REPORT OF PLAINTIFFS' EXPERT
David O. Carpenter, MD

America Unites For Kids, et al.
v. Sandra Lyon, et al.
Case No. 2:15-cv-02124-PA (AJWx)

REPORT
David O. Carpenter, MD

My name is David O. Carpenter, and I am a public health physician, educated at Harvard College where I graduated *magna cum laude* in 1959 and at Harvard Medical School where I graduated *cum laude* in 1964. I have pursued a career in biomedical research and public health rather than patient care. I served as Director of the Wadsworth Center for Laboratories and Research of the New York State Department of Health from 1980-1985, then became the founding Dean of the School of Public Health of the University at Albany, a position I held until 1998. My present position is Director of the Institute for Health and the Environment at the University at Albany. The Institute has been designated as a Collaborating Centre of the World Health Organization. I am also Professor in the Department of Environmental Health Sciences in the School of Public Health. I have over 400 peer-reviewed publications in the general fields of neuroscience and environmental health and am active in research and training. I teach graduate courses in environmental health, radiation biology and neurobiology/neurotoxicology. My *Curriculum vitae* is attached.

QUALIFICATIONS:

Beginning in 1986 I directed a large, interdisciplinary research study on polychlorinated biphenyls (PCBs), funded by the Superfund Basic Research Program of the National Institute of Environmental Health Sciences, one of the National Institutes of Health. Our particular study, supported at over $2 million a year until 2000, was designed around the PCB contamination coming from the General Motors Foundry Site in Massena, NY, which is directly adjacent to the Mohawk Nation of Akwesasne, a Native American community of individuals who traditionally eat fish from waters now heavily contaminated with PCBs. Our investigations included health studies of the Mohawks, animal toxicology studies of effects of PCBs on the nervous, immune and endocrine systems and metabolism, determination of levels of PCBs in human body fluids, animals from the region, soils, sediments, air and water and investigation of several methods for destruction and removal of PCBs from contaminated soils, sediments and water. A laboratory for chemical measurement of PCBs is a central part of our research team and operated under my direction. This study involved over 1,600 members of the Mohawk Nation, where I have reviewed analyses of their PCB blood levels and clinical chemistry results, as well as results taken from questionnaires. The early investigations at Akwesasne were focused entirely on demonstrating that the Mohawks were exposed to PCBs from consumption of contaminated fish. In the grant that was funded in the period 1995-2000 we began to collect health information, the analysis of which and results of which are still being analyzed and published. We have smaller grant-funded studies of the same population ongoing in which we are collecting new data, and continue to submit research grant applications to expand study in this community.

I have also conducted health effects studies of other PCB-exposed populations, including an Alaskan Native population living on St. Lawrence Island, Alaska (funded by the National Institute of Environmental Health Sciences and the US Environmental Protection Agency), residents of Anniston, Alabama who live near to the Monsanto plant that manufactured PCBs

(funded by the Agency for Toxic Substances and Disease Registry) and people living along the PCB-contaminated portions of the Hudson River in New York and the Housatonic River in Massachusetts. I have published numerous articles based on both human exposure to PCBs and experimental studies with animals experimentally exposed to PCBs.

In addition, I have and continue to investigate the distribution of various human diseases in relation to residence near hazardous waste sites. These studies utilize the New York State dataset which records information on every inpatient in state-regulated hospitals, giving age, sex, race, method of payment and zip code of residence, as well as all (up to 15) diseases diagnosed in that patient. We have matched these data to a characterization of every zip code in so far as it has or abuts an identified hazardous waste site that either contains or does not contain persistent organic pollutants (primarily PCBs). While we do not have detailed individual information using these data, we have large numbers that allow us to observe patterns of disease in relation to residence near to waste sites. We have studied diseases such as diabetes, heart disease, stroke, hypertension, thyroid disease, endocrine disease, asthma and infections, and for each find elevations in incidence correlated with residence near to waste sites that contain PCBs. Because simply living in the same zip codes as one containing a hazardous waste site containing PCBs is unlikely to result in exposure coming from dermal contact or ingestion, we conclude that the route of exposure to the PCBs that result in the diseases listed above is inhalation. Because PCBs are semi-volatile chemicals, they are present in the air at low concentrations, varying with temperature. These results provide strong evidence that chronic exposure to PCBs in air is associated with elevations in a variety of human diseases.

I have reviewed medical records and/or serum PCB levels of over 3,000 residents of Anniston, Alabama, Crystal Springs, Mississippi, Chicago, Illinois and Bedford, Indiana who have been exposed to PCBs as a result of residential proximity to industrial contaminated sites, and have provided medical monitoring advice in these and other cases.

In recognition of my contributions in the general field of environmental health, I have and continue to serve on several national and international advisory committees. From January 1997 until February 2001 I was a member of the National Advisory Environmental Health Sciences Council of the National Institute of Environmental Health Sciences. This Committee was appointed by the United States Secretary of Health and Human Services to advise the Director and Staff of the Institute on all matters of policy, scientific direction and priorities. In 1997, I was also appointed to be a member of the Great Lakes Science Advisory Board of the International Joint Commission, which is a binational Commission between Canada and the United States that deals with issues related to the Great Lakes. I was reappointed and served on this board until 2012. In 1999, I was invited to be a member of the Expert Panel Review team for the Agency for Toxic Substances and Disease Registry, a panel assembled to review the newest edition of the publication, *Toxicological Profiles for Polychlorinated Biphenyls*. I have been a member of the Board of Directors of the Pacific Basin Consortium for Environmental and Health Sciences for several years. From 2004 to 2007, I assumed the position of Chair of the Board, and am the Treasurer at present. This is an international organization concerned with health effects of environmental contaminants, remediation of hazardous wastes and other environmental problems such as climate change, with membership open to individuals and organizations from all countries in the Pacific Basin. In 2001, I was appointed as a member of

the Committee on Implications of Dioxins in the Food Supply, a committee of the US National Academy of Sciences, and the report from this committee was released in July, 2003. I was a member of the Children's Health Protection Advisory Committee to the US Environmental Protection Agency between 2003 and 2008. I was a coauthor and the spokesperson for a study of contaminants in farmed and wild salmon first published in Science in January, 2004, and which has received international attention. The study was supported by the Pew Charitable Trust. In November 1999, I was awarded the Homer N. Carver Lecture Award by the Section on the Environment of the American Public Health Association for contributions to the field, and in 2001, I was selected to be the Academic Laureate of the University at Albany Foundation for distinguished service to the University. I serve as Chair of the Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on their joint programs. In 2008 the Institute for Health and the Environment was designated a Collaborating Centre of the World Health Organization in recognition of our international efforts in the area of environmental health. In 2013 I served as an Invited Specialist to the International Agency for Research on Cancer in their review of the carcinogenicity of PCBs, which resulted in PCBs being declared a Group 1, known human carcinogen.

Because of my public health background in environmental health and my research experience in both human and animal studies involving PCBs, I believe that I am qualified to evaluate and review the scientific validity of the plaintiff's complaint of imminent and substantial endangerment to human health posed by the PCB content in the air, dust, soil and school buildings in Malibu, California.

I am a public health physician, not a practicing physician, and study of the health effects resulting from exposure to PCBs and other environmental contaminants is my public health specialty.

COMPENSATION:

My usual compensation for involvement in legal cases is $400.00 per hour, paid to the Research Foundation of SUNY to be used for support of my students and staff, plus personal reimbursement for any travel expenses incurred. Given the nature of this case I will waive all fees unless funds become available, with the exception of any travel costs.

PREVIOUS DEPOSITIONS AND TESTIMONY (past five years):

Ronald Cybart et al., Michael Campanelli, and Donald and Theresa Shea, et al.v. CL&P. Deposed for the plaintiffs. 15 July 2011.

Maria Snoops vs. Lyon Associates, Inc. and Insurance Co of the state of Pennsylvania. Deposed for the plaintiff, 1 November 2011.

John Edward Martinez and Gladys Yolanda Martinez v. Entergy Corporation, et al., Deposed for the plaintiff, 19 December 2011.

AHM and David Mark Morrison vs. Portland Public Schools. Deposed for the plaintiffs,

25 January 2012.

Judy Prescott Barnett v Robert E. Carberry et al. Deposed for the plaintiff, 6 April 2012.

Association Quebecoise de Lutte Contre La Pollution Atmospherique et al. vs. Hydro Quebec, et al. Testified for the plaintiffs, 17-18 May 2012.

FortisBC vs Citizens for Safe Technology. Testified for the plaintiff, 15 March 2013.

John Edward Martinez and Gladys Yolanda Martinez v. Entergy Corporation et al., Deposed for the plaintiff, 21 June 2013.

Village of Stillwater et al. and Saratoga County Water Authority v. General Electric Company. Deposed for the plaintiff, 10 April 2014. Ron Plain and Ada Lockridge v. Director, Ministry of the Environment et al., Deposed for the plaintiff, 13-14 May 2014.

Harry Naeole vs. Alaska Barge & Transport, Employers, continental Insurance Company/CAN, Carrier. Deposed for the plaintiff, 27 May 2014.

Case No U-17767. Before the Michigan Public Service Commission in the matter of the application and request of the Detroit Edison Company seeking approval and authority to implement its proposed Advanced Metering Infrastructure opt out program. Testified to the Commission. 6 July 2015

Edwin Spirer, et al. v. Monsanto Company, et al. Deposed for the plaintiffs, 17-18 December 2015.

POLYCHLORINATED BIPHENYLS (PCBs): HISTORY, ROUTES OF EXPOSURE AND MECHANISMS OF ACTION:

PCBs were manufactured in the US between1929 to 1976 and were useful compounds for a variety of purposes. They are heavy oils that were used in transformers and electrical capacitors on the basis of being relatively good electrical insulators, as hydraulic fluids, as an oil additive to paints, window caulking, ceiling and floor tiles and many other products. PCBs were made by addition of chlorines to biphenyl, and were sold in the US primarily as Aroclor mixtures manufactured by the Monsanto Corporation, with the number of different commercial Aroclor mixtures based on the average degree of chlorination.

There are ten possible positions around the biphenyl molecule where chlorines can be added, and as a consequence there are a theoretical 209 possible PCB congeners. The positions are usually expressed as from 2 to 6, with the 2 and 6 positions being those closest to the biphenyl ring described as "*ortho*", those opposite (4) called "*para*" and the remainder (3 and 5) called "*meta*". Positions around the second ring are usually identified by adding a prime to the number, such that 2, 2' biphenyl is a PCB with two chlorines, one on each ring at the 2 position. The numbers and positions of the chlorines determine both the physical and biological properties of each congener. Those with fewer chlorines are in general more water soluble, more volatile and more easily metabolized. Those with chlorines only in the *meta* and *para* positions tend to assume a planar configuration and have dioxin-like activity. Those with more than one *ortho* chlorine do not show significant dioxin-like activity. PCBs that exist in a planar configuration activate the aryl hydrocarbon receptor (AhR) and have actions similar to those of dioxins and furans. This is the basis of the toxic equivalent factor (TEF) as defined by the World Health Organization (WHO) and many other publications by scientists in this area of research (Safe, 1990; Ahlborg et al., 1992; Walker et al., 2005). While coplanar PCBs are not as potent in

activation of the AhR as 2, 3, 7, 8-tetrachloro-dibenzo-*p*-dioxin, the most toxic dioxin congener, in many circumstances the concentration of the coplanar PCBs is much greater than that of dioxins and furans such that the majority of AhR receptor activity comes from PCBs. This large body of evidence provides proof that the health effects reported for dioxin-exposed populations are directly relevant to individuals exposed to PCBs, although PCBs may exert many other actions via different pathways.

No person exposed to PCBs is exposed only to either the dioxin-like or to the non-dioxin-like PCB congeners, however. Because the non-dioxin-like PCB congeners have different mechanisms of action and result in other diseases, a PCB-exposed person is at risk for all of the diseases caused by dioxin and other diseases caused by the non-dioxin-like congeners.

Each individual PCB congener has its own profile of actions in biological systems. Therefore, it is very important to have measurements of PCBs in any individual's body include measurement of individual congeners. However, we still have inadequate information on the actions of many individual congeners, such that in many cases one is not able to use all of the information that is obtained from measurement of all of the individual congeners (DeCaprio et al., 2005). Various investigators have proposed different patterns of response dependent upon classes of activity of PCB congeners. As mentioned above, those that activate the AhR act like dioxin to induce activity of cytochromes P450 (CYP) 1A1, 1A2 and 1B1. These actions result in proliferation of endoplasmic reticulum in liver, an actual increase in liver size, and alteration in liver function. The size of the thymus gland is reduced, resulting in a reduction in immune function. Since the AhR is one of the greater steroid hormone family of receptors, its activation results in gene induction, with increased or decreased levels of a very large number of gene products. There are four coplanar PCB congeners with at least four chlorines.

The majority of PCB congeners are not AhR activators, but act at several other sites. One important class of congeners is those that cause induction of a different class of cytochrome P450s, that being CYP 2B1 and 2B2. These congeners induce a wasting syndrome and as well as thymic atrophy in experimental animals and presumably also in humans. A third major group of congeners have activities at both of these sites, and are called "mixed" congeners. These are mono-ortho congeners, and activate both the 1A and B and 2B P450s. There are nine major congeners in this group, and they contribute significantly to total TEF because some are present in relatively high concentrations (Erikson, 1997). Fitzgerald et al. (2004) have demonstrated that adults exposed to PCBs are able to more rapidly metabolize caffeine using a caffeine breath test if they have high levels of PCBs. These observations demonstrate that exposure alters liver function in ways that affect metabolism of many substances in addition to PCBs through prolonged induction of the P450s, in this case CYP 1A2.

Some congeners with *ortho* chlorines have a different profile of actions and health effects that appear not to be dependent upon any CYP activity. These PCBs show short-latency effects on the nervous (Kodavanti et al. 1993; Carpenter et al., 1997; Tan et al., 2003) and immune systems (Jeon et al., 2002; Tan et al., 2004a), causing a relatively rapid cell death that is a result of disruption of the membrane structure (Tan et al. 2004b), an effect not seen by coplanar PCBs. These congeners have also been found to stimulate insulin release from a human beta receptor cell line, to reduce synthesis of the neurotransmitter dopamine in neurons, and to

activate neurophils to produce reactive oxygen species (ROS) (Fischer et al., 1998).

Not only do different PCB congeners have unique sites and mechanisms of action, but also their metabolites may have biological activity and may be persistent in living organisms for a period of time (Connor et al., 1997; Sandau et al., 2000). Many of the endocrine disruptive actions of PCBs may be primarily a result of the actions of metabolites, especially hydroxylated metabolites (Garner et al., 1999).

We have demonstrated that we can detect various patterns of PCBs in human blood (DeCaprio et al., 2005). As indicated above, lower chlorinated PCBs are more volatile, and in analysis of a subset of Mohawks we can identify a pattern in their blood that is similar to that in the air. This provides direct evidence that inhalation is an important route of exposure. Others show patterns that correlate with rates of fish consumption. The congener patterns that correlate with fish consumption are not identical to those in the fish, which reflects the differing rates of metabolism of various congeners in the human body. We found two patterns that do not reflect any seen in either exposure pathway, and which we believe to reflect genetic differences in metabolism of PCBs.

Others (Wolff et al., 1997) have classified PCB congeners in groups on the basis of commonality of action, or on the basis of degree of chlorination (Moysich et al., 1998). The coplanar and AhR activating PCBs, for example, are antiestrogenic by virtue of the fact that they induce P450 1A1, which is the P450 which degrades estrogen (Spink et al., 1990). In contrast most other PCBs are estrogenic. A recent report demonstrated estrogenic activity of thirteen congeners, PCBs 17, 18, 30, 44, 49, 66, 74, 82, 99, 103, 110, 128, and 179 (DeCastro et al., 2006), and found that inhalation was the major source of exposure to estrogenic PCB activity. This makes sense since the lower chlorinated congeners are also more volatile, and many of the congeners listed above are lower chlorinated. In the body the hydroxylated metabolites of the PCBs have even greater estrogenic activity than most of the parent PCBs. Since the great majority of PCBs are not AhR activators, the net activity of most PCB mixtures is estrogenic. This variation in action of different PCB congeners is particularly important in developing an understanding of the relationship between exposure to PCBs and risk of breast cancer. Estrogen is by far the best-documented risk factor for breast cancer (Colditz et al., 1995). Thus exposure to AhR-activating PCBs might be expected to be protective from breast cancer on the basis of induction of P450s that degrade estrogen, whereas exposure to the estrogenic congeners might be expected to promote risk. Other categories of PCBs include those with particular persistence, which is characteristic of those congeners with higher degrees of chlorination, especially of the meta and para positions.

It is important to recognize that each individual PCB congener may have multiple sites of action in biological systems, mediated by binding to very different receptor sites or targets. Furthermore, different individual PCB congeners may have the same action, but do so via completely independent mechanisms.

It has usually been assumed that ingestion was the primary route of exposure to PCBs (ATSDR, 2000; IOM, 2003). PCBs are fat soluble, and bioaccumulative. Fish are an especially important route, especially fish from contaminated fresh waters (ATSDR, 2000). However,

inhalation and dermal absorption are also important routes. Animal studies have shown that inhalation of vapor phase PCBs is actually a more efficient route of exposure than ingestion, and that inhaled vapor phase PCBs can bioaccumulate and cause pathological changes (Casey et al., 1999). Vapor phase PCBs are significantly elevated near to PCB-contaminated hazardous waste sites (Chiarenzelli et al., 2000; Hermanson et al., 2003), and our previous investigations have provided strong evidence that inhalation is an important route of exposure (DeCaprio et al, 2005; Fitzgerald et al., 2006) and a cause of health effects among individuals who live near to such contaminated sites (Baibergenova et al., 2003; Kudyakov et al., 2004; Seergev and Carpenter, 2005; Shcherbatykh et al., 2005; Huang et al., 2006). Liebl et al. (2004) have also demonstrated accumulation of lower chlorinated PCB congeners in humans as a result of breathing contaminated indoor air. Because PCBs are lipophilic substances, they are easily absorbed through the skin, a route of exposure that is of particular importance in occupational settings where direct dermal contact occurs with PCBs. In occupational situations exposure is usually a combination of inhalation and dermal absorption (Mallin et al., 2004). I have recently reported that a husband and wife team, related only by marriage, were employed at a plant that was removing waste oils from old transformers and capacitors (Carpenter, 2015). Both were instructed to smell the oils to determine whether or not they contained significant concentrations of PCBs. Both developed thyroid cancer and malignant melanoma, the cancer most strongly associated with PCB exposure. The husband, a non-smoker, also developed primary lung cancer which metastasized to his brain and killed him. These observation indicate strongly that inhalation of PCBs can cause cancer.

PCBs are usually reported as wet weight concentrations, although it may be preferable to report the results as lipid-adjusted values, since all of the PCBs are found in the lipid fraction. In general lipid-adjusted PCB values are 200-250 times larger than wet weight values. Reporting PCB levels after lipid adjustment is especially important if the subject is not fasting, although some (Schisterman et al., 2005) have cautioned that lipid-adjustment may introduce bias. Studies from CDC have shown that there is no difference between fasting and non-fasting results provided that lipid adjustment is made (Phillips et al., 1989). The half-life of PCBs in the human body is long, but varies with the congener in that many of the lower chlorinated congeners are more rapidly metabolized, whereas many of the highly chlorinated congeners persist even for decades. The rates of removal are also a function of body burden. Wolff et al. (1992) reported a half-life of serum PCBs of 3-5 years for individuals with high serum PCBs but of 13-17 years for those with lower values. I usually consider an average half life to be 10 years, but at the same time recognize that the half-life varies greatly among different congeners.

DETERMINATION OF DISEASE CAUSATION FROM EXPOSURE TO PCBs:

The evidence for causality for human disease resulting from exposure to an environmental contaminant is usually evaluated by consideration of the factors identified by Hill (Hill, 1965). These are a) strength of association, b) consistency of association. c) specificity, d) temporality, e) dose-response relationship, f) plausibility, g) coherence, h) experimental evidence and i) analogy. These are appropriate standards for evaluating whether or not there is a relationship between exposure and disease, and together consideration of these factors leads to a general "weight of evidence". Hill did not imply that each of these factors must be met in every

case, but that these were appropriate factors for consideration when considering whether an association was really causation. In fact he states "What I do not believe – and this has been suggested – is that we can usefully lay down some hard-and-fact rules of evidence that *must* be obeyed before we accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*."

The strength of association is usually presented as an odds ratio (OR) or risk ratio (RR) together with a 95% confidence interval (CI). The size of the OR or RR is less important than the CI. The CI provides information about the statistical likelihood that the relationship is real with a likelihood of 95 out of 100 chances. Traditionally the relationship is taken to be "statistically significant" if the lower limit of the 95% CI is greater than 1.0. Clearly the higher the OR or RR the greater the risk, provided that the results are statistically significant on the basis of the lower limit of the 95% CI being greater than 1.0. However Hill (1965) also commented on statistical significance, stating "No formal tests of significance can answer those questions. Such test can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis."

Consistency is a measure of replicability of the observation of a statistically significant relationship in multiple studies, preferably in diverse populations and studies by different investigators. Specificity relates to multiple causes of the same disease, and often causes are not specific to a single exposure. Temporality means that disease must follow exposure, but be related to it. Dose-response relationship means only that more exposure is associated with more risk of disease. Plausibility means that there should ideally be a mechanism that will explain the association. Hill stated "It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day." Coherence means that the interpretation of the data should not seriously conflict with generally known facts relating to the natural history and biology of the disease. Experimental evidence means that there should also be animal or cellular studies that lead to conclusions consistent with the findings. With regard to analogy, Hill states "In some circumstances it would be fair to judge by analogy. With the effects of thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy."

HEALTH EFFECTS OF PCBs:

PCBs are carcinogens that alter immune system function, cause adverse alterations of the nervous system, skin, thyroid and sex steroid hormonal systems, liver, kidney, cardiovascular system and pancreas (ATSDR, 2000; Carpenter, 1998; 2006; IARC, 2015). As a result of these actions on multiple organ systems, humans that are exposed to PCBs are at increased risk of cancer, infections, reduced cognitive function accompanied by adverse behavioral effects, hypothyroidism, infertility, ischemic heart disease, hypertension, diabetes, liver disease, asthma and arthritis, as well as being at increased risk of giving birth to infants of lower than normal birth weight. The scientific evidence in support of these statements as regarding the diseases of

interest in this case is as follows:

Cancer:

PCBs are known to be complete carcinogens, and act as general cancer promoters via generation of ROS and induction of a variety of different genes. These actions occur in all tissues of the body as reported in the review by Cogliano of the USEPA (1998). Therefore one would expect that they would result in an increase in the risk of every kind of cancer. Studies of cancer in human populations are primarily of two major types, occupational studies of workers and case-control studies of individuals with a specific kind of cancer. PCBs are classified as "Group 1, known human carcinogens" by the World Health Organization (International Agency for Research on Cancer, IARC; 2015) on the basis of proof of their carcinogenicity in animals and in humans. IARC considered the evidence in humans to be strongest for malignant melanoma, and strong for breast cancer and non-Hodgkin's lymphoma. There are a number of peer-reviewed publications documenting elevated risks from PCB exposure for other types of cancer (some listed below) and it is likely that exposure to PCBs increases risk to all kinds of cancer. Some of the evidence for PCBs and specific kinds of cancer is given below.

It is important to recognize that each individual PCB congener may have multiple sites of action in biological systems, mediated by binding to very different receptor sites or targets. Furthermore, different individual PCB congeners may have the same action, but do so via completely independent mechanisms. Carcinogenesis is one good example. PCBs are complete carcinogens and tumor promoters (Tharappel et al., 2002), but act via several mechanisms. Chronic exposure to PCBs results in chromosomal aberrations (reviewed by Silberhorn et al., 1990), an action probably mediated primarily by metabolites of the PCBs. This is in spite of the fact that PCBs are not generally viewed as being cancer initiators. As already mentioned some PCB congeners are estrogenic, and therefore will promote risk of cancers related to estrogenic activity. Those PCBs that activate the AhR act as dioxin, a proven human carcinogen (IRAC, 2004). Those that activate the P450 2B1 and 2B2 P450s are also carcinogenic, but act by a different mechanism, perhaps via generation of ROS leading to activation of various transcription factors such as nuclear factor –kB and activator protein-1 (Tharappel et al., 2002). There has been evidence for some time that the dioxin-like congeners and dioxin produce oxidative DNA damage (Oakley et al., 1996). However, in a study investigating the induction of hepatic tumors in rats, Van der Plas et al. (2000) have concluded the "the majority of the tumor promotion potential of PCB mixtures resides in the non-dioxin-like fraction which is not taken into account in the toxic equivalency factor (TEF) approach for risk assessment of PCBs." Therefore one cannot adequately assess cancer risk from PCB exposure by use of TEFs.

Malignant Melanoma: Longnecker et al. (2003) have summarized the various occupational studies that provide evidence for an elevated risk of malignant melanoma in PCB-exposed workers. There is reasonable consistency in this finding in the occupational cohorts, but there have not been investigations in the general population to date.
IARC has declared all PCBs to be known human carcinogens, with the strongest evidence being for malignant melanoma (IARC, 2015). That evidence comes from both occupational and population studies, as referenced in the IARC monograph.

Thyroid Cancer: Thyroid cancer is also commonly induced in animals upon exposure to PCBs (Mayes et al., 1998; Vansell et al., 2004). In the occupational study of capacitor workers by Mallin et al. (2004) they reported a significant SMR of 15.2 for thyroid cancer in men. Thyroid cancer has also been found to be elevated in individuals exposed to dioxin (Saracci et al., 1991). Mallin et al. (2004) reported causes of mortality for workers employed at the Electrical Utilities Company capacitor manufacturing plant in La Salle, Illinois between 1944 and 1981. Among 3,301 workers they reported a significant specific mortality rate (SMR) of 15.2 (95% CI = 3.1-44.5) for death from thyroid cancer in men. The SMR for men with 10 or more years of employment in this capacitor plant was a striking 51.4 (95% CI = 6.2-185.6)! These elevations were similar whether the comparison was made to Illinois or to overall US rates. Thyroid cancer is a relatively rare disease, and the finding of thyroid cancer in two individuals employed in the same laboratory and using PCBs (Carpenter, 2015) is strong evidence for cause and effect. This case is particularly relevant to the situation in Malibu in that both were instructed to "smell" the PCB-contaminated samples, and thus inhalation was their primary route of exposure. Furthermore their blood samples showed elevations in the lower chlorinated congeners, but no significant elevation of more highly chlorinated congeners as compared to the rest of the US population.

Prostate Cancer: Ritchie et al. (2003; 2004) have demonstrated a dose-dependent increase risk of prostate cancer as a function of serum PCB concentrations of three specific groups of PCB congeners. Interestingly, they did not find an increased risk with the dioxin-like congeners, but did find a dose-dependent increased risk of up to more than 2-fold for moderately chlorinated congeners and transient and persistent phenobarbital-like inducers. This study is one of the clearest demonstrations that it is not only dioxin-like congeners that increase risk of cancer. Charles et al. (2003) in an occupational study reported an odds ration of 1.47 (95% CI: 0.97-2.24) for prostate cancer mortality with serum PCB levels. Prince et al. (2006) examined 14,458 workers at two capacitor plants and found a strong exposure-response relationship for prostate cancer mortality (trend p-value = 0.0001).

Non-Hodgkin's Lymphoma: Rothman et al. (1997) report a strong, dose-dependent increase in risk of non-Hodgkin's lymphoma with increasing PCB concentration in serum. They studied 74 cases of non-Hodgkin's lymphoma occurring among 25,802 adults from Washington County, Maryland, from whom blood samples were obtained in 1974 and 147 controls from the same population. They report lipid-adjusted PCB levels. Taking the lowest levels (247-641 ppb) as controls the matched adjusted odds ratio was 1.3 (0.5-3.3) for serum PCB levels of 649-806, 2.7 (0.9-7.8) for serum PCBs of 814-1060, and 4.1 (1.4-11.9) for serum PCBs of 1070-2070. The last value is statistically significant by itself, and there was a significant positive value for p for trend ($p = 0.002$). For those individuals who were not positive for Epstein-Barr virus the level of significance was even greater. His table also gives these PCB levels as serum values (not lipid-adjusted). The mean values of the four groups are 3.8, 5.5, 6.7 and 10.3 ppb.

Hardell et al. (1996) studied 29 cases of B-cell non-Hodgkin's lymphoma and 17 controls. He found that almost all PCB congeners that were analyzed were higher in cases. The mean sum of PCBs for cases was 1,614 ppb (range 637-4,705) (lipid adjusted) while for controls it was 1,213 ppb (range 366-2,282). Congeners elevated included 156, 157, 182+187, 171,172+192, 190, 189, 202, 201, 194 and 208, all $p<0.05$ as compared to controls. He found no

difference for HCB and DDE. In a brief 1997 report Hardell et al. performed logistic regression analysis controlling for age and sex, and found the OR for the sum of PCBs was 1.8 (0.4-7.4). They suggest that the relationship results from immunotoxic actions of PCBs. A later study by Hardell et al. (2001) reported a significant odds ratio of 4.0 for total PCBs in individuals with demonstrable Epstein-Barr virus infection.

Baris et al. (2000) reported that chemotherapy for patients with non-Hodgkin's lymphoma resulted in a 25% decrease in the levels of PCBs. Average interval was 20 months. They studied PCB 138, 153, 156 and total PCBs. Nordstrom et al. (2000) studied 54 cases of hairy cell leukemia (a form of non-Hodgkin's lymphoma) and 54 controls. While there was no significant difference in total PCB concentrations, in a subset of the population who had high serum titers to Epstein-Barr virus (indicating they had been infected), there was a large increase in risk of disease based on the concentration of a subgroup of PCB congeners that are immunotoxic (PCBs 66, 110, 105, 118, 74, 128/167, 156, 138 and 170/190). The odds ratio for low levels of these congeners was 1.7, and for high levels, was 11.3, indicating a 10-fold increased risk.

Hoque et al. (1998) studied various cancers in people exposed to polybrominated biphenyls (PBBs), which are similar to PCBs. They studied 187 exposed persons with cancer and 696 controls. There was no overall increased risk of cancer related to serum PBB level, but a dose-dependent elevated risk for both lymphoma and digestive system cancers. For lymphoma, taking PBB level <3 ppb as control, the odds ratio was 3.85, 19.6 and 48.9 for PBB levels of 4-20, 21-50 and > 50 ppb, respectively. These are highly significant.

Two recent studies add to the evidence for a relationship between non-Hodgkins lymphoma and PCB exposure. Colt et al. (2005) examined 603 cases and 443 controls, and found a significant odds ratio of 1.5 if any of five congeners was detected. There was a significant p for trend for PCB 180, and an odds ratio of 1.7 when comparing highest to lowest tertile. De Roos et al. (2005) report that PCBs 156, 180 and 194 gave odds ratios for highest versus lowest quartile ranging from 2.7 to 3.5, with significant trends.

Pancreatic cancer: Yassi et al. (1994) reported significant elevation of pancreatic cancer mortality in workers in a transformer plant. Hoppin et al. (2001) have shown that exposure to PCBs results in a dose-dependent increased risk of pancreatic cancer. Pancreatic cancer is the fifth leading cause of cancer death in the US. PCBs were measured in serum, and reported as lipid adjusted values. They studied 108 cases and 82 controls. Taken lipid-adjusted PCB levels in serum below 185 ppb as reference, there was a 1.3 fold risk (95% CI 0.6-2.8) for levels between 185-360 ppb, and a 4.2 fold increased risk for levels over 360 ppb (95% CI = 1.9-9.4). Not only is the last number statistically significant, but there also was a significant p for trend, and a significant continuous (odds ratio of 1.003 for each ppb increase in concentration) variable. There were significantly elevated odds ratios for PCB 153 (OR 3.0, 95% CI = 1.4-6.6) and PCB 180 (OR = 8.4, 95% CI = 3.4-21) for the highest tertiles.

Porta et al. (1999) looked at 51 cases of pancreatic cancer, and showed that those cases that had a K-ras mutation had significantly higher concentrations of PCBs 153, 180 and 138 (the only congeners measured). The total concentrations of the three PCBs were higher in the 51

cases than the 26 controls, but differences were significant only for PCB 180. The 51 cases were more than four times more likely to be in the upper tertile of PCB 180 than the 26 controls (OR = 4.6, 1.1-19.0, p for trend = 0.037). The odds ratio for the 34 mutated cases was 7.4 (1.6-34.4, p for trend = 0.012). Considered alone, lipid adjusted, and taking non-detect values as OR = 1.0, for PCB 138 in tertiles the ORs increased from 1.0 to 2.9 (0.5-17.2) to 6.9 (1.1-41.5) with a p for trend of 0.034, for PCB 153 from 1.0 to 1.8 (0.4-7.6) to 7.2 (1.1-45.6), with a p for trend of 0.035, and for PCB 180 from 1.0 to 2.8 (0.6-14.3) to 6.3 (1.0-38.8), with a p for trend of 0.028. Similar results were found for DDT and DDE. Mean concentrations for all cases were 1.45 ppb for PCB 138, 1.59 ppb for PCB 153 and 2.01 ppb for PCB 180.

Breast Cancer: There are many breast cancer studies, some of which suggest a dose-dependent relationship to total PCB exposure but the majority of large studies have not demonstrated a relationship with total PCB levels. As discussed above the conflicting observations may be a result of the different endocrine actions of different PCB congeners. However there is evidence for a relationship between PCB exposure and risk of breast cancer in certain individuals with exposure to certain PCB congeners.

Moysich et al. (1999) looked at risk of breast cancer in relation to both serum PCB levels and the presence of one specific polymorphism of P450 1A1. They report that women with serum PCB levels above the median of the distribution in the control group (control low level, 0.75-3.72 ppb, high levels 3.73-19.04 ppb) who also were homozygous for the isoleucine allele had elevated risk (OR 2.96, 95% CI = 1.18-7.45). For heterozygous women OR = 2.87 (1.19-7.30). There was no significantly elevated risk for high PCBs alone (OR=0.88, 0.29-2.70) or homozygous genotype alone (1.07, 0.68-1.70). This observation has been generally confirmed by two other groups (Laden et al., 2002; Zhang et al., 2004). Millikan et al. (2000) studied plasma DDE and PCB levels in women in relation to breast cancer in African-American and white women. ORs for highest to lowest third for total PCBs were 1.74 (1.00-3.01) for black women and 1.03 (0.68-1.56) for white women. For obese black women the OR was 4.92 (1.63-14.83). These observations are consistent with there being sub-populations which are genetically at greater risk of developing breast cancer as a result of exposure to PCBs.

Aronson et al. (2000) examined breast tissue from 217 cancers and 213 controls. They found no relation with total PCBs, but significant correlations with PCB 105 and 118. The odds ratios for these two congeners increased linearly across categories (p for trend <0.01). The elevated risk associated with PCBs 105 and 118 were higher among premenopausal women, and for postmenopausal women the risks were elevated with PCBs 170 and 180. Demers et al. (2002) studied 315 cases of breast cancer and 523 controls. They report significant elevations in risk of disease in relation to the levels of three individual PCB congeners, PCB 99, 118 and 156, with odd ratios between 1.6 and 1.8. They also found a significant relationship with the sum of PCB 105, 118 and 156 (odds ratio = 2.02). These observations suggest that some congeners or combinations of congeners cause a dose-dependent increase of risk of breast cancer, whereas others do not. While there is less information on PCB-induced risk of other estrogen-dependent cancers, it is likely that what is true for breast is also true for the others.

Gastrointestinal Cancers: Mallin et al. (2004) reported a mortality study of workers employed at a capacitor manufacturing plant using PCBs, and found an elevated incidence of

intestinal cancer in women employed 5 or more years (SMR 2.2), and an elevated risk of stomach cancer in men (SMR 2.2). Howsam et al. (2004) reported an elevated risk of colorectal cancer with levels of the mono-orthe congeners, PCBs 28 and 118. In the top tertile the OR was 2.94 (95% CI = 1.39-6.20). Gastrointestinal cancers have been reported significantly elevated in some but not all occupational studies (Bertazzi et al., 1987). As mentioned above, Hogue et al. (1998) reported a dose-dependence increase in gastrointestinal cancers in the Michigan cohort exposed to PBBs. For PBB levels of 4-20, 21-50 and >50 ppb the ratios were 8.23, 12.3 and 22.9.

Liver/biliary Cancers: Liver and biliary cancers are some of the most common cancers induced in experimental animals exposed to PCBs (Kimbrough et al., 1975; Mayes et al., 1998). They are been reported in human occupational studies (Brown, 1987; Gustavsson and Hogstedt, 1997). In the capacitor study of Mallin et al. (2004) a SMR of 6.2 was found for liver/biliary cancer in women employed for 10 or more years in the plant.

Lung Cancer: An elevation in lung cancer has been reported in one occupational cohort after control for other factors (Greenland et al., 1994). Animal studies have shown that exposure of mice to Kanechlor-400 (a Japanese PCB product) resulted in various kinds of lung neoplasms (Nakanishi et al., 1999).

PCB Exposure Also Increases Risk of a Variety of Non-Cancer Diseases:

Neurobehavioral Effects:

The effects of PCBs on the nervous system are probably some of the most damaging insofar as effects are on the general population, and because PCBs adversely and apparently permanently reduce IQ and cause adverse behavioral changes. While these have been primarily studied in children and in animal models, there is evidence that even adult exposure is associated with adverse central nervous system effects. Smith et al. (1982) reported that there was a statistically significant association between serum PCB levels in occupationally exposed workers and systemic malaise and altered peripheral sensation. Emmett et al. (1988) found exposed workers to suffer from more eye irritation, increased tearing, chest pain on walking, wheezing, loss of appetite, frequent headaches, trouble sleeping and memory trouble than controls.

The mechanisms responsible for the nervous system effects are still not certain, although several specific actions of PCBs on nervous tissue are known. One of the most important is the demonstration that some PCB congeners block the synthesis of dopamine (Shain et al., 1991), a neurotransmitter known to be reduced in patients with depression and to be involved in most forms of mental illness (Dolan et al., 1995). PCBs also block the uptake of dopamine into the synaptic vesicles in the nerve terminals (Mariussen et al., 1999). PCBs also induce hypothyroidism, which is also characterized by depression (Duval et al., 1999). While the reduced dopamine and thyroid function connection is probably responsible for the depression and abnormal behavior seen in exposed persons, the mechanism for the reduced IQ is probably through the ability of PCBs to block the process of long-term potentiation (LTP) (Hussain et al.,

2000), known to be a critical component of learning and memory. Animals fed PCBs also show hyperactivity, impulsiveness and a reduced ability to deal with frustration (Berger et al., 2001; Carpenter et al., 2002b; Daly et al., 1996).

Jacobson and Jacobson (1996) followed 212 children from birth to 11 years of age. This paper reports IQ at age 11 years. PCB levels were determined in mother's serum and breast milk, umbilical cord blood and the child's blood at age 11 years. The average mother's serum level at the time of the child's birth was 6 ppb, while the child's at 11 years was 1 ppb. These values are not lipid adjusted. The breast milk on average contained 841 ppb of PCBs, lipid adjusted. They show a significant reduction in full scale IQ in children whose mother's breast milk had PCB concentrations of 1,250 ppb (lipid adjusted) or greater and poorer performance on reading mastery in children whose mother had 1,000 ppb or greater PCBs in breast milk. They conclude that perinatal exposure to PCBs causes irreversible decrement of IQ.

Chen et al. (1992) reported on the cognitive development of the children born to mothers who ate rice oil contaminated with PCBs and dibenzofurans in Taiwan in 1978. The problem continued for about 6 months until some of the adults developed skin lesions (chloracne). These authors studied 118 children born to exposed mothers (some born years after the poisoning) and 118 matched controls. The exposed children scored approximately 5 points lower on the Wechsler Intelligence Scale for Children. The children were followed at ages 4, 5, 6 and 7, and there was no obvious improvement with time. This study did not obtain PCB levels, but reports that after the exposure the average levels were 49.3 in adults (range 2.0-456 ppb) (while the median level was 25.5 ppb) in the exposed population, while the mean PCB level in 92 Taiwanese blood donors (not the controls in this study, but perhaps reflective of the population) was 9.8 ppb.

Lonky et al. (1996) attempted to replicate the studies by Jacobson through study of children born to mothers who ate PCB-contaminated Lake Ontario fish. Their paper does not give PCB values (they only studied cord blood, but this data is in a different paper), but categorizes mothers as a) no fish", "low fish" and "high fish". There were over 150 mother-infant pairs in each group. They used the Neonatal Behavioral Assessment Scale to study neurologic development over the first 48 hours of life, and found that infants in the "high fish" category showed more abnormal reflexes, greater responses to stress and less habituation to repeated stimuli than did the "no fish" and "low fish" babies. These results are consistent with effects of PCBs on the brain before birth. These children have since been studied at older ages. At 38 and 54 months, cord blood PCB levels were related to a statistically significant decrement in cognitive performance (Stewart et al., 2003a). Stewart et al. (2003b; 2005) found that response inhibition was reduced in children in relation to their prenatal PCB exposure. Exposed children were characterized by showing excessive and impulsive responding at ages 4.5 and 9.5 years of age. Poor response inhibition is characteristic of ADHD (Barkley, 1997). Exposed children also show a shortened attention span, greater frustration and antisocial behavior (May, 2000), all symptoms of ADHD as well.

Schantz et al. (2001) studied memory function in 572 adults who ate a great amount of contaminated Great Lakes fish, and 419 people who did not. These individuals were of a sufficient age that their exposure could not have come from early life exposure. Three memory

tests were used, as well as some visual tests. They measured serum PCBs and a number of other contaminants. None of the other contaminants were associated with a poorer performance on memory tests, but there was clear reduction in relation to serum PCB levels. The PCB levels were divided into four groups: ND-4.6 ppb, 4.7-7.8 ppb, 7.9-13.8 ppb and 13.9-75.0 ppb. There was a decrease in performance as dose was increased in all three tests. This study is important because it is the only one clearly showing that even adults can suffer from loss of IQ and memory upon exposure. Thus while prenatal exposure may result in permanent cognitive and neurobehavioral changes, exposure at any age can cause cognitive decrements.

Studies in animals indicate that exposure to PCBs also results in behavioral changes that are similar to those of ADHD (Carpenter et al, 2002b; Sagvolden et al., 2005). Rice (2000) found that monkeys exposed to PCBs showed deficits on discrimination reversal and spatial delayed alternation performance, and deficits in ability to change an already established response strategy and to inhibit inappropriate responses. With PCB exposure monkeys performed differently from controls on a fixed interval schedule of reinforcement, which requires the temporal organization of behavior using only internal cues. Studies in rats fed PCB-contaminated fish have shown hyperactivity and frustration when a reward is delayed as well as reduction of electrophysiological indicators of cognition (Carpenter et al., 2002b). These findings are also consistent with PCB induction of the symptoms of ADHD in animals.

The general conclusion is that the higher the child's early life exposure to PCBs the lower the IQ, the more the child exhibits anti-social behavior, depression and ADHD symptoms. The effect is found over the full range of IQ, however, and even the bright kids would have been brighter had they not been exposed. The decrement in IQ appears to be permanent. The study by Schantz et al. (2001) and our observations with the Mohawks show that there is a similar reduction of memory in adults exposed to PCBs (see below). Although cognitive function and attention are governed by different brain regions, it is striking to note that exposure to PCBs appears to alter both functions in parallel (Carpenter, 2006). Thus both animal and human studies are consistent with the conclusion that exposure to PCBs not only results in a reduced IQ and learning disabilities, but also causes ADHD.

The adverse neurobehavioral effects of PCBs are of particular concern when PCBs are present in schools, since that is where children come to learn. Regardless of route of exposure, PCBs result in reduced IQ and altered behavior, especially reduced attention span (Newman et al., 2009; Stewart et al., 2008; Haase et al., 2009; Sagiv et al., 2012).

Infections More Often and More Serious:

Experimental exposure of animals to PCBs results in a reduction in the function of the immune system (Tryphonas, 1995), and immunosuppression is one of the most sensitive indicators of PCB exposure according to scientists from the federal ATSDR and EPA (Johnson et al., 1999). PCBs are known to inhibit the immune system by at least two different mechanisms. Dioxin-like PCBs and dioxins/furans activate the Ah receptor, and this action induces a variety of genes. Through this mechanism the immune cells in the thymus gland are triggered to die by the process of apoptosis (McConkey and Orrenius, 1989). In addition, there are non-Ah receptor-mediated forms of immunosuppression (Kerkvliet et al., 1990) and ortho-

substituted PCBs rapidly kill thymocytes by a different process which depends upon insertion of the PCB molecule into the cellular membranes (Tan et al., 2003). Both of these mechanisms cause suppression of lymphocytes, the white blood cells responsible for certain kinds of immunity. In addition granulocytes, another type of white blood cell, have been shown to release their protective granules upon exposure to PCBs (Tithof et al., 1996; Voie et al., 1998). This results in their not being available to kill bacteria.

Suppression of the immune system is important because individuals with subnormal immunity are more susceptible to infections and to cancer. Thus frequent infections may be a direct result of PCB exposure. Until recently the ATSDR considered immune suppression to be the biologic effect that occurred at the lowest PCB concentration, although they now believe that effects on neurobehavior occur at even lower doses. A study with a good range of concentrations is Weisglas-Kuperus et al. (2000), and this study shows clear immunosuppression at levels of the order of 1-5 ppb. This demonstrates that the immune system is compromised at any detectible level of PCB exposure.

Human studies have clearly shown that people exposed to PCBs have a greater incidence of infection of all kinds. Lu and Wu (1985) studied patients in Taiwan who were exposed to PCB-contaminated rice oil in 1979 one year after the event. As compared to controls, the exposed patients had more infections, especially respiratory tract and skin. The initial PCB levels in the patients varied between 3 to 1156 ppb with a mean of 89 ppb. When a sub-group was tested later they showed elevated IgA and IgM, reduced response to a skin test to an antigen, and a reduced number of several classes of white blood cells. This study does not report PCB levels in these specific subjects, but the differences are significant relative to unexposed controls.

Weisglas-Kuperus et al. (1995) showed that Dutch children exposed to a mixture of PCBs and dioxins had lower levels of monocytes and granulocytes at 3 months of age than less exposed children. In a later study (Weisglas-Kuperus et al., 2000) they studied 207 Dutch mother-infant pairs, measuring the sum of PCBs 118, 138, 153 and 180 in mother's serum, cord blood and breast milk. They state that these four congeners constitute 46% of total PCBs. The range of PCB levels in children at 42 months of age was from 0.08 to 5.90 ppb, with the average well below 1 ppb. Adjusted for confounders, higher PCB levels were associated with a higher incidence of recurrent middle-ear infections (elevated 3-fold) and of chicken pox (elevated 7.6-fold), and a lower prevalence of asthma (which is due to a hyperactive immune system). The children with the higher PCB levels had more coughing, chest congestion and phlegm. They conclude that the higher the PCB level the greater the frequency and severity of infections, but the lower the frequency of allergic diseases. These children were followed to school age, and were found to still have a higher prevalence of ear infections (Weisglas-Kuperus et al., 2004). Dewailly et al. (2000) found that one-year old infants fed milk contaminated with PCBs had a 20-fold higher incidence of infectious diseases such as measles, meningitis and middle ear infections than children less exposed. Van Den Heuvel et al. (2002), in a study of Belgian adolescents, found that as levels of three PCB congeners and total dioxin equivalents (TEQs) increased, there was an increase in serum IgA, one of the immunoglobins made by lymphocytes, and a decrease in IgG and IgE. These are all good markers for the integrity of the immune system.

We have recently studied rates of hospitalization for infectious diseases in relation to residence near to PCB contaminated sites. Carpenter et al. (2003) have reported a 30% greater hospitalization for young children for several different infectious diseases if they live in a zip code that contains or abuts a PCB-contaminated site. Kudyakov et al., (2004) have found that hospitalization rates for acute infectious and chronic respiratory diseases (such as chronic bronchitis and chronic obstructive pulmonary disease) are significantly elevated in persons living in PCB-contaminated zip codes in upstate New York. As a control for other well-documented confounders, they reported that while individuals living along the Hudson River have a greater family income than other upstate residents, and on the basis of the Behavioral Risk Factor Surveillance System smoke less, exercise more and eat more fruits and vegetables than other New Yorkers, yet they show significantly higher rates of hospitalization for these acute and chronic respiratory infections. We attribute the elevated rates to inhalation exposure to PCBs coming from the Hudson River, resulting in suppression of immune function. We (Ma et al., 2007) have recently published a report that deal specifically with hospitalization of children for respiratory infections and found that there was a 15% elevation in rates of hospitalization of children ages 0-9 for respiratory infectious diseases if they lived in the zip code containing a waste site containing persistent organic pollutants such as PCBs as compared to children living in a zip code without a waste site. This was after adjusting for gender, race, urban, rural residence and medium household income.

Thyroid Disease:

Thyroid hormone has two rings, structures which have iodines on them, much like PCBs which have two rings but with chlorines. It is clear from a number of animal studies that PCBs interfere with thyroid hormone at multiple sites (Langer, 1997; Porterfield, 2000), and disrupt thyroid function through actions at multiple sites (Brouwer et al., 1998). In the absence of normal thyroid function during development one would expect reduced growth and reduced IQ (a "cretin" is a grossly retarded person without a functioning thyroid). In adults hypothyroidism is characterized by dullness, lack of energy, excessive weight, dry skin and excessive sleeping, while hyperthyroidism is characterized by excessive energy, weight loss, inability to sleep and hyperactivity. Thyroid function is usually determined by measurement of thyroid stimulating hormone (TSH), the brain hormone that tells the thyroid to work harder, and thyroxin (T4) or triiodothyronine (T3), the hormones released from the thyroid gland. Thus, in hypothyroidism TSH is elevated and T3 and/or T4 are low, while in hyperthyroidism these changes are opposite.

Schell et al. (2002; 2004) studied PCBs and thyroid hormones in Mohawk adolescents. The mean PCB level was 1.82 ppb, the maximum level 4.75 ppb. They found a statistically significant positive relationship and dose-dependent relationship between TSH levels and total PCBs, and a significant negative relation with both free and total thyroxin. There was a negative relation to T3, but not at a level that was significant. This study shows clearly that PCBs at levels common in the population reduce thyroid function, that this occurs at a very low concentration and that there is no threshold for this effect.

A later study Schell et al. (2008) found that PCB levels in adolescents that were breast fed (median 0.95 ppb) were significantly higher than in those that were not (0.68 ppb). They found that the relationships between PCB levels and altered thyroid function was greater in

adolescents who were not breast fed than in those who were, even though the PCB levels were lower in non-breastfed adolescents. This observation requires further study, but indicates again that prenatal exposure to PCBs alters thyroid function. This either indicates a beneficial effect of breast feeding, or that PCB exposure via breast feeding obscures the relationship to an adverse effect of PCBs on thyroid hormone homeostasis by adding to the serum PCB level even at these later ages. Since these are adolescents, ages 10-16 years, they have had a relatively long period of time in which to get rid of the PCB intake before and immediately after birth, but still have elevated levels from this period of time. Furthermore this is the most critical period of time for brain development, cognitive potential and behavior. These observations in the Mohawks are consistent with studies from other groups. For example, Skaare and Polder (1990) have reported that primipara women have 1.5 to 2.1 times levels of PCBs than those nursing their second or third child. Abraham et al. (1998) report that infant concentration of organochlorine contaminants at the end of the first year of life can be 1.5 to 3.6 times higher than those in the mother due to decreasing maternal levels during gestation and lactation.

We also have evidence that there is a direct inverse relationship between serum PCB levels and thyroid function in adults. This has been the subject of a doctoral dissertation by Dr. Serban Negoita (2008). He has demonstrated that adults with a serum PCB concentration in the $>20^{th}$ percentile as compared to the $<20^{th}$ percentile of his sample have a 6-8 fold elevated risk of being hypothyroid, depending upon which PCB congeners are present.

The relationship between serum PCBs and thyroid disease is important because being hypothyroid is associated with a variety of characteristics, including being mentally dull, lacking energy, and gaining weight. Thus being hypothyroid increases risk of being less productive in the society.

Animal studies have demonstrated that acute PCB exposure causes an elevation of thyroid hormones and histological changes in thyroid gland structure (Casey et al., 1999). Elevated thyroid hormones levels have also been reported in some human studies (Murai et al., 1987), but in most reports PCB exposure is associated with hypothyroidism. Koopman-Essenboom et al. (1994) studied thyroid function in mother-infant pairs in the Dutch study, and found that higher organochlorine levels in mother's milk correlated with lower plasma maternal T3 and T4 levels, and higher TSH levels in the infants. Osius et al. (1999) studied 1,091 second grade children in Germany living near an incinerator. They obtained blood samples where they measured thyroid hormones and eight PCB congeners. They found one congener (PCB 118) correlated positively with thyroid stimulating hormone (TSH), the brain hormone that makes the thyroid work, while five other congeners were negatively correlated with free T3, the active form of thyroid hormone. The sum of the PCBs measured had a mean of 0.49 ppb, and a maximum of 4.48 ppb. Turyk et al. (2006) reported studies of thyroid function in adult male Great Lakes fish consumers, and found significant negative associations with thyroid function.

Carpenter et al. (2001) investigated hospitalization for thyroid disease in women in Western New York to test the hypothesis that exposure to PCBs and dioxins would increase this class of disease. They investigated rates of hospitalization with a diagnosis of thyroid disease in individuals living near PCB contaminates sites as compared to not living near to PCB-contaminated sites. They found a highly significant increased diagnosis of thyroid disease in

women at all ages greater than 25 years, but not in men. Thyroid disease is much more frequent in women than men.

Hypertension:

Kreiss et al. (1981) reported that people living in Triana, Alabama, who had a high serum PCB concentration from eating local, contaminated fish, showed a significantly higher incidence of high blood pressure. The increase in blood pressure was independent of age, sex, body mass index and social class. Rates of borderline or definite hypertension were 30% higher than expected from national rates in a population of 458 persons with an average serum PCB concentration of 17.2 ppb. The relationship between PCB concentrations was highly significant for diastolic blood pressure and of borderline significance for systolic blood pressure. Excess hypertension has also been reported in several occupational studies (Morgan et al., 1980; Sandifer and Keil, 1972; Stehr-Green et al., 1985). We (Huang et al., 2006) have found that rates of hospitalization for hypertension are 19.2% greater for individuals living in a zip code containing a waste site with PCBs, dioxins or chlorinated pesticides than those living in a zip code without any waste sites. The publication of Goncharov et al. (2008) also demonstrated that exposure of adult Mohawks to PCBs resulted in an increase in reported rates of high blood pressure. It shows that there is no threshold for elevated risk of hypertension, and that the risk increases with dose. In subsequent studies of residents of Anniston, Alabama, who live near to the Monsanto plant that manufactured PCBs from 1929-1971, we (Goncharov et al., 2009; 2010) that serum PCB levels were a greater risk factor for hypertension than any other factor but age, including BMI, race, sex, and serum lipid levels.

Hyperlipidemia: Relation to Cardiovascular Disease:

The best understood risk factors for ischemic heart disease are hypertension (see above) and elevation of serum lipid levels (Wilsgaard et al., 2001). Animal studies dating from the 1970s and later have demonstrated that exposure to dioxin is associated with an elevation in the levels of low-density plasma triglycerides (see Lovati et al., 1984). Mochizuki et al. (1998; 2000) showed that rats fed PCBs have significantly higher HDL-cholesterol and lipids as compared to control rats. Other studies in rats indicated that PCBs induce the liver to make more of the enzymes that synthesize lipids (Boll et al., 1998; Oda et al., 1994). Bell et al. (1994) exposed 67 female rhesus monkeys to Aroclor 1254, and found that the PCBs caused an increase in plasma triglycerides and a decrease in HDL-cholesterol (the "good" cholesterol). These changes would be expected to increase risk of cardiovascular disease. Surprisingly, they found a decrease in total cholesterol, which is not what is usually seen in humans. In a recent animal study Riecke et al. (2002) found that a single dose of dioxin at a level corresponding to the current average human concentration resulted in direct damage to the heart muscle, which they interpret as being the result of disturbance of various growth factors.

Baker et al. (1980) first reported that workers exposed to PCBs showed a significant direct correlation between serum PCB levels and plasma triglyceride levels. They found that blood levels of PCBs in people working in a sewage sludge plant in Indiana and their families

were between 17.4 and 75.1 ppb, and that there was a highly significant relationship between plasma triglyceride levels and serum PCB concentrations. Kreiss et al (1985) determined serum lipids as a function of serum PCB levels in 458 people over 12 years of age in Triana, Alabama, where there was significant contamination. They found the higher the PCB level, the higher the serum cholesterol level, and the higher the blood pressure. The mean PCB level was 17.2 ppb, with a range from 3.2 to 157.9. Sixty of these people had levels greater than 30 ppb. Chase et al. (1982) found significantly elevated serum triglyceride levels in exposed workers. Martin (1984) reported that workers exposed to dioxin in an accident still had statistically significant elevations of both serum cholesterol and triglyceride concentrations ten years later. Gustavsson and Hogstedt (1997) studied 242 male capacitor workers exposed to PCBs, and found a significantly increased incidence of death from cardiovascular disease, with a latency of 20 years. A number of other studies report similar results (see Hay and Tarrel, 1997), but do not really provide any exposure data. Occupational exposure to dioxins/furans has also been correlated with an excess incidence of cardiovascular disease (Calvert et al., 1998; Flesch-Janys et al., 19956; Vena et al., 1998).

Moysich et al., (2002) reported a significant correlation between total lipids and serum PCB levels even after age adjustment in a study of anglers in New York. Tokunaga and Kataoka (2003) recently reported on the relationship between PCB exposure and serum lipids in the Japanese population exposed through contaminated rice oil in the 1960s. They found a ten-fold elevation of PCB levels was associated with an elevation of serum total cholesterol by 18.4 mg/dL ($p<0.001$) in men and 17.5 mg/dL in women, and of serum triglycerides by 43.3% in men and 42.8% in women.

In addition to elevation of serum lipids, PCBs also directly damage the endothelial cells which line the arteries. Hennig et al. (1999) have demonstrated that dioxin-like PCBs induce oxidative stress in endothelial cells through the generation of reactive oxygen species (Slim et al., 1999), and that in the presence of certain kinds of lipids (in this case linoleic acid) this results in damage to the endothelial cell. Recently, they have expanded their studies and have demonstrated that non-dioxin-like PCBs also induce oxidative stress in endothelial cells, and that this is mediated via stimulation of inflammatory processes (Choi et al., 2003). In some cases this actually results in the death of the endothelial cells (Lee et al., 2003), which would obviously have devastating effects on the cardiovascular system. There is overwhelming evidence that both dioxins and dioxin-like PCBs induce oxidative stress in a variety of tissues from animal studies (Hassoun et al., 2002). There are also clearly other effects of dioxin-like PCBs on the cardiovascular system that are just now getting significant attention. Jokinen et al. (2004) reported that both dioxin and PCB 126 cause cardiomyopathy, a degenerative cardiac muscle disease and chronic active arteritis. One organ that appears to be particularly vulnerable to arteritis is the pancreas, which may also have relevance to increased risk of diabetes.

Sergeev and Carpenter (2005) have recently reported hospitalization discharge diagnosis rates for coronary heart disease and myocardial infarction among New York State residents who live in a zip code that contains or abuts a hazardous waste site containing persistent organic pollutants (POPs), of which PCBs are the most frequent contaminant. They found that rates of diagnosis of coronary heart disease were 15% higher in residents who live in zip codes containing or abutting POPs sites, while rates of myocardial infarction were 20% higher in these

zip codes. They also studied a subset of zip codes along the Hudson River, which is 200 miles of a National Priority Site highly contaminated with PCBs, and where average income is higher, smoking rates lower, and where residents exercise more frequently and consume fruits and vegetables more regularly than in the other parts of the state. In spite of a higher socio-economic status and healthier life style, they found a 35.8% higher frequency diagnosis of coronary heart disease and a 39.1% more frequent diagnosis of myocardial infarction in this population. Shcherbatykh et al. (2005) found a similar elevation in rates of stroke using the same kind of analysis, with a 15% elevation in individuals living near waste sites with PCBs and other persistent chlorinated compounds. Somewhat similar results were reported by Ha et al (2007) using the National Health and Nutrition Examination Survey. They found that levels of both dioxin-like and non-dioxin-like PCBs were correlated with prevalence of cardiovascular disease in women.

Our studies in the Mohawks have clearly shown a relationship between serum PCB level and cardiovascular disease. Goncharov et al. (2008) have demonstrated that there is a direct relationship between adult Mohawk levels of serum PCBs with levels of serum cholesterol and triglycerides. Furthermore they find a statistically significant relationship between reported rates of heart disease and serum PCB levels. The analysis shows that the effect of PCBs on heart disease is secondary to the elevation in serum lipids, and is almost certainly not a direct effect on the heart. The report shows that the effect of PCBs on serum lipids increased over the full range of serum PCB levels. This clear dose-response relationship indicates that there is no threshold of PCB level that is safe, and that the greater the exposure the greater the risk of elevated serum lipids and cardiovascular disease.

Infertility and Disorders of the Reproductive System:

As with thyroid hormone, PCBs alter sex hormones and therefore affect a number of diseases dependent on sex hormone activity (Faroon et al., 2001). As mentioned above, some PCBs and their break-down products are estrogenic, mimicking the female sex hormone. Others have exactly the opposite action, being antiestrogenic. Therefore, the composition of the mixture to which one is exposed can alter the disease. This is a very active area of research, with emotive things such as sperm counts, general fertility and sexual preference all being studied in relation to prenatal exposure to PCBs. Diseases shown clearly in animals to result from exposure to PCBs include endometriosis, reduced fertility in both males and females, feminization of sexual behavior in males, and smaller genitalia in males.

One of the best documented effects of PCB exposure is on male sexual function. PCBs are potent inhibitors of the synthesis of the male sex hormone, testosterone (Kovacevic et al., 1995). Testosterone is the basis of sexual arousal and secondary sexual characteristics (Bardin and Catterall, 1981). Animals exposed to PCB-containing transformer fluids show decreased levels of testosterone (Andric et al., 2000). In addition, PCBs compete with testosterone for binding at the testosterone receptor (Portigal et al., 2002), which even further reduces masculinity. Serum levels of PCB 153 have been shown to be inversely correlated with free testosterone levels in healthy, young humans (Richthoff et al., 2003). In addition higher PCB levels have been correlated with reduced sperm mobility (Bush et al., 1986; Richthoff et al.,

2003). Men in Taiwan exposed to PCBs in 1978-1979 were studied by Hsu et al. (2003) in 1999-2002, and were found to have more abnormal sperm than controls, and the sperm they did have showed a reduced capability to bind and penetrate oocytes. In occupational studies of exposed workers, Emmett et al. (1988) reported a significantly greater incidence of fertility problems in the exposed group. For both dioxins (Mocarelli et al., 2000) and PCBs (Weisskopf et al., 2003) there is clear evidence that the ratio of male to female births is strikingly reduced following parental exposure.

Exposure to these compounds also alters female reproduction. Both PCB (Denham et al., 2005 in a study of Mohawk adolescents) and PBB (Blanck et al., 200) exposure causes an earlier menarche in girls. There is clear evidence that exposure of monkeys to dioxins and dioxin-like PCBs results in endometriosis (Rier et al., 1993; 2001; Rier and Foster, 2002). However, it has proven difficult to confirm this observation in humans because endometriosis is rarely diagnosed definitively by surgery. One recent study did show an elevated incidence of endometriosis in individuals with higher dioxin toxic equivalents (Pauwels et al., 2001).

Diabetes:

While diabetes is rarely considered to be an environmentally-induced disease, there is strong evidence that environmental exposure contributes to incidence of this disease. Some of the earliest evidence came from study of the US Air Force personnel who dropped Agent Orange contaminated with dioxin on Vietnam during the War. A highly significant relationship between exposure to dioxin and onset and severity of diabetes was found in those individuals with the greatest exposure (Henrikson et al., 1997). This led to a committee of the National Academy of Sciences' Institute of Medicine report (IOM, 2000), which concluded that there was suggestive evidence of an association between dioxin exposure and diabetes. Pesatori et al. (1998) and Bertazzi et al. (1998) studied the individuals exposed to dioxins at Severso, Italy, and found elevated diabetes in exposed individuals. A similar conclusion was drawn by Vena et al. (1998) from a study of phenoxyacid herbicides and chlorophenol production workers exposed to dioxins. Cranmer et al. (2000) studied a population of individuals exposed to dioxin from a Superfund site, and demonstrated that plasma insulin concentrations were significantly higher in individuals with elevated dioxin levels, and concluded that high serum dioxins levels cause insulin resistance.

Longnecker et al (2001) studied 2,245 pregnant women, 44 of whom had diabetes. The mean serum PCB level in the women with diabetes (3.77 ppb) was 30% higher than the controls (2.79 ppb), and the relationship of PCB level to adjusted odds ratio for diabetes was linear. Taking PCB levels < 2.50 ppb to have an odds ratio of 1.0, the odds ratio was 2.9 for PCB levels 2.50-3.75, 4.4 for PCB levels 3.75-5.00 and 5.1 for PCB levels greater than 5.0. All values were statistically significant. This is an excellent study showing a dose-response relationship. Strong support for this relationship between exposure and diabetes is also found in the study of Fierens et al. (2003) from a population-based study in which they found after adjustment for age and other covariates that total TEF and 12 marker PCB concentrations were 62% and 39% higher, respectively than in controls. The ORs were 5.1 (95% CI = 1.18-21.7) for dioxins, 13.3 (95% CI = 3.31-53.2 for coplanar PCBs and 7.6 (95% CI = 1.58-36.3) for 12 marker PCBs. Vasiliu et al.

(2006) investigated a Michigan cohort that had elevated exposure to polybrominated biphenyls (PBB) in relation to risk of diabetes, and found a significant relationship with PCB concentrations (odds ratio 2.33, 95% CI = 1.25-4.34), but not with PBB concentrations.

The strongest evidence for the relationship between PCB and other organochlorine exposure and diabetes has come from use of the data from the US National Health and Examination Survey. Lee et al. (2006) studied PCB 153, two dioxin congeners and three pesticide levels in relation to risk of diabetes in a study of 2,016 adults. For PCB 153 the odd ratio for individuals with serum levels in the greater than 90th percentile was 6.8 (95% CI = 3.0-15.5), and the p for trend was <0.001 with increasing concentration. Rylander et al. (2005) have also reported a significant elevated risk of diabetes in relation to concentrations of PCB 153 in Swedish fisherman and their wives. Everett et al. (2007) independently analyzed similar data to that used by Lee et al. (2006) and investigated 2,090 persons in relation to levels of PCB 126. PCB 126 levels >83.8 pg/g showed an odds ratio for diabetes of 3.68 (95% CI=2.09-6.49) as compared to PCB 126 levels < 31.2 pg/g. The results of Lee et al. (2006) and Everett et al. (2007) are of particular interest because PCB 153 is not active at the dioxin receptor, whereas PCB 126 is. Therefore it may be that both dioxin-like and non-dioxin-like PCBs increase risk of diabetes. A striking finding in the Lee et al. (2006) study was that obese individuals who did not have elevated levels of organochlorines were not at increased risk of diabetes, consistent with the possibility that it is the PCBs and related compounds that cause diabetes, not obesity (see Porta, 2006).

Animal studies are consistent with the evidence that PCB and dioxin exposure increases risk of diabetes. Nishizume et al. (1995) showed that rats given Kanechlor-400 (a Japanese equivalent to Aroclor) showed depressed insulin sensitivity which increased with the duration of PCB exposure, as well as disturbed glucose and lipid metabolism and elevated serum lipids. Stahl (1995) reported that dioxin alters enzyme activity related to glucose metabolism in rat liver cells. Several older studies have demonstrated morphological changes in the structure of the beta cells in the pancreas (where insulin is made) upon PCB exposure (Kimbrough et al., 1972; Wassermann et al., 1975). Boll et al. (1998) demonstrated that gluconeogenic enzymes in rat liver are altered upon PCB exposure.

Liver Disease:

The draft EPA Dioxin Reassessment document gives the statement "Increased liver size is consistently reported in treated animals after exposure to 2,3,7,8-TCDD...Among exposed human populations, four case reports in three populations, but not controlled epidemiologic studies, described evidence of enlarged livers or hepatomegaly." With regard to enzyme changes following dioxin exposure there is the following statement: "Laboratory studies have demonstrated changes in hepatic enzyme levels after 2,3,7,8-TCDD exposure, although there is considerable interspecies variation in the observed effect. … Epidemiologic studies and case reports describe elevated liver enzymes among exposed TCP production workers and among Severso residents." While this is still not an official report, the references to the studies indicated are included in the draft document.

In studies focused specifically on PCBs, Kreiss et al. (1981) reported that the exposed

residents of Triana, Alabama showed a positive relationship between serum PCBs levels and gamma-glutamyl transpeptidase (GGTP) levels. Chase et al. (1982) and Fischbein (1985) both reported significantly elevated serum glutamic oxaloacetic transaminase (SGOT) levels in 120 workers exposed to PCBs. Smith et al. (1982) surveyed three groups of PCB workers and found significant elevations of both SGOT and GGTP positively correlated with serum PCB levels. Maroni et al (1981) found that 16 of 80 workers exposed to PCBs had hepatomegaly with an increase in serum GGT, AST, ALT and OCT. The studies of Fitzgerald et al. (2005) demonstrated that PCB exposed increased rates of metabolism of caffeine, providing proof that PCB exposure results in induction of elevated levels of liver enzymes.

All of these effects are an indication of liver injury and are likely contributing factors leading to the development of liver cancer and liver failure.

Asthma and Other Respiratory Disease:

While dioxin exposure is known to reduce the incidence of asthma (Weisglas-Kuperus et al., 2000), PCB exposure is associated with a significant increase in risk of asthma (Van Den Heuvel, et al., 2002). The latter report demonstrated and increased risk of 2.12 fold based on concentrations of PCBs 138, 153 and 180, while they confirmed the opposite effect of dioxin-like compounds using the CALUX assay, which measures total dioxin like activity, including that of the dioxin-like PCBs. It is likely that the specific PCB effect on asthma is secondary to the immune system alterations induced by the ortho-substituted PCBs, as have been studied by Tan et al. (2003). As mentioned above, Kudyakov et al. (2004) reported significant elevations in rates of hospitalization for chronic bronchitis and chronic obstructive pulmonary disease among residents living near to PCB-containing hazardous waste sites. This may be secondary to suppression of the immune system, but they could not rule out direct harmful affects on the lung. We (Ma et al., 2007) have found a 1.7-fold elevation in rates of hospitalization for asthma among children living in upstate New York in zip codes containing PCB, dioxin or chlorinated pesticide-containing hazardous wastes sites, as compared to children living in zip codes without an identified waste site. The children also showed elevated rates of hospitalization for respiratory infections.

Osteoarthritis:

An elevation in the incidence of joint disease has been seen in both of the Asian populations who were exposed to PCBs in cooking oil. Kuratsune (1980) reported elevated incidence of joint inflammation in the Yushu exposed persons in Japan. Guo et al. (1999) report that Taiwanese men exposed to PCBs in the same accident had a 4.1-fold elevated risk of developing arthritis. Guo et al. (1999) also reported that Taiwanese men exposed to PCBs had a 2.9-fold increased risk of developing back problems because of intervertebral disc disease. As with asthma, the mechanism responsible is not known, but both of these studies show highly significant effects in relation to PCB exposure.

Low Birth Weight as a Factor Increasing Risk of Diabetes, Cardiovascular Disease and Hypertension:

Several studies (see Baibergenova et al., 2003, for references) have shown that PCB exposure increases the risk of babies having low birth weight. Taylor et al. (1984) showed that women working in a capacitor plant in areas where they were presumed to be exposed to PCBs gave birth to children 153 gm lower in weight and with an average of 6.6 days shorter gestation. This effect has been seen in a number of other studies and the effect appears greater in male than female infants.

We (Baibergenova et al., 2003) have shown that maternal residence in a zip code containing or abutting a PCB-contaminated site significantly increases risk of giving birth to a low birth weight infant, and that the risk is greater for male than female infants. This is particularly important in relation to adult diseases because it has been clearly shown that low birth weight increases risk of several chronic diseases in adulthood, including cardiovascular disease (Barker, 1999), hypertension (Law et al., 2001), and diabetes (Rich-Edwards et al., 1999).

Although we have not collected birth weight information specifically in the Mohawk population, these previous studies indicate that elevated exposure causes elevated risk of giving birth to an infant of low birth weight. Low birth weight is a risk factor for early infant death, as well as of all of the diseases identified above, including cardiovascular disease, hypertension and diabetes.

Deficits in Hearing:

A number of investigators have demonstrated that developmental exposure of laboratory animals to PCBs results in impaired auditory function (Goldey et al., 1995; Crofton and Rice, 1999). This appears to be secondary to hypothyroidism, which impair ear development. Rylander and Hagmar (2000) found that Swedish boys born on the eastern coast of Sweden, where there is high rates of consumption of PCB-contaminated fish from the Baltic Sea, had significantly greater hearing impairments than those born on the west coast, and suggest that this is secondary to PCB exposure, although they did not obtain PCB concentrations.

The Situation in Malibu Schools:

In March, 2015, the school district released results of testing of caulk in the Juan Cabrillo Elementary (JCES), Malibu middle & high schools (known collectively as Malibu Schools) . Values ranged from 330 ppm in room 7 to 220,000 ppm in room 505 of the middle/high school and 130,000 ppm in room 22 to 570,000 ppm in room 19 of JCES. These levels in the caulking grossly exceed the levels set in the Toxic Substance Control Regulations for PCBs as hazardous waste (50 ppm), which require removal to an accredited hazardous waste site. This followed the November, 2013, tests of the building materials which showed PCBs in the caulking, paint, air and dust. But the school district had first learned there was PCB contamination in the soil in 2009, which presumably came from the building materials.

There are multiple routes of exposures to PCBs at the Malibu schools including inhalation, incidental ingestion, and direct dermal contact. The most serious is inhalation of vapor-phase PCBs. PCBs from building material slowly volatilize, and when PCBs in the air are breathed they are readily absorbed by the lung. Because everyone in the school must breathe continuously, PCBs in air constitute a continuous exposure. The risk of incidental ingestion is high based on the levels of PCBs in caulk found in the Malibu schools and the likelihood of kids and teachers coming into contact with it every day through the normal course of work and attending school. Incidental ingestion can occur by kids who have a habit called PICA, which is when non-food sources are eaten. This can also occur by putting their hands in their mouth after touching something in the classroom with PCBs on it. It can also occur when eating lunch outside under contaminated windows. Without identifying the locations of PCBs and performing a full risk assessment detailing how the school is used by teachers and kids, there can be no assertion of protection from incidental ingestion.

Direct contact occurs by touching PCB contaminated items and absorbing PCBs through the skin. It can also occur from vapor-phase PCBs in the air absorbing through your skin. Any PCB-contaminated material should not be touched because of the danger that PCBs will be absorbed. Once again, without identifying the locations of PCBs and performing a full risk assessment detailing how the school is used by teachers and kids, there can be no assertion of protection from direct contact and dermal exposure.

PCB exposure cannot be accessed by looking at one pathway at time, as this is not a scientifically sound approach nor is it indicative of real life exposures. All exposure pathways must be addressed and added together, as PCB exposure is additive and cumulative. PCBs can also have synergistic effects with other chemicals to which the children are exposed. A synergistic effect is when two chemicals have a greater effect than the sum of each alone or by simply adding them together.

Air PCB levels monitored in the various classrooms of the Malibu schools were found to be from 3ng/m^3 (with open windows) to 480 ng/m^3. It is important to note that all the air tests were not taken the same way, with the same control measures, and these tests were performed under two different EPA analysis methods. Based on the proven health risks of PCBs in the air at low levels, I would recommend that the reporting limit be set as low as background air whenever possible. The district's current reporting limit was set at approximately 70 ng/mg^3. Setting this high limit misses the important low levels of PCBs in the air that cause harm to health and allow the district to inaccurately claim that there are no finding of PCBs in the air.

Malibu Schools first used a threshold of 200 ng/m^3 for PCBs in the air, based on a draft site specific risk document from New York City's school created using EPA's internal PCB tool for PCBs. But EPA uses a general guidance policy on PCBs in the air ranging from 100-600 ng/m^3 depending on age. EPA's PCB tool and PCB policy is based on human health effects of Aroclor 1254 from 1994 (22 years ago), ingestion studies and not inhalation studies, and does not account for the full range of 209 congeners. Without this full range many important risk factors are excluded. The RfD (reference dose factor) for Aroclor 1254 is derived only from immunotoxic effects. For all of these reasons there is no scientific basis for the EPA's guideline. Furthermore this calculation is based solely in inhalation in the classroom, and does not take into

consideration of the possible additional exposures from ingestion and dermal uptake from the school vicinity nor excessive dietary and other possible sources of exposure from home. This EPA tool is completely inadequate to protect humans from the harmful effects of PCBs. Several EPA scientists have admitted this and have written it in scientific publications (Lehmann et al., 2015).

The major health concerns from PCB exposure at the Malibu schools are cancer, neurobehavioral toxicity and endocrine disruptive effects that are known to influence onset of puberty in girls (Denham et al., 2005) and levels of male sex hormones in boys (Schell et al., 2014). Furthermore, there is no consideration in the EPA draft guideline for effects of the lower chlorinated PCB congeners, which are the ones most clearly associated both with cancer and with neurobehavioral decrements. It is the lower chlorinated congeners that are most volatile, and these are not dominant in the pattern of Aroclor 1254, even though that mixture does contain some of them. This is again a reason that the EPA tool is inadequate to protect health.

Ampleman et al (2015) have reported that inhalation can account for up to one-third of total exposure to PCBs in children, and that the majority of this exposure comes from schools. The average concentration of PCBs in air in East Chicago schools was 6.4 ng/m^3, and this concentration was found to contribute significantly to the exposure of school children and add to their body burden. The PCBs levels found at the Malibu schools exceed those reported by Ampleman et al. (2015). Several other studies have documented elevations, especially of lower chlorinated congeners, in the blood of teachers and/or students working in schools with PCB contamination (Gabrio et al., 2000; Liebl et al., 2004; Herrick et al., 2011). Other studies of residents and/or workers in PCB-contaminated buildings have shown concentrations of lower chlorinated congeners four (Meyer et al., 2013) to ten (Pedersen et al., 2015) times higher than among control groups. There is definitive evidence that teachers working at PCB-contaminated schools have elevated concentrations of the lower-chlorinated, more volatile PCBs in their blood (Herrick et al., 2011) and the same will apply to children, who are more vulnerable to environmental contaminant exposures (Sly and Carpenter, 2012). Lower chlorinated, more volatile PCB exposure from indoor sources has been shown to be associated with increased risk of developmental abnormalities of the brain and altered neurobehavior in children (Wang et all., 2015). Inhalation of PCBs has been clearly shown to increase of cancer (Carpenter, 2015).

A recent publication from USEPA scientists acknowledges that inhalation may contribute more to total PCB exposure than previously assumed (Lehmann et al., 2015). However this information has not been translated into a guideline or a reference dose factor that reflects the large body of evidence that low concentrations of PCBs in air, under circumstances where children and teachers in schools are breathing that air for many hours per day, results in neurobehavioral decrements, elevated risk of cancer and disruption of endocrine systems. Because children and teachers are in PCB-contaminated environments every day during the school year, this constitutes a long-term exposure. This is yet another reason why EPA's policy on "safe" air levels is completely invalid and is not protective of children's and teachers' health.

As documented above and in the several publications from my group, individuals living near to hazardous waste sites containing PCBs are at excess risk of being hospitalized by cardiovascular disease (Sergeev and Carpenter, 2005), diabetes (Kouznetsova et al., 2007),

stroke (Shcherbatykh et al., 2005), hypertension (Huang et al., 2006), asthma (Ma et al., 2007) and respiratory infections (Kudyakov et al., 2004; Ma et al., 2007). We have concluded that the route of exposure to PCBs responsible for the excess rates of these diseases is inhalation. Although we do not have measurements of air concentrations at these sites, the air concentrations are certainly much lower than the EPA's acceptable range of 100 to 600 ng/m^3. Fitzgerald et al. (2008) have reported decrements in verbal learning and an increase in depressive symptoms in residents living near to the Hudson River, even though total PCB concentrations were not significantly different from controls.

Thyroid cancer is a relatively rare disease. According to the 2015 publication "The global burden of cancer" (Global Burden of Disease Cancer Collaboration, 2015) the incidence of thyroid cancer worldwide in 2013 was 1.54 cases per 100,000 persons. I have been informed by my client that there are self-reported cases of cancer and disease from those who work or have attended the Malibu schools. This information states that six teachers and four young alumni have developed thyroid cancer. While the total population from which these cases were drawn is uncertain, the number is certainly very much smaller than 100,000. Thus this is clearly a thyroid cancer cluster. Three teachers who were diagnosed with thyroid cancer within months of one another teach at the middle school, which has a total of approximately 30 teachers. These teachers taught in rooms where the PCB concentrations in caulk were greater than 50 ppm. In addition, at least 25 teachers have self-reported to have thyroid disease, including 14 of 30 middle school teachers. Two teachers have been diagnosed with malignant melanoma, the cancer most strongly associated with PCB exposure by IARC (2015). These data are not a result of a systematic survey of teachers and alumni, but rather are self-reported, suggesting that even these results may be an underestimation of the actual rates. These results are particularly striking as they support another similar report by Carpenter (2015) of a husband and wife team, both employed by a company dealing with waste oils that might or might not contain PCBs, both of whom were told to smell the oils to determine whether PCBs were present at significant concentrations. Both developed thyroid cancer when in their early '30s. The husband developed malignant melanoma, while the wife developed a dysplastic nevus, a precursor to malignant melanoma. The serum PCBs from samples from each of them showed elevations in the lower chlorinated congeners, but not in those with more chlorines, indicating that inhalation was their primary route of exposure.

Sensitive populations, such as pregnant teachers and children with special needs, are even more susceptible to the harm caused by PCBs. It is known that PCBs cross the placenta and they concentrate in breast milk. PCBs are known to have adverse health effects on the fetus, infants, and children. While it is not possible to identify a "safe" level of PCBs exposure for anyone, these sensitive populations are at even greater risk for harm from PCB exposure.

PCBs have no beneficial effect, and there is no evidence that there is any "safe" level of PCB exposure. This is particularly the case for PCB inhalation, since not breathing is not an option.

The individual congener profile in the air and in the caulking was done in early 2013 by the district's environmental consultant, Mark Katchen. These congener profiles can be related to Aroclor products sold for particular uses in building construction (ATSDR, 2000). Based on the

pattern of congeners in the air and caulk samples detected in classrooms in the same building, it can be reasonably determined, that the rest of the classrooms with similar patterns of PCBs in the air, contain similar PCB-contaminated caulking. In conclusion, if the air in rooms have the same or similar PCB profile, then it is reasonably certain that the rooms will also have the same PCB contaminated building materials.

On March 2014 the County of Los Angeles Public Health Department wrote to Sandra Lyon, Superintendent of the Santa Monica-Malibu Unified School District, and stated that the only risk factors for thyroid cancer are ionizing radiation, iodine deficiency, obesity, family history and history of thyroid conditions. This view reflects extraordinary ignorance of what is known about thyroid cancer and PCB exposure. Scientists at the General Electric Corporation exposed rats to various Aroclor mixtures, and found the development of thyroid cancer from Aroclor 1242, 1254, and 1260 (Mayes et al., 1998). PCB inhalation studies in rats showed altered thyroid hormones and cause histopathologic changes in thyroid structure (Casey et al., 1999). Studies reported above in the section on cancer, and especially the husband and wife who both developed thyroid cancer after being required to "smell" PCB-contaminated oil (Carpenter, 2015), provide evidence that PCBs cause thyroid cancer in humans, and indicate that inhalation is one important route of exposure. This response from the County of Los Angeles Public Health Department is wrong, totally unjustified and questionably ethical, especially since they apparently made no effort to document the rates of thyroid cancer and thyroid disease among teachers and former students.

Conclusions:

PCBs are known human carcinogens according to the World Health Organization. There is clear evidence that 50 ppm in building materials represents a real health risk, particularly to children. PCBs in indoor caulk lead to PCBs in air, PCBs in the dust, and PCBs migrating from the source material into other surrounding porous materials. The dangers of exposure to PCBs come from a variety of routes of exposure. Until the PCBs are identified, mitigated and removed, students and teachers will not be protected from their harm.

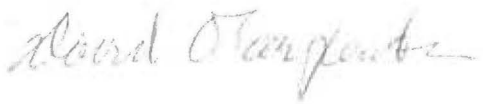
Based on the air, dust and materials testing that I have reviewed, students and teachers at the Malibu elementary, middle and high schools are exposed to dangerous levels of PCBs continuously from many different exposure pathways every moment they spend at the school. This means the students and staff have had long term exposure. The caulk contamination at the Malibu schools has been known since November 2013, and existed long before that. The toxic body burden in students and teachers is increasing on a daily basis from being at the Malibu schools. Because of this exposure they are at elevated risk of developing several different diseases. There is overwhelming documentation that PCB levels are excessive and wide-spread at these schools. Levels in caulk at up to 570,000 ppm that have been found in the schools greatly exceed the 50 ppm health regulations requiring removal under Federal law. Yet the school has not conducted a proper characterization of the PCB building material identifying the extent of contamination, and most classrooms' building materials have not been tested. By not identifying the source material, there is no way to protect children from PCB exposure. This means that children and teachers remain in continued serious risk for a variety of adverse health effects. Concentrations in air at Malibu Schools are in excess of those which have been shown by

peer reviewed studies to increase the risk of several human diseases and increase body burden. Children, whose minds and bodies are developing, are especially at risk. No child should be exposed to known and dangerous levels of PCBs, such as those that have been documented within the Malibu schools, for even one day, let alone for many years.

There is no logical scientific reasoning that warrants the actions of the school district to refuse PCB source identification, removal and other remedial actions if their goal is to protect the health of children and teachers. There is simply no scientific evidence for the district or their consultants to claim the school is safe based on air and dust testing alone. To date, there is no evidence that proves any level of PCBs in the air is safe, in fact, to the contrary, the few inhalation studies that are available show that even low levels, from 2-30 ng/mg^3 cause harm. The school district is misinforming the parents and teachers which is putting children and teacher's health at risk.

With no evidence of safe levels of PCBs in the air, the EPA's PCBs in Schools policy on "safe" air levels is scientifically unsubstantiated and is certainly inadequate to protect children and teachers from the harmful effects of PCBs.

Based on my knowledge of the dangers of PCBs, I would not want my wife, who is a teacher or my son as a student to work or attend these schools. The PCBs in the Malibu schools must be removed.



David O. Carpenter, MD
24 January 2016

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Vena J, Boffetta P, Becher H, Benn T, Bueno-de-Mesquita HB, et al. (1998) Exposure to dioxin and noneoplastic mortality in the expanded IARC international cohort study of phenoxy herbicide and chlorophenol production workers and sprayers. Environ Health Perspect 106 (Suppl 2): 645-653.

Walker NJ, Crockett P, Nyska A, Brix A, Jokinen MP, Sells DM, Hailey JR, Easterling M, Haseman JK, Yin M, Whde ME, Bucher JR, and Portier CJ (2005) Dose-additive carcinogenicity of a defined mixture of "dioxin-like compounds". Environ Health Perspect 113: 43-48.

Wilsgaard T, Jacobsen BK, Schirmer H, Thune I, Lochen ML, Njolstad I and Arnesen E (2001) Tracking of cardiovascular risk factors. Am J Epidemiol 154: 418-426.

Wolff MS, Camann D, Gammon M, and Stellman SD (1997) Proposed PCB congener groupings for epidemiological studies. Environ Health Perspect 105: 13-14.

Wolff MS, Fischbein A and Selikoff IJ (1992) Changes in PCB serum concentrations among capacitor manufacturing workers. Environ Res 59: 202-216.

David O. Carpenter, M.D.
Curriculum Vitae

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-Pres.	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:	Journal of Local and Global Health Sciences 2012-present
Editor-in-Chied:	Environmental Pollution 2015-present
Editorial Advisor:	Cellular and Molecular Neurobiology, 1987 – Present
Academic Editor:	Journal of Environmental and Public Health, 2009-2013
Academic Editor:	PLoS ONE 2014-present
Editorial Boards:	Journal of Public Health Management and Practice, 1995 - 2002
	International Journal of Occupational Medicine & Environmental Health 1996 – Present
	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-present
	Global Health Perspective, 2012-present
	Environment International 2013-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-Pres.	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
2003-2008	Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
2003-Pres.	Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
2004-Pres.	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-present	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-present	Member, Board of Directors, Regenerative Research Foundation

Honors, Awards and Fellowships:

1959	B.A. awarded magna cum laude. Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, Ambystoma tigrinum" (Thesis advisor, Professor George Wald)
	Elected to Phi Beta Kappa and to Sigma Xi
1964	M.D. awarded cum laude for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
1964	Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
1970	Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
1971	Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months
1982, 1986	Visiting Professor of Physiology, Department of Physiology, Kyushu

1987	University, Fukuoka, Japan, for a period of three months each
1989	Awarded Jacob Javits Neuroscience Investigator Award from the National Institute of Neurological and Communicative Diseases and Stroke
1999	Awarded Homer N. Calver Award from the American Public Health Association for studies in environmental health.
2001	Awarded 2001 Academic Laureate from the University at Albany Foundation.
2010	Awarded the Albion O. Bernstein, M.D. Award in recognition of an outstanding contribution to public health and the prevention of disease though lifelong research of environmental health hazards and for limitless devotion to medical education by the Medical Society of the State of New York.
2011	Awarded the Rodney Wylie Eminent Visiting Fellowship 2011 at the University of Queensland, Brisbane, Australia for a period of four weeks.
2013	Awarded the Annual Kenneth V. Dodgson, M.D., Lectureship at the University of Rochester Department of Occupational and Environmental Medicine Grand Rounds.

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

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 – present.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.

6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibitibility of motoneurons of different sizes. J. Neurophysiol., 28:599-620, 1965.

7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. J. Neurophysiol., 28:560-580, 1965.

8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J. Pharmacol., 148:380-385, 1965.

9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. J. Neurophysiol., 28:958-965, 1965.

10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. Arch. Ital. Biol., 104:73-85, 1966.

11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. J. Neurophysiol., 29:353-368, 1966.

12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in Aplysia neurons. J. Gen. Physiol., 50:1469-1484, 1967.

13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv. Pharmacol., 6A:351-364, 1968.

14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na^+ pump to membrane potential in Aplysia neurons. J. Gen. Physiol., 52:1-21, 1968.

15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch. Neurol., 19:591-597, 1968.

16. Carpenter, D.O. Membrane potential produced directly by the Na^+ pump in Aplysia neurons. Comp. Biochem. Physiol., 35:371-385, 1970.

17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of Aplysia neurons upon Na^+ and Ca^{++}. J. Cell. Physiol., 75:121-127, 1970.

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

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

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

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

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

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