in welts or your eyes didn't water too bad, they would use it. And uh it turns out that Charlie and I feel, from our observation, that the latency period of Pyranol, PCB Pyranol is around fifteen to twenty years. You really got to be exposed to it, and you did get itching out of it and you got sore eyes out of it ... and this we would heat up to 55 degrees C or around 120 degrees when we were running tests on transformers. And the odor of it was unbelievable. It'd choke you but, you know, there was nothing published ... It wasn't until 1969 that it became noted, and it was noted by the GE in a document, that it was a real problem ... but it wasn't until 1975 and 76 that they started to use silicon and phase out Pyranol.

Reginald Meunier began working for GE in 1946:

They never forewarned us of any dangers of handling any liquids there ... And you know they never gave us anything to protect ourselves which was ridiculous. They never told anyone there was any dangers with any of the oil we used or anything ... If you would have seen some of these rashes on my legs that I developed from having the oil drop on my legs you wouldn't believe it. Even developed on my back — my wife would keep saying where did you get all these terrible rashes and sores on your back ...

But they never told us wear any special clothing, which they should have done. When we complained that the oil was dripping all over our clothes, they got us some rubber aprons which was great for the front but it would run down and oil would seep into our shoes and at the end of the day you'd walk around and hear this squooshing going on ... But as far as being forewarned or forearmed, never. They never divulged to us what we were working with. Never told us the hazards. Never put any signs around. The only signs that we saw were hazards against electrical shock or — that was it ...

The Environmental Working Group (EWG) has [aggregated important internal Monsanto documents](#). Monsanto claimed again and again that, until the 1960s, the company didn't know PCBs were harmful to human health or even that they persisted in the environment: "And the truth is that in 1966 when we found out that PCBs were in the environment, we started an investigation journey and we tried to gather information and we acted responsibly." (Trial Transcript, Owens v. Monsanto CV-96-J-440-E, (N.D. Alabama April 4, 2001), pg. 454, line 6.)

In fact, Monsanto and GE knew from the very beginning that the PCBs they were manufacturing and using were toxic. [The Washington Post reveals](#) that after purchasing the Swann Anniston plant, "the company learned that PCBs, in the double negative of one company memo, 'cannot be considered non-toxic.'"

By 1936, there were already reports of serious health effects amongst workers who were exposed to these chemicals. United States Public Health Service Senior Surgeon Dr. Louis Schwartz wrote [an article in the American Journal of Public Health](#), in which he discussed treating not only workers who had been contaminated but a wife and child who had developed chloracne, a severe skin rash, from contact with work clothes. Dr. Schwartz wrote:

In addition to these skin lesions, symptoms of systemic poisoning have occurred among workers inhaling these fumes. Those working with the chloro diphenyls have complained of digestive disturbances, burning of the eyes, impotence and hematuria. The latter symptom developed among a number of men making amino diphenyl, which is used in the manufacture of a rubber antioxidant. Cases of death from yellow atrophy of the liver have been reported among workers exposed to the fumes of the chloro naphthalenes.

Louis Schwartz, "Dermatitis from Synthetic Resins and Waxes," June 1936. Highlighting added.

Schwartz urged that a series of preventative measures be taken, including isolating the areas the chemicals were used in, or if that wasn't possible, utilizing suction exhaust systems to vent the fumes. Floors and walls needed to be washed often, and he proposed lockers for all the workers so that they could change out of their street clothes and into work clothes, then reverse the process with available showers. He wrote: "It has been estimated at one point that 6 cents a day per worker will take care of furnishing clean clothes each day." Most importantly, he wrote:

There should be periodic medical examination of workers to detect cases of dermatitis and workers in chlorinated naphthalenes and diphenyls should be periodically examined for symptoms of systemic poisoning. Laws should be passed making it compulsory for factories where there are skin hazards to adopt these measures. (pp. 591-592) (Emphasis added.)

And in a second 1936 article, "Skin Hazards in American Industry Part II," Schwartz reiterated: "Workers in chlorinated naphthalenes and diphenyls should be periodically examined for symptoms of systematic poisoning." (p. 10) (Emphasis added.)

Dr. Cecil Drinker of Harvard University had been hired by the Halowax Corporation to investigate mounting health problems among its workers exposed to chlorinated naphthalenes and diphenyl. Halowax used these chemicals to coat electric wire. GE began using Halowax's products. In September 1937, Dr. Drinker, MF Warren, and GA Bennett, published an article entitled "The Problem of Possible Systemic Effects from Certain Chlorinated Hydrocarbons." The authors wrote:

For years it has been known that many of these compounds cause a troublesome acne, and there is a large literature upon this phase of the subject. Our investigations have not been concerned with chloracne but with the possibility of systemic effects following ingestion or inhalation of such products. In the spring of 1936, the Halowax Corporation, a division of the Bakelite Corporation, called our attention to three fatal cases of jaundice in workmen using chlorinated naphthalenes and chlorinated diphenyl, and requested that the subject be investigated as rapidly and thoroughly as possible. (Emphasis added.)

Drinker's article quoted extensively from a conference held by the Harvard School of Public Health attended by representatives from Halowax, Monsanto, GE, and the U.S. Public Health Service. Dr. Drinker wrote: "the chlorinated diphenyl is certainly capable of doing harm in very low concentrations and is probably the most dangerous ... **These experiments leave no doubt as to the possibility of systemic effects from the chlorinated naphthalenes and chlorinated diphenyls.**" (Emphasis added.)

According to Drinker, Mr. F.R. Kaimer, the assistant manager of GE's York, Penn. Wireworks factory recounted:

*It is only 1 1/2 years ago that we had in the neighborhood of 50 to 60 men afflicted with various degrees of this acne about which you all know. Eight or ten of them were very severely afflicted — horrible specimens as far as their skin conditions was concerned. **One man died and the diagnosis may have attributed his death to halowax vapors, but we are not sure of that ...** (Emphasis added.)*

R. Emmett Kelly, who represented Monsanto, described the results of their human experiments: "A more or less extensive series of skin eruptions which we were never able to attribute as to cause, whether it was impurity in the benzene we were using or to the chlorinated diphenyl."

A [Monsanto memo](#), dated October 13, 1937, by L.A. Watt acknowledges:

***Experimental work in animals shows that prolonged exposure to Aroclor vapors involved at high temperatures or by repeated ingestion will lead to systemic toxic effects. Repeated bodily contact with the liquid Aroclors may lead to an acne-form skin eruption. Suitable draft ventilation to control the vapors, as well as protect by suitable garments from extensive bodily contact with the liquid Aroclors, should protect any untoward effect.** (Emphasis added.)*

Workers told me that none of these protections were ever implemented by GE in Pittsfield.

Problems with chlorinated hydrocarbons were well known by users. A July 18, 1939 document marked "[Confidential, Not for Publication](#)" was issued by the National Paint Varnish and Lacquer Association to its members: "Chlorinated Hydrocarbons. Carbon tetrachloride, trichlor ethylene, tetrachlorethane, ethylene dichloride, and similar chlorinated aliphatic hydrocarbons **are said to be very dangerous toxics ... some chlorinated aromatics are frequently the source of skin troubles.**" (Emphasis added.) They reminded members of the Manufacturing Chemists' Association Legal Principles: "A manufacturer who puts out a dangerous article or substance without accompanying it with a warning as to its dangerous properties is ordinarily liable for any damage which results from such failure to warn." (Emphasis added.)

Monsanto's [October 1944 "Salesmen's Manual for Aroclor"](#) stated clearly: "All chlorinated hydrocarbons have measurable degrees of toxicity" and that Aroclor poisoning could result in atrophy of the liver ..." (Emphasis added.)

MONSANTO CHEMICAL COMPANY

10-1-44

2. **Acute yellow atrophy of the liver in which the liver cells show swelling, hypergranulation, hyaline inclusions and vacuolation as a result of extensive exposure over long periods of time.**

Monsanto's October 1, 1944 "Manual for Salesmen of Aroclor." Highlighting added.

A 1947 article in The Chemist Analyst by Robert M. Brown, chief of the industrial hygiene section of the Health Department of St. Louis, Mo., acknowledged the toxicity of the Aroclors and the dangers of coming in contact with the liquid, as well as breathing the vapors:

The Chemist Analyst, Vol. 36, No. 2, page 33,
J. T. Baker Chemical Co., Phillipsburg, N. J.
September 1947

ON THE TOXICITY OF THE "AROCHLORS" (sic)

Robert M. Brown, Chief
Industrial Hygiene Section, Division of Health
Dept. of Public Welfare, City of St. Louis, Mo.

A recently published article (Maglio, M. Martin, Chemist Analyst, 22 94 (1946)), has recommended the substitution of one of the "Arochlors" as the melting-point bath liquid in preference to the customary sulphuric acid. As stated in that article "Arochlors" are a group of chlorinated diphenyls produced by the Monsanto Chemical Company.

There is need therefore to give warning. For the toxicity of these compounds has been repeatedly demonstrated, both from the standpoints of their absorption from the inspired air, as well as from their effects in producing a serious and disfiguring dermatitis when allowed to remain in contact with the skin. Since these effects have been repeatedly observed, industrial hygienists have taken care to see that the proper controls have been established wherever these products are used. For example, the maximum allowable concentration of chlorinated diphenyl for an 8-hour working day is 1 milligram per cubic meter of air.

"On the Toxicity of the Aroclors," September 1947. Highlighting added.

The Environmental Working Group adds:

In response to a 1947 inquiry from one of its Aroclor customers about possible liver damage, Monsanto referred the customer to Dr. Drinker's published work, promoting it as the best information available on the subject: 'The best published information about the toxicity of Aroclor vapors with reference to possible damage to the liver is in a series of three articles written for the Journal of Industrial Hygiene and Toxicology ...'

EWG continues:

At the conclusion of this letter highlighting Drinker's findings and praising it as the most accurate available science on PCBs, Monsanto wrote to a customer that:

'Based on our practical experience in the manufacture and sale of millions of pounds of Aroclors annually, the point that we would emphasize is that workers should not be exposed to Aroclor vapors and that the men working with Aroclors should observe "good housekeeping" rules about keeping their clothing and skins free of the material and avoid ingestion of it.' [Monsanto to Celanese; December 30, 1947.]

A 1952 memo refers to Monsanto's and other manufacturers' agreement with United States Public Health System about labelling:

Since the wording of the label was formulated and put into use, skin patch tests run under competent medical supervision indicated that Aroclors are neither skin irritants nor skin sensitizers. This is indicated in our literature, but of course is not intended to mean that people should bathe in Aroclor. There are instances in the commercial use of Aroclor where people have immersed their arms up to the elbow, day in and day out, in the liquid Aroclors, and of course we do not approve of this, and to avoid such misuse of Aroclor, the wording on our label, to avoid repeated contact with the skin seems highly significant. Back in 1938 or thereabouts, when the Aroclor applications were relatively few and the customers about equally few, there was indeed the prize application of using Aroclor 1254 as a chewing gum plasticizer. The wording of our label would not be compatible with this sort of thing.

Since so many of the new Aroclors involve their use at elevated temperatures, the wording of the warning label to avoid inhalation of fumes is especially significant. We constantly keep this factor in mind in our development work, and emphasize it in direct contacts as well as indirect contacts such as our literature. While the toxicity hazard of Aroclor's fumes is well established and should be thoroughly understood by all, yet as we go along we find that we are always confronted with violations in one degree or another, and indeed, regard keeping in touch with these things to a major responsibility in the promotion of Aroclors.
(Emphasis added.)

A simple site visit to GE Power Transformer would have immediately revealed how grievously Monsanto was failing in its responsibilities to safely promote its Aroclors.

On September 1, 1953, Monsanto Medical Director Dr. Emmet Kelly responded to Mr. Lewis W. Spoiler, director of the Division of Industrial Health at the Indiana State Board of Health. Dr. Kelly balanced Monsanto's admission that Aroclors couldn't be classified as "non-toxic" with a denial that Aroclors had caused long-standing health problems:

From **MONSANTO CHEMICAL COMPANY**

At St. Louis

- 4 SEP 1953

Date September 1, 1953

To Mr. E. Mather

At Rusben

Reference Your memo to
ATB - 8/11/53

Subject AROCLORS: TOXICITY

cc Mr. T.K. Smith - 7
Mr. A.T. Beauregard - 7
Mr. P. O. Benignus - 7
Dr. J.W. Barrett - London
Dr. J.A. Gardner, - Fulmer
Mr. J.P. Stickleley - KXOK
Dr. N.B. Dyson - Newport

Mr. Beauregard has asked the Medical Department to comment on your letter referred to above.

As I am sure you know, Aroclors cannot be considered nontoxic. The interpretation of the toxic properties of a compound, however, determine whether or not there is any hazard associated with the specific use of a compound. To my knowledge, there is no hazard involved in the use of transformers containing Aroclors as a substitute for other materials. To my understanding, in the United States this application of Aroclors is widely accepted and has not resulted in any difficulty from a toxicological standpoint.

These denials were very often contradicted by further study. The Environmental Working Group published [this 1954 acknowledgment](#) that one of Monsanto's customers had recently discovered that seven of its workers had health problems after working with Aroclor:

Lesions of chloracne developed in seven workers employed in an organic acid manufacturing plant when Aroclor was used. An unusual feature of this outbreak of dermatitis was the long period of exposure before any cases were recognised. Sudden recognition of seven cases after 19 months was a result of the especially careful examination of the exposed employees after discovery of the first case. Of 14 exposed or potentially exposed, seven developed chloracne. The fact that air tests, even in the presence of vapors, showed only negligible amounts of chlorinated hydrocarbons indicates that this type of intermittent but fairly long continued mild exposure is not innocuous. (Emphasis added.)

An April 1955 document, "[Process for the Production of Aroclors, Pyranols, Etc. at the Anniston and at the WM. G. Krummrich Plant,](#)" notes:

There are many literature references to harmful effects of the type of 'chlor acne' resulting from exposure to chlorinated diphenyls, especially in cases where people working with small electrical components have been exposed to the fumes of hot, highly chlorinated Aroclors. Chloracne is sometimes accompanied by gastric troubles, and there are literature references to liver troubles.

From the start of Aroclor manufacture at the Krummrich plant the operators have been supplied a clean change of clothes every day, and time has been allowed at the end of the shift for bathing. Operators are advised to wash hands and face before eating. The Anniston operators do not have the same issue of clean clothes. At Anniston, no special protective clothing is provided for the Diphenyl and Aroclors operators. A daily change of clothing was provided in the past but this practice ceased before the war ... the men are expected to take a bath, in their own time, at the end of the shift.

In September 1955, [Dr. J. Emmet Kelly wrote Dr. J. W. Barret in London](#) to discuss "Aroclor Toxicity," suggesting that the company had already invested a lot of money in testing its Aroclors and urging that more tests weren't really necessary. But most important of all, Dr. Kelly acknowledged the toxicity of Aroclors and advised that, should these issues come to trial, a jury wouldn't particularly be interested in the details of "Maximum Allowable Concentrations":

I don't know how you would get any particular advantage in doing more work. What is it that you want to prove? I believe your work should be directed towards finding out what the concentrations are of Aroclor during different operations whether it is industrial or painting. The reports you have seen from Kettering Laboratory are the result of approximately \$15,000 to \$20,000 expenditure by MCC.

MCC's position can be summarized in this fashion. We know Aroclors are toxic but the actual limit has not been precisely defined. It does not make too much difference, it seems to me, because our main worry is what will happen if an individual develops any type of liver disease and gives a history of Aroclor exposure. I am sure the juries would not pay a great deal of attention to MACs.

As the Environmental Working Group explains, Monsanto's history with the U.S. Navy is instructive:

In 1956, the U.S. Navy considered using one of Monsanto's products which contained PCBs, called Pydraul 150, as a hydraulic fluid in Navy submarines. But after conducting their own toxicity tests, which showed that skin applications of Pydraul 150 killed all rabbits tested and that a statistical model on inhalation of Pydraul 150 indicated 'definite liver damage,' the Navy decided not to use Monsanto's product due to its potentially harmful effects. In Dr. Kelly's own words: 'No matter how we discussed the situation, it was impossible to change their thinking that Pydraul 150 is just too toxic for use in a submarine.'

The Environmental Working Group adds:

The Navy's decision to do its own toxicity tests, despite having been supplied Monsanto's tests, greatly bothered a member of Monsanto's Medical Department, Elmer Wheeler, who wrote on December 26, 1956 to a Monsanto colleague in Washington, D.C.: 'Out of all of this it appears quite certain that in the future we will not spend one nickel to develop toxicity data on hydraulic fluids for the Navy. We will continue to get information to satisfy ourselves that the use of our fluids is safe under any normal foreseeable conditions. This is generally enough to satisfy non-military customers. If the Navy has interest in any of these fluids and wishes to accept them toxicity wise on the information available, they are welcome to do so. If the fluids are not acceptable toxicity wise on the basis of such data, then perhaps we can save a lot of time and effort by advising the Navy to look elsewhere for their requirements.'

In spite of the tone of the above memo, Emmet [Dr. Kelly] and I wish you the happiest of New Years!' [Wheeler to Sido; December 26, 1956] ...

Despite all the company knew of the toxic effects of PCBs, Monsanto consistently failed to adequately disclose its full knowledge to its customers. Most customers didn't take the initiative to run their own tests as the Navy had, and therefore, remained in the dark about the potential danger of exposure to PCBs.

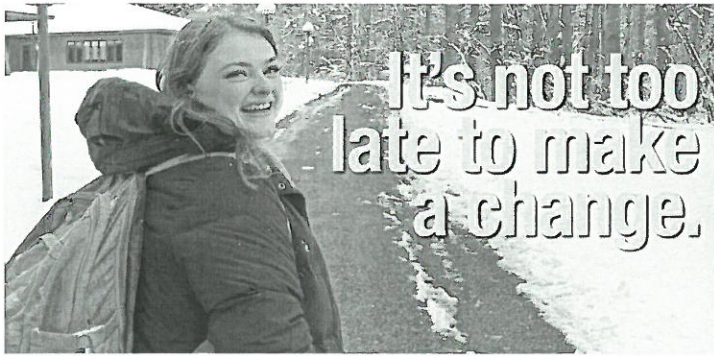
Despite these medical studies and Monsanto's acknowledgment that animal tests revealed "prolonged exposure to Aroclor vapors involved at high temperatures or by repeated ingestion will lead to systemic toxic effects," GE workers spoke in "Good Things To Life," of inadequate ventilation and constant contact with PCB-contaminated oil without protective gear. Those working with transformers and capacitors often put their arms in PCB oil up to their elbows to tighten bolts by hand. They explained that they couldn't do the job while wearing gloves. Crane operators talked about the clouds of vapor that extended to the extraordinarily high ceiling of Building 100. Other workers talked about the puddles of PCB oil that covered the floors, slowly eating through the rubber soles of their boots, and the coupons GE offered them for replacement boots. Everyone I interviewed worked in their street clothes and brought their oil-soaked shirts and pants home to be washed with the family's other clothes.

Monsanto assured the United States Public Health System in 1952 that it regarded inhalation of fumes as highly significant. Perhaps Monsanto failed to deliver the message because GE obviously never got the message or they disregarded it. And GE workers in Berkshire County and the rest of have paid the price.

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Smoke billows from GE's PCB-contaminated-oil-burning thermal oxidizer, which was affectionately known by workers as "Puff the Magic Dragon." Still courtesy of Mickey Friedman.

IN FOCUS

THE OTHER SIDE: Sue Monsanto, save Lee — a modest proposal (Part Two)

Thanks to our share of Aroclor, we in Berkshire County have been victims of GE and Monsanto's gross incompetence, remarkable negligence, and their unrelenting impulse to put profit before worker safety, and the public health and our environment.

BY **MICKEY FRIEDMAN**

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Despite the repeated concern of medical authorities with the vaporization and inhalation of PCBs—realities Monsanto and GE were well aware of—for 22 years, beginning in 1973, GE's thermal oxidizer continuously burned PCB-contaminated oil. Crane operator Al Bertelli told me they used to refer to the oxidizer as "Puff the Magic Dragon." Puff stood adjacent to the Newell Street/Lakewood residential neighborhood. And as Ron Desgroseilliers of GE's Area Environmental and Facility Operations admitted to Pittsfield residents, most of the toxic oil GE burned came from out of town.

On April 14, 1993, Desgroseilliers revealed that 20 percent of the PCB-contaminated oil burned in the thermal oxidizer came from GE Pittsfield, another 40 percent from other GE plants, and 40 percent from other companies across the United States. Plumes of smoke and vapor would infiltrate the entire surrounding area where many of the GE workers lived and many small businesses were located. As Ed Bates told me:

"They say that it's 99 percent—you know, 99.5 percent effective, which is fine, but we just got word, we got some figures in there from The Advocate, the North Adams paper, where last year they burned a million, 800,000 pounds of PCBs. Well if, uh, that's good, but if you're losing two tenths of one percent, it isn't much, but it adds up to about 3,600 pounds. Where does it go? Is it left in the ash? Does it go in the air?"

Joe Carr, who worked for 30 years at GE, told me:

"[Puff] burned a lot of materials which were hazardous, and it was supposed to be regulated by the Environmental Protection Agency and several other agencies, even our local agencies with GE. And they had rods in there which were called sensors. I mean that thing was a hundred some-odd feet tall, and there must have been two, three hundred rods in there that would measure what was coming out for smoke ... It tells exactly how hot you can burn that fire, and then I had a person I know from a government agency go in there and go up to the top and there was no rods in there. They were all burned out. They used to burn it so hot. And there were no rods to determine what the hell is all over Lakewood."

When it came to the human costs of the continuing use of Aroclor, one of the great frustrations experienced by Ed Bates was the great gap between the rhetoric of safety from GE and Monsanto and the reality of sickness. He felt responsible for the medical problems and premature deaths of those who worked under him at Power Transformer. And after much pressure, GE hired Dr. Wegman of Harvard to do an occupational health study. When Ed Bates and Charles Fessenden examined the data Wegman was working with, they found numerous instances where researchers misidentified where some GE workers had worked, and made errors with their death certificates and cause of death:

"Charles and I've been very close since we were in school, and it would seem to me that every other week Charlie would say, 'OK we got to go to a funeral ... a wake' ... And it wasn't just the Tests where we worked, the building itself where we used to mix the Pyranol, and deliver it out through a series, a system of pipes. There was a Shipping group. There was a Maintenance group. There was an Assembly group. There was a Test group. All these people were in the same building, and it seemed to me that we were—you know, it started to get on you. Charlie and I had reached the age of 40 and 50 and say, gee, why come all these people are dying, but we didn't do anything about it. Maybe we were wrong."

As Reginald Meunier put it:

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As for the rhetoric, in 1962, then-Director of the Monsanto Medical Department Dr. R. Emmet Kelly wrote Dr. Marcus Key at the Division of Occupational Health of the United States Public Health Service:

“As I told you on the telephone, our experience and the experience of our customers over a period of nearly 25 years, has been singularly free of difficulties. To our knowledge, there have been only three instances where chloracne has occurred. In view of the millions of pounds which have been produced and used in many and varied applications, the low frequency of any difficulties has been gratifying.” (*Emphasis added.*)

GE, the EPA, and the towns who allied themselves with the Rest of River Committee have crafted a compromise that “will excavate PCB contamination from 45 acres of floodplain and 300 acres of river sediment, resulting in removal of over one-million cubic yards of PCB-contaminated material.” But the crucial question that remains for us is: How much PCB contamination will be left in the river sediments and bank soils? And what about the massive amounts that will be transferred from one part of our environment to another, to a lined dump the authorities swear will be safe from the likely increasing threats of the climate crisis? All of which remains a continuing and constant threat to the public health and the environment. And while the negotiated settlement crafted by GE and the EPA and the river towns, coupled with the decision of the court, have preempted us from taking action against them, the toxic Aroclors from Monsanto remain.

Upon publication of “Sue Monsanto, save Lee — a modest proposal in three parts (Part One),” a source reached out and sent me Monsanto documents I had never seen before. I learned I was off by several years and several significant symptoms. Yes, Dr. Louis Schwartz of the United States Public Health Service had warned in 1936 about the systemic effects—including chloracne, hematuria, and impotence—from working with and inhaling PCBs. But the Swann Company, which Monsanto had purchased in 1930, had already acknowledged the dangers of Aroclor’s vapors. Of course, there is always a however:

VAPORIZATION LOSS

The clear resins have rather low vaporization losses. At 65.6°C (150°F) these losses are less than 0.04 g. per square inch of surface per month.

TOXICITY

The Aroclors, so far as is known, have no harmful physiological action. When they are applied to the skin, there is no evidence that they are vesicants. The vapors, however, will irritate the membranes of the nose and throat.

“Monsanto: A Brief Description of Aroclors,” February 20, 1931. Highlighting added.

In fact, GE workers in Pittsfield were experiencing the very real “physiological” consequences daily. Bill Kowalczyck shared his experience:

“I was a group leader as a crane operator, I overseen most of the moving operations of not only the transformers and their core units ... GE is constantly saying there’s no health effects, ill effects with this PCBs or anything attributed with the PCBs. In my own case, I have chloracne on my legs. I have 21 ppb PCBs in my blood. My wife has health conditions. My daughter has tumors ... There are my friends that I have worked with over the years that I have noticed now they are dying. Pancreatic explosions ... seven of my friends right now that have a blood vessel disorder, which is a collapsing of blood vessels. It seems awful strange that we all worked together and we all have the same problem. ... Too many instances of my friends dying over the last, you know, five, six years. I’ll be 55 in

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"[W]hen the transformer blew for whatever reason or failed Test, they would pull it out, take the iron off, which was covered with that Pyranol, bring it back upstairs, and we had to re-lace it into a new coil. And we couldn't wear gloves—we had to put it in bare-handed because the oil, you couldn't pick the iron up ... in the Tank it was even worse ... [Y]ou drop the transformer down into the tank, it's full of Pyranol, or oil whatever they were going to use, and it would set on two pins, and you had to reach down into that oil and put two nuts and a washer, a lock washer, so you're up to about here in it, putting it in ... And the stuff wasn't bad when they first put it through. It's when it blew up—it would come back, and you lace one in the morning, you'd pick up the first leg of iron, and I'd have a headache the whole day ..."

In public, Monsanto would routinely deny its Aroclors were harmful. In fact, those working in the Monsanto factory in Anniston, Ala. were suffering greatly. Thanks to the series of court battles that began in 1996, [Walter Owens v. Monsanto](#) and [Abernathy v. Monsanto](#), many internal Monsanto documents, some of which I have already referenced, some marked "Confidential: Read and Destroy," were produced in discovery.

During his opening statement in the Owens v. Monsanto trial, on April 4, 2001, Monsanto's counsel claimed:

"The truth is that PCBs are everywhere. They are in meat, they are in everyone in the courtroom, they are everywhere and they have been for a long time ... The truth is that the men and women who have worked around PCBs the most over forty, fifty, sixty years, people in our plant, people in the electrical industry, have not experienced any significant health problems which can be associated or tied into or caused by PCBs other than a serious skin condition called chloracne, which is easily treatable." (*Emphasis added.*) (Trial Transcript, Owens v. Monsanto CV-96-J-440-E, [N.D. Alabama April 4, 2001], pg. 453, line 16.)

With the release of the documents, the extent of Monsanto's knowledge of PCB toxicity was revealed. As [The Washington Post](#) wrote on January 1, 2002: "Monsanto Hid Decades Of Pollution."

The workers demanded a jury trial before the Seventh Judicial Circuit Court of Alabama:

"The plaintiff avers that as a proximate result of defendant's negligence and his subjection to said fumes and dust, he has sustained the following injuries: He has been made sick, his body has become covered with sores, the skin of his whole body has deteriorated, his digestion has become impaired, his nervous system has been disordered, and other organs of his body have become affected and he has been caused to suffer physical pain and mental anguish and to lose sleep. He has become impotent and has lost his vigor and vitality. He has become less able to earn a livelihood and plaintiff avers that he has been permanently injured." (*Emphasis added.*)

Symptoms far beyond chloracne. Having known of this worker's complaint, and the dangers to the nervous system caused by

**WORKERS FILE
DAMAGE SUITS**

**\$1,200,000 Asked Of Swann
And Allied Plants**

Damage suits aggregating \$1,200,000 have been filed against the Swann Chemical Company and allied corporations by 12 Anniston chemical plant workers. It became known here Thursday.

The plaintiffs, each seeking \$100,000 apiece, charge in their bill that because of their employers' negligence they were permitted to work in chemical plants and inhale vapors from chemicals that have caused their bodies to be covered with sores, their nervous systems have been affected, they have suffered mental anguish and physical pain and have become "less able to earn a livelihood."

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Instead, in 1935, E.H. Buford of Monsanto wrote a report entitled "Diphenyl and Chlorinated Diphenyl Derivatives." Buford acknowledged that in 1933, thanks to inadequate ventilation, workers exposed to dust and fumes from heated Aroclors began to develop "a severe type of dermatitis." Bur explained that, simultaneously, the "electrical properties of the distilled Aroclors began to fall below specifications" which led them to conclude there was some impurity in the solution, and that styrene dichloride entered the pores of the workers along with the Aroclor dust and fumes causing irritation and an infection. In the face of worker litigation, Monsanto's solution was to replace the workers most readily susceptible to the fumes and contact with Aroclors and swap them with workers they hoped were more hardy.

Skin specialists were consulted, but cure of the disease was found to be slow and difficult. The company became involved in an expensive litigation since suit was entered by the majority of the men affected.

As a result of this trouble the following precautionary measures were taken,

1. All affected men were removed from the operation and new men substituted as rapidly as they could be trained.

2. Adequate ventilation facilities were installed.

DSW 001398

Monsanto's 1935 "Diphenyl and Chlorinated Diphenyl Derivatives." Highlighting added.

Then they implemented a set of protocols we know never lasted: a change of clothes, towels, soap, and cold cream which resulted in "no recurrence of dermatitis among the operators."

And yet the problems persisted. On March 16, 1936, the Commonwealth of Pennsylvania published "A Preliminary Report on the Dermatological and Systemic Effects of Exposure to Hexachloro-Napthalene and Chloro-Diphenyl":

"The relatively high percentage of these individuals affected ... in a group of one hundred and twenty-six persons examined, indicates a need for preventive measures ... In those processes where the compounds are heated to a temperature sufficient to cause volatilization and subsequent condensation in air, either closed methods, operating under partial vacuum or exhaust ventilation should be instituted."

In 1936, Dr. Jones and Dr. Alden published "An Acneform Dermatogosis":

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REPORT OF A CASE

History.—O. D., a Negro aged 26, began work in the distillation of chlorinated di-phenyl in April 1930 and worked regularly until the latter part of the year 1933. About May of 1933, he noticed the appearance of blackheads on his face, neck, arms and legs. These areas itched slightly. In a short time blackheads began to appear on the chest, back and lower part of the abdomen, around the navel and on the scrotum and penis. Many of these blackheads swelled and became infected, discharging thick pus. The areas healed with difficulty and often left scars. The condition seemed to be progressive until November 1933. When seen in December 1933 the patient complained of lassitude, loss of appetite and loss of blith and said that his cutaneous condition seemed to be improving.

Physical Examination.—On examination he seemed in good general health. His complaint of lassitude was not borne out by anything more than the usual temperament of the Negro toward work. On the forehead, extending within the hair line, and on the cheeks, chin, nose and neck were numerous small, very black, tenacious comedones, their distribution best described as being "peppered" within the skin. Many of the comedones surmounted firm shotlike cysts, which in some areas contained viscid yellow pus. The pustular elements were more noticeable on the neck. Similar shotlike comedones and cysts had appeared on the shoulders, midportion of the back and chest, with an occasional large cyst. A peculiar leppering of the skin with tenacious carbon-colored comedones was apparent around the umbilicus and lower portion of the abdomen. The scrotum and penis were involved in a similar process, the former being given more to the formation of cysts. The outer surfaces of the forearms and anterior thighs showed similar but fewer comedones. The whole eruption was acneform but differed from

"An Acneform Dermatogosis," 1936. Highlighting added.

Sadly, there is the egregious racism of these doctors: "His complaint of lassitude was not born out by anything more than the usual temperament of the Negro toward work." But far more relevant to the issue at hand is the evidence of yet another case of someone who worked with PCBs as early as 1930 to 1933 experiencing a wide range of systemic effects.

And as Monsanto endangered its workers, so too did it expose those who lived adjacent to its Anniston, Ala. factory. As The Washington Post explained:

"[F]or nearly 40 years ... Monsanto Co. routinely discharged toxic waste into a west Anniston creek and dumped millions of pounds of PCBs into oozing open-pit landfills ... In 1966, Monsanto managers discovered that fish submerged in that creek turned belly-up within 10 seconds, spurting blood and shedding skin as if dunked into boiling water. They told no one. In 1969, they found fish in another creek with 7,500 times the legal PCB levels. They decided 'there is little object in going to expensive extremes in limiting discharges.' In 1975, a company study found that PCBs caused tumors in rats. They ordered its conclusion changed from 'slightly tumorigenic' to 'does not appear to be carcinogenic.' Monsanto enjoyed a lucrative four-decade monopoly on PCB production in the United States, and battled to protect that monopoly long after PCBs were confirmed as a global pollutant. 'We can't afford to lose one dollar of business,' one internal memo concluded." (*Emphasis added.*)

In its January 1, 2002 story "[In Dirt, Water and Hogs, Town Got Its Fill of PCBs](#)," The Post focused on one of the plaintiffs:

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The first time the man from Monsanto Co. knocked on Ruth Mims's door, he announced that her hogs were trespassing on company property. "We'll give y'all 24 hours to move those hogs," he warned her. She panicked. She had no room for hogs in her little yard.

But the man knocked again a few minutes later. Tell you what, he told Mims. We'll buy the hogs for \$25 a head, plus a pint of white corn liquor. Mims didn't drink, but Christmas was coming and she was short on cash, so she said yes.

That was in December 1970. It wasn't until Mims told that story in federal court last April that she saw Monsanto's secret "Hog Analysis Results" from 30 years earlier. The company had dissected some hogs from the west Anniston area and found PCB levels as high as 19,000 parts per million. There were no legal limits then, because the idea that PCBs could end up in hogs was pretty new, but that would be more than 90,000 times the legal maximum in some states today.

The Washington Post, Jan. 1, 2002. Highlighting added.

On December 15, 1966, London's New Scientist journal published a brief notice of the work of Swedish scientist Sören Jensen, "[Report of a New Chemical Hazard](#)":

"A Swedish research worker has expressed concern over the increased evidence of polychlorinated biphenyl (PCB) entering the air, presumable from industrial smoke and rubbish dump smoke and being absorbed by water and taken up by fish and later humans. PCB which is related to and as poisonous as DDT was detected by Mr. Sören Jensen of the Institute of Analytical Chemistry, University of Stockholm, in 200 pike taken from different parts of Sweden, fish and fish-spawn throughout the country, an eagle which was found dead in the Stockholm Archipelago, and in his own, his wife, and his baby daughter's hair. As the baby is only five months old her father concludes she got her dose of PCB with her mother's milk."

Then, in 1968, [more than 1,300 people got sick in Yusho, Japan](#) after consuming rice bran oil that had been contaminated with more than 2,000 parts per million (ppm) of PCB oil. "Among males, excess mortality was observed for all cancers ... Among females, increased mortality was observed for liver cancer ..."

On December 14, 1968, Nature magazine published "[Polychlorinated Biphenyls in the Global Ecosystem](#)," by R. W. Risebrough et al, citing research that showed the dangerous effects of PCBs on the thickness of the eggs of raptorial and fish-eating birds in America and Great Britain:

"A widespread change in the chemical environment which affected the calcium physiology of these species evidently occurred at that time. The chlorinated hydrocarbons, which came into general use in the 1940s may now be the most abundant synthetic pollutants present in the global environment." (*Emphasis added.*)

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It seems that, by April 1969, Monsanto was aware of studies being done on shrimp and oysters in the Escambia River and Pensacola Bay, Fla. The results of this research, "A Polychlorinated Biphenyl (Aroclor 125) in the Water, Sediment, and Biota of Escambia Bay," Florida was published in the Bulletin of Environmental Contamination and Toxicology in March–April 1970:

"We have detected a polychlorinated biphenyl (PCB), Aroclor 1254, in the biota, sediment, and water of estuarine area near Pensacola, Florida. Only one source of the chemical, an industrial plant on the Escambia River, has been located. However, the chemical occurs in tissues of pelagic and sessile organisms that are widely distributed within the estuary. This distribution of Aroclor 1254 could be due to dispersion of the chemical from the river by currents and biota to other parts of the system ...

"Our laboratory studies showed that juvenile shrimp were the most sensitive, and were killed when exposed to 5.0 ppb of Aroclor 1254 in flowing sea water. The Aroclor content in water from Escambia Bay, even near the mouth of the river, contained less than 1 ppb. Shrimp collected from the bay contained a maximum of 2.5 ppm. Thus, the shrimp in Escambia Bay probably were not exposed to lethal levels of the chemical during the sampling period.

"This study demonstrates the urgent need for continued surveillance of our estuaries in order to preserve these nursery grounds for our valuable fishery resources. Also, the study shows a need for conducting long-term tests on the effect of sub-lethal concentrations of Aroclor 1254 on estuarine organisms in sensitive stages of their life history." (*Emphasis added.*)

By the way, "5 ppb" is five parts per billion—a very small amount. And so what had been kept private all along—the knowledge of the systemic toxicity of Monsanto's PCB Aroclors and, beginning with even small doses, their tendency to bioaccumulate—was now becoming public, and impossible to ignore. Monsanto began a public relations campaign to respond.

Here is a [handwritten note](#) from a meeting of Monsanto's PCB Committee on August 25, 1969, which gathered to weigh strategies to deal with the ever-increasing appreciation of the dangers of PCBs. They posed two alternatives: Go out of business, or sell the living hell out of their Aroclors as long as they can.

Subject is Snowballing:
Where do we go from here →
Alternatives:
Possibly 1254 } identified
1260 }
1248 }
1242 }
1.) GO OUT OF BUSINESS
2.) Sell the Hell out of them as long as we can and
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Here are some notes from Monsanto's September 5, 1969 first meeting of its "Aroclor Ad Hoc Committee":

Objectives: (Agreed to by the Committee) Submit recommendations for action which will:

1. Permit continued sales and profits, of Aroclors and Terphenyls.
2. Permit continued development of uses and sales.
3. Protect image of Organic Division and of the Corporation ...

Background Discussion of Problem:

2. PCB has been found in:

a. Fish, oysters, shrimp, birds.

b. Along coastlines of industrialized areas such as Great Britain, Sweden, Rhine River, low countries. Lake Michigan, Pensacola Bay, in Western wild life (eagles). It may be a global contaminant.

3. PCB has been tied to DDT in effects on disappearance of wild birds which have fish diets. Ratio of PCB to DDT has been about 40-50:1 generally. Dr. Reisboro reported almost 1:1 ratio. PCB may be contributing to or exaggerating the effects of other chlorinated aromatics. (Emphasis added.)

Then, close to home, there was the Escambia River problem:

6. Escambia River Problem:

For a clearer understanding of the general problem, the situation at Pensacola was reviewed. From a relatively negligible discharge of 1-3 gal/day into a large river, 1/4 mile downstream levels of 42 ppb in water and 476 ppm in mud were found. Although use of Aroclor was halted immediately, we can expect the water contamination to continue for a lengthy period by leaching from the contaminated mud. No downstream samples have yet been taken to measure the decrease in contamination (as of 9/5/69).

September 5, 1969, first meeting of the "Aroclor Ad Hoc Committee." Highlighting added.

Monsanto acknowledges—but most probably underestimates—the loss of Aroclor (one to three gallons a day) from its factory to the surrounding waterway. It then explains that preventing such losses from continuing probably wasn't worth the money it would cost:

"The question of exactly how far to reduce (how much money to spend) is not yet clear and expenditures to date have been comparatively small. It was agreed that, until the problems of gross environmental contamination by our customers have been alleviated, there is little object in going to expensive extremes in limiting discharges from the plants." (Emphasis added.)

On March 3, 1969, Monsanto issued [this statement](#):

"On February 24, the San Francisco Chronicle carried a major feature about 'a menacing new pollutant' found in the San Francisco Bay area. The article was based on marine life research carried out by Dr. Robert Risebrough of the University of California. It stated that residues of polychlorinated biphenyl (PCB) were killing certain birds and implied a long-term threat to humans. Monsanto manufactures polychlorinated biphenyl and markets it under our

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"Dr. Risebrough's more recent work reports the identification of PCB, along with DDT and DDE pesticides, in the tissues of birds and fish on the West Coast. The conclusions of these scientists are puzzling from several aspects. Polychlorinated biphenyls are stable chemical compounds which are essentially insoluble in water. Their use does not make them easily released into the natural environment ... It has also been implied that polychlorinated biphenyls are 'highly toxic' chemicals. This is not true. The toxicity of any material, whether it be chemicals, drugs, natural plants or even foods, is relative. Just like other industrial chemicals and home products now in widespread use, PCBs are not hazardous when properly handled and used." (*Emphasis added.*)

On October 2, 1969, Monsanto issued its confidential "Report of 'Aroclor' Ad Hoc Committee," and it is interesting to see how they rephrased one of their objectives to make it more eco-friendly: "Protect the image of the Organic Division and the Corporation as members of the business community recognizing their responsibilities to prevent and/or control contamination of the global ecosystem." (*Emphasis added.*)

But, most significantly, the committee counseled the development of a strategy to maintain Monsanto's profitability. With little likelihood that Monsanto could control the growing realization of the risks of their Aroclors throughout the world, it was most critical that they continue sales.

PROBABILITY OF SUCCESS

The committee believes there is little probability ~~(~~to~~ ~~be~~ ~~achieved~~)~~ that any action that can be taken will prevent the growing incrimination of specific polychlorinated biphenyls (the higher chlorinated--e.g. Aroclors 1254 and 1260) as nearly global environmental contaminants leading to contamination of human food (particularly fish), the killing of some marine species (shrimp), and the possible extinction of several species of fish eating birds.

Secondly, the committee believes that there is ~~no possible~~ ^{practical} ~~course of action~~ that can so effectively police the uses of these products as to prevent ~~environmental~~ ^{completely some} contamination. _{in order}

There are, however, a number of ~~possible~~ actions which must be undertaken ^{in order} to prolong the manufacture, sale and use of these particular Aroclors as well as to protect the continued use of other members of the Aroclor series.

The ultimate that can be expected is ^(less than 5 chlorines) the continued use of the lower chlorinated biphenyls and the chlorinated terphenyls in applications amenable to such control that there is practically zero losses to the environment. In the interim we would hope to establish by appropriate research efforts "tolerance" or safe levels for particular Aroclors in the environment.

Monsanto's confidential October 2, 1969 "Report of 'Aroclor' Ad Hoc Committee." Highlighting added.

They are, however, keenly aware of their liability:

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BASIS FOR RECOMMENDATIONS (Continued)

As the alarm concerning the contamination of the environment grows it is almost certain that a number of our customers or their products will be incriminated. The company could be considered derelict, morally if not legally, if it fails to notify all customers of the potential implication.

sept. ~~August~~ A case in point is the recent determination (mid-August) that milk to be marketed by the Maryland Cooperative Milk Producers, Inc. in Baltimore was contaminated with polychlorinated biphenyls. The source of the PCB's was isolated to six dairy herds in Martinsburg, West Virginia. Investigation by the Producers Association is continuing but to our knowledge the specific source of the PCB has not been pin-pointed.

Monsanto's confidential October 2, 1969 "Report of 'Aroclor' Ad Hoc Committee." Highlighting added.

This is a critically significant document: While Monsanto acknowledges the growing tendency to regard its Aroclors as "nearly global contaminants leading to contamination of human food (particularly fish), the killing of some marine species (shrimp), and the possible extinction of several species of fish eating birds," it claims there is no practical way to effectively police the use of their Aroclors or prevent environmental contamination. There are, however, ways to prolong sales.

Considering the many billions of dollars Monsanto had made marketing Aroclors, the claim they couldn't actively protect the environment seems palpably false. Unsurprisingly, the actions the Aroclor Ad Hoc Committee doesn't contemplate is to make a public admission of their liability along with a pledge to finance immediate remediation of contaminated waterways.

Rather, Monsanto acted to protect itself from claims that "it was derelict morally if not legally." How? By reaching out in an almost ex post facto fashion to notify customers to take steps to prevent their Aroclors from entering the environment. The confidential October 2, 1969 "Report of 'Aroclor' Ad Hoc Committee" refers to the letters they sent:

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On ~~September~~ 24, 1969 the San Francisco Chronicle published a "scare" story following an interview with Dr. Robert Risebrough of the University of California. The latter had recently published in Nature the finding of polychlorinated biphenyls in fish, birds and eggs in the California coastal areas.

On March 3, 1969, the Functional Fluids group sent a letter to the 31 major Aroclor customers in the transformer and capacitor applications. The letter included a copy of the Chronicle story and a Monsanto statement concerning the situation. This was intended to announce to these customers that the polychlorinated biphenyls might be in trouble and implied that the customers should make every effort to prevent loss of these materials to the environment. There has been subsequently some follow-up with at least General Electric and Westinghouse.

It has been recognized from the beginning that other functional fluid uses could lead to losses of the Aroclors to liquid waste streams from the customers' plants. Losses could occur from spills, unusual leakage of large volumes and daily losses of smaller volumes.

It has also been recognized that there could be vapor losses but it has been felt that these were perhaps of less significance than the vapor losses in plasticizer applications. The concern for vapor losses rises from the published proposed theory that even minute quantities of vapors are eventually transferred to the water environment and accumulated therein.

Monsanto's confidential October 2, 1969 "Report of 'Aroclor' Ad Hoc Committee." Highlighting added.

On November 17, 1969, Monsanto made a "PCB Presentation to Corporate Development Committee." The presentation first acknowledged, "Certain PCB's have recently been identified by various scientists along with DDT in fish, birds, and other wildlife. From the standpoint of reproduction, the PCB's are highly toxic to birds," then moved to secure guidance and approval on "a recommended plan of action."

This is a serious matter, not only from the pollution viewpoint, but also because of the \$22 M worldwide customer business involved with resultant gross profits of \$10 M and a net investment of approximately \$9 M. In addition, there could be possible adverse legal and public relations problems leveled against Monsanto.

Monsanto's Nov. 17, 1969 "PCB Presentation to Corporate Development Committee." Highlighting added.

Three alternative strategies were offered:

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Alternative 2: Go out of total Aroclor business was considered unacceptable from a Divisional viewpoint, but from a Corporate viewpoint may be necessary. Only-you-can-make-that-decision. All Aroclor products are not serious pollutants – many degrade, there is too much customer/market need and selfishly too much Monsanto profit to go out ...

Alternative 3: Go out of Aroclor 1254 and 1260. This was seriously considered and may eventually occur by our actions and customer actions, nevertheless, we feel that segments of this business are defensible or are so confined in use that specific plans of action are called for this portion ... (Emphasis added.)

Not only were Aroclors contaminating our environment, but when Monsanto hired Industrial Bio-Test Laboratories (IBT) in 1969 to undertake toxicity studies of its products, it began a decade-and-a-half-long joint effort to falsify test results. In "Ashamed to Put His Name to It: Monsanto, Industrial Bio-Test Laboratories, and the Use of Fraudulent Science, 1969-1985," David Rosner and Gerald Markowitz tell the story of how Monsanto and IBT tried to falsely reassure customers and the regulatory agencies that their Aroclors were safe.

Here is an excerpt from Monsanto's notes of a January 21 and 22, 1970 meeting in St. Louis with GE:

D. Status of Aroclor Studies At Industrial Bio-Test. Table 2.

In essence results reported by Mr. Wheeler on chronic animal toxicity tests and animal reproducibility studies underway are not as favorable as we had hoped or anticipated. Particularly alarming is evidence of effect on hatchability and production of thin egg shells regards white leghorn chickens. The studies involved Aroclor 1242, 1254 and 1260. Some of the studies will be repeated to arrive at better conclusions.

Excerpt from Monsanto's "The PCB-Pollution Problem." Highlighting added.

Rosner and Markowitz explain:

"Monsanto sent IBT new samples that they claimed were 'clean[ed] up' and told IBT they hoped to 'find a higher 'no effect level,' a potential "safe" level below which the experimental animals would not show symptoms of damage.' Indeed, their collaboration with IBT to downplay the hazards of PCBs appears to have been successful. By 1973, they claimed that 'the most important data which has led the government agencies to permit the continued but constricted use of polychlorinated biphenyl are the extensive animal toxicity studies which we have completed in the last two years.'" (Emphasis added.)

Except that the most important data was the falsified study data.

Then, in the meeting with GE, Monsanto emphasized the critical need for transformers and the Aroclor fluid:

"[W]ithout availability of askarel transformers large cities like New York would be shut down with no power ... Without Aroclor capacitors most of the lights across our country would go out and motors in air conditioners and many industrial applications would not run." (Emphasis added.)

And we learned from Monsanto that, while expressing the continuing need for Aroclor, GE refused Monsanto's wish that GE accept sole liability for its use:

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L. What GE Desires

1. GE seeks that Monsanto take no precipitous reaction to the PCB problem that would result in withdrawing supply of Aroclor 1254 or 1260 to GE.
2. The consensus is that no suitable replacement for transformer askarel fluid is foreseen.
3. In event of development of a suitable fire-resistant fluid replacement for askarel, Mr. Raab emphasized that a minimum of 2 years testing work would be required before commercial use could be adopted.
4. In reply to Monsanto's legal question whether with continued use of Aroclor 1254 and 1260 GE would assume sole and complete liability -- Mr. Raab answered, No! To substantiate his reply, Mr. Raab cited case examples involving GE where damages were sought and collected, even though GE was only the third party. He further stated that any arrangement seeking to delegate and confine liability to GE relative to the PCB problem would be worthless.

Excerpt from Monsanto's "The PCB-Pollution Problem." Highlighting added.

GE needed Monsanto's Aroclors. As we will see soon, some things are not what they seem. As for the IBT studies, Rosner and Markowitz explain:

"[T]he reliability of those studies was belied by two facts: first, the actual conditions in the IBT labs that tested PCBs for Monsanto were soon found to be compromised, and second, data were found to be fabricated and sent to the government as ostensible 'proof of their chemicals' safety ... Central to these activities was Paul Wright, who was the link between IBT and Monsanto. Wright was employed at Monsanto beginning in 1965 as a senior research chemist and from 1968 until 1970 as a research group leader. In 1970, as IBT began its two-year chronic testing of PCBs for Monsanto, Wright moved to IBT, where he directed the toxicology lab that oversaw these studies. In late 1972, he returned to Monsanto as the toxicology manager and stayed at Monsanto until 1984 ... Philip Smith, the lab assistant in the IBT PCB studies, gave vivid descriptions of how Wright had falsified data that ended up in the report sent to the government ..."

Rosner and Markowitz continue.

"On May 4, 1981, a federal grand jury handed down an indictment ... [and] detailed that Monsanto's Wright made 'false, fictitious and fraudulent statements and representations ... In August 1983, Paul Wright, Moreno L. Keplinger, and James B. Plank, former assistant toxicology manager, were convicted of fraud and sentenced to jail. But even following conviction, Monsanto gave Wright a 'golden parachute,' providing him with full retirement benefits, accrued vacation time, one month's severance, and the services of a recruitment specialist to help him find future jobs when he was released from prison."

According to the EPA, the Monsanto plant in Anniston, Ala. "produced Aroclors between 1929 and 1971... [EPA] estimated that the amount of Aroclors produced at the Anniston plant was about 680 million pounds."

Monsanto poisoned the shrimp of Escambia. GE poisoned the fish and ducks of the Housatonic. The EPA revealed in 1999:

"Waterfowl samples collected last summer on the Housatonic River in western Massachusetts show elevated

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3,700 parts per million adjusted for fat—were found in the breast tissue of a six-month old wood duck." (*Emphasis added.*)

Thanks to our share of Aroclor, we in Berkshire County have been victims of GE and Monsanto's gross incompetence, remarkable negligence, and their unrelenting impulse to put profit before worker safety, and the public health and our environment. GE and Monsanto have made us pay a fearsome price, and they are determined to leave us all, but especially Lee with a toxic legacy. Hopefully we can find a possible solution.

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Woods Pond in Lee in the late afternoon. Photo by Mickey Friedman.

IN FOCUS

THE OTHER SIDE: Sue Monsanto, save Lee — a modest proposal (Part Three)

I have read too many words, written too many. For me, there is always the reality of a poisoned Rising Pond, but the enduring dream of a fishable, swimmable river.

BY MICKEY FRIEDMAN
POSTED ON DECEMBER 26, 2023



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For a moment, I want to go back to what turns out to be a very important year: 1970. The scientific reality that hydrocarbons like Aroclor were poisoning the global environment began to overwhelm Monsanto's rhetoric, and environmental regulators were slowly beginning to take notice. Monsanto reached out to its customers in a coordinated attempt to maintain its profits and buy some time. Elmer Wheeler of Monsanto met with GE on January 21 and 22, 1970:

A. Presentation and Discussion of Published Articles About Chlorinated Aromatic Hydrocarbon Insecticides, (DDT etc.) and PCBs

Mr. Wheeler presented to Mr. Raab a booklet containing most of the pertinent publications, to date, and indicated that additional articles will appear shortly.

He mentioned that manufacturers of DDT and chlorinated aromatic hydrocarbon insecticides will tend to emphasize the finding and interference of PCBs as the Government hearings limiting or banning use of the insecticides are held.

This lead GE to seek understanding of the scope, reproducibility, reliability and validity of the analytical procedures used by various investigators who reported finding PCB in concentrations as low as parts per billion.

B. The Analytical Procedures

Drs. Keller and Tucker presented details of Monsanto's GLC - Mass Spectrometric analytical capability and apparatus, as portrayed in Table 1. The sophistication of our analytical capability was emphasized to assure that our approach is the ultimate and is not surpassed. On this basis our views of the validity of results given in various publications are indicated in the attachments to Table 1.

General Electric were impressed and completely satisfied with the scope of our analytical capability and work.

"The PCB-Pollution Problem," the Monsanto-GE meeting of January 21-22, 1970. Highlighting added.

Monsanto states:

In essence results reported by Mr. Wheeler on chronic animal toxicity tests and animal reproducibility studies underway are not as favorable as we had hoped or anticipated. Particularly alarming is evidence of effect on hatchability and production of thin egg shells regards white leghorn chickens. The studies involved Aroclor 1242, 1254 and 1260. Some of the studies will be repeated to arrive at better conclusions. (Emphasis added.)

If these notes are accurate, and if GE was completely satisfied, it appears that Monsanto never fully shared with GE its knowledge of—and GE never fully appreciated—the reliability of several studies showing why its Aroclor (Pyranol) needed to be kept from leaving their factories, or the details of what actually happened with the PCBs that made it to the environment.

On February 18, 1970, Donald A. Olson, director of sales at the Monsanto Functional Fluids Group, wrote to other customers:

It is claimed that the PCBs found strongly resemble chlorinated biphenyls containing 54 [percent] and 60 [percent] chlorine by weight. Products which are sold by Monsanto under the trade names of Aroclor® 1254 and 1260 do contain chlorinated biphenyls ... As your supplier of Aroclor® 1254 and 1260 and formulated products containing 1254, we wish to alert you to the potential problem of environmental contamination as referred to in the

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We feel that all possible care should be taken in the application, processing and effluent disposal of these products to prevent them becoming environmental contaminants. Of interest to you may be an article in Chemical Week, October 29, 1969, regarding water pollution standards set by each state in the Union. It is attached. This article reflects that good manufacturing practice in the future may require that no product used by any company should find their way into waterways.

We realize that you have marketed or may now market transformers and other electrical equipment containing dielectric fluids which include Aroclor® 1254 and 1260. Although these fluids are sealed into such equipment it is recognized that occasionally the fluid may be lost through leaks resulting from equipment misuse or equipment repair necessitating replacement of the fluid. Since the dielectric fluid contained in this equipment is only an incidental part of the over-all unit manufactured by you, we are not notifying the purchasers of such equipment of the potential environmental contamination problem described in this letter. We do recommend, however, that you notify such equipment users of this problem.

Monsanto's February 18, 1970 letter to its customers. Highlighting added.

As always, Monsanto parsed its words and actual environmental contamination became potential. Here is Chemical Weekly's [1969 listing of water quality standards](#) for Massachusetts:

Radioactivity: None in concentrations harmful to human, animal or aquatic life. Turbidity, Color, Taste and Odor: None in concentrations that would impair industrial use. Solids: None allowed except that which may result from the discharge from waste-treatment-facilities providing appropriate treatment. Toxic Substances: None in concentrations or combinations harmful to human, animal or aquatic life.

"For the first time, a summary of water quality standards set by all 50 states," Chemical Weekly, October 29, 1969. Highlighting added.

No toxic substances should be released in Massachusetts that would prove harmful to human, animal, or aquatic life.

Monsanto was trying to have it both ways: acknowledging the PCB problem to some while denying it to others. After all, almost all this guidance to be extra careful had been advocated by Dr. Schwartz in June 1936—then rigorously disregarded. That same day, [Papageorge wrote Dr. Continelli](#) at the Buffalo Children's Hospital asserting that Monsanto had no knowledge of the harmful effects of their Aroclor:

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August 31, 1970

Dr. Basil M. Continelli
Drug Information Center
Buffalo Children's Hospital
125 Hodge Street
Buffalo, New York 14222

Dear Dr. Continelli:

Your letter to Monsanto Company dated August 24, 1970 was forwarded to me for response since I have been assigned the responsibility for studying the total PCB environmental problem.

I appreciate the concern you have regarding the possible toxic effects of PCB.

Monsanto has manufactured Aroclors for about 40 years and throughout the period we have not observed any harmful effects on our employees or our customer's employees. Extensive animal testing being conducted for Monsanto has not revealed any evidence of deformity in the usual laboratory animals. We are therefore disturbed by the article's reference to birth defects in animals. To our knowledge no laboratory has evidence to confirm this statement.

W. B. Papageorge to Dr. Basil M. Continelli, August 31, 1970. Highlighting added.

Meanwhile, the February 1971 "[Conference in the Matter of Pollution of the Interstate Waters of Escambia River Basin](#)" named the Monsanto Chemical Plant as the source for PCBs in Escambia:

Our first transect was about two miles above the Monsanto Plant and we found no Aroclor 1254 in the surface or bottom water sediments. At the Monsanto weir in Escambia River we found 0.4 of a part per billion in the surface water and 0.2 in the bottom water. Fifty yards below this point, 0.5 parts per billion on the surface and it was not detectable in the bottom.

The document continues:

... it does appear from these first samples that Aroclor is contained in sediments a little deeper than the surface ... At zero to 2 inches from the surface we had 78.0 parts per million at the weir, 2 to 4 inches 30.0 parts per million, 4 to 6 inches, 6.1, and 6 to 8 inches 0.4.

MR. REED: Mr. Chairman, may I interrupt?

MR. WHITE: Yes, Mr. Reed.

MR. REED: Tom, this doesn't sound like a short-term leak to me if we are getting stratification levels like this. This sounds like a long and continued discharge into the bay to build up. Admitted, these are preliminary figures, but this is not — this is a stratification that is deep. (Emphasis added.)

With Escambia, two realities became crystal clear: Monsanto's Aroclors had made it to the waterway, and while levels

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let the Aroclors make their way to water. But what they didn't make clear was that the Aroclors weren't moving through the water column and out to sea—as most might have assumed—but were congregating in the sediments and bank soils where they contaminated the living things that came in contact with them.

It is also clear that, despite this now undeniable knowledge, Monsanto never did anything to take effective action to remediate this problem—a problem they had caused for decades and continue to cause as long as their toxic Aroclors remain present.

A revelatory [1972 interview with Elmer Wheeler](#) provides insight into what was going on within Monsanto:

[W]e were told—that the material that the Swedes had identified was specifically polychlorinated biphenyl in probably March or April 1967 ... Initially, we ignored the significance of the Swedish work. We just couldn't believe that based on the uses of the Aroclor, based on the stability of it and based on the lack of solubility, that these things could conceivably begin to show up in the environment as did the chlorinated pesticides ... Almost a year following April 1967 the British published information that tended to confirm the Swedish work—again, however, without being so specific in their analytical techniques that true identification was without question. I guess it was late February 1969 that the you-know-what hit the fan when Professor Risebrough out at Berkeley published his paper indicating that he had found PCB's in birds and fish on the California coast ...

Wheeler also talks about the new understanding that came with the 1968 acquisition and use of “a gas liquid and spectrophotometer ... able to detect the presence of PCBs in all types of samples at low levels ...”

Here is an excerpt from that interview:

INTERVIEWER: So, as I understand it, he took water, things of that sort, and then tested them to see if he could determine if PCBs were present in them?

WHEELER: Yes. And in order to do that, he deliberately contaminated them and then ran them through the instrument to see if his numbers matched the input ...

INTERVIEWER: Did he do any analysis on material that he hadn't contaminated deliberately with PCBs? ...

WHEELER: ... They did go out and get some soil samples around the plants, soil near the operation and so on.

INTERVIEWER: And did they find that these soils had PCBs in them?

WHEELER: Yes ... [and] they were able to feel more confident of the numbers ...

INTERVIEWER: Was he surprised that there were PCBs in the environment?

WHEELER: Oh, yes ...

INTERVIEWER: So as I understand your answer, Dr. Keller's surprise was with respect to the extent that the PCBs had migrated from the factories? ...

WHEELER: That was one of the surprises.

INTERVIEWER: What other surprises did he have?

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Wheeler described a 1969 pesticide symposium at Oregon State University:

[S]ome of Risebrough's more vocal adherents were there ... Real hippy types with long hair, clean but raggedy jeans, sandals or bare feet and any time Risebrough would take a crack at industry, they would applaud and of course the local TV and press were just swarming around these activists every opportunity they had ...

The upshot of this meeting was that, on the evening of the first day, Marsh and I were having a drink before we went to dinner and Marsh said, 'Elmer, these people are going to put Monsanto out of the PCB business' and I said 'I'm beginning to think you are right.' Fancher had the same reaction. We came back from that meeting and sat down with ... the people that were involved in what was going on in the PCB area at that time and out of that came a task force of 5 or 6 of us to put together the then current status of our knowledge about PCB's and predictions as to the future. We issued a report which was intended to be a preliminary draft of the deliberations of that group. That was about October 15. It got fairly wide distribution and subsequently we got a call from one of Dick Stohr's counterparts in the Law Department which said, 'call back all of those reports and burn them.' It was redistributed to some 12 people with the legend on the front 'ATTORNEY CLIENT PRIVILEGE.' This led to a review in November of the whole situation before the Corporate Management Committee which was the agency of the Board ...

Yes, of course, burn all reference to the inconvenient reports or acknowledgment of what was really happening with Monsanto's Aroclors. But Monsanto didn't exactly have to worry yet about the EPA. The U.S. Interdepartmental Task Force on PCBs submitted its report "[Polychlorinated Biphenyls and The Environment](#)" in May 1972. It was clear U.S. regulatory agencies still didn't fully appreciate what Monsanto knew. And it is evident they still hadn't adequately done their own definitive tests or accurately extrapolated all the medical and scientific data available from the late 1930s on up. The EPA was still underestimating the severe risks that PCBs posed to human health and the environment:

PCBs should be restricted to essential or non-replaceable uses which involve minimal direct human exposure since they can have adverse effects on human health. There currently are no toxicological or ecological data available to indicate that the levels of PCBs currently known to be in the environment constitute a threat to human health, but additional experiments are underway to evaluate the impact of low level, long-term exposure to PCBs.

EPA then made a series of statements based as much on politics and economics as on environmental science: "PCBs have been used so widely over such a long period that they are ubiquitous. Even a total cessation of manufacturing and use of PCBs would not result in the rapid disappearance of the material, and ultimate disappearance from the environment will take many years ... Prohibition of PCB discharges into water will result in the reduction of such residues ...

The use of PCBs should not be banned entirely. Their continued use for transformers and capacitors in the near future is considered necessary because of the significantly increased risk of fire and explosion and the disruption of electrical service which would result from a ban on PCB use. Also, continued use of PCBs in transformers and capacitors presents a minimal risk of environmental contamination. The Monsanto Company, the sole domestic producer, has reported voluntarily eliminating its distribution of PCBs to all except manufacturers of electrical transformers and capacitors. (Emphasis added.)

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Several developments affected us in Berkshire County. As the world better understood the growing devastation of PCB contamination on the environment, corporate lawyers focused on issues of liability. As Monsanto contemplated withdrawing from the manufacture and sale of its Aroclors, GE still had a thriving business and still depended on Aroclor 1254. Monsanto decided to leverage that dependency. While GE had earlier refused Monsanto's request to accept all liability, it now relented. An [April 18, 1972 Monsanto document](#) explains:

Since early 1970, Monsanto Chemical Company, sole supplier of Pyranol, has advised that this material, under the generic term askarel, contains polychlorinated biphenyls (PCB). Polychlorinated biphenyls are highly stable compounds and are not readily biodegradable. Therefore, when placed in the environment, they may be considered contaminants and may adversely affect some species of animal and marine life.

At that time all customers were advised through Sales Channels to take every precaution to prevent any entry of this material into the environment through spills, usage, leakage, disposal, vaporization or otherwise.

In the past we have supplied Pyranol to transformer repair shops, and to companies requiring these products for top-up of existing transformers. Pyranol was also available from Monsanto under GE Specification A1333 without restriction regarding its usage.

These sales have been discontinued by Monsanto as of January 15, 1972, except to those who have entered into special agreement to indemnify Monsanto with respect to this product for use in transformers.

General Electric has agreed to indemnify Monsanto for this use, as have several other major manufacturers. Some manufacturers have chosen not to indemnify Monsanto and have decided to discontinue manufacturing askarel-filled transformers.

Monsanto's April 18, 1972 letter to customers. Highlighting added.

As Monsanto put it:

These sales have been discontinued by Monsanto as of January 15, 1972, except to those who have entered into special agreement to indemnify Monsanto with respect to this product in transformers ... General Electric has agreed to indemnify Monsanto for this use ...

Not surprisingly [GE passed on the indemnification requirement](#) to its customers:

Mr. A. P. Engel
LPD
Erie Works

Mr. J. K. McCall
TSPD
Erie Works

Consistent with our prior discussions you are aware of the considerable concern that has been expressed over the effects of polychlorinated biphenyls (PCB) on the environment. In an effort to protect the General Electric Company from unreasonable risks in this area, we are forwarding the attached information. This information must be communicated to your customers which have purchased Pyranol-filled transformers as part of your equipment.

The first attachment is an indemnification clause which we suggest you use with your ultimate customer on all new proposals. This indemnification clause will be included in all new Power Transformer Products Department proposals for Pyranol transformers. The second attachment is a

GE's October 1973 document discussing Pyranol indemnification agreements. Highlighting added.

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This incinerator was designed, and was demonstrated, to destroy PCB wastes at 99.9998 percent efficiency. By September, 1971, this unit was in continuous operation and was destroying both waste from our production and PCBs returned by customers as they converted to non-PCB replacements. When phaseout from nonelectrical applications was complete, the incinerator was used to destroy waste PCB which was collected from ongoing dielectric use.

GE built Puff and burned its own Pittsfield Pyranol and Pyranol from other GE plants and then charged others to burn their PCB oil. According to [a deposition with Monsanto's Papageorge](#) on April 27, 1999, by the middle of 1977, "the amount of material being returned had reduced to the point where it was perceived not to be needed any longer because there were commercial units available ..."

In its "[PCBs: A Report on Uses, Environmental and Health Effects and Disposal](#)," Monsanto offers itself the benefit of every doubt as it explains its last years in the PCB business:

At least as late as a 1975 press conference, the EPA reiterated it would not ask Monsanto to stop production because that would shut down the electrical power industry and the railroads in short order. The EPA objective was to find a way to proceed without shutting down the country. That was precisely the objective to which Monsanto had been committed since 1970 ...

Because PCBs were reported in the environment and tests indicated they could accumulate in the food chain, Monsanto voluntarily decided to terminate sales of chlorinated biphenyls to open applications — those which could result in losses to the environment. Plasticizer and carbonless paper applications fell into this category ... High operating pressures cause hydraulic systems to leak. Monsanto therefore alerted users to manage wastes to eliminate PCB discharge to the environment. As new hydraulic fluids were developed in 1971, we terminated sales of those containing PCBs ...

*Although a variety of effects of PCBs has been postulated from research of widely varying quality, **there is no evidence of environmental levels of PCBs being a major human health hazard.** However, it became apparent that the public perceived risks associated with continued PCB dielectric use and preferred to forego its undoubted functional benefits. Therefore, in October of 1976, we advised dielectric customers that it was our intent, consistent with their progress with substitute products, to terminate chlorinated biphenyl production and sale by October, 1977 ... Monsanto's decision to withdraw from the PCB business was based on concerns about environmental presence rather than health effects ... (Emphasis added.)*

Monsanto, all those years ago, seemed to have come to the conclusion that Mother Earth couldn't sue them, that the shrimp, oysters and eagles wouldn't ever be able to hold the company accountable. I am, I must remind you, a journalist and a story-teller, not an attorney, but I wonder if an indemnification agreement signed by a party ignorant of all the relevant facts still prevails. Or might GE have a case that Monsanto hadn't been completely forthcoming about what it had learned at Escambia, about what would happen if/when its Aroclors had left GE's factories and made it to the Hudson and Housatonic Rivers. Would GE have agreed to indemnification if it had known Monsanto's Aroclors could move through the water column then gather in the bank soils and river sediments and with modest amounts poison fish, mink, eagles and ducks?

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In December 1996, EPA issued a Unilateral Administrative Order under the Superfund law to GE to remove highly contaminated sediment and bank soil in the area adjacent to GE's Building 68, which is on the banks of the River in the Upper ½ Mile Reach ... in 1997 and 1998 GE excavated and disposed of 5,000 cubic yards of heavily contaminated sediment (average PCB concentration of approximately 1,534 parts per million, or 'ppm') from a 550-foot section of the river and 2,230 cubic yards of heavily contaminated bank soil (average concentration of surficial soil of 720 ppm and average concentration of subsurface soil of 5,896 ppm) from a 170-foot stretch of the river bank.

At Enclambia, researchers found contamination levels of 78.0 parts per million. Here in Pittsfield, the Building 68 site had average levels of 1,534 ppm.

Having sat across from and engaged with some of them, I know how very talented GE's attorneys are. Perhaps because it was Monsanto's Aroclors that settled in the sediments and bank soils, they might argue that Monsanto reimburse GE for some of the many hundreds of millions of dollars the company has already spent remediating the first two miles of the Housatonic and for what [the EPA estimates will be \\$576 million](#) to clean up the next installment of 1.13 million cubic yards of [contaminated soil and sediment from the Rest of River](#). Couldn't GE divert some of that money to underwrite the costs of shipping Housatonic River PCBs out of state?

As for us, the ordinary citizens of Berkshire County, there is a growing understanding of PCB exposure. As the World Health Organizations' 2016 "[Assessment of Polychlorinated Biphenyls and Polybrominated Biphenyls](#)" explains:

*The reason that PCB and PBB mixtures in the environment today differ from the original commercial products is that after release into the environment, the congener composition changes through partitioning, chemical transformation, and bioaccumulation. **Partitioning refers to processes by which different congeners separate into air, water, sediment, and soil. Some congeners tend to volatilize or disperse as aerosols, providing an effective vehicle for long-range transport. Congeners with low chlorine or bromine content tend to be more volatile, and also somewhat soluble in water.*** (Emphasis added.)

And, in fact, previous epidemiological studies of PCB exposure focused on workers "exposed to the 'fresh' product, by inhalation or dermal contact, while studies in the general population assessed individuals exposed primarily through intake of contaminated food, for which the exposure profile is difficult to assess." But as time went on, we have learned so much more about bioaccumulation and volatilization.

Today, and this is particularly relevant to those living near the Housatonic River and other PCB-contaminated waterways in the Commonwealth, "exposure is to complex mixtures originating from commercial products that have been altered by environmental processes (i.e. weathering, transport, and bioaccumulation)."

And WHO notes: "[Harrad et al. \(2006\)](#) have suggested that inhalation may account for 4-63 [percent] (median, 15 [percent]) of overall exposure in humans." (Emphasis added.)

Dr. David Carpenter of the Institute for Health and the Environment at the University of Albany was an invited specialist consultant for the WHO Assessment and testified as well for the plaintiffs in the Monsanto Anniston, Ala. cases. Dr. Carpenter's recent research has focused on the volatilization of PCBs and the marked increase in disease shown in those who live near PCB-contaminated waterways like the Hudson River and toxic-waste sites.

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In previous studies, PCB exposure in Mohawk adults has been associated with an increased risk of thyroid disease, reproductive abnormalities in both men and women, diabetes, hypertension, obesity, hyperlipidemia, and metabolic syndrome [9]. While earlier studies monitored total PCBs in blood, more recent studies investigated the effects of different PCB congener groups. This is important because high-chlorinated congeners are more persistent in both the environment and the human body, and their primary route of exposure is from the ingestion of animal fats, especially contaminated fish [10]. However, lower chlorinated congeners are more easily metabolized and, therefore, are less persistent and more volatile, leading to inhalation as a major route of exposure. We found that for some diseases, such as diabetes [11] and the abnormalities of menstruation and ovulation [12,13], the biological effects were primarily due to the low-chlorinated congeners. However, for other outcomes, such as hyperlipidemia and obesity, high-chlorinated congeners are more strongly associated, implicating ingestion as the major route of exposure [9].

PCB exposure also adversely affects cognitive function. Mohawk adolescents at Akwesasne were studied in an investigation of the adverse effects of PCB exposure on cognition, and it was found that the elevated concentrations of total PCBs were negatively associated with delayed recall, long-term retrieval, and comprehension knowledge scores [14]. Dioxin-like, non-dioxin-like, persistent, and low-persistent PCB congener groupings were all associated with reduced long-term memory [15]. Auditory processing was associated only with the persistent congener group, while the non-persistent congener group was associated with the scores of delayed recall, long-term retrieval, and comprehension knowledge [15]. Therefore, Mohawk adolescents who were highly exposed to PCBs showed significant detriments to cognitive skills.

Excerpt from Dr. Carpenter's "Study of PCBs and their Effect on Cognitive Function in Mohawk Adults at Akwesasne." Highlighting added.

Remember Monsanto's surprise in the late 1960s when their newly sophisticated testing regimes revealed that "the composition of that PCB would change, indicating that something was affecting the lower chlorinated PCBs."

Here is another study written by Dr. Carpenter and his colleagues: "[Exocrine pancreatic cancer and living near to waste sites containing hazardous organic chemicals, New York State, USA – an 18-year population-based study.](#)" And here is a selection where [Dr. David Carpenter highlights](#) the health risks of volatilization and its impact on cancer, heart disease, hypertension, and diabetes:

[T]hese 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 ... (Emphasis added.)

Close to home, Dr. Carpenter and his team [conducted indoor air testing](#) coupled with PCB blood testing in Pittsfield:

Pittsfield still has large landfills with PCBs, and air, soil and water still contain high concentrations of PCBs. While most monitoring of PCBs levels in environmental and human samples have focused on the more persistent congeners, our group has increasingly become concerned about vapor phase PCBs and with inhalation of PCBs as being a significant route of exposure. While many of the more volatile congeners are not persistent in the human body, if PCBs are present in air, especially indoor air, the exposure will be continuous and may pose health hazards that are not adequately identified by measurement of more persistent congeners in blood. (Emphasis added.)

Most Berkshire residents are unaware that they and their families may very well have paid—and continue to pay—a

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Let's revisit the troubling results of the 1997 [Housatonic River Area PCB Exposure Assessment Study](#) conducted by the Massachusetts Department of Public Health. A quick note: While PCB levels in fish tissue and river sediments are often measured in parts per million (ppm), PCB levels in human blood is measured in parts per billion (ppb).

Sixty-nine of 1,529 participants agreed to blood testing.

Total serum PCBs, which were classified as Aroclor 1260, ranged from non-detectable to 35.81 ppb, with a mean of 5.44 ppb and a median of 3.93 ppb ... In addition, residents who were not chosen for the study but who were concerned about exposure to PCBs were offered the opportunity to volunteer to participate in a separate effort ...

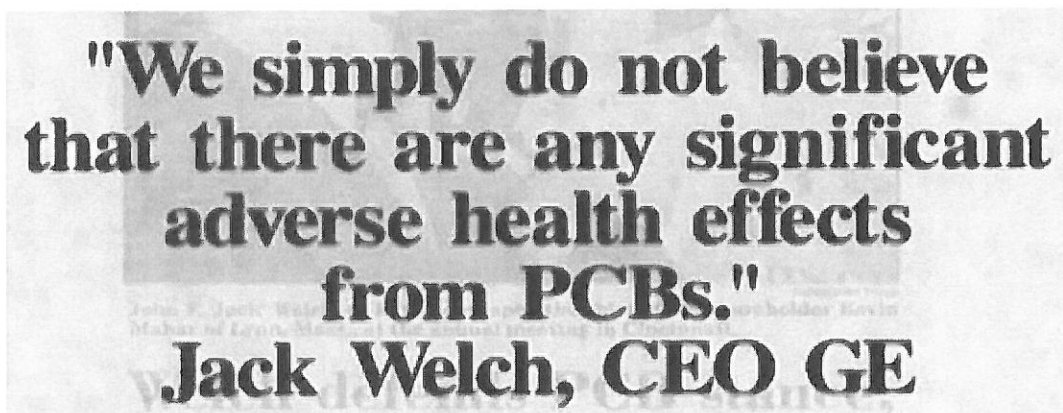
The mean PCB blood level for Pittsfield adults aged 18 to 64 was 4.22 ppb, and for those older than 65, it was 10.56 ppb. The mean PCB blood levels for those aged 18 to 64 living nearby the Housatonic was 3.56 ppb, and for those 65 and older, it was 5.78 ppb.

MDPH stated:

The serum PCB levels found among participants of both studies were generally within typical background estimates for a non-occupationally exposed U.S. population. ATSDR reports that, for U.S. populations without occupational exposure, mean serum PCB levels were usually between 4 and 8 ppb, with 95 [percent] of the individuals having concentrations less than 20 ppb. Since the results of this study represented individuals with the highest risk of exposure, it is reasonable to assume that serum PCB levels of most non-occupationally exposed residents in the HRA communities are within the US background range, though individual differences may likely occur. (Page 2, emphasis added.)

But, in fact, MDPH misstated the national background level for serum PCBs. Soon after the report, James Cogliano, former chief of the EPA's Quantitative Risk Methods Group, told a Pittsfield audience that the accurate figure for the national background level of PCBs in the blood was actually 0.9 to 1.5 ppb. So, in fact, those 18 to 64 years old in Pittsfield had more than three to four times the national background level of PCBs in their blood, while those in Pittsfield 65 and older had more like seven to 10 times the levels of PCBs in their blood. So, contrary to what MDPH and GE were telling them, there were many reasons to be concerned.

This inaccuracy has provided false comfort for too many. And GE took immediate advantage, publishing a series of full-page ads in The Berkshire Eagle, including this patently false assertion by GE CEO Jack Welch that there was absolutely no reason to be concerned about PCBs.



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Recently, a *Berkshire Eagle* article (dated Tuesday, April 25) declared the Allendale School 'safe' from PCBs ... While it is true that the most likely way to become exposed to PCBs is through our food supply (and that probably holds true even for those of us who live next to a PCB contaminated site), those who live or go to school next to a PCB hazardous waste site face an additional threat from airborne PCBs. This may occur from breathing PCBs, both in the form of suspended particles in the air (dust) and as an invisible vapor (volatilized).

Average 'background' PCB air levels in uncontaminated regions are about 1 nanogram/m³. A number of PCB air levels measured at GE's On Plant Consolidation Areas have been substantially higher than that. The EPA claims that PCB air levels less than 50 nanograms/m³ (that is 50 times higher than background) do not expose children to excessive health risk. We do not share their comfort level. We feel that the EPA's risk assessments are based on limited data. This data does not consider the latent health effects of endocrine disruption nor the fact that children will be exposed to additional PCBs through their food. In addition, no safe PCB exposure level has yet been determined for children ... (Emphasis added.)

As for "Sue Monsanto," the issue of volatilization [came to a head in November 2023](#):

Three schoolteachers in Washington state who sued the chemical company Monsanto over exposure to materials in fluorescent lights have been awarded \$185m ... The teachers, who worked at the Sky Valley education center in Monroe, Washington, said they suffered brain damage from exposure to polychlorinated biphenyls, or PCBs, in the fluorescent lighting at the school. 'This is a big step in holding Monsanto accountable,' the teachers' attorney, Rick Friedman, said in a statement.

Law firm [Friedman Rubin](#) wrote:

... the Washington State jury found Pharmacia LLC liable for selling polychlorinated biphenyls (PCBs) without providing adequate warnings, resulting in neurological injuries, endocrine disruption and cognitive impairment to the plaintiffs, which included five teachers, a librarian and a custodian. All seven plaintiffs recovered damages including two of the teachers who developed rare cancers ... The verdict comes after nearly two months of trial and two weeks of deliberations. Of the \$165 million verdict, \$49.8 million is for compensatory damages, while \$115.3 million is for punitive damages.

They noted that in July "Friedman Rubin and PCVA were instrumental in securing a \$72 million verdict on behalf of another set of former Sky Valley employees who also sustained life-long injuries from PCB exposure at the school."

According to [Courtroom View Network](#):

The teachers blamed their health problems, which include severe fatigue and cognitive difficulties, on exposure to PCBs from 2011 to 2015. They claim Monsanto, the sole company to manufacture PCBs, did so despite knowing they posed a risk to the public prior to being banned in the 1970's [sic].

As for other successful legal actions against Monsanto, the state of Washington [filed a complaint in 2016](#) alleging that there was a long list "of water bodies within the state of Washington that are contaminated with Monsanto's PCBs, as measured in fish tissue or sediment PCBs in the environment."

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3. Monsanto Company was the sole manufacturer of PCBs in the United States from 1935 to 1979, and trademarked the name "Aroclor" for certain PCB compounds. Although Monsanto knew for decades that PCBs were toxic and knew that they were widely contaminating all natural resources and living organisms, Monsanto concealed these facts and continued producing PCBs until Congress enacted the Toxic Substances Control Act ("TSCA"), which banned the manufacture of PCBs by January 1, 1979.

4. PCBs were used in many industrial and commercial applications such as paint, caulking, transformers, capacitors, coolants, hydraulic fluids, plasticizers, sealants, inks, lubricants, and other uses. PCBs regularly leach, leak, off-gas, and escape their intended applications, contaminating runoff during naturally occurring storm and rain events.

5. As a result, PCBs contaminate waterways, waterbodies, sediment, fish, and other land, water, and wildlife throughout the state of Washington. PCBs bioaccumulate in fish and humans, causing PCB levels hazardous to human health.

State of Washington v. Monsanto. Highlighting added.

The complaint continued to list the many waterways, large and small, whose fish and river sediment are now contaminated by Monsanto's PCBs.

8. Monsanto's PCBs have caused and will continue to cause direct injury to Washington's public natural resources.

9. The State of Washington has incurred significant costs to identify and reduce sources of Monsanto's PCBs entering and contaminating public natural resources. The State has also incurred significant costs towards monitoring, investigation, analysis, and remediation of Monsanto's PCBs in the environment. The State's residents have borne costs of treating and managing PCB-contaminated water.

10. The State incurs and will continue to incur significant costs into the future due to the presence of Monsanto's PCBs.

State of Washington v. Monsanto. Highlighting added.

The following claims of Washington are particularly relevant to the contamination of the Housatonic River, along with the extensive lands owned by the Commonwealth, including the 874 acres of the George Darey Housatonic Valley Wildlife Management Area that borders the Housatonic, and our interests in protecting the Housatonic ACEC:

16. Monsanto's PCB contamination constitutes injury to the State's public natural resources and to other property and waters of the State, for which the State seeks damages, including on behalf of itself and on behalf of its residents in its parens patriae capacity.

17. The State has a quasi-sovereign interest in and trustee obligation to protect the State's public natural

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18. *The State has a proprietary interest in protecting all property owned by the State and has an interest in remediating the contamination of its exclusive property and in preventing future contamination ...*

20. *Injury to public natural resources caused by Monsanto's PCBs has resulted in loss of public use and enjoyment of those resources. The economic value of these natural resources, as well as the cost of restoring them, is substantial.*

Washington explains: "The State brings this suit pursuant to RCW 7.48.010, et seq. and any other applicable codes or forms of relief available for monetary damages and removal of the public nuisance caused by Monsanto's PCBs."

Washington also makes clear that as "trustee of certain public natural resources, including certain lands, aquatic lands, wildlife, and state waters within state boundaries including but not limited to 3 million acres of state trust lands and 2.6 million acres of state-owned aquatic lands in public trust," they have "standing to bring this lawsuit as trustee of all aforementioned public natural resources."

As the state of Washington maintained, so too is our Commonwealth's property contaminated by Monsanto's PCBs and located throughout the state, including in Berkshire County. And yes, Monsanto's products containing PCBs were sold and used in Berkshire County.

Utilizing some of the internal memos I have referred to previously, the state of Washington asserted that Monsanto's interest was not in adequately addressing the ramifications of its careless distribution of PCBs but in maintaining the market they had come to rely upon. On June 24, 2020, [the state of Washington announced](#) that they had won a judgment of \$95 million dollars.

In January 2018, [the state of Oregon sued Monsanto](#) for damages related to PCB contamination of Oregon's land, waters, fish, and wildlife. They stated:

Between 1929 and 1977, Monsanto was the only company in the United States to manufacture PCBs for widespread commercial use. Monsanto distributed PCBs widely, including throughout Oregon, for use in a broad array of products ranging from electrical equipment to lighting ballasts, from paint to caulking.

[Oregon Attorney General Ellen Rosenblum won](#) "a historic \$698 million dollar settlement with the Monsanto Company (Monsanto) for their role in polluting Oregon with PCBs (polychlorinated biphenyls) for the past 90 + years ..." The statement on the Oregon Department of Justice's website reads:

'This is a huge win for our state,' said AG Rosenblum. 'PCBs are still present throughout Oregon — especially in our landfills and riverbeds — and they are exceedingly difficult to remove, because they 'bioaccumulate' in fish and wildlife.

You can read the Consent Judgment [here](#).

[Pennsylvania filed suit in December 30, 2021](#) claiming:

Despite that Monsanto knew early on of dangers associated with PCBs, and/or knew or should have known that PCBs 'substantially persist in the natural environment rather than break down over time'; ... that they 'would inevitably volatilize and leach, leak, and escape their intended applications, contaminating runoff during naturally

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soils, and plants, as well as fish and other wildlife'; and 'that PCBs bio-accumulate and bio-magnify in animal tissue, including in fish tissue and human tissue, and [pose] an increasingly hazardous threat to the health of the Commonwealth's residents ... Monsanto nevertheless continued to market and sell its products containing PCBs ...'

As the [Pennsylvania Pressroom reports](#):

The Shapiro Administration has secured \$100 million through a consent agreement with the Monsanto Company, Solutia INC., and Pharmacia LLC to resolve claims related to their production of products containing polychlorinated biphenyls (PCBs), which damaged waterways and other natural resources across Pennsylvania ...

In Virginia, on September 12, 2023, [WRIC-TV reported](#), "Attorney General Miyares has announced an \$80 million settlement with Monsanto ..."

On June 16, 2023, [the state of Vermont sued Monsanto](#), with the legal help of Attorney Matthew Pawa, who represented our Rest of River Committee and managed to give the U.S. Court of Appeals for the First Circuit quite the false impression that the citizens of Lee still supported the confidential agreement he negotiated. Having worked hard to deny the Housatonic River Initiative's desire for a more extensive cleanup and to pressure GE and the EPA to opt for transporting PCB-contaminated waste off site rather than burden Lee with a massive PCB dump, Pawa now seems to be as interested in helping Vermont as he was in thwarting Lee.

Vermont's suit claims:

All ten sections of Lake Champlain and the entire 7-mile reach of the Hoosic River is considered impaired for PCBs on Vermont's most current 303(d) Impaired Waters List. Because of this, Vermont has a fish consumption advisory for all of Lake Champlain and the Hoosic River. The accumulation of PCBs in natural resources, and fish in particular, poses a public health threat to the citizens of Vermont ...

Monsanto never advised the State or the public that Old Monsanto's PCB mixtures or products would inevitably leach, leak, off-gas, emit, discharge, and release PCBs from their ordinary and intended applications and from disposal sites, regardless of the nature of the application, to contaminate Vermont's waters, sediments, soils, lands, air, fish, and wildlife. Monsanto issued no public warning or instruction about such issues or the health and environmental hazards presented and, indeed, as alleged above, denied that such hazards exist in their communications with public entities and the public more generally ...

Causes of Action – Count 1 – Public Nuisance: Defendants or their predecessors intentionally designed, manufactured, distributed, marketed, and sold PCBs and PCB-containing products with the knowledge that they inevitably and foreseeably caused or created environmental contamination, indoor air contamination, property damage, and unreasonable health risks when used as intended ...

Defendants' or their predecessors' conduct causes and continues to cause harm to Plaintiff. Plaintiff has suffered and will continue to suffer damage from Defendants' PCBs and PCB-containing products. This harm is severe and greater than Plaintiff should be required to bear without compensation ...

Defendants are under a continuing duty to act to correct and remediate the injuries their conduct, or that of their predecessors, has introduced, and to warn Plaintiff and the public about the human health risks posed by their

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Attorney General Andrea Joy Campbell's Office is an advocate and resource for the people of Massachusetts in many ways, including protecting consumers, combating fraud and corruption, investigating and prosecuting crime, and protecting the environment, workers, and civil rights.

The citizens of Berkshire County have suffered for decades from the irresponsible misuse of PCBs. These PCBs were manufactured by Monsanto and then irresponsibly used by GE. Monsanto and GE lied about the ongoing threats to public health and the environment posed by their toxic Aroclors. Clearly, both GE and Monsanto share responsibility for our PCB contamination.

Meanwhile, many citizens of Berkshire County regard the revised CERCLA order as insufficient. GE will save \$200 million in transportation costs with the decision to force Lee to host the UDF, a massive PCB landfill. Now, in addition to the ongoing public nuisance posed by the continuing dislocation of the proposed remediation, and with constant volatilization of the remaining PCBs, we will all suffer again because of GE's insistence on transporting PCB-contaminated soils and sediments through our towns and city streets by truck to the UDF. These realities will continue to present a continuing and imminent danger to our health. A continuing public nuisance, an environmental trespass.

While many of us in Berkshire County might not have fully appreciated the price we were paying all the years GE was misusing Monsanto's Aroclors, we know now, thanks to studies by Dr. David Carpenter, that we suffer and will suffer from a continuing imminent danger.

Is it too much to imagine that the attorney general—taking into account all we have learned about the continuing public health threat of PCBs from the Housatonic—will act on our behalf.

Is it too much to imagine that the attorney general will act to enforce the following provision of [Mass Law Chapter 21E](#) and its provision, Section 142A, Pollution or contamination of atmosphere; prevention; regulations; violation; enforcement:

"Whoever violates any such regulation or any permit or plan approval or order issued thereunder: (a) shall be punished for each violation by a fine of not more than twenty-five thousand dollars, or by imprisonment for not more than one year, or both such fine and imprisonment; or (b) shall be subject to a civil penalty of not more than twenty-five thousand dollars for each violation. Each day or part thereof that such violation occurs or continues shall be a separate violation. The civil penalty may be assessed in an action brought on behalf of the commonwealth in the superior court. The commonwealth may also bring an action for injunctive relief in the superior court for any such violation, and the superior court shall have jurisdiction to enjoin such violation and to grant such further relief as it may deem appropriate.

Certainly, the residents of Lee will suffer the greatest nuisance as a result of Monsanto's negligence as their property values decline at the very same time their public health is threatened more than most.

I have attended dozens and dozens of public meetings beginning in the early 1980s with the Massachusetts Department of Environmental Quality Engineering (DEQE), then its successor agency, the Massachusetts Department of Environmental Protection (DEP), and, of course, the EPA, and I have never heard them address Monsanto's significant responsibility. These agencies spent decades negotiating with GE but not Monsanto. And while GE is removing a portion of the PCBs that contaminated the Housatonic River, too many will remain. As GE acts to fulfill its

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I have read too many words, written too many. For me, there is always the reality of a poisoned Rising Pond, but the enduring dream of a fishable, swimmable river.

Isn't it time for Massachusetts Attorney General Andrea Joy Campbell and her staff of fine lawyers to follow the lead of other states? Isn't it time for the Commonwealth to Sue Monsanto and Save Lee?

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EXHIBIT C

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 subspecialties 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 Adjudicatory 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 versus 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 chlorine that volatilize 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 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 volatilize, 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 volatilize 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 1×10^{-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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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.

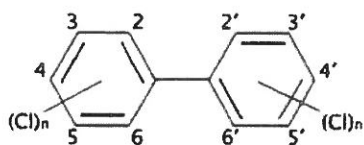


Figure 1: The structure of PCBs. There can be any number of chlorines around the biphenyl ring between one and ten. The convention for labelling the position is shown by the numbers, where the 2 and 6 positions are *ortho*, the 3 and 5 positions are *meta*, and the 4 position is *para*. The prime sign 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/m ³	Log octanol/Water coefficient	Evaporation rate, g/m ³ /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 volatilize. 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 volatilize 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

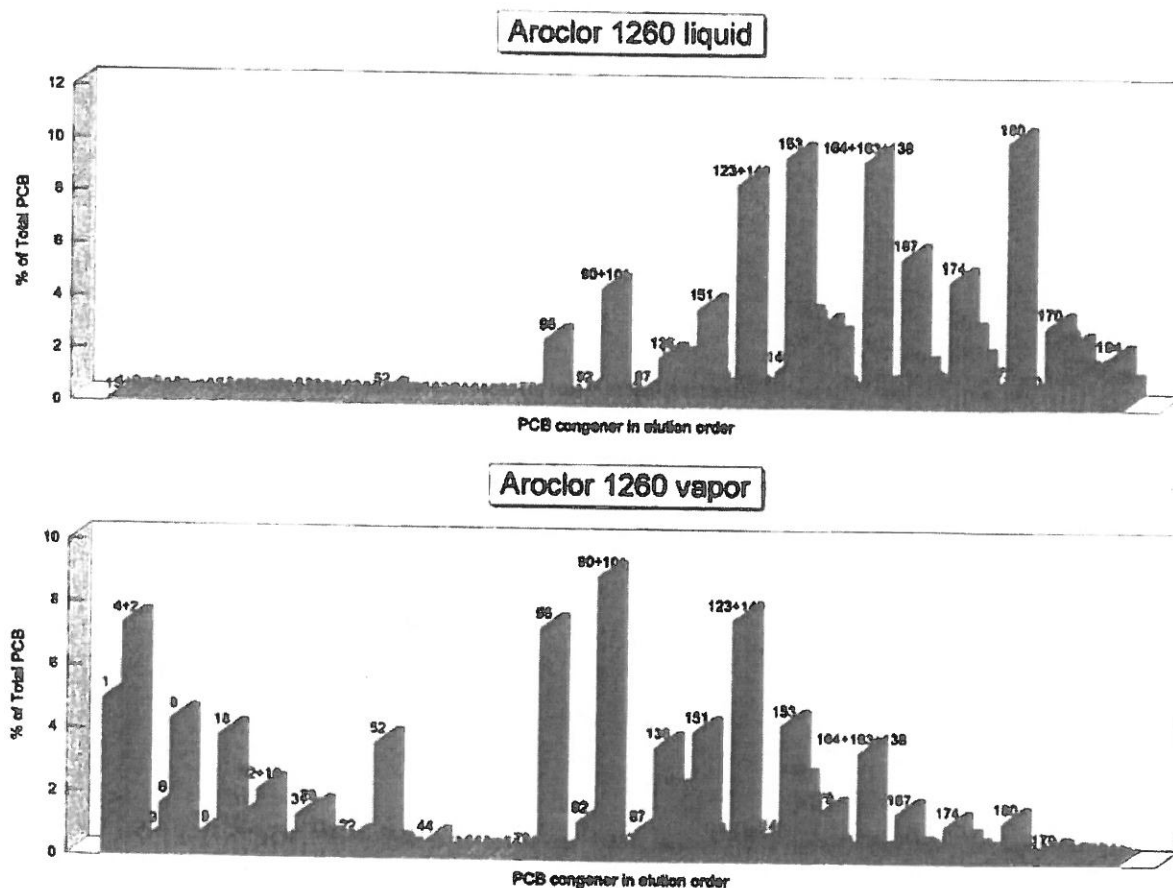


Figure 2: The congener patterns in Aroclor 1260 liquid (top) and the congener pattern seen when passing air over the liquid and collecting and analyzing the vapor-phase PCBs. Peaks are shown in the order they elute from the column. The numbers above the peaks identify individual congeners or groups of 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' Hexachlorobiphenyl	10.8
153	2,2',4,4',5,5' Hexachlorobiphenyl	14.4
170	2,2',3,3',4,4',5 Heptachlorobiphenyl	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

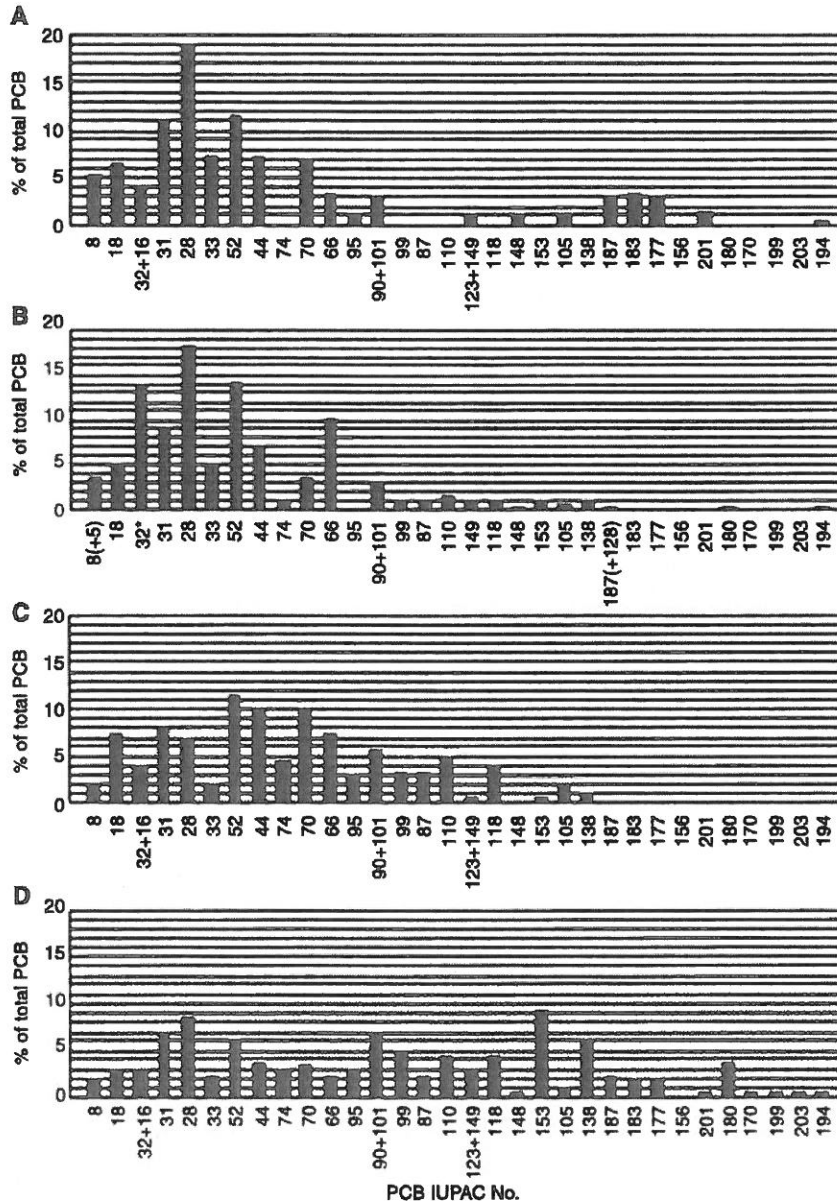


Figure 3: Congener compositions of (A) End-member (EM)-1 as determined by polytopic vector analysis (PVA) of serum PCB congener data for 702 adult Mohawks, (B) air sampled above "Contaminant Cove" at the western boundary of Akwesasne in summer 1993,²⁷ (C) native commercial A1248 liquid, and (D) serum 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 in the 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 mCi ^{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.

There are several remarkable findings in this tragic story. For two persons who are not blood relatives to

Table 3: PCB concentrations ($\mu\text{g}/\text{kg}$ 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.

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 lower 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 or 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.

References

- Hansen LG. The ortho side of PCBs: occurrence and disposition. Boston, MA: Kluwer Academic Publishers, 1999.
- Carpenter DO, Welfinger-Smith G. The Hudson River: a case study of PCB contamination. In: Selendy JM, editor. Water and sanitation-related diseases and the environment: challenges, interventions and preventive measures. Hoboken, NJ: Wiley-Blackwell, 2011:303–27.
- Erickson MD. Introduction: PCB properties, uses, occurrence, and regulatory history. In: Robertson LW, Hansen LG, editors. PCBs: recent advances in environmental toxicology and health effects. Lexington, Kentucky: The University Press of Kentucky, 2001:xi–xxx.
- Chiarenzelli J, Scudato R, Arnold G, Wunderlich M, Rafferty D. Volatilization of polychlorinated biphenyls from sediment during drying at ambient conditions. *Chemosphere* 1996;33:899–911.
- Chiarenzelli J, Scudato RJ, Wunderlich ML. Volatile loss of PCB aroclors from subaqueous sand. *Environ Sci Technol* 1997;31:597–602.
- Chiarenzelli J, Bush B, Casey A, Barnard E, Smith B, et al. Defining the sources of airborne polychlorinated biphenyls: evidence for the influence of microbially dechlorinated congeners from river sediment? *Can J Fish Aquat Sci* 2000;57:86–94.
- Jeremiason JD, Hornbuckle KC, Eisenreich SJ. PCBs in Lake Superior, 1978–1992: decreases in water concentrations reflect loss by volatilization. *Environ Sci Technol* 1994;28:903–14.
- Vorhees DJ, Cullen AC, Altshul LM. Exposure to polychlorinated biphenyls in residential indoor air and outdoor air near a superfund site. *Environ Sci Technol* 1997;31:3612–8.
- Sandy AL, Guo J, Miskewitz RJ, McGillis WR, Rodenburg LA. Fluxes of polychlorinated biphenyls volatilizing from the Hudson River, New York measured using micrometeorological approaches. *Environ Sci Technol* 2012;46:885–91.
- Bremle G, Larsson P. PCB in the air during landfilling of a contaminated lake sediment. *Atmos Environ* 1998;32:1011–9.
- Hermanson MH, Scholten CA, Compher K. Variable air temperature response of gas-phase atmospheric polychlorinated biphenyls near a former manufacturing facility. *Environ Sci Technol* 2003;37:4038–42.
- Ritter R, Scheringer M, MacLeod M, Moeckel C, Jones KC, et al. Intrinsic human elimination half-lives of polychlorinated biphenyls derived from the temporal evolution of cross-sectional biomonitoring data from the United Kingdom. *Environ Health Perspect* 2011;119:225–31.
- James MO. Polychlorinated biphenyls: metabolism and metabolites. In: Robertson LW, Hansen LG, editors. PCBs: recent advances in environmental toxicology and health effects. Lexington, KY: The University Press of Kentucky, 2001:35–46.
- Hu X, Adamcakova-Dodd A, Lehmler H-J, Hu D, Kania-Korwell I, et al. Time course of congener uptake and elimination in rats after short-term inhalation exposure to an airborne polychlorinated biphenyl (PCB) mixture. *Environ Sci Technol* 2010;44:6893–900.
- Hu X, Adamcakova-Dodd A, Thorne PS. The fate of inhaled ¹⁴C-labeled PCB11 and its metabolites in vivo. *Environ Int* 2004;63:92–100.
- Letcher RJ, Klasson-Wehler E, Bergman Å. Methyl sulfone and hydroxylated metabolites of polychlorinated biphenyls. In: Paasivirta J, editor. The handbook of environmental chemistry Vol. 3 Part K. Berlin Heidelberg: Springer Verlag, 2000.
- Matthews HB, Tvey DB. The effect of chlorine position on the distribution and excretion of four hexachlorobiphenyl isomers. *Toxicol Appl Pharmacol* 1980;53:377–88.
- Johnson CD, Balagurunatham Y, Tadesse MG, Fatahatpisheh MH, Brun M, et al. Unravelling gene-gene interactions regulated by ligands of the aryl hydrocarbon receptor. *Environ Health Perspect* 2004;112:403–12.
- Vezina CM, Walker NJ, Olson JR. Subchronic exposure to TCDD, PeCDF, PCB126, and PCB153: effect on hepatic gene expression. *Environ Health Perspect* 2004;112:1636–44.
- Maier MSV, Legare ME, Hanneman WH. The aryl hydrocarbon receptor agonist 3,3',4,4',5-pentachlorobiphenyl induces distinct patterns of gene expression between hepatoma and glioma cells: chromatin remodeling as a mechanism for selective effects. *NeuroToxicology* 2007;28:594–612.
- DeCaprio AP, Johnson GW, Tarbell AM, Carpenter DO, Chiarenzelli JR, et al. Polychlorinated biphenyls (PCB) exposure assessment by multivariate statistical analysis of serum congener profiles in an adult Native American population. *Environ Res* 2005;98:284–302.
- Herrick RF, Meeker JD, Altshul L. Serum PCB and congener profiles among teachers in PCB-containing schools: a pilot study. *BMC Environ Health* 2011;10: 56. Available at: <http://www.ehjournal.net/content/10/1/56>.
- Meyer HW, Frederiksen M, Göen T, Ebbehøj NE, Gunnarsen L, et al. Plasma polychlorinated biphenyls in residents of 91 PCB-contaminated and 108 non-contaminated dwellings – an exposure study. *Int J Hyg Environ Health* 2013;216:755–62.
- Lauby-Secretan B, Loomis D, Grosse Y, Grosse Y, El Ghissassi F, et al. Carcinogenicity of polychlorinated biphenyls and polybrominated biphenyls. *Lancet Oncol* 2013;14:287. doi: 10.1016/S1470-2045(13)70104-9.

25. Carpenter DO. Polychlorinated biphenyls (PCBs): routes of exposure and effects on human health. *Rev Environ Health* 2006;21:1–24.
26. van der Plas SA, Sundberg H, van den Berg H, Scheu G, Wester P, et al. Contribution of planar (0-1 ortho) and nonplanar (2-4 ortho) fractions of aroclor 1260 to the induction of altered hepatic foci in female Sprague-Dawley rats. *Toxicol Appl Pharmacol* 2000;169:255–68.
27. Sandal S, Yilmaz B, Carpenter DO. Genotoxic effects of PCB 52 and PCB 77 on cultured human peripheral lymphocytes. *Mutation Res* 2008;654:88–92.
28. Yilmaz B, Sandal S, Carpenter DO. PCB 9 exposure induces endothelial cell death while increasing intracellular calcium and ROS levels. *Environ Toxicol* 2010;27:185–91.
29. Zhu Y, Mapuskar KA, Marek RF, Xu W, Lehmler HJ, et al. A new player in environmentally induced oxidative stress: polychlorinated biphenyl congener, 3,3'-dichlorobiphenyl (PCB11). *Toxicol Sci* 2013;136:39–50.
30. Ludewig G, Lehmann L, Esch H, Robertson LW. Metabolic activation of PCBs to carcinogens in vivo – a review. *Environ Toxicol Pharmacol* 2008;25:241–6.
31. Tan Y, Chen C-H, Lawrence D, Carpenter DO. Ortho-substituted PCBs kill cells by altering membrane structure. *Toxicol Sci* 2004;80:54–9.
32. Mayes BA, McConnell EE, Neal BH, Brunner MJ, Hamilton SB, et al. Comparative carcinogenicity in Sprague-Dawley rats of the polychlorinated biphenyl mixtures Aroclors 1016, 1242, 1254, and 1260. *Toxicol Sci* 1998;41:62–76.
33. Vansell NR, Muppidi JR, Habeebu SM, Klaassen CD. Promotion of thyroid tumors in rats by pregnenolone-16 α -carbonitrile (PCN) and polychlorinated biphenyl (PCB). *Toxicol Sci* 2004;81:50–9.
34. Greenland S, Salvan A, Wegman DH, Hallock MF, Smith TJ. A case-control study of cancer mortality at a transformer-assembly facility. *Int Arch Occup Environ Health* 1994;66:49–54.
35. Nakanishi Y, Bai F, Takayama K, Pei XH, Inoue K, et al. Effect of PCBs on mouse lung tumorigenesis induced by 1-nitropyrene: a preliminary report. *Fukuoka igaku zasshi = Kukuoka acta medica* 1999;90:231–7.
36. Robertson LW, Ludewig G. Polychlorinated biphenyl (PCB) carcinogenicity with special emphasis on airborne PCBs. *Gefahrst Reinhalt Luft* 2011;71:25–32.
37. Maddox C, Wang B, Kirby PA, Wang K, Ludewig G. Mutagenicity of 3-methylcholantrene, PCB3, and 4-OH-PCB3 in the lung of transgenic BigBlue® rats. *Environ Toxicol Pharmacol* 2008;25:260–6.
38. Xie W, Wang K, Robertson LW, Ludewig G. Investigation of mechanism(s) of DNA damage induced by 4-monochlorobiphenyl (PCB3) metabolites. *Environ Int* 2010;36:950–61.
39. Sergeev AV, Carpenter DO. Hospitalization rates for coronary heart disease in relation to residence near areas contaminated with persistent organic pollutants and other pollutants. *Environ Health Perspect* 2005;113:756–61.
40. Shcherbatykh I, Huang X, Lessner L, Carpenter DO. Hazardous waste sites and stroke in New York State. *BMC Environ Health* 2005;4:18. Available at: <http://www.jstor.org/stable/4133069>.
41. Goncharov A, Haase RF, Santiago-Rivera A, Morse G, Akwesasne Task Force on the Environment, McCaffrey RJ, et al. High serum PCBs are associated with elevation of serum lipids and cardiovascular disease in a Native American population. *Environ Res* 2008;106:226–39.
42. Aminov Z, Haase RF, Pavuk M, Carpenter DO, Anniston Environmental Health Research Consortium. Analysis of the effects of exposure to polychlorinated biphenyls and chlorinated pesticides on serum lipid levels in residents of Anniston, Alabama. *BMC Environ Health* 2013;12:108. Available at: <http://www.ehjournal.net/content/12/1/108>.
43. Hennig B, Meerarani P, Slim R, Toborek M, Daugherty A, et al. Proinflammatory properties of coplanar PCBs: in vitro and in vivo evidence. *Toxicol Appl Pharmacol* 2002;181:174–83.
44. Ha M-H, Lee D-H, Jacobs DR Jr. Association between serum concentrations of persistent organic pollutants and self-reported cardiovascular disease prevalence: results from the national health and nutrition examination survey, 1999–2002. *Environ Health Perspect* 2007;115:1204–9.
45. Huang X, Lessner L, Carpenter DO. Exposure to persistent organic pollutants and hypertensive disease. *Environ Res* 2006;102:101–6.
46. Goncharov A, Bloom M, Pavuk M, Birman I, Carpenter DO. Blood pressure and hypertension in relation to levels of serum polychlorinated biphenyls in residents of Anniston, Alabama. *J Hypertens* 2010;28:2053–60.
47. Goncharov A, Pavuk M, Foushee HR, Carpenter DO. Blood pressure in relation to concentrations of PCB congeners and chlorinated pesticides. *Environ Health Perspect* 2011;119:319–25.
48. Everett CJ, Mainous AG III, Frithsen IL, Player MS, Matheson EM. Association of polychlorinated biphenyls with hypertension in the 1999–2002 national health and nutrition examination survey. *Environ Res* 2008;108:94–7.
49. Ha M-H, Lee D-H, Son H-K, Park S-K, Jacobs DR Jr. Association between serum concentrations of persistent organic pollutants and prevalence of newly diagnosed hypertension: results from the national health and nutrition examination survey 1999–2002. *J Hum Hypertens* 2009;23:274–86.
50. Kouznetsova M, Huang X, Jing M, Lessner L, Carpenter DO. Increased rate of hospitalization for diabetes and residential proximity of hazardous waste sites. *Environ Health Perspect* 2007;115:75–9.
51. Codru N, Schymura MJ, Negoita S; The Akwesasne Task Force on the Environment, Rej R, Carpenter DO. Diabetes in relation to serum levels of polychlorinated biphenyls and chlorinated pesticides in adult Native Americans. *Environ Health Perspect* 2007;115:1442–7.
52. Aminov Z, Haase R, Rej R, Schymura MJ, Santiago-Rivera A, et al. Low chlorinated, non-dioxin-like polychlorinated biphenyls are strongly associated with elevated prevalence of diabetes in a Native American population. Submitted.
53. Newman J, Aucompaugh AG, Schell LM, Denham M, DeCaprio AP, et al. PCBs and cognitive functioning of Mohawk adolescents. *Neurotoxicol Teratol* 2006;28:439–45.
54. Haase RF, McCaffrey RJ, Santiago-Rivera AL, Morse GS, Tarbell A. Evidence of an age-related threshold effect of polychlorinated biphenyls (PCBs) on neuropsychological functioning in a Native American population. *Environ Res* 2009;109:73–85.
55. Seegal RF. Neurochemical effects of polychlorinated biphenyls: a selective review of the current state of knowledge. In: Robertson LW, Hansen LG, editors. *PCBs: recent advances in environmental toxicology and health effects*. Lexington, KY: The University Press of Kentucky, 2001:241–55.

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, Negolta 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. Balbergenova 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, Pöpke 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 J 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. Ruokojärvi 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, Phillips 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 University, Fukuoka, Japan, for a period of three months each
- 1987
- 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 through 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 Health 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. A Central/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, A Indoor 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, A Cooperative 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. A Multidisciplinary 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 (1R01ES019620), "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 (2R01ES19620-06A1), "Protecting the Health of Future Generations: Assessing and Preventing Exposures to Endocrine Disrupting 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 inhibibility 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 norepinephrine 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. Intl. 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. H₁ and H₂ 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 resistivity 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, 192:808-809, 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. Keababian, P.R., Keababian, 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.
52. Pellmar, T.C. and Carpenter, D.O. Voltage-dependent calcium current induced by serotonin. Nature, 277:483-484, 1979.
53. Ruben, P.C., Swann, J.W. and Carpenter, D.O. Neurotransmitter receptors on gill muscle fibers and the gill peripheral nerve plexus in Aplysia. Canad. J. Physiol. Pharmacol., 57:1088-1097, 1979.
54. Pellmar, T.C. and Carpenter, D.O. Serotonin induces a voltage-sensitive calcium current in neurons of Aplysia californica. J. Neurophysiol., 44:423-439, 1980.
55. Parver, L.M., Auker, C. and Carpenter, D.O. Choroidal blood flow as a heat dissipating mechanism in the macula. Am. J. Ophthalmol., 89:641-646, 1980.
56. Mell, L.D., Jr. and Carpenter, D.O. Fluorometric determination of octopamine in tissue homogenates by high-performance liquid chromatography. Neurochem. Res., 5:1089-1096, 1980.
57. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Thyrotropin-releasing hormone has multiple actions in cortex. Brain Res., 194:244-248, 1980.
58. Meszler, R.M., Auker, C.R. and Carpenter, D.O. Fine structure and organization of the infrared receptor relay, the lateral descending nucleus of the trigeminal nerve in pit vipers. J. Comp. Neurol., 196:571-584, 1981.
59. Auker, C.R., Parver, L.M., Doyle, T. and Carpenter, D.O. Choroidal blood flow: I. Ocular tissue temperature as a measure of flow. Arch. Ophthalmol., 100:1323-1326, 1982.

60. Parver, L.M., Auker, C., Carpenter, D.O. and Doyle, T. Choroidal blood flow: II. Reflexive control in the monkey. Arch. Ophthalmol., 100:1327-1330, 1982.
61. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Lateral olfactory tract transmitter: Glutamate, aspartate or neither? Cell. Mol. Neurobiol., 1:115-120, 1981.
62. Scappaticci, K.A., Dretchen, K.L., Carpenter, D.O. and Pellmar, T.C. Effects of furosemide on neural mechanisms in Aplysia. J. Neurobiol., 12:329-341, 1981.
63. Pellmar, T.C. and Carpenter, D.O. Cyclic AMP induces a voltage-dependent current in neurons of Aplysia californica. Neurosci. Lett., 22:151-157, 1981.
64. Parver, L., Auker, C. and Carpenter, D.O. Stabilization of macular temperature: The stabilizing effect of the choroidal circulation on the temperature environment of the macula. Retina, 2:117-120, 1982.
65. Green, R.W. and Carpenter, D.O. Biphasic responses to acetylcholine in mammalian reticulospinal neurons. Cell. Molec. Neurobiol., 1:401-405, 1981.
66. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Pharmacologic sensitivity of amino acid responses and synaptic activation of in vitro prepyriform neurons. J. Neurophysiol., 48:1289-1301, 1982.
67. Slater, N.T. and Carpenter, D.O. Blockade of acetylcholine-induced inward currents in Aplysia neurons by strychnine and desipramine: effect of membrane potential. Cell. Molec. Neurobiol., 2:53-58, 1982.
68. Swann, J.W., Sinback, C.N., Pierson, M.G. and Carpenter, D.O. Dopamine produces muscle contractions and modulates motoneuron-induced contractions in Aplysia gill. Cell. Molec. Neurobiol., 2:291-308, 1982.
69. Swann, J.W., Sinback, C.N., Kebabian, P.R. and Carpenter, D.O. Motoneurons which may utilize dopamine as their neurotransmitter. Cell. Molec. Neurobiol., 2:309-324, 1982.
70. Auker, C.R., Meszler, R.M. and Carpenter, D.O. Apparent discrepancy between single unit activity and ¹⁴C-deoxyglucose labelling in the optic tectum of the rattlesnake. J. Neurophysiol., 49:1504-1516, 1983.
71. Slater, N.T., Carpenter, D.O., Freedman, J.E. and Snyder, S.H. Vipoxin both activates and antagonizes three types of acetylcholine response in Aplysia neurons. Brain Res., 278:266-270, 1983.
72. French-Mullen, J.M.H., Hori, N., Nakanishi, H., Slater, N.T. and Carpenter, D.O. Assymmetric distribution of acetylcholine receptors and M channels on prepyriform neurons. Cell. Molec. Neurobiol., 3:163-182, 1983.
73. Carpenter, D.O., Briggs, D.B. and Strominger, N. Responses of neurons of canine area postrema to neurotransmitters and peptides. Cell. Molec. Neurobiol., 3:113-126, 1983.
74. Slater, N.T. and Carpenter, D.O. Blocking kinetics at excitatory acetylcholine responses on Aplysia neurons. Biophys. J., 45:24-25, 1984.
75. Chesnut, T.J. and Carpenter, D.O. Two-component desensitization of three types of responses to acetylcholine in Aplysia. Neurosci. Lett., 39:285-290, 1983.
76. Haas, H.L., Jeffreys, J.G.R., Slater, N.T. and Carpenter, D.O. Modulation of low calcium induced field bursts in the hippocampus by monoamines and cholinomimetics. Pflugers Arch., 400:28-33, 1984.
77. Parvar, L.M., Auker, C.R. and Carpenter, D.O. Choroidal blood flow. III. Reflexive control in human eyes. Arch. Ophthalmol., 101:1604-1606, 1983.

78. Slater, N.T., Haas, H.L. and Carpenter, D.O. Kinetics of acetylcholine-activated cation channel blockade by the calcium antagonist D-600 in *Aplysia* neurons. Cell. Molec. Neurobiol., 3:329-344, 1983.
79. McCreery, M.J. and Carpenter, D.O. Modulation of neuronal responses to L-glutamate in *Aplysia*. Cell. Molec. Neurobiol., 4:91-95, 1984.
80. Carpenter, D.O., Briggs, D.B. and Strominger, N. Peptide-induced emesis in dogs. Behav. Brain Res., 11:277-281, 1984.
81. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. N-methyl-D-aspartate and L-aspartate activate distinct receptors in prepyriform cortex. Cell. Molec. Neurobiol., 4:185-189, 1984.
82. Slater, N.T. and Carpenter, D.O. A study of the cholinolytic actions of strychnine using the technique of concentration jump relaxation analysis. Cell Molec Neurobiol 4:263-271,1984.
83. Slater, N.T., Hall, A.F. and Carpenter, D.O. Kinetic properties of cholinergic desensitization in *Aplysia* neurons. Proc. Roy. Soc. Lond. B, 223:63-78, 1984.
84. Akaike, N., Hattori, K., Oomura, Y. and Carpenter, D.O. Bicuculline and picrotoxin block gamma-aminobutyric acid-gated Cl⁻ conductance by different mechanisms. Experientia, 41:70-71, 1985.
85. Slater, N.T., Carpenter, D.O., Freedman, J.E. and Synder, S.H. Dual effects of the snake venom polypeptide vipoxin on receptors for acetylcholine and biogenic amines in *Aplysia* neurons. Neurosci., 14:723-733, 1985.
86. Mizuno, Y., Oomura, Y., Hori, N. and Carpenter, D.O. Action of vasopressin on CA1 pyramidal neurons in rat hippocampal slices. Brain Res., 309:241-246, 1984.
87. Slater, N.T., Hall, A.F. and Carpenter, D.O. Trifluoperazine and calcium antagonists accelerate cholinergic desensitization in *Aplysia* neurons. Brain Res., 329:275-279, 1985.
88. French-Mullen, J.M.H., Koller, K., Zaczek, R., Coyle, J.T., Hori, N. and Carpenter, D.O. N-acetylaspartylglutamate: Possible role as the neurotransmitter of the lateral olfactory tract. Proc. Nat. Acad. Sci., 82:3897-3900, 1985.
89. Greene, R.W. and Carpenter, D.O. Actions of neurotransmitters on pontine medial reticular formation neurons of the cat. J. Neurophysiol., 54:520-531, 1985.
90. Hori, N., French-Mullen, J.M.H. and Carpenter, D.O. Kainic acid responses and toxicity show pronounced Ca²⁺ dependence. Brain Res., 358:380-384, 1985.
91. Gaillard, W.D. and Carpenter, D.O. Spectra of neurotransmitter receptors and ionic responses on cerebral A and B neurons in *Aplysia californica*. Brain Res., 373:303-310, 1986.
92. Gaillard, W.D. and Carpenter, D.O. On the transmitter at the A-to-B cell in *Aplysia californica*. Brain Res., 373:311-315, 1986.
93. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. A comparison on the effects of quinolinate and N-methyl-aspartate on neurons in rat piriform cortex. Neurosci. Lett., 63:66-70, 1986.
94. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. Receptors for the excitatory amino acids on neurons in rat pyriform cortex. J. Neurophysiol., 55:1283-1294, 1986.
95. Slater, N.T., David, J.A. and Carpenter, D.O. Relaxation studies on the interaction of hexamethonium with acetylcholine-receptor channels in *Aplysia* neurons. Cell. Molec. Neurobiol., 6:191-211, 1986.
96. Leung, M.K., S.-Rozsa, K., Hall, A., Kuruvilla, S., Stefano, G.B. and Carpenter, D.O. Enkephalin-like substance in *Aplysia* nervous tissue and actions of leu-enkephalin on single neurons. Life Sci., 38:1529-34, 1986.
97. Slater, N.T., Filbert, M. and Carpenter, D.O. Multiple interactions of anticholinesterases with *Aplysia* acetylcholine responses. Brain Res., 375:407-412, 1986.

98. Carpenter, D.O. and Briggs, D.B. Insulin excites neurons of the area postrema and causes emesis. Neurosci. Lett., 68:85-89, 1986.
99. Carpenter, D.O., Briggs, D.B., Knox, A.P. and Strominger, N.L. Radiation-induced emesis in the dog: Effects of lesions and drugs. Rad. Res., 108:307-316, 1986.
100. Briggs, D.B. and Carpenter, D.O. Excitation of neurons in the canine area postrema by prostaglandins. Cell. Molec. Neurobiol., 6:421-426, 1986.
101. Chesnut, T.J., Carpenter, D.O. and Strichartz, G.R. Three effects of venom from conus striatus on the delayed rectifier potassium current of molluscan neurons. Toxicon, 25:267-278, 1987.
102. Yakushiji, T., Tokutomi, N., Akaike, N. and Carpenter, D.O. Agonists of GABA responses, studied using internally perfused frog dorsal root ganglion neurons. Neuroscience 22:1123-1133, 1987.
103. Akaike, N., Yakushiji, T., Tokutomi, N. and Carpenter, D.C. Multiple mechanisms of antagonism of GABA responses. Cell. Molec. Neurobiol., 7:97-103, 1987.
104. Hori, N., Galeno, T. and Carpenter, D.O. Responses of pyriform cortex neurons to excitatory amino acids: Voltage dependence, conductance changes and effects of divalent cations. Cell. Molec. Neurobiol., 7:73-90, 1987.
105. Oyama, Y., King, W.M. and Carpenter, D.O. Edrophonium-induced membrane current in single neurons physically isolated from Aplysia californica. Brain Res., 438:95-100, 1988.
106. Jahan-Parwar, B., S.-Rozsa, K., Salanki, J., Evans, M.L. and Carpenter, D.O. In vivo labeling of serotonin containing neurons by 5,7-dihydroxytryptamine in Aplysia. Brain Res., 426:173-178, 1987.
107. King, W.M. and Carpenter, D.O. Distinct GABA and glutamate receptors may share a common channel in Aplysia neurons. Neurosci. Lett., 82:343-348, 1987.
108. Carpenter, D.O., Briggs, D.B., Knox, A.P. and Strominger, N. Excitation of area postrema neurons by transmitters, peptides and cyclic nucleotides. J. Neurophysiol., 59:358-369, 1988.
109. Carpenter, D.O., Hall, A.F. and Rahmann, H. Exogenous gangliosides induce direct voltage and conductance changes on isolated neurons. Cell. Molec. Neurobiol., 8:245-250, 1988.
110. Hori, N., Carpenter, D.O. and Katsuda, N. Effect of acetylcholine on the pyramidal cell in the rat piriform cortex in vitro. Neurosciences, 13:172-174, 1987 (in Japanese).
111. Hori, N. and Carpenter, D.O. Excitatory amino acid receptors in piriform cortex do not show receptor desensitization. Brain Res., 457:350-354, 1988.
112. Allen, C.N., Brady, R., Swann, J., Hori, N. and Carpenter, D.O. N-methyl-D-aspartate (NMDA) receptors are inactivated by trypsin. Brain Res., 458:147-150, 1988.
113. Oyama, Y., Akaike, N. and Carpenter, D.O. Strychnine decreases the voltage-dependent Ca²⁺ current of both Aplysia and frog ganglion neurons. Cell. Molec. Neurobiol., 8:307-314, 1988.
114. Oyama, Y., King, W.M., Allen, C.N., Hori, N. and Carpenter, D.O. Characterization of an inward current elicited by edrophonium in physically isolated and internally perfused Aplysia neurons. Brain Res., 463:124-132, 1988.
115. Hori, N., Akaike, N. and Carpenter, D.O. Piriform cortex brain slices: Techniques for isolation of synaptic inputs. J. Neurosci. Methods, 25:197-208, 1988.
116. Oyama, Y., Evans, M.L., Akaike, N. and Carpenter, D.O. Electrophysiological detection of acetylcholinesterase activity using concentration clamp on physically isolated Aplysia neurons. Neuroscience Res., 6:174-180, 1988.
117. Tsuda, Y., Oyama, Y., Carpenter, D.O. and Akaike, N. Effects of Ca²⁺ on the transient outward current of single isolated Helix central neurones. Brit J. Pharmacol., 95:526-530, 1988.

118. Oyama, Y., Hori, N., Evans, M.L., Allen, C.N. and Carpenter, D.O. Electrophysiological estimation of the actions of acetylcholinesterase inhibitors on acetylcholine receptor and cholinesterase in physically isolated *Aplysia* neurones. Brit. J. Pharmacol., 96:573-582, 1989.
119. King, W.M. and Carpenter, D.O. Voltage-clamp characterization of Cl⁻ conductance gated by GABA and L-glutamate in single neurons of *Aplysia*. J. Neurophysiol., 61:892-899, 1989.
120. Evans, M.L. and Carpenter, D.O. Desensitization kinetics of a chloride acetylcholine response in *Aplysia*. Brain Res., 495:309-318, 1989.
121. Salanki, J., Evans, M.L. and Carpenter, D.O. Desensitization kinetics of a K⁺ acetylcholine response in *Aplysia*. Brain Res., 495:298-308, 1989.
122. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Effects of exogenous ganglioside and cholesterol application on excitability of *Aplysia* neurons. Membrane Biochemistry, 8:19-26, 1989.
123. Carpenter, D. Neural mechanisms of emesis. Canad. J. Physiol. Pharmacol., 68:230-236, 1990.
124. Oyama, Y., Hori, N., Allen, C.N., and Carpenter, D.O. Influences of trypsin and collagenase on acetylcholine responses of physically-isolated single neurons of *Aplysia californica*. Cell. Molec. Neurobiol., 10:193-205, 1990.
125. Büsselberg, D., Evans, M.L., Rahmann, H., and Carpenter, D.O. Lead inhibits the voltage-activated calcium current of *Aplysia* neurons. Toxicol. Lett., 51:51-57, 1990.
126. Doi, N., Carpenter, D.O. and Hori, N. Differential effects of baclofen and GABA on rat piriform cortex pyramidal neurons *in vitro*. Cell. Molec. Neurobiol., 10: 559-564, 1991.
127. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Zn²⁺ blocks the voltage activated calcium current of *Aplysia* neurons. Neurosci. Letts., 117:117-122, 1990.
128. Büsselberg, D., Carpenter, D.O., Sugita, M., Araki, S., Satake, M. and Rahmann, H. Effects of exogenous lipid application on excitability of *Aplysia* neurons. Biomed. Res., 11:77-86, 1990.
129. Evans, M.L., Kadan, M.J., Hartig, P.R. and Carpenter, D.O. Correlation of ¹²⁵I-LSD autoradiographic labelling with serotonin voltage clamp responses in *Aplysia* neurones. Synapse, 8:22-29, 1991.
130. S.-Rozsa, K., Stefano, G., Salanki, J. and Carpenter, D.O. Characterization of responses to enkephalins and FMRFamide on B neurons of the cerebral ganglion of *Aplysia*. Comp. Biochem. Physiol., 99C:403-412, 1991.
131. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Lead and zinc block a voltage activated calcium channel of *Aplysia* neurons. J. Neurophysiol., 65:786-795, 1991.
132. Hori, N., Doi, N., Miyahara, S., Shinoda, Y. and Carpenter, D.O. Appearance of NMDA receptors triggered by anoxia independent of voltage *in vivo* and *in vitro*. Exp. Neurol., 112:304-311, 1991.
133. Büsselberg, D., Evans, M.L., Rahmann, H. and Carpenter, D.O. Effects of inorganic and triethyl lead and inorganic mercury on the voltage activated calcium channel of *Aplysia* neurons. NeuroToxicology, 12:733-744, 1991.
134. Evans, M.L., Büsselberg, D. and Carpenter, D.O. Pb²⁺ blocks calcium currents of cultured dorsal root ganglion cells. Neurosci. Letts., 129:103-106, 1991.
135. Kemenes, G., S.-Rozsa, K., Stefano, G. and Carpenter, D.O. Distinct receptors for leu- and met-enkephalin on the metacerebral giant cell of *Aplysia*. Cell. Molec. Neurobiol., 12:107-119, 1992.
136. Ayrapetyan, S.N. and Carpenter, D.O. Very low concentrations of acetylcholine and GABA modulate transmitter responses. NeuroReport 2:563-565, 1991.
137. Carpenter, D.O. and Hori, N. Neurotransmitter and peptide receptors on medial vestibular nucleus neurons. Ann. NY Acad. Sci., 656:668-686, 1992.

138. Hernadi, L., S.-Rozsa, K., Jahan-Parwar, B. and Carpenter, D.O. A topography and ultrastructural characterization of *in vivo* 5,7-dihydroxytryptamine-labelled serotonin-containing neurons in the central nervous system of *Aplysia californica*. Cell. Molec. Neurobiol., 12:317-326, 1992.
139. Carpenter, D.O., Fejtli, M., Ayrapetyan, S., Szarowski, D. and Turner, J.N. Dynamic changes in neuronal volume resulting from osmotic and sodium transport manipulations. Acta Biologica Hungarica, 43:39-48, 1992.
140. Ayrapetyan, S.N. and Carpenter, D.O. On the modulating effect of ultralow transmitter concentrations on the functional activity of the neuron membrane. J. Evol. Biochem. Physiol., 27:110-116, 1991.
141. Büsselberg, D., Michael, D., Evans, M.L., Carpenter, D.O. and Haas, H.L. Zinc (Zn^{2+}) blocks voltage gated calcium channels in cultured rat dorsal root ganglion cells. Brain Res., 593:77-81, 1992.
142. Matthews, M.R., Parsons, P.J. and Carpenter, D.O. Solubility of lead as lead (II) chloride in HEPES-Ringer and artificial seawater (Ca-ASW) solutions. NeuroToxicology, 14:283-290, 1993.
143. Hori, N., Büsselberg, D., Matthews, R., Parsons, P.J. and Carpenter, D.O. Lead blocks LTP by an action not at NMDA receptors. Exp. Neurol., 119: 192-197, 1993.
144. Büsselberg, D., Evans, M.L., Haas, H.L. and Carpenter, D.O. Blockade of mammalian and invertebrate calcium channels by lead. NeuroToxicology, 14:249-258, 1993.
145. Riepe, M., Hori, N., Ludolph, A.C., Carpenter, D.O., Spencer, P.S. and Allen, C.N. Inhibition of energy metabolism by 3-nitropropionic acid activates ATP-sensitive potassium channels. Brain Res., 586:61-66, 1992.
146. Hori, N., Hirotsu, I., Davis, P.J. and Carpenter, D.O. Long-term potentiation is lost in aged rats but preserved by calorie restriction. NeuroReport, 3:1085-1088, 1992.
147. Knox, A.P., Strominger, N.L., Battles, A.H. and Carpenter, D.O. Behavioral studies of emetic sensitivity in the ferret. Brain Res. Bull., 31:477-484, 1993.
148. Allen, C.N., Spencer, P.S. and Carpenter, D.O. β -N-methylamino-L-alanine in the presence of bicarbonate is an agonist at non-N-methyl-D-aspartate-type receptors. Neuroscience 54:567-574, 1993.
149. Elekes, K., Stefano, G.B. and Carpenter, D.O. Enkephalin-like immunoreactive neurons in the central nervous system of gastropods (*Helix pomatia*, *Lymnaea stagnalis*, *Aplysia californica*): A comparative immunocytochemical study. Cell Tiss. Res. 272:329-41, 1993.
150. Büsselberg, D., Platt, B., Haas, H.L. and Carpenter, D.O. Voltage gated calcium channel currents of rat dorsal root ganglion (DRG) cells are blocked by Al^{3+} . Brain Res. 622:163-168, 1993.
151. Strominger, N.L., Knox, A.P. and Carpenter, D.O. The connectivity of the area postrema in the ferret. Brain Res. Bull., 33:33-47, 1994.
152. Knox, A.P., Strominger, N.L., Battles, A.H. and Carpenter, D.O. The central connections of the vagus nerve in the ferret. Brain Res. Bull., 33:49-63, 1994.
153. Lin, Y. and Carpenter, D.O. Medial vestibular neurons are endogenous pacemakers whose discharge is modulated by neurotransmitters. Cell. Molec. Neurobiol., 13:601-613, 1993.
154. Kemenes, G., S.-Rózsa, K. and Carpenter, D.O. Cyclic-AMP-mediated excitatory responses to leucine enkephalin in *Aplysia* neurones. J. Exp. Biol. 181: 321-328, 1993.
155. Büsselberg, D., Platt, B., Michael, D., Carpenter, D.O. and Haas, H.L. Mammalian voltage-activated calcium channel currents are blocked by Pb^{2+} , Zn^{2+} and Al^{3+} . J. Neurophysiol., 71:1491-1497, 1994.
156. Hori, N. and Carpenter, D.O. Transient ischemia causes a reduction of Mg^{2+} blockade of NMDA receptors. Neurosci. Letts., 173:75-78, 1994.

157. Riepe, M.W., Hori, N., Ludolph, A.C. and Carpenter, D.O. Failure of neuronal ion exchange, not potentiated excitation, causes excitotoxicity after inhibition of oxidative phosphorylation. Neuroscience, 64:91-97, 1995.
158. Hori, N. and Carpenter, D.O. Functional and morphological changes induced by transient *in vivo* ischemia. Exp. Neurol., 129:279-289, 1994.
159. Lin, Y. and Carpenter, D.O. Direct excitatory opiate effects mediated by non-synaptic actions on rat medial vestibular neurons. Eur. J. Pharmacol., 262:99-106, 1994.
160. Carpenter, D.O. Epidemiological evidence for an association between exposure to 50 and 60 Hz magnetic fields and cancer. James Bay Publication Series, Hydro-Electric Development: Environmental Impacts - Paper No. 6, pp. 2-31, 1994.
161. Carpenter, D.O. Communicating with the public on issues of science and public health. Environ. Health Perspect, 103:127-130, 1995.
162. Fejtl, M., Gyori, J. and Carpenter, D.O. Hg²⁺ increases the open probability of carbachol-activated Cl⁻ channels in *Aplysia* neurons. NeuroReport, 5:2317-2320, 1994.
163. Carpenter, D.O. The public health significance of metal neurotoxicity. Cell. Molec. Neurobiol., 14:591-597, 1994.
164. Gyori, J., Fejtl, M. and Carpenter, D.O. Effect of HgCl₂ on acetylcholine, carbachol and glutamate currents of *Aplysia* neurons. Cell. Molec. Neurobiol., 14:653-664, 1994.
165. Fejtl, M., Gyori, J. and Carpenter, D.O. Mercuric (II) chloride modulates single channel properties of carbachol activated Cl⁻ channels in cultured neurons of *Aplysia californica*. Cell. Molec. Neurobiol., 14:665-674, 1994.
166. Carpenter, D.O., Matthews, M.R., Parsons, P.J. and Hori, N. Long-term potentiation in piriform cortex is blocked by lead. Cell. Molec. Neurobiol., 14:723-733, 1994.
167. Salanki, J., Gyori, J. and Carpenter, D.O. Action of lead on glutamate-activated chloride currents in *Helix Pomatia L.* neurons. Cell. Molec. Neurobiol., 14:755-768, 1994.
168. Carpenter, D.O. How hazardous wastes affect human health. Cent. Eur. J. Publ. Hlth. 2:6-9, 1994.
169. Oyama, Y., Carpenter, D.O., Ueno, S., Hayashi, H. and Tomiyoshi, F. Methylmercury induces Ca²⁺-dependent hyperpolarization of mouse thymocytes: A flow-cytometric study using fluorescent dyes. Eur. J. Pharmacol., 293:101-107, 1995.
170. Fejtl, M., Szarowski, D.H., Decker, D., Buttle, K., Carpenter, D.O. and Turner, J.N. Three-dimensional imaging and electrophysiology of live *Aplysia* neurons during volume perturbation: confocal light and high-voltage electron microscopy. JMSA 1(2):75-85, 1995.
171. Carpenter, D.O., Kemenes, G., Elekes, K., Leung, M., Stefano, G., S.-Rozsa, K. and Salanki, J. Opioid peptides in the nervous system of *Aplysia*: A combined biochemical immunocytochemical, and electrophysiological study. Cell. Molec. Neurobiol. 15:239-256, 1995.
172. Riepe, M. and Carpenter, D.O. Delayed increase of cell volume of single pyramidal cells in live hippocampal slices upon kainate application. Neurosci. Letts. 191:35-38, 1995.
173. Son, H. And Carpenter, D.O. Protein kinase C activation is necessary but not sufficient for induction of LTP at the synapse of mossy fiber-CA3 in the rat hippocampus. Neuroscience 72:1-13, 1996.
174. Iwase, T., Hori, N., Morioka, T. and Carpenter, D.O. Low power laser irradiation reduces ischemic damage in hippocampal slices *in vitro*. Lasers Surg. Med., 19:465-450, 1996.
175. Carpenter, D.O., King, W.M. and McCreery, M.J. The role of glutamate reuptake in regulation of glutamate responses in *Aplysia* neurons. Acta Biologica Hungaria 46:363-373, 1995.

176. Saghian, A.A., Ayrapetyan, S.N. and Carpenter, D.O. Low concentrations of ouabain stimulate Na/Ca exchange in neurons. Cell. Molec. Neurobiol., 16:489-498, 1996.
177. Platt, B., Carpenter, D.O., Büsselberg, D., Reymann, K.G. and Riedel, G. Aluminum impairs hippocampal long-term potentiation in rats in vitro and in vivo. Exp. Neurol., 134:73-86, 1995.
178. Rubakhin, S.S., Gyori, J., Carpenter, D.O. and Salanki, J. HgCl₂ potentiates GABA activated currents in *Lymnaea stagnalis* L. neurons. Acta Biologica Hungaria, 46:431-444, 1995.
179. Fejtl, M. and Carpenter, D.O. Neurite outgrowth is enhanced by conditioning factor(s) released from central ganglia of *Aplysia californica*. Neurosci. Letts., 199:33-36, 1995.
180. Riepe, M.W., Niemi, W.N., Megow, D., Ludolph, A.C. and Carpenter, D.O. Mitochondrial oxidation in rat hippocampus can be preconditioned by selective chemical inhibition of SDH. Exp. Neurol., 138:15-21, 1996.
181. Son, H. and Carpenter, D.O. Interactions among paired-pulse facilitation and post-tetanic and long-term potentiation in the mossy fiber-CA3 pathway in rat hippocampus. Synapse, 23:302-311, 1996.
182. Carpenter, D.O., Suk, W.A., Blaha, K. and Cikrt, M. Hazardous wastes in Eastern and Central Europe. Environ. Health Perspect., 104:244-248, 1996.
183. Son, H., Davis, P.J. and Carpenter, D.O. Time course and involvement of protein kinase C-mediated phosphorylation of F1/GAP-43 in area CA3 after the mossy fiber stimulation. Cell. Molec. Neurobiol., 17:171-194, 1997.
184. Dyatlov, V.A., Platoshin, A.V., Lawrence, D.A. and Carpenter, D.O. Mercury (Hg²⁺) enhances the depressant effect of kainate on Ca-inactivated potassium current in telencephalic cells derived from chick embryos. Toxicol. Appl. Pharmacol., 138:285-297, 1996.
185. Carpenter, D.O. and Conway, J.B. Optimizing professional education in public health. J. Public Health Management Practice, 2:66-72, 1996.
186. Carpenter, D.O. Great Lakes contaminants: A shift in human health outcomes. Health and Environment Digest, 10:17-19, 1996.
187. Boldyrev, A.A., Stvolinsky, S.L., Tyulina, O.V., Koshelev, V.B., Hori, N. and Carpenter, D.O. Biochemical and physiological evidence that carnosine is an endogenous neuroprotector against free radicals. Cell. Molec. Neurobiol., 17:259-271, 1997.
188. Szűcs, A., Angiello, C., Salánki, J. and Carpenter, D.O. Effects of inorganic mercury and methylmercury on the ionic currents of cultured rat hippocampal neurons. Cell. Molec. Neurobiol., 17:273-288, 1997.
189. Niemi, W.D., Slivinski, K., Audi, J., Rej, R. and Carpenter, D.O. Propylthiouracil treatment reduces long-term potentiation in area CA1 of neonatal rat hippocampus. Neurosci. Letts., 210:127-129, 1996.
190. Son, H., Madelian, V. and Carpenter, D.O. The translocation and involvement of protein kinase C in mossy fiber-CA3 long-term potentiation in hippocampus of the rat brain. Brain Res., 739:282-292, 1997.
191. Oyama, Y., Carpenter, D.O., Chikahisa, L. and Okazaki, E. Flow-cytometric estimation on glutamate- and kainate-induced increases in intracellular Ca²⁺ of brain neurons. Brain Research, 728:121-124, 1996.
192. Carpenter, D.O., Stoner, C.R.T. and Lawrence, D.A. Flow cytometric measurements of neuronal death triggered by PCBs. NeuroToxicology, 18:507-514, 1997.
193. Azatian, K.V., Ayrapetyan, S.N. and Carpenter, D.O. Metabotropic GABA receptors regulate acetylcholine responses on snail neurons. Gen. Pharmacol., 29:67-72, 1997.

194. Carpenter, D.O., Stoner, C.T., Lawrence, D.A., Niemi, W.D., Shain, W. and Seegal, R. Multiple mechanisms of PCB neurotoxicity. *Proceedings of the 1996 Pacific Basin Conference on Hazardous Waste, Kuala Lumpur, Malaysia, CONF-9611157*, pp. 404-918.
195. Carpenter, D.O. New Dimensions in our understanding of the human health effects of environmental pollutants. *Proceedings of the 1996 Pacific Basin Conference on Hazardous Waste, Kuala Lumpur, Malaysia, CONF-9611157*, pp. 37-53.
196. Carpenter, D.O. Possible effects of electromagnetic fields on the nervous system and development. *Men. Retard. Dev. Dis. Res. Rev.* 3:270-274, 1997.
197. Chiarenzelli, J., Scrudato, R., Bush, B., Carpenter, D. and Bushart, S. Do large-scale remedial and dredging events have the potential to release significant amounts of semi-volatile compounds to the atmosphere? *Environ. Hlth. Perspect.*, 106:47-49, 1998.
198. Dyatlov, V.A., Dytlova O.M., Parsons, P.H., Lawrence, D.A. and Carpenter, D.O. Lipopolysaccharide and interleukin-6 enhance lead entry into cerebellar neurons: Application of a new and sensitive flow cytometric technique to measure intracellular lead and calcium concentrations. *NeuroToxicology*, 19:293-302, 1998.
199. Dyatlov, V.A., Platoshin, A.V., Lawrence, D.A. and Carpenter, D.O. Lead potentiates cytokine- and glutamate-mediated increases in permeability of the blood-brain barrier. *NeuroToxicology*, 19:283-292, 1998.
200. Niemi, W.D., Audi, J., Bush, B. and Carpenter, D.O. PCBs reduce long-term potentiation in the CA1 region of rat hippocampus. *Exper. Neurol.*, 151:26-34, 1998.
201. Carpenter, D.O. Health effects of metals. *Cent. Eur. J. Publ. Hlth.*, 6:160-163, 1998.
202. Carpenter, D.O., Bláha, K., Buekens, A., Cikrt, M., Damstra, T., Dellinger, B., Sarofim, A., Suk, W.A., Wyes, H. and Zejda, J. Remediation of hazardous wastes in Central and Eastern Europe: Technology and health effects. *Cent. Eur. J. Publ. Hlth.*, 6:77-78, 1998.
203. Carpenter, D.O. Human health effects of environmental pollutants: New Insights. *Environ. Monitor. Assess. J.*, 53:245-258, 1998.
204. Dyatlov, V.A., Makovetskaia, V.V., Leonhardt, R., Lawrence, D.A. and Carpenter, D.O. Vitamin E enhances Ca²⁺-mediated vulnerability of immature cerebellar granule cells to ischemia. *Free Rad. Biol. Med.*, 25: 793-802, 1998.
205. Fitzgerald, E.F., Schell, L.M., Marshall, E.G., Carpenter, D.O., Suk, W.A. and Zejda, J.E. Environmental pollution and child health in Central and Eastern Europe. *Environ. Health Persp.*, 106:307-311, 1998.
206. Carpenter, D.O., Arcaro, K.F., Bush, B., Niemi, W.D., Pang, S. and Vakharia, D.D. Human health and chemical mixtures: An overview. *Environ. Health Perspect.*, 106: 1263-1270, 1998.
207. Carpenter, D.O., Cikrt, M. and Suk, W.A. Hazardous wastes in Eastern and Central Europe: Technology and health effects. *Environ. Health Perspect.*, 107: 3-4, 1999.
208. Carpenter, D.O. Polychlorinated biphenyls and human health. *Int. J. Occup. Med. Environ. Hlth.* 11: 291-303, 1998.
209. Boldyrev, A.A., Johnson, P., Yanzhang, W., Tan, Y. and Carpenter, D.O. Carnosine and taurine protect rat cerebellar granular cells from free radical damage. *Neurosci. Letts.*, 263: 169-172, 1999.
210. Boldyrev, A.A., Carpenter, D.O., Huentelman, M.J., Peters, C.M. and Johnson, P. Sources of reactive oxygen species production in excitotoxin-stimulated neurons. *Biophys. Biochem. Res. Commun.*, 256: 320-324, 1999.
211. Ayrapetyan, S.N., Ayrapetyan, G. and Carpenter, D.O. The electrogenic sodium pump activity in *Aplysia* neurons is not potential dependent. *Acta Biologica Hungarica*, 50: 27-34, 1999.

212. Boldyrev, A., Song, R., Lawrence, D. and Carpenter, D.O. Carnosine protects against excitotoxic cell death independently of effects on reactive oxygen species. Neuroscience, 94: 571-577, 1999.
213. Boldyrev, A., Song, R., Dyatlov, V.A., Lawrence, D.A. and Carpenter, D.O. Neuronal cell death and reactive oxygen species. Cell. Molec. Neurobiol., 20:433-450, 2000.
214. Gyori, J., Platoshyn, O., Carpenter, D.O. and Salanki, J. Effect of inorganic- and organic tin compounds on ACh- and voltage-activated Na currents. Cell. Molec. Neurobiol. 20:591-604, 2000.
215. Hussain, R.J., Gyori, J., DeCaprio, A.P. and Carpenter, D.O. *In vivo* and *in vitro* exposure to PCB 153 reduces long-term potentiation. Environ. Hlth. Perspect., 108 :827-831, 2000.
216. Negoita, S., Swamp, L., Kelley, B. and Carpenter, D.O. Chronic diseases surveillance of St. Regis Mohawk health service patients. J. Public Health Management Practice, 7:84-91, 2001.
217. Hussain, R.J., Parsons, P.J., Carpenter, D.O. Effects of lead on long-term potentiation in hippocampal CA3 vary with age. Dev. Brain Res., 121: 243-252, 2000.
218. Tanji, M., Katz, B.H., Spink, B.C. and Carpenter, D.O. Growth inhibition of MCF-7 cells by estrogen is dependent upon a serum factor. Anticancer Res., 20: 2779-2784, 2000.
219. Tanji, M. and Carpenter, D.O. A steroid-binding protein mediates estrogen-dependent inhibition of growth of MCF-7 breast cancer cells. Anticancer Res., 20:2785-2790, 2000.
220. Gyori, J., Hussain, R., Carpenter, D.O. Long-term potentiation in CA1 region of rat brain slices is blocked by PCB 153. Cent. Europ. J. Publ. Hlth., 8: 21-22, 2000.
221. Carpenter, D.O. Human health effects of polychlorinated biphenyls. Cent. Eur. J. Public Health, 8: 23-24, 2000.
- 221a. Sukdolova, V., Negoita, S., Hubicki, L., DeCaprio, A., and Carpenter, D.O. The assessment of risk to acquired hypothyroidism from exposure to PCBs: a study among Akwesasne Mohawk women. Cent. Eur. J. Public Health, 8: 167-168, 2000.
222. Carpenter, D.O., Chew, F.T., Damstra, T., Lam, L.H., Landrigan, P.J., Makalinao, I., Peralta, G.L. and Suk, W.A. Environmental threats to the health of children: The Asian perspective. Environ. Hlth. Perspect., 108: 989-992, 2000.
223. Boldyrev, A.A., Carpenter, D.O. and Johnson, P. Natural mechanisms of protection of neurons against oxidative stress. Recent Res. Devel. Comparative Biochem. & Physiol. 1: 91-103, 2000.
224. Strominger, N.L., Hori, N., Carpenter, D.O., Tan, Y. and Folger W.H. Effects of acetylcholine and GABA on neurons in the area postrema of *Suncus murinus* brainstem slices. Neurosci. Letts. 309: 77-80, 2001.
225. Strominger, N.L., Brady, R., Gullikson, G. and Carpenter, D.O. Imiquimod-elicited emesis is mediated by the area postrema, but not by direct neuronal activation. Brain Res. Bull. 55: 445-451, 2001.
226. Hori, N., Tan, Y., Strominger, N.L. and Carpenter, D.O. Intracellular activity of rat spinal cord motoneurons in slices. J. Neurosci. Meth. 112: 185-191, 2001.
227. Sukocheva, O.A., Abramov, A.Y., Levitskaya, J.O., Gagelgans, A.I. and Carpenter, D.O. Modulation of intracellular Ca concentration by vitamin B12 in rat thymocytes. Blood Cells. Mol. Dis. 27: 812-824, 2001.
228. Gilbertson, M., Carpenter, D. and Upshur, R. Methodology for assessing community health in Areas of Concern: Measuring the adverse effects on human health. Environ. Health Perspect. 109 (Suppl 6): 811-812, 2001.
229. Carpenter, D.O., Shen, Y., Nguyen, T., Le, L. and Lininger, L.L. Incidence of endocrine disease among residents of New York Areas of Concern. Environ. Health Perspect. 109: (Suppl 6) 845-851, 2001.

230. Suk, W.A., Carpenter, D.O., Cirkt, M. and Smerhovsky, Z. Metals in Eastern and Central Europe: Health effects, sources of contamination and methods of remediation. Internat. J. Occup. Med. Environ. Health 14, 151-156, 2001.
231. Carpenter, D.O. Effects of metals on the nervous system of humans and animals. Internat. J Occup. Med. Environ. Health 14: 209-218, 2001.
232. Carpenter, D.O., Arcaro, K. and Spink, D.C. Understanding the human health effects of chemical mixtures. Environ. Health Perspect. 110 (Suppl 1), 25-42, 2002.
233. Carpenter, D.O., Nguyen, T., Le, L., Kudyakov, R. and Lininger, L. Human disease in relation to residence near hazardous waste sites. Proceedings of The 10th Pacific Basin Conference on Hazardous Waste, Okayama, Japan, December 5-7, 2001.
234. Carpenter, D.O., Tarbell, A., Fitzgerald, E., Kadlec, M.J., O'Hehir, D.O. and Bush, B. University-community partnership for the study of environmental contamination at Akwesasne. In: Biomarkers of Environmentally Associated Disease, S.H. Wilson and W.A. Suk, editors, CRC Press/Lewis Publishers, 507-523, 2002.
235. Carpenter, D.O., Hussain, R.J., Berger, D.F., Lombardo, J.P., Park, H-Y. Electrophysiological and behavioral effects of perinatal and acute exposure of rats to lead and polychlorinated biphenyls. Environ. Health Perspect., 110: 377-386, 2002.
236. Hori, N., Tan, Y. King, M., Strominger, N.L. and Carpenter, D.O. Differential actions and excitotoxicity of glutamate agonists on motoneurons in adult mouse cervical spinal cord slices. Brain Res., 958: 434-438, 2002.
237. Laemle, L.K., Hori, N., Strominger, N.L., Tan, Y. and Carpenter, D.O. Physiological and anatomical properties of the suprachiasmatic nucleus of an anophthalmic mouse. Brain Res., 953: 73-81, 2002.
238. Hori, N., Tan, Y., Strominger, N.L. and Carpenter, D.O. Rat motoneuron cell death in development correlates with loss of N-methyl-D-aspartate receptors. Neurosci. Letts., 330:131-134, 2002.
239. Carpenter, D.O., Morris, D.L. and Legator, M. Initial attempts to profile health effects with types of exposure in Anniston, Alabama. FEB, 12: 191-195, 2003.
240. Carpenter, D.O., Nguyen, T., Le, L., Baibergenova, A. and Kudyakov, R. Profile of health effects related to proximity to PCB-contaminated hazardous waste sites in New York. FEB, 12: 173-180, 2003.
241. Hori, N., Carp, J.S., Carpenter, D.O. and Akaike, N. Corticospinal transmission to motoneurons in cervical spinal slices from adult rats. Life Sci., 72: 389-396, 2002.
242. Carpenter, D.O. and Hussain, R.J. Cell-to-cell communication of neurons is impaired by metals. Mat.-wiss. U. Werkstofftech. 34: 1-8, 2003.
243. Tan, Y., Hori, N. and Carpenter, D.O. The mechanism of presynaptic long-lasting-depression mediated by group 1 metabotropic glutamate receptors. Cell. Molec. Neurobiol., 23: 187-203, 2003.
244. Baibergenova, A., Kudyakov, R., Zdeb, M., and Carpenter, D.O. Low birth weight and residential proximity to PCB-contaminated waste sites. Environ. Health Perspect., 111: 1352-1357, 2003.
245. Nishizaki, Y., Oyama, Y., Sakai, Y., Hirama, S., Tomita, K., Nakao, H., Umabayashi, C., Ishida, S., Okano, Y. and Carpenter, D.O. PbCl₂-induced hyperpolarization of rat thymocytes: Involvement of charybdotoxin-sensitive K⁺ channels. Environ. Toxicol., 18(5): 321-326, 2003.
246. Hussain, R.J. and Carpenter, D.O. The effects of protein kinase C activity on synaptic transmission in two areas of rat hippocampus. Brain Res., 990: 28-37, 2003.
247. Suk, W.A., Ruchirawat, K., Balakrishnan, K., Berger, M., Carpenter, D., Damstra, T., Pronczuk de Garbino, J., Koh, D., Landrigan, P.J., Makalinao, I., Sly, P.D., Xu, Y. and Zheng, B.S.

- Environmental threats to children's health in Southeast Asia and the Western Pacific. Environ. Health Perspect. 111: 1340, 2003.
248. Carpenter, D.O. The need for global environmental health policy. New Solutions, 13(1): 53-59, 2003.
 249. Tan, Y., Li, D., Song, R., Lawrence, D. and Carpenter, D.O. Ortho-substituted PCBs kill thymocytes. Toxicol. Sci., 76: 328-337, 2003.
 250. Boldyrev, A., Bulygina, E., Carpenter, D.O. and Schoner, W. Glutamate receptors communicate with Na⁺/K⁺-ATPase in rat cerebellum granule cells: Demonstration of differences in the action of several metabotropic and ionotropic glutamate agonists on intracellular reactive oxygen species and the sodium pump. J. Molec. Neurosci., 21:213-222, 2003.
 251. Hites, R.A., Foran, J.A., Carpenter, D.O., Hamilton, M.C., Knuth, B.A. and Schwager, S.J. Global assessment of organic contaminants in farmed salmon. Science 303: 226-229, 2004.
 252. Sandal, S., Yilmaz, B., Chen, C-H and Carpenter, D.O. Comparative effects of technical toxaphene, 2,5-dichloro-3-biphenylol and octabromodiphenylether on cell viability, [Ca²⁺]_i levels and membrane fluidity in mouse thymocytes. Toxicol. Letts., 151: 417-428, 2004.
 253. Tan, Y., Chen, C-H., Lawrence, D. and Carpenter, D.O. Ortho-substituted PCBs kill cells by altering membrane structure. Toxicol. Sci., 80: 54-59, 2004.
 254. Tan, Y., Song, R., Lawrence, D. and Carpenter, D.O. Ortho-substituted but not coplanar PCBs rapidly kill cerebellular granule cells. Toxicol. Sci., 79: 147-156, 2004.
 255. Ozcan, M., Yilmaz, B., King, W.M. and Carpenter, D.O. Hippocampal long-term potentiation (LTP) is reduced by a coplanar PCB congener. NeuroToxicology, 25: 981-988, 2004.
 256. Ssempebwa, J.C., Carpenter, D.O., Yilmaz, B., DeCaprio, A.P., O=Hehir, D.J. and Arcaro, K.F. Waste crankcase oil: an environmental contaminant with potential to modulate estrogenic responses. J. Toxicol. Environ. Hlth. Part A, 67: 1081-1094, 2004.
 257. Foran, J.A., Hites, R.A., Carpenter, D.O., Hamilton, M.C., Mathews-Amos, A. and Schwager, S.J. A survey of metals in tissues of farmed Atlantic and wild Pacific salmon. Environ. Toxicol. Chem., 23: 2108-2110, 2004.
 258. Oenga, G.N., Spink, D.C. and Carpenter, D.O. TCDD and PCBs inhibit breast cancer cell proliferation in vitro. Toxicol. In Vitro, 18: 811-819, 2004.
 259. Hussain, R.J. and Carpenter, D.O. A comparison of the roles of protein kinase C in long-term potentiation in rat hippocampal areas CA1 and CA3. Cell. Molec. Neurobiol., 25: 649-661, 2005.
 260. Hites, R.A., Foran, J.A., Schwager, S.J., Knuth, B.A., Hamilton, M.C. and Carpenter, D.O. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. Organohalogen Compounds, 66: 3826-3829, 2004.
 261. Kudyakov, R., Baibergenova, A., Zdeb, M. and Carpenter, D.O. Respiratory disease in relation to patient residence near to hazardous waste sites. Environ. Toxicol. Pharmacol., 18: 249-257, 2004.
 262. Gilbertson, M. and Carpenter, D.O. An ecosystem approach to the health effects of mercury in the Great Lakes basin ecosystem. Environ. Res. 95: 240-246, 2004.
 263. Hites, R.A., Foran, J.A., Schwager, S.J., Knuth, B.A., Hamilton, M.C. and Carpenter, D.O. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. Environ. Sci. Technol., 38: 4945-4949, 2004.
 264. DeCaprio, A.P., Johnson, G.W., Tarbell, A.M., Carpenter, D.O. Chiarenzelli, J.R., Morse, G.S., Santiago-Rivera, A.L., Schymura, M.J., and the Akwesasne Task Force on the Environment. PCB exposure assessment by multivariate statistical analysis of serum congener profiles in an adult Native American population. Environ. Res., 98: 284-302, 2005.

265. Boldyrev, A.A., Kazey, V.I., Leinsoo, T.A., Mashkina, A.P., Tyulina O.V., Tuneva, J.O., Chittur, S. and Carpenter, D.O. Rodent lymphocytes express functionally active glutamate receptors. Biochem. Biophys. Res. Comm., 324: 133-139, 2004.
266. Boldyrev, A.A., Koudinov, A., Berezov, T. and Carpenter, D.O. Amyloid- β induced cell death is independent of free radicals. J. Alzheimer's Dis., 6: 633-638, 2004.
267. Neagu, B., Strominger, N.L. and Carpenter, D.O. Use of bipolar parallel electrodes for well-controlled microstimulation in a mouse hippocampal brain slice. J. Neurosci. Meth., 144: 153-163, 2005.
268. Suk, W.A., Avakian, M.D., Carpenter, D., Groopman, J.D., Scammell, M. and Wild, C.P. Human exposure monitoring and evaluation in the Arctic: The importance of understanding exposures to the development of public health policy. Environ. Health Perspect. 112: 113-120, 2004.
269. Neagu, B., Neagu, E.R., Strominger, N.L. and Carpenter, D.O. A new fast electro-physiological response measured extracellularly in a mouse hippocampal brain slice. Neurosci. Letts., 381: 179-184, 2005.
270. Sergeev, A.V. and Carpenter, D.O. Hospitalization rates for coronary heart disease in relation to residence near areas contaminated with POPs and other pollutants. Environ. Health Perspect., 113: 756-761, 2005.
271. Foran, J.A., Carpenter, D.O., Hamilton, M.C., Knuth, B.A. and Schwager, S.J. Risk-based consumption advice for farmed Atlantic and wild Pacific salmon contaminated with dioxins and dioxin-like compounds. Environ. Health Perspect. 113: 552-556, 2005.
272. Shaw, S.D., Bourakovsky, A., Brenner, D., Carpenter, D.O., Tao, L., Kannan, K. and Hong, C-S. Polybrominated diphenyl ethers (PBDEs) in farmed salmon from Maine and Eastern Canada. In: Proceedings of 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs (DIOXIN 2005), August 21-26, 2005, Toronto, Canada.
273. Carpenter, D.O., DeCaprio, A.P., O=Hehir, D., Akhtar, F., Johnson, G., Scudato, R.J., Apatiki, L., Kava, J., Gologergen, J., Miller, P.K. and Eckstein, L. Polychlorinated biphenyls in serum of the Siberian Yupik people from St. Lawrence Island, Alaska. Int. J. Circumpolar Health, 64(4): 322-335, 2005.
274. Foran, J.A., Good, D.H., Carpenter, D.O., Hamilton, M.C., Knuth, B.A. and Schwager, S.J. Quantitative analysis of the benefits and risks of consuming farmed and wild salmon. J. Nutr 135: 2639-2643, 2005.
275. Huang, X., Hites, R.A., Foran, J.A., Hamilton, C., Knuth, B.A., Schwager, S.J. and Carpenter, D.O. Consumption advisories for salmon based on risk of cancer and non-cancer health effects. Environ. Res., 101: 263-274, 2006.
276. Shcherbatykh, I., Huang, X., Lessner, L. and Carpenter, D.O. Hazardous waste sites and stroke in New York State. Environ. Health, 4:18, 2005.
277. Hamilton, M.C., Hites, R.A., Schwager, S.J., Foran, J.A., Knuth, B.A. and Carpenter, D.O. Lipid composition and contaminants in farmed and wild salmon. Environ. Sci. Tech., 39: 8622-8629, 2005.
278. Yilmaz, B., Sandal, S., Chen, C-H. and Carpenter, D.O. Effects of PCB 52 and PCB 77 on cell viability, $[Ca^{2+}]_i$ levels and membrane fluidity in mouse thymocytes. Toxicology, 217: 184-193, 2006.
279. Tan, Y., Hori, N., and Carpenter, D.O. Electrophysiological effects of three groups of glutamate metabotropic receptors in rat piriform cortex. Cell. Molec. Neurobiol., 26: 915-924, 2006.
280. Boldyrev, A.A., Carpenter, D.O. and Johnson, P.A., Emerging evidence for a similar role of glutamate receptors in the nervous and immune systems. J. Neurochem., 95: 913-918, 2005.

281. Sandal, S., Yilmaz, B., Godekmerdan, A., Kelestimur, H. and Carpenter, D.O. Effects of PCBs 52 and 77 on Th1/Th2 balance in mouse thymocyte cell cultures. Immunopharmacol. Immunotoxicol. 27: 601-613, 2005.
282. Carpenter, D.O. Environmental contaminants and learning and memory. International Congress Series, 1287: 185-189, 2006.
283. Carpenter, D.O. Polychlorinated biphenyls (PCBs): Routes of exposure and effects on human health. Rev. Environ. Health, 21: 1-23, 2006.
284. Huang, X., Lessner, L. and Carpenter, D.O. Exposure to persistent organic pollutants and hypertensive disease. Environ. Res., 102: 101-106, 2006.
285. Carpenter, D.O., El-Qaderi, S., Fayzieva, D., Gilani, A., Hambartsumyan, A., Herz, K., Isobaev, M., Kasymov, O., Kudyakov, R., Majitova, Z., Mamadov, E., Nemer, L., Revich, B., Stege, P., Suk, W., Upshur, R., Yilmaz, B. and Zaineh K. Children's environmental health in Central Asia and the Middle East. Int. J. Occup. Environ. Health, 12: 362-368, 2006.
286. King, W.M., Sarup, V., Sauve, Y., Moreland, C.M., Carpenter, D.O. and Sharma, S.C. Expansion of visual receptive fields in experimental glaucoma. Visual Neurosci. 23: 137-142, 2006.
- 286b. Laemle LK, Strominger NL, and Carpenter DO. Cross-modal innervation of primary visual cortex by auditory fibers in congenitally anophthalmic mice. Neurosci Lett 396: 108-112: 2006.
287. Tuneva, J., Chittur, S., Boldyrev, A.A., Birman, I. and Carpenter, D.O. Cerebellar granule cell death induced by aluminum. Neurotox. Res., 9: 297-304, 2006.
288. Trasande, L., Boscarino, J., Graber, N., Falk, R., Schechter, C., Dunkel, G., Geslani, J., Moline, J., Kaplan-Liss, E., Miller, R.K., Korfmacher, K., Carpenter, D., Balk, S.J., Laraque, D., Frumkin, H. and Landrigan, P.J. The environment in pediatric practice: A study of New York pediatricians' attitudes, beliefs, and practices towards children's environmental health. J. Urban Health, 2006, DOI: 10.1007/s11524-006-9071-4.
289. Surdu, S., Montoya, L.D., Tarbell, A. and Carpenter, D.O. Childhood asthma and indoor allergens in Native Americans in New York. Environ. Health: A Global Access Science Source, 5:22, 2006. DOI: 10.1186/1476-069X-5-22.
290. Ozcan M., Yilmaz, B. and Carpenter, D.O. Effects of melatonin on synaptic transmission and long term potentiation in two areas of mouse hippocampus. Brain Res., 1111: 90-94, 2006.
291. Shaw, S.D., Brenner, D., Berger, M.L., Pulser, E.L., Carpenter, D.O., Hong, C-W and Kannan K. PCBs, dioxin-like PCBs, dioxins, and organochlorine pesticides in farmed salmon (*Salmo salar*) from Maine and Eastern Canada. Environ. Sci. Technol. 40: 5347-5354, 2006.
292. Yilmaz, B., Ssempebwa J., Mackerer, C.R., Arcaro, K.F. and Carpenter, D.O. Effects of polycyclic aromatic hydrocarbon-containing oil mixtures on generation of reactive oxygen species and cell viability in MCF-7 breast cancer cells. J. Toxicol. Environ. Health, Part A: 70: 1-8, 2007.
293. Kouznetsova, M., Huang, X., Ma, J., Lessner, L. and Carpenter, D.O. Increased rate of hospitalization for diabetes and residential proximity of hazardous waste sites. Environ. Health Perspect., 115:75-79, 2007.
294. Yilmaz, Y., Seyran, A.D., Sandal, S., Aydin, M., Colakoglu, N., Kocer, M. and Carpenter, D.O. Modulatory effects of Aroclors 1221 and 1254 on bone turnover and vertebral histology in intact and ovariectomized rats. Toxicology Letts., 166: 276-294, 2006.
295. Shcherbatykh, I. and Carpenter, D.O. The role of metals in the etiology of Alzheimer's disease. J. Alzheimer's Dis., 11: 191-205, 2007.
296. Surdu S, Neamtii I, Gurzau E, Kasler I and Carpenter D. Blood lead levels and hand lead contamination in children ages 4-6 in Copsa Mica, Romania. In: *Environmental Health in Central and Eastern Europe*. KC Donnelly and LH Cizmas, Eds. Springer Netherlands. pp. 123-134, 2007.

297. Carpenter D.O. The importance of the Great Lakes Water Quality Agreement. J Public Health Policy 28: 216-220, 2007.
298. Codru N, Schymura MJ, Negoita S, the Akwesasne Task Force on the Environment, Rej R and Carpenter DO. Diabetes in relation to serum levels of polychlorinated biphenyls (PCBs) and chlorinated pesticides in adult Native Americans. Environ Health Perspect. 115: 1442-1447, 2007.
299. Carpenter DO. Biomarcadores de efectos neuroconductuales. Acta Toxicol Argent 14 (Suplemento): 11-12, 2006.
300. Hennig B, Ormsbee L, Bachas L, Silverstone A, Milner J, Carpenter D, Thompson C and Suk WA. Introductory comments: nutrition, environmental toxins and implications in prevention and intervention of human diseases. J Nutrit Biochem 189: 161-163, 2007.
301. Arnold R, Armour MA, Barich J, Cebrian M, Cifuentes L, Kirk D, Koh D, Lewis ND, Ling B, Makalinao I, Maiden T, Paz-y-Mino C, Peralta G, Singh K, Sly P, Suk W, Woodward A, Zheng B and Carpenter DO. Threats to human health and environmental sustainability in the Pacific Basin: The 11th International Conference of the Pacific Basin Consortium. Environ Health Perspect. 115: 1770-1775, 2007.
302. Parrish RR, Horstwood M, Arnason JG, Chenery S, Brewer T, Lloyd NS and Carpenter DO (2008) Depleted uranium contamination by inhalation exposure and its detection after approximately 25 years: Implications for health assessment. Sci Total Environ 390: 58-68.
303. Goncharov A, Haase RF, Santiago-Rivera A, Morse G, Akwesasne Task Force on the Environment, McCaffrey RJ, Rej R and Carpenter DO. (2008) High serum PCBs are associated with elevation of serum lipids and cardiovascular disease in a Native American population. Environ Res. 106: 226-239.
304. Ma J, Kouznetsova M, Lessner L and Carpenter DO. Asthma and infectious respiratory disease in children – correlation to residence near hazardous waste sites. Paediatr Respir Rev 8: 292-298, 2007.
305. Schell LM, Gallo MV, Denham M, Ravenscroft J, DeCaprio AP and Carpenter DO (2008) Relationship of thyroid hormone levels of polychlorinated biphenyls, lead, p,p'-DDE and other toxicants in Akwesasne Mohawk youth. Environ Health Perspect. 116: 806-813.
306. Ssempebwa J and Carpenter DO (2009) The generation, use and disposal of waste crankcase oil in developing countries: A case for Kampala District, Uganda. J Hazard Materials 161: 835-841.
307. Carpenter DO (2008) Environmental contaminants as risk factors for developing diabetes. Rev Environ Health 23: 59-74.
308. Shaw SD, Berger ML, Brenner D, Carpenter DO, Lao L, Hong CS and Kannan K (2008) Polybrominated diphenyl ethers (PBDEs) in farmed and wild salmon marketed in the Northeastern United States. Chemosphere 71: 1422-1431.
309. Sandel S, Yilmaz B and Carpenter DO (2008) Genotoxic effects of PCB 52 and PCB 77 on cultured human peripheral lymphocytes. Mutation Res. 654: 88-92.
310. Carpenter DO and Sage C (2008) Setting prudent public health policy for electromagnetic field exposures. Rev Environ Health 23: 91-117.
311. Neagu B, Strominger NL and Carpenter DO (2008) Contribution of NMDA receptor-mediated component to the EPSP in mouse Schaffer collateral synapses under single pulse stimulation protocol. Brain Res. 1240: 54-61.
312. Holdren J, Tao S and Carpenter DO (2008) Environment and health in the 21st Century: Challenges and solutions. Ann NY Acad Sci. 1140:1-21.
313. Carpenter DO, Ma J and Lessner L (2008) Asthma and infectious respiratory disease in relation to residence near hazardous waste sites. Ann NY Acad Sci. 1140: 201-208.

314. Sandal S, Tuneva J, Yilmaz B and Carpenter DO (2009) Effects of cholesterol and docosahexaenoic acid on cell viability and (Ca^{2+}) levels in acutely isolated mouse thymocytes. *Cell Biochem Funct* 27: 155-161.
315. Steele RE, de Leeuw, E and Carpenter DO (2009) A novel and effective treatment modality for medically unexplained symptoms. *J Pain Management* 1: 402-412
316. Sage C and Carpenter DO (2009) Public health implications of wireless technologies. *Pathophysiology* 16: 233-246.
317. Sly PD, Eskenazi B, Pronczuk J, Sram R, Diaz-Barriga F, Machin DG, Carpenter DO, Surdu S and Meslin EM (2009) Ethical issues in measuring biomarkers in children's environmental health. *Environ Health Perspect.* 117: 1185-1190.
318. Goncharov A, Rej R, Negoita S, Schymura M, Santiago-Rivera A, Morse G, Akwesasne Task Force on the Environment and Carpenter DO (2009) Lower serum testosterone associated with elevated polychlorinated biphenyl concentrations in Native American men. *Environ Health Perspect.* 117:1454-1460.
319. Tuneva JO, Karpova LV, Shittur SV, Carpenter DO, Johnson P and Boldyrev AA (2009) Amyloid- β and aluminum ions enhance neuronal damage mediated by NMDA-activated glutamate receptors. *Biochemistry (Moscow) Supplement Series A: Membrane and Cell Biology* 4: 466-471.
320. Carpenter DO and Nevin R (2009) Environmental causes of violence. *Physiol Behavior* 99: 260-268.
321. Goncharov A, Bloom MS, Pavuk M, Carpenter DO for the Anniston Environmental Health Research Consortium. (2009) Exposure to PCBs and hypertension in the Anniston Community Health Survey. *Organohal Comp* 71: 0-136.
322. Sergeev AV and Carpenter DO (2010) Residential proximity to environmental sources of persistent organic pollutants and first-time hospitalizations for myocardial infarction with comorbid diabetes mellitus: A 12-year population-based study. *Int J Occup Med Environ Health* 23: 5-13.
323. Carpenter DO (2010) Electromagnetic fields and cancer: The cost of doing nothing. *Rev Environ Health* 25: 75-80.
324. Sergeev AV and Carpenter DO (2010) Exposure to persistent organic pollutants increases hospitalization rates for myocardial infarction with comorbid hypertension. *Primary Prevention Insights.* 2: 1-9.
325. Hori N, Kadota MT, Watanabe M, Ito Y, Akaike N and Carpenter DO (2010) Neurotoxic effects of methamphetamine on rat hippocampus pyramidal neurons. *Cell Mol Neurobiol.*30: 849-856.
326. Hardell, S, Tilander H, Welfinger-Smith G and Carpenter DO (2010) Levels of polychlorinated biphenyls (PCBs) and three organochlorine pesticides in fishes from the Aleutian Islands of Alaska. *PLoS ONE*, 5:e12396.
327. Carpenter, DO. (2010) Human health effects of EMFs: The cost of doing nothing. *IOP Conf. Series: Earth and Environmental Science* 10: 012004. doi:10:1088/1755-1315/10/1/10/012004.
328. Goncharov A, Bloom M, Pavuk M, Birman I and Carpenter DO for the Anniston Environmental Health Research Consortium. Blood pressure and hypertension in relation to levels of serum polychlorinated biphenyls in residents of Anniston, Alabama. *J Hypertension.* 28: 2053-2060..
329. Prasad A, Ahs M, Goncharov A and Carpenter DO (2010) Omega-3 and omega-6 fatty acids kill thymocytes and increase membrane fluidity. *The Open Cell Development & Biology Journal* 3: 1-8
330. Sergeev AV and Carpenter DO (2010) Increased hospitalizations for ischemic stroke with comorbid diabetes and residential proximity to source of organic pollutants: A 12-year population-based study. *Neuroepidemiology* 35:19.
331. Prasad A, Bloom M and Carpenter DO (2010) Role of calcium and ROS in cell death induced by polyunsaturated fatty acids in murine thymocytes. *J Cell Physiol.* 225: 829-836.

332. Sergeev AV and Carpenter DO (2010) Geospatial patterns of hospitalization rates for stroke with comorbid hypertension in relation to environmental sources of persistent organic pollutants: Results from a 12-year population-based study. *Environ Sci Pollut Res Int* 18: 576-585.
333. Brown D, Goncharov A, Paul E, Simonin H and Carpenter DO. (2010) The relationships between Adirondack lake pH and levels of mercury in yellow perch. *J Aquat Animal Health*. 22:280-290.
334. Gavidia T, Brune M-N, McCarty KM, Pronczuk J, Etzel R, Neira M, Carpenter DO, Suk WA, Arnold RG, Ha EH, and Sly PD (2010) Children's environmental health – from knowledge to action. *Lancet* 377:1134-1136.
335. Bushkin-Bedient S and Carpenter DO (2010) Benefits versus risks associated with consumption of fish and other seafood. *Rev Environ Health* 25: 161-191.
336. Goncharov A, Pavuk M, Foushee HR and Carpenter DO for the Anniston Environmental Health Consortium (2010) Blood pressure in relation to concentrations of PCB congeners and chlorinated pesticides. *Environ Health Perspect*. 119:319-325.
337. Yilmaz B, Sandal S and Carpenter DO (2012) PCB 9 exposure induces endothelial cell death while increasing intracellular calcium and ROS levels. *Environ Toxicol*.27: 185-191.
338. Sly PD, Arnold RG and Carpenter DO (2011) Environmental exposures in the era of climate change. *Rev Environ Health* 26: 1-4.
339. Carpenter DO (2011) Health effects of persistent organic pollutants: The challenge for the Pacific Basin and for the World. *Rev Environ Health* 26: 61-69.
340. Sergeev AV and Carpenter DO (2011) Increase in metabolic syndrome-related hospitalizations in relation to environmental sources of persistent organic pollutants. *Int J Environ Res Public Health* 8:762-776.
341. Carpenter DO, Miller PK, Waghiyi, Welfinger-Smith G (2011) Environmental contamination of the Yupik people of St. Lawrence Island, Alaska. *J Indigenous Res* In Press.
342. Carpenter DO (2010) Human health effects of EMFs: The cost of doing nothing. *IOP C Ser Earth Env* 10:1-6.
343. Kamalov J, Carpenter DO, Birman I (2011) Cytotoxicity of environmentally relevant concentrations of aluminum in murine thymocytes and lymphocytes. *J Toxicol*. Doi:10.1155/2011/796719.
344. Silbernagel S, Carpenter DO, Gilbert SG, Gochfeld M, Groth E, Hightower JM, Schiavone FM. (2011) Recognizing and preventing over exposure to methylmercury from fish and seafood consumption: Information for physicians. *J Toxicol*, 2011; doi:10.1155/2011/983072
345. Welfinger-Smith G, Minholz JL, Byrne S, Waghiyi V, Gologergen J, Kava J, Apatiki M, Ungott E, Miller PK, Arnason J and Carpenter DO. (2011) Organochlorine and metal contaminants in traditional foods from St. Lawrence Island, Alaska. *J Toxicol Environ Health A*. 74: 1-20.
346. Ahs M, Prasad A, Aminov Z and Carpenter DO (2011) Mechanisms of cell death of thymocytes induced by polyunsaturated, monounsaturated and trans-fatty acids. *J Cell. Biochem*. 112: 3863-3871.
347. Boberg E, Lessner L and Carpenter DO. (2011) The role of residence near hazardous waste sites containing benzene in the development of hematologic cancers in upstate New York. *Int J Occup Med Environ Health*. 24: 1-12..
348. Turyk ME, Bhazsar SP, Bowerman W, Boysen E, Clark M, Diamond M, Mergler D, Pantazopoulos P, Schantz S and Carpenter DO (2012) Risks and benefits of consumption of Great Lakes fish. *Environ Health Perspect*. 120: 11-18.
349. Ma J, Lessner L, Schreiber J and Carpenter DO (2009) Association between residential proximity to PERC dry cleaning establishments and kidney cancer in New York city. *J Environ Public Health* doi:10.1155/2009/183920.

350. Morse GS, Duncan G, Noonan C, Carrouette E, Santiago-Rivera A, Carpenter DO and Tarbell A (2011) Environmental toxins and depression in an American Indian Community. *J Indigen Res* 1: (1) Article 6. <http://digitalcommons.usu.edu/kicjir/vol1/iss1/6>.
351. Liu X, Lessner L and Carpenter DO (2012) Association between residential proximity to fuel-fired power plants and hospitalization rate for respiratory diseases. *Environ Health Perspect* 120: 807-810.
352. Ruzzin J, Lee D-H, Carpenter DO and Jacobs DR Jr. (2012) Reconsidering metabolic disease: The impact of persistent organic pollutants. *Atherosclerosis* 224: 1-3.
353. Florea A-M, Busselberg D and Carpenter D (2012) Metals and disease. *J Toxicol* 2012. Doi:10.1155/2012/825354.
354. Smolyaninova LV, Carpenter DO, Dergalev AA, Kulebyakin KY and Boldyrev AA (2013) Carnosine prevents necrotic and apoptotic death of rat thymocytes via ouabain sensitive Na/K-ATPase. *Cell Biochem Funct.* 20: 30-35.
355. Khwaja HA, Fatmi Z, Malashock D, Aminov Z, Siddique A and Carpenter DO (2012) Effect of air pollution on daily morbidity in Karachi, Pakistan. *J Local Global Health Science*, 2012:3 <http://dx.doi.org/10.5339/jlghs.2012.3>
356. Scudato RJ, Chiarenzelli FR, Miller PK, Alexander CR, Arnason J, Zamzow K, Zweifel K, Gologergen J, Kava J, Waghiyi V and Carpenter DO. (2012) Contaminants at Arctic formerly used defense sites. *J Local Global Health Science*. 2012:2 <http://dx.doi.org/10.5339/jlghs.2012.2>
357. Hoover E, Cook K, Plain R, Sanchez K, Waghiyi V, Miller P, Dufault R, Sislin C and Carpenter DO (2012) Indigenous peoples of North America: Environmental exposures and reproductive justice. *Environ Health Perspect.* 120: 1645-1649.
358. Pantazopoulos P, Sawyer JM, Turyk ME, Diamond M, Bhavsar SP, Mergler D, Schantz S, Ratnayake N and Carpenter DO (2012) Fatty acids in Great Lakes lake trout and whitefish. *J Great Lakes Res.* 39: 120-127.
359. Sly JL and Carpenter DO (2012) Special vulnerability of children to environmental exposures. *Rev Environ Health* 27: 151-157.
360. Bushkin-Bedient S and Carpenter DO (2013) Exposure to chemicals and radiation during childhood and risk for cancer later in life. *J Adolesc Health*, 52: S21-S29.
361. Surdu S, Fitzgerald EF, Bloom MS, Boscoe FP, Carpenter DO et al. (2013) Occupational exposure to ultraviolet radiation and risk of non-melanoma skin cancer in a multinational European study. *PLoS One*. In press.
362. Surdu A, Fitzgerald EF, Bloom MS, Boscoe FP, Carpenter DO et al. (2013) Occupational exposure to arsenic and risk of non-melanoma skin cancer in a multinational European study. *Int J Cancer*. 133:2182-2191.
363. Norman RE, Carpenter DO, Scott J, Brune MN and Sly PD (2013) Environmental exposure: an under-recognized contribution to noncommunicable diseases. *Rev Environ Health* 28: 59-65.
364. Sly PD, Chen L, Gangell CL, Scott J, Zubrick S, Whitehouse A and Carpenter DO (2012) Polychlorinated biphenyls but not chlorinated pesticides are associated with externalizing behavior in adolescents. *Organohal Comps*.
365. Grant KL, Carpenter DO, Sly LJ and Sly PD (2013) Environmental contributions to obesity and type 2 diabetes. *J Environ Immunol Toxicol* 1: 80-91.
366. Miller PK, Waghiyi V, Welfinger-Smith G, Byrne SC, Kava J, Gologergen J, Eckstein L, Scudato R, Chiarenzelli J, Carpenter DO and Seguinot-Medina S. (2013) Community-based participatory research projects and policy engagement to protect environmental health on St. Lawrence Island, Alaska. *Int J Circumpolar Health* 72: 1-11. <http://dx.doi.org/10.3402/ijch.v72i10.21656>.

367. Gore AC, Balthazart J, Bikle D, Carpenter DO, Crews D et al. (2013). Policy decisions on endocrine disruptors should be based on science across disciplines: a response to Dietrich et al. *Eur J Endocrinol* 169: E1-E4.
368. Nayebare SR, Wilson LR, Carpenter DO, Dziwulski DM, and Kannan K (2014) A review of potable water accessibility and sustainability issues in developing countries – case study of Uganda. *Rev Environ Health*. In press.
369. Carpenter DO (2013) Human disease resulting from exposure to electromagnetic fields. *Rev Environ Health* 28: 159-172.
370. Aminov Z, Haase RF, Pavuk M and Carpenter DO (2013) Analysis of the effects of exposure to polychlorinated biphenyls and chlorinated pesticides on serum lipid levels in residents of Anniston, Alabama. *Environ Health* 12: 108
371. Musoke D, Carpenter D, Kasasa S, Bazeyo W and Ssempebwa JC (2013) Water, sanitation and hygiene status of two urban slums in Uganda: a baseline survey. *Environ Health Internat* 14: 24-36.
372. Carpenter DO (2014) Environmental exposure in indigenous communities: an international perspective. *Rev Environ Health* 29: 3-4.
373. Falfushynska HI, Gnatyshyna LL, Osadchuk OY, Farkas A, Vehovszky A, Carpenter DO, Gyori J and Stoliar OB. (2014) Diversity of the molecular responses to separate wastewater effluents in freshwater mussels. *Comp Biochem Physiol*, 164: 51-58.
374. Neff MR, Bhavsar SP, Ni F, Carpenter DO, Drouillard K, Fisk AT, and Arts MT (2014) Risk-benefit of consuming Lake Erie fish. *Environ Res*. 134: 57-65.
375. Wang H, Liddell CA, Coates MM, Mooney MD et al. (2014) Global, regional, and national levels of neonatal, infant, and under-5 mortality during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* [http://dx.doi.org/10.1016/S0140-6736\(14\)60497-9](http://dx.doi.org/10.1016/S0140-6736(14)60497-9).
376. Lu X, Lessner L and Carpenter DO (2014) Association between hospital discharge rate for female breast cancer and residence in a zip code containing hazardous waste sites. *Environ Res*. 134: 375-381.
377. Aminov Z, Haase R, Olson JR, Pavuk M and Carpenter DO (2014) Racial differences in levels of serum lipids and effects of exposure to persistent organic pollutants on lipid levels in residents of Anniston, Alabama. *Environ Internat* 73: 216-223..
378. Carpenter DO (2014) Excessive exposure to radiofrequency electromagnetic fields may cause the development of a syndrome of electro-hypersensitivity. *Alt Therap* 20: 80-82379.
379. Macey GP, Breech R, Chernaik M, Cox C, Larson D, Thomas D and Carpenter DO (2014) Air concentrations of volatile compounds near oil and gas production: a community-based exploratory study. *Environ Health* 13: 82.
380. Surdu S, Fitzgerald EF, Bloom MS, Boscoe FP, Carpenter DO, Haase RF, Gurzau E, Rudnai P, Koppova K, Vahter M, Leonardi G, Goessler W, Kumar R and Fletcher T (2014) Polymorphisms in DNA repair genes XRCC1 and XRCC3, occupational exposure to arsenic and sunlight, and the risk of non-melanoma skin cancer in a European case-control study. *Environ Res* 134: 382-389.
381. Chacko A, Carpenter DO, Callaway L and Sly PD (2015) Early life risk factors for chronic non-respiratory diseases. *Eur Resp* 45: 244-259
382. GBD 2013 Mortality and Causes of Death Collaborators (2014) Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: A systematic analysis for the Global Burden of Disease. *Lancet* [http://dx.doi.org/10.1016/S0140-6736\(14\)61682-2](http://dx.doi.org/10.1016/S0140-6736(14)61682-2).

383. Brune M-N, Goldizen FC, Neira M, van den Berg M, Lewis N, King M., Suk WA, Carpenter DO, Arnold RG and SlyPD (2013) Health effects of exposure to e-waste. *Lancet Global Health* 1(2): e70.
384. Goodson, W III, Lowe L, Carpenter D, Gilbertson M, Ali A et al. (2015). Assessing the carcinogenic potential of low dose exposures to chemical mixtures in the environment: The challenge ahead. *Carcinogenesis*. 36, Suppl 1, S254-S296.
385. Carpenter DO (2015) Exposure to and health effects of volatile PCBs. *Rev Environ Health* 30:81-92..
386. Byrne S, Miller P, Vaghiyi V, Buck CL, von Hippel F and Carpenter DO (2015) Persistent organochlorine pesticide exposure related to a formerly used defense site on St. Lawrence Island, Alaska: Data from sentinel fish and human sera. *J Toxicol Environ Health A* 78: 976-992.
387. GBD 2013 Disease and Injury Incidence and Prevalence Collaborators (2015) Global, regional, and national incidence, prevalence and YLDs for 301 acute and chronic diseases and injuries for 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*
388. Pavuk M, Olson JR, Wattigney WA, Dutton ND, Sjodin A et al. (2014) Predictors of serum polychlorinated biphenyl concentrations in Anniston residents. *Sci Total Environ* 496: 624-634.
389. Sly PD, Neira M, Collman G, Carpenter DO, Landrigan PJ, van den Berg M, Barriga FD, Ruchirawat M, Laborde A, Pascale A, Heacock M, Calmau MT and Suk WA (2014) Networking to advance progress in children's environmental health. *Lancet Global Health* e129-130.
390. Wang, G, Liddell CA, Coates MM, Mooney MD, Levitz CE et al. (2014) Global, regional, and national levels of neonatal, infant, and under-5 mortality during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 384: 957-979.
391. Global Burden of Disease Cancer Collaboration (2015) The Global Burden of Cancer. *JAMA Oncol* 1(4): 505-527. Doi:10.1001/jamaoncol.2015.0735.
392. DeNicola E, Aburizaiza OS, Siddique A, Khwaja H and Carpenter DO (2015) Obesity and public health in the Kingdom of Saudi Arabia. *Rev Environ Health* 30: 191-205.
393. Bwesigye DA, Loneck BM, Sherman BR and Carpenter DO (2015) Theoretical application assessing adaptation of District Health Information System (DHIS 2) for HIZ/AIDS surveillance in Uganda. *Health Sys Policy Res* 2: 1-7.
394. Lombardo JP, Berger DF, Hunt A and Carpenter DO (2015) Inhalation of polychlorinated biphenyls (PCB) produces hyperactivity in rats. *J Toxicol Environ Health A* 00: 1-12. DOI 10.1080/15287394.2015.1060913.1395.
395. GBD (2015) Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death. 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 385: 117-171.
396. GBD (2015) Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic disease and injuries in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 388: 743-800.
397. Carpenter DO (2015) The microwave syndrome or electro-hypersensitivity: Historical background. *Rev Environ Health*, 30: 217-222.
398. Saxton DI, Brown P, Segininot-Medina S, Eckstein L, Carpenter DO, Miller P and Waghiyi V (2015) Environmental health and justice and the right to research: institutional review board denials of community-based chemical biomonitoring of breast milk. *Environ Health* DOI: 10.1186/s12940-015-0076-x
399. DeNicola E, Aburizaiza OS, Siddique A, Khwaja H and Carpenter DO (2015) Climate change and water scarcity: The case of Saudi Arabia. *Ann Global Health* 81: 342-353.

400. GBD (2015) Global, regional, and national levels of neonatal, infant and under-5 mortality during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 384: 957-979.
401. GBD (2015) Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: a systematic analysis for the Global of Disease Study 2013. *Lancet* 386: 2287-2323.
402. GBD (2015) Global, regional and national incidence, prevalence and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 386: 743-800.
403. GBD (2015) Global, regional, and national disability-adjusted life years (DALYs) for 306 diseases and injuries and healthy life expectancy (HALE) for 188 countries, 1990-2013: quantifying the epidemiological transition. *Lancet* 386: 2145-2101.
404. Portier CJ, Armstrong B, Baguley BC, Baur X, Belyaev I et al. (2016) Differences in the carcinogenic evaluation of glyphosate between the International Agency for Research on Cancer (IARC) and the European Food Safety authority (EFSA). *J Epidemiol Comm Health*. 10.1136/jech-2015-207005.
405. Carpenter DO (2016) Hydraulic fracturing for natural gas: Impact on health and environment. *Rev Environ Health*, 31: 47-52.
406. Heacock M, Kelly CB, Asante KA, Birnbaum LS, Bergman AL et al. (2016) E-Waste and harm to vulnerable populations: A growing Global problem. *Environ Health Perspect*. 124: 550-555.
407. Aminov Z, Haase R and Carpenter DO (2016) Diabetes in Native Americans: Elevated risk as a result of exposure to polychlorinated biphenyls (PCBs). *Rev Environ Health*, 31: 115-120
408. Mukama T, Ndejjo R, Musoke D, Musinguzi G, Halaga AA, Carpenter DO and Ssempebwa J (2016) Practices, concerns and willingness to participate in solid waste management in two urban slums in central Uganda. *J Environ Public Health*. <http://dx.doi.org/10.1155/2016/6830163>.
409. Aminov Z, Haase R, Rej R, Schymura MJ, Santiago-Rivera A, Morse G, DeCaprio A et al. (2016) Diabetes prevalence in relation to serum concentrations of polychlorinated biphenyl (PCB) congener groups and three chlorinated pesticides in a Native American population. *Environ Health Perspect*. 124: 13761383.
410. Suk WA, Ahanchian H, Asante KA, Carpenter DO, Diaz-Barriga F et al. (2016) Environmental pollution: an under-recognized threat to children's health, especially in low- and middle-income countries. *Environ Health Perspect* 124: A41-A45.
411. GBD Mortality and Causes of Death Collaborators (2016) Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015. *Lancet* 388: 1459-1544.
412. Nayebare SR, Aburizaiza OS, Khwaja HA, Siddique A, Hussain MM, Zeb J, Khatib F, Carpenter DO and Blake DR (2016) Chemical characterization and source apportionment of PM_{2.5} in Rabigh, Saudi Arabia. *Aerosol Air Qual Res* 16: 3114-3129
413. Sly PD, Carpenter DO, Van den Berg M, Stein RT, Landrigan PJ, Brune-Drisse M-N and Suk W (2016) Health consequences of environmental exposure: Causal thinking in global environmental epidemiology. *Ann Global Health* 82: 3-9.
414. Suk W, Ruchirawat M, Stein RT, Diaz-Barriga F, Carpenter DO, Neira M and Sly PD (2016) Health consequences of environmental exposures in early life: Coping with a changing world in the post-MDG era, *Ann Global Health* 82: 20-27
415. Gallo MV, Ravenscroft J, Carpenter DO, Frye CA, Akwesasne Task Force on the Environment, Cook B and Schell LM (2016) Endocrine disrupting chemicals and ovulation: Is there a relationship? *Environ Res*. 151: 410-418.

416. DeNicola E, Aburizaize OS, Siddique A, Khwaja H and Carpenter DO (2016) Road traffic injury as a major public health issue in the Kingdom of Saudi Arabia: A review. *Front Public Health* 4:215.
417. GBD 2015 Child Mortality Collaborators (2016) Global, regional, national, and selected subnational levels of stillbirths, neonatal, infant, and under-5 mortality, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 388: 1725-1774.
418. GBD 2015 Mortality and Causes of Death Collaborators (2016) Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 388: 1459-1544.
419. GBD Disease and Injury Incidence and Prevalence Collaborators (2016) Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 388: 1545-1598.
420. GBD 2015 Risk Factors Collaborators (2016) Global, regional and national comparative risk assessment of 79 behavioral, environmental and occupational, and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 388: 1659-1724.
421. Fazzo L, Bianchi F, Carpenter D, Martuzzi M and Comba P (2017) Hazardous waste: A challenge for public health. *Public Health Panorama* 3: 247-252.
422. Nayebare SR, Aburizaiza OS, Siddique A, Carpenter DO, Zeb J, Aburizaiza AJ, Pantea C, Hussain MM and Khwaja HA (2017) Association of fine particulate air pollution with cardiopulmonary morbidity in Western Saudi Arabia. *Saudi Med J* 38: 179-189.
423. Lee D-H, Jacobs DR Jr., Park HY and Carpenter DO (2017) A role of low dose chemical mixtures in adipose tissue in carcinogenesis. *Environ Int* 108: 170-175.
424. Byrne S, Seguinot-Medina S, Miller P, Waghiyi V, von Hippel FA, Buck CL and Carpenter DO (2017) Exposure to polybrominated diphenyl ethers and perfluoroalkyl substances in a remote population of Alaska Natives. *Environ Poll* 231: 387-395.
425. Zhang X-Y, Carpenter DO, Song Y-J, Chen P, Qin Y, Wei N- Y and :om S-C(2017) Application of the IEUBK model for linking childrens' blood lead with environmental exposure in a mining site, south China. *Environ Poll* 231: 971-978.
426. Fatmi SS, Zehra R and Carpenter DO (2017) Powassan Virus: A new re-emerging tick-borne disease. *Frontiers in Public Health* 5:342.
427. Asad N and Carpenter DO (2017) Effects of climate change on spread of zika virus. *Rev Environ Health*. 28: 31-42.
428. von Hippel F, Miller P, Carpenter D, Dillon D, Smayda L, Katsiadaki I, Titus T, Batzel P, Postlethwait J, and Buck C (2017) Endocrine disruption and differential gene expression in sentinel fish on St. Lawrence Island, Alaska: Health implications for indigenous residents. *Environ Pollution* 234: 279-287.
429. Byrne SC, Miller P, Seguinot-Medina S, Waghiyi V, Buck CL, von Hippel FA and Carpenter DO (2018) Associations between serum polybrominated diphenyl ethers and thyroid hormones in a cross-sectional study of a remote Alaska Native population. *Sci Reports* 8: 2198.
430. GBD Study Group 2016 (2017) Global, regional, and national disability-adjusted life-years (DALYs) for 333 diseases and injuries and healthy life expectancy (HALE) for 195 countries and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study, 2016. *Lancet* 390: 1260-1344.
431. GBD Study Group 2016 (2017) Global, regional, and national under-5 mortality, adult mortality, age-specific mortality and life expectancy, 1970-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 390: 1084-1160.

432. GBD Study Group 2016 (2017) Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 390; 1211-1259.
433. Moradi-Lakeh, ElBcheraoui C, Khalil I, Charara R et al. (2017) Diabetes mellitus and chronic kidney disease in the Eastern Mediterranean Region: findings from the Global Burden of Disease 2015 study. *Int J Public Health* doi:10.1007/s00038-017-1014-1.
434. GBD 2015 Chronic Respiratory Disease Collaborators (2017) Global, regional, and national deaths, prevalence, disability-adjusted life years, and years lived with disability for chronic obstructive pulmonary disease and asthma, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet Respir Med* 5: 691-706.
435. Musoke D, Ndeejjo R, Halage AA, Kasasa S, Ssempebwa J and Carpenter DO (2017) Drinking water supply, sanitation and hygiene promotion interventions in two slum communities in central Uganda. *J Environ Public Health*, doi:10.1155/2018/3710120.
436. Gallo MV, Ravenscroft J, Carpenter DO, Schell LM and the Akwesasne Task Force on the Environment. (2018) Persistent organic pollutants as predictors of increased FSH:LH ratio in naturally cycling, reproductive age women. *Environ Res* 164: 556-564.
437. Bandara P and Carpenter DO (2018) Planetary electromagnetic pollution: It is time to assess its impact on life on earth. *Lancet Planetary Health*, 2: e512-e514.
438. Byrne SC, Miller P, Seguinot-Medina S, Waghiyi V, Buck CL, von Hippel FA and Carpenter DO (2018) Exposure to perfluoroalkyl substances and associations with serum thyroid hormones in a remote population of Alaska Natives. *Environ Res* 166: 537-543.
439. Belpomme D, Hardell L, Belyaev I, Burgio E and Carpenter DO (2018) Thermal and non-thermal health effects of non-ionizing radiation: an international perspective. *Environ Poll* 242: 643-658.
440. Heacock M, Trottier B, Adhikary S, Asante KA, Basi M et al. (2018) Prevention-intervention strategies to reduce exposure to e-waste. *Rev Environ Health* 33: 219-228..
441. Nayebare SR, Aburizaiza OS, Siddique A, Carpenter DO, Pope AC, Hussain MM, Zeb J, Aburizaiza AJ and Khwaja HA (2018) Fine particulate (PM_{2.5}) air pollution is a major risk factor for cardiopulmonary morbidity in Jeddah, Saudi Arabia. *Science of the Total Environment*, In press.
442. GBD 2016 Disease and Injury Incidence and Prevalence Collaborators (2018) Global, regional, and national incidence, prevalence and year lived with disability for 328 disease and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 390: 1211-1259.
443. Nayebare SR, Aburizaiza OS, Siddique A, Carpenter DO, Hussain MM, Zeb J, Aburizaiza AJ and Khwaja HA (2018) Ambient air quality in the holy city, Makkah: a source apportionment with elemental enrichment factors (EFs) and factor analysis (PMF). *Environ Poll.* 243: 1791-1801.
444. Malashock D, Khwaja H, Fatmi Z, Siddique A, Lu Y, Lin S, and Carpenter DO (2018) Short-term association between black carbon exposure and cardiovascular diseases in Pakistan's largest megacity. *Atmosphere*, In press.
445. Sly PD, Trottier B, Carpenter D, Cha'on U, Cormier S, Galluzzo B et al. (2019) Children's environmental health in south and southeast Asia: Networking for better child health outcomes. *Ann global Health* 85: 1-11. DOI:<https://doi.org/10.5334/aogh.2403>.
447. Fatmi SS, Zehra R and Carpenter DO (2018) Dietary magnesium intake and heart health. *J Food Nutri Metab* <http://dx.doi.org/10.31487/j.JFNM.2018.01.001>.
448. Lurie K, Nayebare SR, Fatmi Z, Carpenter DO, Siddique A, Malashock D, Khan K, Zeb J, Hussain MM, Khatib F, Khwaja HA (2019) PM 2.5 in a megacity of Asia (Karachi): Source apportionment and health effects. *Atmospheric Environment* 202: 223-233.

449. GBD2016 (2019) Global, regional, and national burden of Alzheimer's disease and other dementias, 1990-2026: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Neurol* 18: 88-106.
450. Zehra R, Fatmi S and Carpenter DO (2018) Pathophysiological mechanisms of mercury's effect on thyroid gland. *Int J Thyroid Disorders Therapy* 1(1): 1-6.
451. Russo PN and Carpenter DO (2019) Occupational exposures to carcinogens in an industry: Identifying and communicating chemical risk. *Int J Poll Res* IJPE-105. DOI:10.29011/IJPR-105.000005.
452. Russo PN and Carpenter DO (2019) Air emissions from natural gas facilities in New York State. *Internat J Environ Res Pub Health* 16: 1591: doi:10.3390/ijerph16091591.
453. Lu Y, Lin S, Fatmi Z, Malashock D, Hussain MM, Siddique A, Carpenter DO, Lin Z and Khwaja HA (2019) Assessing the association between fine particulate matter (PM2.5) constituents and cardiovascular diseases in a mega-city of Pakistan. *Environ Poll* 252: 1412-1422.
454. Carpenter DO (2019) Extremely low frequency electromagnetic fields and cancer: How source of funding affects results. *Environ Res* 178: 108688.
455. Kerr K, Morse G, Graves D, Zuo F, Lipowicz A and Carpenter DO (2019) A detoxification intervention for Gulf War Illness: A pilot randomized controlled trial. *Int J Environ Res Public Health* 16, 4143.
456. Bandara P and Carpenter DO (2019) Causes of Cancer: Perceptions vs. Scientific Evidence. *Eur J Cancer* <https://doi.org/10.1016/j.ejca.2019.08.016>.
457. Zheng G, Miller P, von Hippel FA, Buck CL, Carpenter DO and Salamova A (2020) Legacy and emerging semi-volatile organic compounds in sentinel fish from an Arctic formerly used defense site in Alaska. *Environ Poll* 259: 113872.
458. Aminov Z, Carpenter DO and the Akwesasne Task Force on the Environment (2020) Serum concentrations of persistent organic pollutants and the metabolic syndrome in Akwesasne Mohawks, a Native American community. *Environ Poll* 260: 114004
459. Graves DF, Morse GS, Kerr K and Carpenter DO (2020) Review of study of novel treatment of Gulf War Illness. *J Ment Health Clin Psychol* 4: 1-4.
460. Goodson WH III, Lowe L, Gilbertson M and Carpenter DO (2020) Testing the low dose mixtures hypothesis from the Halifax Project. *Rev Environ Health*. In press.
461. Graves DF, Morse GS, Kerr K, and Carpenter DO (2020) A pilot study to examine psychological and neuropsychological outcomes and a novel detoxification program for Gulf War Illness. *Mil Med* In press.
462. Mohammad A, Alshamari T, Adeyeye T, Lazariu V, McNutt LA and Carpenter DO (2020) A comparison of risk factors for osteo- and rheumatoid arthritis using NHANES data. *Prev Med Rep*, 20: 101242.
463. Weinstein B, da Silva AR, Kouzoukas DE, Bose T, Kim GJ, Correa PA, Pondugula S, Lee YJ, Kim J and Carpenter DO (2021) Precision mapping of COVID-19 vulnerable locales by epidemiological and socioeconomic risk factors, developed using South Korean data. *Int J Environ Res Public Health* 18: 604.
464. Emeny RT, Carpenter DO and Lawrence DA (2021) Health disparities: Intracellular consequences of social determinants of health. *Toxicol Appl Pharmacol* 416: 115444 <https://doi.org/10.1016/j.taap.2021.115444>.
465. Weinstein B, Muresan B, Solano S, Vaz de Macedo A, Lee Y, Kim GJ, Camargo C, Su YC, Henriquez-Luthje G, Ahn YS and Carpenter DO (2021) Comparative efficacy and safety of experimental versus approved CAR T-cell therapies in large B-cell lymphoma using matching

- adjusted indirect comparison: A systematic review and meta-analysis. *Principal and Practice of Clinical Research Journal*, In press.
466. Bella D, Carpenter DO and the Akwesasne Task Force on the Environment (2021) Interactions among thyroid hormones and serum lipid levels in association with PCB exposure in the Mohawk Akwesasne population. *Environ Res* 200: 111334
 467. Moslehi R, Stagnar C, Srinivasan S, Radziszowski P and Carpenter DO (2020) The possible role of arsenic and gene-arsenic interactions in susceptibility to breast cancer: a systematic review. *Rev Environ Health* <https://doi.org/10.1515/reveh-2020-0080>.
 468. Belpomme D, Carlo GL, Irigaray P, Carpenter DO, Hardell L, Kundi M Belyaev I et al. (2021) The critical importance of molecular biomarkers and imaging in the study of electrohypersensitivity. A scientific consensus international report. *Int J Mol Sci* 22: 7321.
 469. Saheb T, Sabeb, T and Carpenter DO (2021) Mapping research strands of ethics of artificial intelligence in healthcare: A bibliometric and content analysis. *Computers Biol Med* 135: 104660..
 470. Lebbie T, Omosehin O, Asante, KA, Fobil JN, Brune-Drisse MN, Suk WA, Sly PD, Gorman JF and Carpenter DO (2021) E-Waste in Africa: A serious threat to the health of children. *Int J Environ Res Public Health* 18: 8488 <https://doi.org/10.3390/ijerph181648488>.
 471. Hori N, Xu Z, Akaike N, Tan Y and Carpenter DO (2021) Physiological and pharmacological studies on cervical motor neurons in slices prepared from neonatal and aged mice. *J Brain Nerves*. <http://dx.doi.org/10.31487/j.JBN.2021.01.02>.
 472. Hori N, Tan Y, Xu Z, Akaike N and Carpenter DO (2021) Electrophysiological and morphological studies of SOD1 transgenic mice: An animal model of ALS. *J Brain Nerves* <http://dx.doi.org/10.31487/j.JBN.2021.01.03>.
 - 473.
 474. Begum T and Carpenter DO (2021) Health effects associated with phthalates activity on nuclear receptors. *Rev Environ Health*, In press.
 475. Weinstein B, Muresan B, Solano S, Vaz de Macedo A, Lee Y, Su YC, Ahn Y, Henriquez G, Camargo C, Kim G and Carpenter DO (2021) Efficacy and safety of innovative experimental chimeric antigen receptor (CAR) T-cells versus axicabtagene ciloleucel (Yescarta) for the treatment of relapsed/refractory large B-cell lymphoma (LBCL): Matching adjusted indirect comparisons (MAICs) and Syst. *Innovations in Pharmacology*, 12 (4): Article 18.
 476. Parvez SM, Johan F, Brune MN, Gorman J, Rahman MJ, Carpenter D, Islam Z, Raahman M, Aich N, Knibbe LD and Sly PD (2021) Health consequences of exposure to e-waste: an updated systematic review. *The Lancet Planetary Health* 4: e905-e920.
 477. Weinstein B, da Silva A and Carpenter DO (2022) Exocrine pancreatic cancer and living near to waste sites containing hazardous organic chemicals, New York State, USA. An 180year population-based study. *Int J Occup Med Environ Health* 35 (4). <https://doi.org/10.13075/ijomed.1896.01886>.
 478. Casey A, Bush B and Carpenter DO (2022) PCBs in indoor air and human blood in Pittsfield, Massachusetts. *Chemosphere* 293: 13551.
 479. Begum T, Morse GS, Buchwald D and Carpenter DO (2021) Assessing the psychometric properties of the WHO-DAS 2.0 in an American Indian Community. *J Indig Res* 9 (12): Article 12.:
 480. Sasaki N and Carpenter D (2022) Associations between metal exposures and cognitive function in American older adults. *Int J Environ Res Public Health* 19: 2327. <https://doi.org/10.3390/ijerph19042327>.
 481. Jordan-Ward R, von Hippel FA, Zheng G, Salamova A, Dillon D, Gologergen J, Immingan T, Dominguez E, Miller P, Carpenter DO, Postlethwait JH, Byrne S and Buck CL (2022) Elevated

- mercury and PCB concentrations in Dolly Varden (*Salvelinus malma*) collected near a formerly used defense site on Sivuqaq, Alaska. *Sci Total Environ* s.
482. Begum TF, Carpenter DO and the Akwesasne Task Force on the Environment (ATFE) (2022) Review of environmental contamination at Akwesasne and associated health outcomes. *J Indig Res* 10: Issue 2022, Article1.
 483. Nayebare N, Aburizaiza OS, Siddique A, Hussain MM, Zeb J, Khatib F, Carpenter DO, Blake DR and Khwaja HA (2022) Understanding the sources of ambient fine particulate matter (PM2.5) in Jeddah, Saudi Arabia. *Atmosphere* 1681315.
 484. Moyebi OD, Frank BP, Tang S, LaDuke G, Carpenter DO and Khwaja HA (2022) Ambient size-segregated particulate matter characterization from a Port in Upstate New York. *Atmosphere* 13: 984. <https://doi.org/10.3390/atnis13060984>.
 485. Mohammad A and Carpenter DO (2022). PCBs and dioxins either cause or make worse both osteo and rheumatoid arthritis. *Med Res Arch* 10(7) <https://doi.org/10.1813/mra.v107.2900>.
 486. Madani NA and Carpenter DO (2022) Effects of glyphosate and glyphosate-based herbicide like Roundup™ on the mammalian nervous system. *Environ Res* In press.
 487. Sasaki N, Jones LE, Morse GS, Carpenter DO and on behalf of Akwesasne Task Force on the Environment (2022) Mixture effects of polychlorinated biphenyls (PCBs) and three organochlorine pesticides on cognitive function in Mohawk adults at Akwesasne. *Int J Environ Res Public Health* 20: 1148: <https://doi.org/10.3390/ijerph200211148>.
 488. Moyebi OD, Fatmi Z, Carpenter DO, Santoso M, Siddique A, Khan K, Zeb J, Hussain MM and Khwaja HA (2023) Fine particulate matter and its chemical constituents' levels: A troubling environmental human health situation in Karachi, Pakistan. *Sci Total Environ* 868: 161474.
 489. Moyebi OD, Sannoh F, Fatmi Z, Siddique A, Khan K, Zeb J, Hussain MM, Carpenter DO and Khwaja HA (2023) State of gaseous air pollutants and resulting health effects in Karachi, Pakistan. *Environ Monit Assess* 195: 266. <https://doi.org/10.1007/s10661-022-10787-1>.
 490. Madani NA and Carpenter DO (2023) Patterns of emergency room visits for respiratory diseases in New York State in relation to air pollution, poverty and smoking. *Int J Environ Res Public Health* 20: 3267.

Books:

1. Cellular Pacemakers I: Mechanisms of Pacemaker Generation, David O. Carpenter, editor; John Wiley & Sons, New York, 1982.
2. Cellular Pacemakers II: Function in Normal and Disease States, David O. Carpenter, editor; John Wiley & Sons, New York 1982.
3. Biologic Effects of Electric and Magnetic Fields, Volume I: Sources and Mechanisms of Biologic Effects, David O. Carpenter and Sinerik Ayrapetyan, editors; Academic Press, California, 1994.
4. Biologic Effects of Electric and Magnetic Fields, Volume II: Beneficial and Harmful Effects, David O. Carpenter and Sinerik Ayrapetyan, editors; Academic Press, California, 1994.
5. Environmental Challenges in the Pacific Basin, David O. Carpenter, ed. New York Academy of Sciences, Vol 1140, 457 pp, 2008.
6. Effects of Persistent and Bioactive Organic Pollutants on Human Health, David O. Carpenter, ed. Wiley-Blackwell, In press, 2013.

Reviews and Book Chapters:

1. Carpenter, D.O. Ionic mechanisms and models of endogenous discharge of *Aplysia* neurons. Proceedings of the Symposium on Neurobiology of Invertebrates: Mechanisms of Rhythm Regulation. Tihany, Hungary, August 2-5, 1971, Hungarian Academy of Sciences, pp. 35-58, 1973.
2. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Measurements of intracellular conductivity in *Aplysia* neurons: Evidence for organization of water and ions. Ann. NY Acad. Sci., 204:502-533, 1973.
3. Carpenter, D.O., Hubbard, J.H., Humphrey, D.R., Thompson, H.K. and Marshall, W.H. CO₂ effects on nerve cell function. In: Topics in Environmental Physiology and Medicine: Carbon Dioxide and Metabolic Regulation. (Eds.: G. Nahas and K.A. Schaefer), Springer-Verlag, New York, pp. 49-62, 1974.
4. Parmentier, J. and Carpenter, D.O. Blocking action of snake venom neurotoxins at receptor sites to putative central nervous system transmitters. In: Animal, Plant and Microbial Toxins (Eds.: A. Ohaska, K. Hayashi, and Y. Sawai), Plenum Press, London, Vol. 2, pp. 179-191, 1976.
5. Pierau, Fr.-K. and Carpenter, D.O. Metabolic control of peripheral temperature receptors in the scrotal skin of the rat. Israel J. Med. Sci., 12:1044-1046, 1976.
6. Carpenter, D.O. Membrane Excitability: In: Mammalian Cell Membranes Vol. 4, Membranes and Cellular Functions, (Eds.: G.A. Jamieson and D.M. Robinson), Butterworth & Co., London, pp. 184-206, 1977.
7. Carpenter, D.O., Myers, P.R., Shain, W., Sinback, C.N. and Swann, J.W. Interchangeable association of neurotransmitter receptors and ionophores in vertebrate and invertebrate cells. Proc. Symposium: "Iontophoresis and Transmitter Mechanisms in the Mammalian Central Nervous System", Cambridge, England, Raven Press, pp. 203-205, 1978.
8. Carpenter, D.O., McCreery, M.J., Woodbury, C.M. and Yarowsky, P.J. Modulation of endogenous discharge in neuron R-15 through specific receptors for several neurotransmitters. In: Abnormal Neuronal Discharges, (Eds: N. Chalazonitis and M. Boisson), Raven Press, New York, pp. 189-203, 1978.
9. Tsien, R.W. and Carpenter, D.O. Ionic mechanisms of pacemaker activity in cardiac purkinje fibers. Fed. Proc., 37:2127-2131, 1978.
10. Keabian, P.R., Keabian, J.W. and Carpenter, D.O. Serotonin causes accumulation of cyclic AMP in *Aplysia* heart. The Proceedings of the Fourth International Catecholamine Symposium, (Eds: E. Usdin and I. Kopin), Pergamon Press, New York, pp. 1167-1169.
11. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Direct and modulatory actions of thyrotropin-releasing hormone (TRH) in sensorimotor cortex. Proc. EMBO Workshop on Drug Receptors in the Central Nervous System, Weizman Institute of Science, Rehovot, Israel, February 10-14, 1980.
12. Carpenter, D.O. Ionic and metabolic bases of neuronal thermosensitivity. Fed. Proc., 40:2808-2813, 1981.
13. Carpenter, D.O. and Reese, T.S. Chemistry and Physiology of Synaptic Transmissions. In: Basic Neurochemistry, 3rd Edition, (Eds.: Siegel, Albers, Agranoff and Katzman), Little, Brown and Company, pp. 161-168, 1981.
14. Shain, W. and Carpenter, D.O. Mechanisms of synaptic modulation. Intl. Rev. Neurobiol., 22:205-247, 1981.
15. Wiederhold, M.L. and Carpenter, D.O. Possible Role of Pacemaker Mechanisms in Sensory Systems. In: Cellular Pacemakers II: Function in Normal and Disease States, (Ed.: D.O. Carpenter), John Wiley & Sons, New York, pp. 27-58, 1982.
16. Carpenter, D.O. The generator potential mechanism in cold afferents may be an electrogenic sodium pump. Workshop on Mechanisms of Thermal Regulations. J. Therm. Biol., 387-390, 1983.

17. Carpenter, D.O. and Gregg, R.A. Functional significance of electrogenic pumps in neurons. In: Electrogenic transport: Fundamental Principles and Physiological Implications, (Eds.: M. Blaustein and M. Liebermann), Raven Press, pp. 253-270, 1984.
18. Carpenter, D.O., Briggs, D.B. and Strominger, N. Behavioral and electrophysiological studies of peptide-induced emesis in dogs. Fed. Proc., 43:16-18, 1984.
19. Coyle, J.T., Blakeley, R.D., Zaczek, R., Ory-Lavallee, L., Koller, K., French-Mullen, J.M.H. and Carpenter, D.O. Acidic peptides in brain: Do they act at putative glutamatergic synapses. In: Excitatory Amino Acids and Epilepsy, (Eds.: Y. Ben-Ari and R. Schwarcz), Plenum Press, New York, pp. 375-384.
20. Carpenter, D.O., French-Mullen, J.M.H., Hori, N., Sinback, C.N. and Shain, W. Segregation of synaptic function on excitable cells. In: Neural Mechanisms of Conditioning, (Eds.: D. Alkon and C.D. Woody), Plenum Press, NY, pp. 355-369, 1985.
21. Carpenter, D.O. and Hall, A.F. Responses of Aplysia cerebral ganglion neurons to leucine enkephalin. In: Comparative Aspects of Opioid and Related Neuropeptide Mechanisms, (Eds.: M. Leung and G. Stefano), CRC Press, pp. 49-57.
22. Zaczek, R., Koller, K., Carpenter, D.O., Fisher, R., French-Mullen, J.M.H. and Coyle, J.T. Interactions of acidic peptides: Excitatory amino acid receptors. In: Excitatory Amino Acids, (Ed.: P.J. Roberts), Macmillan, London, 1987.
23. Carpenter, D.O. Central nervous system mechanisms in deglutition and emesis. In: Handbook of Physiology, Section 6: The Gastrointestinal System. Vol. I, Motility and Circulation, (Ed.: J.D. Wood), American Physiological Society, Chapter 18, pp. 685-714, 1989.
24. Carpenter, D.O., Briggs, D.B. and Strominger, N. Mechanisms of radiation-induced emesis in the dog. Pharmacol. Ther., 39:367-371, 1988.
25. Carpenter, D.O. Comparative biology of neurotransmitter functions. Biology International, 15:2-9, 1987.
26. Carpenter, D.O. Electromagnetic Fields: Do We Know Enough to Act? In: Health and Environmental Digest, Vol. 2, pp. 3-4, 1988.
27. Carpenter, D.O. The New York State Power Lines Project: Summary and Conclusions. In: 20th Annual National Conference on Radiation Control, CRCPD Publication 88-6, Nashville, Tennessee, May 15-19, 1988, pp. 399-409.
28. S.-Rozsa, K., Carpenter, D.O., Stefano, G.B. and Salanki, J. Distinct responses to opiate peptides and FMRFamide on B-neurons of the Aplysia cerebral ganglia. In: Comparative Aspects of Neuropeptide Function, (Eds. E. Florey and G.B. Stefano), Manchester University Press, Chapter 6, pp. 73-86, 1991.
29. Carpenter, D.O. A common mechanism of excitation of area postrema neurons by several neuropeptides, hormones and monoamines. In: Comparative Aspects of Neuropeptide Function, (Eds. E. Florey and G.B. Stefano) Manchester University Press, Chapter 21, pp. 260-270, 1991.
30. Carpenter, D. O., Hirotsu, I., Katsuda, N. and Hori, N. The effects of acetylcholine and aging on electrical excitability of the central nervous system. In: Neuroregulatory Mechanisms in Aging, Pergamon Press LTD, pp. 5-23, 1993.
31. Turner, J.N., Swann, J.W., Szarowski, D.H., Smith, K.L., Shain, W., Carpenter, D.O. and Fejtli, M. Three-dimensional confocal light and electron microscopy of neurons: fluorescent and reflection stains. Methods in Cell Biology, 38:345-366, 1993.
32. Deno, D. and Carpenter, D.O. Sources and characteristics of electric and magnetic fields in the environment. In: Biologic Effects of Electric and Magnetic Fields. Volume I: Sources and Mechanisms of Biologic Effects, David O. Carpenter and Sinerik Ayrapetyan, editors, Academic Press, California, pp. 3-59, 1994.

33. Carpenter, D.O. The public health implications of magnetic field effects on biological systems. In: Biologic Effects of Electric and Magnetic Fields, Volume II: Beneficial and Harmful Effects, David O. Carpenter and Sinerik Ayrapetyan, editors, Academic Press, California, pp. 321-329, 1994.
34. Carpenter, D.O. Multidisciplinary study of hazardous wastes at a Great Lakes Superfund Site. Great Lakes Research Review, 1: 37-39, 1994.
35. Fejtl, M. and Carpenter, D.O. Single-channel studies in molluscan neurons. In: Ion Channels, Vol. 4, Toshio Narahashi, ed., Plenum Press, New York, pp. 333-376, 1996.
36. Turner, J.N., Swann, J.W., Szarowski, D.H., Smith, K.L., Shain, W., Carpenter, D.O. and Fejtl, M. Three-dimensional confocal light and electron microscopy of central nervous system tissue, and neurons and glia in culture. In: International Review of Experimental Pathology, V.J. Savin and T.B. Wiegmann, editors, Volume 36, Academic Press, pp. 53-72, 1996.
37. Boldyrev, A., Lawrence, D. and Carpenter, D. Effect of carnosine and its natural derivatives on apoptosis of neurons induced by excitotoxic compounds. In: Peptide Science-Present and Future, Y. Shimonishi, editor, Kluwer Academic Publishers, Great Britain, pp. 424-426, 1998.
38. Carpenter, D.O., Hussain, R., Tan, Y., Niemi, W. and Hori, N. Long-term potentiation and long-term depression: Relevance to learning and memory. In: Modern Problems of Cellular and Molecular Biophysics. S.N. Ayrapetyan and A.C.T. North, editors, Nayan Tapan, pp. 83-94, 2001.
39. Carpenter, D.O. NMDA receptors and molecular mechanisms of excitotoxicity. In: Oxidative Stress at Molecular, Cellular and Organ Levels, A. Boldyrev and P. Johnson, editors, Research Signpost, pp. 77-88, 2002.
40. Carpenter, D.O. Clearing the air: Asthma an indoor exposure. JNMA 96: 1-2, 2004.
41. Carpenter DO. Environmental contaminants and human health: The health effects of persistent toxic substances. Firat Tip Dergisi 10: ____: 2005.
42. Hermanson MH, Johnson GW and Carpenter DO. Routes of human exposure to PCBs in Anniston, Alabama. ACS Division of Environmental Chemistry, 232rd National Meeting, 46: 1117-1122, 2006
43. Carpenter DO and Welfinger-Smith G. The Hudson River: A case study of PCB contamination. In: Water and Sanitation-Related diseases and the Environment: Challenges, Interventions, and Preventative Measures. Janine M.H. Selendy, Ed., Wiley & Sons, Inc. 2011, pp 303-327.
44. Welfinger-Smith G and Carpenter DO. Addressing sources of PCBs and other chemical pollutants in water. In: Water and Sanitation-Related diseases and the Environment: Challenges, Interventions, and Preventative Measures. Janine M.H. Selendy, Ed., Wiley & Sons, Inc. 2011, pp 359-384.
45. Carpenter DO. Human health effects of nonionizing electromagnetic fields. In: Patty's Toxicology. Sixth Edition. 2012 Volume 6. E Bingham and B Cohrssen, editors. Chapter 100, pp 109-132
46. Carpenter DO Introduction: Why should we care about organic chemicals and human health? In: Effects of Persistent and Bioactive Organic Pollutants on Human Health. 2013 DO Carpenter, editor. Chapter 13, pp 1-7..
47. Carpenter DO Organic chemicals and the Immune System. In: Effects of Persistent and Bioactive Organic Pollutants on Human Health. 2013 DO Carpenter, editor. Chapter 13, pp 362-382.
48. Carpenter DO Intellectual developmental disability syndromes and organic chemicals. In: Effects of Persistent and Bioactive Organic Pollutants on Human Health. 2013 DO Carpenter, editor. Chapter 16, pp 421-447.
49. Carpenter DO. Mechanisms of the neurotoxic actions of organic chemicals. In: Effects of Persistent and Bioactive Organic Pollutants on Human Health. 2013 DO Carpenter, editor. Chapter 17 pp 448-470.

50. Carpenter DO. How much human disease is caused by exposure to organic chemicals? In: Effects of Persistent and Bioactive Organic Pollutants on Human Health. 2013 DO Carpenter, editor. Chapter 13, pp 557-569..

Other Publications:

1. Barker, J.L. and Carpenter, D.O. Neuronal thermosensitivity. Science, 172:1361-1362, 1971.
2. Carpenter, D.O. Cellular Pacemakers. Fed. Proc., 37:2125-2126, 1978.
3. Carpenter, D.O. Membrane biophysics and general neurobiology in Japan. ONR Tokyo Scientific Bulletin, 3:23-27, 1978.
4. Carpenter, D.O. Research on the primate nervous system in Japan. ONR Tokyo Scientific Bulletin, 3:28-32, 1978.
5. Carpenter, D.O. Report on the Sixth International Biophysics Congress, Kyoto, Japan. ONR Tokyo Scientific Bulletin, 3:38-40, 1978.
6. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Research Bulletin, 4:149-152, 1978.
7. Carpenter, D.O. and Ahlbom, A. Power lines and cancer: Public health and policy implications. Forum, 3:96-101, 1988.
8. Carpenter, D.O. Setting Health Policy When the Science and the Risk are Uncertain. In: The Scientific Basis of Health Policy in the 1990s, Proceedings of the School of Public Health's Fifth Anniversary Symposium, 54-63, 1990.
9. Carpenter, D.O. Integrating public health in professional education. Optometry and Vision Science, 70: 699-702, 1993.
10. Bowerman, W.W., Carey, J., Carpenter, D.O., Colborn, T., DeRosa, C., Fournier, M., Fox, G.A., Gibson, B.L., Gilbertson, M., Henshel, D., McMaster, S. and Upshur, R. Is it time for a Great Lakes Ecosystem Agreement separate from the Great Lakes Water Quality Agreement? J. Great Lakes Res. 25:237-238, 1999.
11. Carpenter, D.O. Editorial Comment of "Primary hypoxic tolerance and chemical preconditioning during estrus cycle". Stroke, 30:1262, 1999.
12. Carpenter, D.O. Bring environmental health back into public Health. J. Pub. Health Mgmt. Pract., 5:vii-viii, 1999.
13. Carpenter, D.O. Should children and women of childbearing age eat Great Lakes fish? Great Lakes Commission Advisor, 13: 8, 2000.
14. Hites, R.A., Foran, J.A., Schwager, S.J., Knuth, B.A., Hamilton, M.C. and Carpenter, D.O. Response to comment on "Global Assessment of Polybrominated Diphenyl Ethers in Farmed and Wild Salmon". Environ. Sci. Technol. 39: 379-380.
15. Carpenter, D.O. Blood lead and IQ in older children. Letter to the editor. Environ. Health Perspect., 113: A581-A582, 2005.
16. Foran, J.A., Carpenter, D.O., Good, D.H., Hamilton, M.C., Hites, R.A., Knuth, B.A. and Schwager, S.J. Risks and benefits of seafood consumption. Letter to the editor. Am. J. Prev. Med. 30: 438-439, 2006.
17. Kouznetsova, M, Huang X, Lessner L and Carpenter DO. Zip code and GIS Studies: Kouznetsova et al. Respond. Environ Health Perspect 116: A18-A19, 2008.

18. Bolte G, Kohlhuber M, Carpenter DO and Tamburlini G Environmental inequalities among children and adolescents in Europe. Report prepared and submitted to the World Health Organization, 2009.
19. Gavidia T, Brune M-N, McCarty KM, Pronczuk J et al. (2010) Children's environmental health – from knowledge to action. *The Lancet* DOI:10.1016/S0140-6736(10)60929-4.
20. Toxins and the Brain. PSR's Environmental Health Policy Institute, 9 April 2012
21. Carpenter DO Electromagnetic fields: The effect on human health. *San Francisco Medicine* 85: 30-31: 2012
22. Carpenter DO and Sly P (2012) New leadership for Reviews on Environmental Health. *Rev Environ Health* 27: 65.
23. Florea A-M, Busselberg D and Carpenter (2012) Metals and disease. *J Toxicol* doi:1155/2012/825354
24. Brune M-N, Goldizen FC, Neira M, van den Berg M et al. (2013) Health effects of exposure to e-waste. *The Lancet* 1:e70
25. Sage C, Carpenter DO and Hardell L (2015) Comment on SCENIHR: Opinion on potential health effects of exposure to electromagnetic fields. *Bioelectromagnetics* 36: 480-484.
26. Carpenter DO and Belpomme D (2015) Idiopathic environmental intolerance. *Rev Environ Health* 30: 207.
27. Carpenter DO (2016) What constitutes environmental health? *Rev Environ Health* 31: 193.
28. Carpenter DO and Sly PD (2016) Environmental chemicals as endocrine disruptors. *Rev Environ Health* 31: 389.
29. Sly PD and Carpenter DO (2016) Traditional and emerging environmental hazards in South-East Asia: double-trouble in the 21st Century. *Rev Environ Health* 31: 1.
30. Carpenter DO (2017) Beware: New dangers from the chemicals all around us. *Bottom Line Health* May: 3-5.