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What's NOT to eat- Food adulteration in the context of human biology

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Abstract

Food has nutritional and non-nutritional components. The latter are not well studied despite the fact that food adulteration has been common. Food adulteration may have reached its peak in cities of western Europe and the US in the 18th and 19th centuries when foods were often purposely contaminated with additives to increase bulk, attractiveness, disguise spoilage and increase profit. Effective regulation of food began in the late 19th and 20th centuries. Nevertheless, today food recalls for bacterial contamination are common, while pesticides and compounds from manufacturing are detected in many foods. Foods with strong reputations for healthiness, such as salmon, may have sizable contaminant contents. The contaminant content of many foods varies by origin and season. Nearly all commercially raised salmon has higher contaminant levels than wild caught salmon. Opting out of the commercial food distribution system is an option, but the value depends on the habitat in which the food is obtained.

Traditionally, the Akwesasne Mohawk Nation has depended on local fish and wildlife for their diet. Now pollution of local waterways has led to the contamination of many local foods, and levels of the contaminant PCBs in the Akwesasne Mohawk people reflect current or past dietary patterns. Many other communities in non-urban settings are exposed to contaminants through long-trail distribution of contaminants in food, air, and/or water. Human biologists considering nutrition, disease, growth, reproduction, aging, to name a few areas, may consider the non-nutritional components of food as many have the ability to alter physiological functioning.

Keywords

Food; diet; pesticides; PCBs; persistent organic pollutants; Mohawk; Akwesasne

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Introduction

Food adulteration has a long history. In any urbanized society, most people eat food raised by others, and people depend on a chain of commerce that raises, transports, and sells food to consumers who are far from the countryside where most food originates. In the US, which has a population of nearly 300 million people, fewer than one million people claim farming as their occupation. The 299 million non-producers depend, rely and trust that the food is healthy, or at least not harmful, and regulated to a certain degree.

Until regulations were passed and enforced, the system of food production and distribution provided ample opportunities for modifications, both in adulteration and/or improvement. The term chosen to describe may depend on where one is in the system of food production and distribution, producer, distributor or consumer. Adulteration is the dilution of the quality of products through the addition of extraneous, impure or inferior ingredients, and improvements in profitability were often achieved through adulteration. Food adulteration is one of the chief characteristics of urban society, and the history of urban populations is intertwined with the history of food adulteration and the attempts to minimize it.

A very brief history of food adulteration

In the 18th and 19th centuries, food adulteration was commonplace in European and North America cities (Wilson, 2008). In Europe, rural to urban immigration increased and urban populations grew rapidly. Small-scale food production that had existed in earlier medieval towns became impossible, and consumers depended entirely on providers who were middlemen, or were middlemen to other middlemen. In the US, a combination of immigration from both rural areas and European cities increased population size and density (Macionis and Parrillo, 2001). More than ever before urban residents had less personal knowledge of how food was produced and handled and so had little or no knowledge of the food's quality (Wallis and North, 1986). Food swindles were especially common among the large immigrant population of urban society as they often were the poorest and most powerless (Wilson, 2008).

Among the first to draw public attention to food adulteration was Frederick Accum. In 1820 he published an exposé, *Adulterations of Food and Culinary Poisons*, which was a seminal event in the history of adulteration and reform (Wilson, 2008). The book itself features a small skull and cross-bones motif on the cover with the underlying words, “There is death in the pot”, summarizing Accum's conclusion. A professional chemist better known in his day for work demonstrating the safety of gas illumination in comparison to candlelight, Accum exposed numerous examples of dangerous food adulteration (Wilson, 2008). By devising methods to detect chemical additives, he proved their common existence in a large variety of foods. Among the problems he observed were the frequent use of metals to color or modify food. Candies and sweets generally were marketed in bright colors, some of which were achieved using red lead or copper, both poisonous. Pickles and many green vegetables were made greener with copper washes. Imitation tea was boiled with verdigris (produced by the oxidation of copper) and painted with it too to secure the impression of real tea. Complaints of stomach ailments were often heard and fatalities were noted in newspaper accounts of the day. The reasons for these complaints are well known now. Copper and lead are toxic at the high doses that were used in food adulteration. Each produces a wide range of effects depending on dose including death, neurotoxicity, kidney and liver damage, as well as mundane stomach upset (nausea, vomiting). Although the free use of chemical additives was regarded with repulsion by 19th century food consumers and modifiers of public opinion, Accum's exposé did not stop this practice.

That honor goes as much or more to an early retired physician named Arthur Hill Hassail who applied the simple microscope to the problem (Wilson, 2008). By doing so, he was able to visualize many of the non-chemical contaminants that were purposefully added to foods to improve profits but had escaped Accum's methods of chemical analysis. These additions included different kinds of plant leaves added to tea to increase bulk at low cost, chicory to coffee, and most famously the addition of alum to flour to increase the whiteness of bread. Most of Hassail's investigations were published in a new journal, the Lancet, and were widely read. Eventually laws to protect consumers from adulterated foods were passed in England in 1860, 1872 and later in the US (Coppin and High, 1999; Wilson, 2008). (It should be noted that before their passage, there had been many laws promulgated by monarchs in Europe to regulate wine and bread production.)

An additional group of non-nutritional elements added to food are the organic pollutants and these undoubtedly contributed to the high rate of gastrointestinal infections and deaths. The supply chain from farm to city to plate, being rather long, there has provided ample opportunity for bacterial contamination of milk and other easily spoiled foods. Tobias Smolett's novel, *The Expedition of Humphrey Clinker*, provides a taste of conditions. Milk was carried in open pails through the streets and became contaminated with the "spewing's of infants," and with "spittle, snot and tobacco-quids from foot passengers," and called bread "a deleterious paste, mixed up with chalk, alum and bone-ashes; insipid to the taste and destructive to the constitution" (Smollett and Machen, 1929).

In many cities, the worst, most spoiled food was offered for sale cheaply to factory workers after they were released from their long labors. They had no choice but to buy cheap, spoiled meat, fish, etc., as the better goods had been purchased earlier by those not confined to factories during the day. Since it was usually late in the day when they were released from the factory, it was often too dark to see signs of spoilage and purveyors had numerous methods of disguising spoiled foods (Wilson, 2008; Wohl, 1983).

In the US, it was the knowledge that spoiled food and additives to food were typically part of the diet that led to the passage of the first legislation to protect the food supply (Wilson, 2008). Sinclair Lewis was one of many reformers in this era. His effort to expose the wretched lives of working class immigrants in the Chicago meatpacking industry and the industry itself is well known. He described in most unflattering ways the making of sausage stating that it included rats, rat feces and rat poison. When then President Theodore Roosevelt established an investigation, Lewis' observations were validated. In 1906 Roosevelt signed the Meat Inspection Act and the Pure Food and Drug act (Wilson, 2008).

Food Adulteration Today

The extent of food adulteration has changed over and over, and continues to change. Most believe our food is sufficiently clean and pure as we base our food handling techniques on sound knowledge of bacteriology. Now that there are no more roguish boys spitting into milk pails carried through urban streets, our food must be free of contamination, we think. In fact though, there are numerous recalls of foods even in recent memory reflecting contamination of food. Many may remember recent recalls of raw spinach owing to contamination with *E. coli* and earlier contamination of eggs with salmonella. In the U.S., the safety of food, drugs, animal health, biologics, and medical devices is maintained by the US Food and Drug Administration (USFDA), which presents a website of recent recalls in each of those areas (http://www.fda.gov/Safety/Recalls/default.htm).

Currently, the most common causes of food recalls are elevated lead levels, some type of bacteria, and either undeclared or mislabeled contents. Foods thought to be relatively non-nutritious or unhealthy, such as the dessert/snack "Twinkies", do not contain any of the

metals or other overtly toxic ingredients that Accum would have objected to, although they do contain several different types of sweeteners and other items many would be concerned about to some degree.

Food adulteration, in the sense of ingredients added for the purpose of increasing profits, has morphed subtly into food modification. For example, many people no longer regard the addition of ingredients to extend shelf-life as adulteration. Whether considered adulteration or improvement, additives to consumables are ubiquitous. Even something as simple as cigarettes contain some 600 ingredients in addition to tobacco.

Today some additives are used in the growing rather than the preparation or distribution process. Many of these are pesticides that increase productivity by increasing resistance to pests and increasing crop yields. Other additions are accidental. Common non-nutrients in foods include pesticides, persistent organic pollutants (POPs), metals and plasticizers (Table 1). As conditions of growing and distributing food have changed, the question that Accum asked and answered in his day, still resonates, what exactly is really in our food?

What is in our food?

Pesticides

It would seem to be a simple matter to list the pesticides found in vegetables, fruits, and other foods, but it is not. One influential variable is the season of the year. Tomatoes sampled in US markets in the summer and fall are far more likely to be produced locally, or at least domestically (United States Department of Agriculture, 2009; 2011). Pesticide use can vary with location of production so the extra-contents of tomatoes sampled in the summer are quite different from those in the winter.

The monitoring of pesticide contents of food is managed in the US by three distinct agencies with different missions but overlapping operations. Inspection, analysis and report of pesticide levels are the responsibility of the US Department of Agriculture (USDA). The Environmental Protection Agency (EPA) registers the use of pesticides, and sets limits of the maximum allowed on or in a food. The US Department of Agriculture's Food Inspection Service is responsible for some egg products, meat and poultry products transported into the country or across states, and carries out a market basket survey in its Total Diet Study (United States Department of Agriculture, 2009; 2011). The USFD's Agricultural Marketing Service operates the Pesticide Data Program that tests pesticides for residues focusing on raw agricultural products and some processed foods. These monitoring programs are not necessarily intended to provide a representative picture of pesticide residues in the food supply. They concentrate on foods that are suspected of high levels, and on sources that are believed to produce products with high levels.

There are 473 pesticides tested for by the USDA at this time. Many fruits and vegetables do not have detectable pesticide residues, but strawberries, celery, nectarines, and peaches have the highest residues as of the report released in 2009 (Figure 1, http://www.ams.usda.gov/pdp) (United States Department of Agriculture, 2009; 2011). Sweet potatoes and asparagus have only negligible levels (0.3 and 0.1% respectively), and of the fruits and vegetables tested, only sweet corn has no detectable pesticide residue.

Having the most residues does not necessarily make a food the most dangerous. The type of pesticide residue found is important. Figure 2 shows the foods with chlorpyrifos residues detected most often. Peaches, nectarines, and broccoli have the highest residues (≥8%).

Chlorpyrifos is an organophosphate insecticide, acaricide and miticide used on a variety of food and feed crops. Approximately 10 million pounds are applied annually on farms, and

the single largest agricultural market is corn, which enters the diet in industrialized countries in many forms(Pollan, 2006). It also is used in homes to counter cockroaches, fleas, and termites, and it is included in some pet flea and tick collars. Thus, exposure to chlorpyrifos is through multiple pathways. A major effect of chlorpyrifos is through the nervous systems as a cholinesterase inhibitor (Farahat et al., 2010; Lovasi et al., 2011; Viswanath et al., 2010). Effects are dose-dependent, and exposure can cause nausea, dizziness, confusion, and, at very high exposures, respiratory paralysis and death. Evidence for other routes of effect such as a direct action on cellular processes involved in brain development is being evaluated (Slotkin, 2004; 2011). According to the ATSDR, its influence on the fetus and children are not well characterized. However, several studies have described effects on children's development, either neuropsychological or physical or both, that may stem from either pre- or postnatal exposure to the pesticide alone or in combination with other materials (Perera et al., 2002; Perera et al., 2005; Whyatt et al., 2004; Zhao et al., 2005)

Chlorpyrifos is just one pesticide and is not the most common one found. Listing each food by pesticide residue type or frequency would produce a voluminous table, but a succinct picture is obtained by looking at commodity groups (Figure 3). Among domestically produced foods, fruits and vegetables have the most samples with detectable pesticide residues along with the heterogeneous ”other” category that includes nuts, seeds, oils, honey, candy, spices, and dietary supplements. Few domestic commodities have residue levels that are violative (violate the pesticide residue limit set by the USDA) yet 1.3% of vegetables are in violation, and 2.6% of our nuts, oils, etc. have levels above the acceptable limit (Figure 3). Among commodity groups of imported foods, the percent with detectable residues, and levels in violation, is uniformly higher, with 1.2% of grains in violation, and fruits, vegetables, and `other' with violative residues above 4%.

The pesticides most commonly found in our foods currently are DDT (in all its forms), malathion and (Agency for Toxic Substances Disease Registry, 2002; 2003; Food and Drug Administration, 2001; United States Department of Agriculture, 2009) (Table 2). DDT is one of the “dirty dozen” of persistent organic pollutants as identified by the US Center for Disease Control (CDC) and EPA. Widely used to reduce insect populations and very effective against mosquitos, it was banned in the US in 1972 (Agency for Toxic Substances Disease Registry, 2002). As it is still the most common pesticide in/on food, it is clearly a very persistent pollutant. In 2001, it was banned by a UN convention, and in 2004 a global treaty on Persistent Organic Pollutants banned its use worldwide, except for controlling diseases such as malaria. DDT has been linked to numerous ill effects on reproduction, beginning with Rachel Carson's exposé in 1962 showing that DDT was responsible for reproductive failure in some important bird species. Epidemiologists have linked DDT to accelerated maturation in humans, different patterns of weight gain at puberty, and other significant effects (Denham et al., 2003; Elobeid et al., 2010; Lyche et al., 2010; Lyche et al., 2009; Roy et al., 2009; Schell et al., 2010; Schell and Gallo, 2010; Schell et al., 2009a).

Malathion, the second most commonly found pesticide is an organophosphate that was first registered for use as an insecticide in the United States in 1956. It is applied widely in agriculture, home gardens, greenhouses, public parks, and publicly operated pest control programs. It is also used to treat head lice on humans, fleas on pets, and kill mosquitos and Mediterranean fruit flies. As with chlorpyrifos, the dose largely determines the effects. High exposure can impair respiration, cause cramps, diarrhea, tightening in the chest, blurred vision, and even loss of consciousness and death. Effects on child physical or neuropsychological development have been reported (Cupul-Uicab et al., 2010; Eskenazi et al., 2009; Eskenazi et al., 2007; Lopez-Espinosa et al., 2011). More information on this pesticide, and many others, is available from the CDC's ToxFaqs source

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(http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=521&tid=92) (Agency for Toxic Substances Disease Registry, 2003).

Fortunately, the levels of many pesticides in the environment have declined over the last few decades. A common approach to assessing levels is by monitoring index species that interact with the environment closely, particularly avian and aquatic ones that can reflect pesticide levels in that environment. Levels in the eggs of herring gulls from the Great Lakes region have been monitored since 1974. DDE levels (DDE is the metabolite of DDT that is commonly measured as body burden) have declined in each year since then. In 1974, DDE levels in the herring gull eggs were above 24 ug/g (wet weight). Over 30 years later in 2005 (the most recent year for which data are available), DDE levels were one twentieth of this level at less than 1.0 ug/g (wet weight). The percent decline of was nearly 97% (Figure 4).

Unfortunately, not all contaminants have decreased by as much, and further, a new generation of research finds many effects at far lower levels than seen before (Colburn et al. 2006). Although lead is not a pesticide, its history of regulation provides the best example of the relationship between research and the setting of tolerance levels. Like pesticides, most lead exposure is through ingestion. Until 1970 the blood tolerance level was 60 ug/dL then it was reset to 30 until 1985 when it was further reset to 25 and finally in 1991 it was set at 10 by the Centers for Disease Control and Prevention. Each resetting developed from research that observed effects below the tolerance limits. Now with the action level set to 10, several studies have shown that the current action level of 10 μg/dL cannot be supported. Considerable evidence exists for a continuum of effects on child development and cognition extending well below 10 μg/dL (Pocock et al., 1994; Schwartz, 1994). As larger samples are engaged to understand the effects of low levels of many contaminants, the effects become clearer and the likelihood that no level is without effects becomes more evident.

NON-PESTICIDE CONTAMINANTS FROM INDUSTRIAL PROCESSES—The herring gull egg monitoring program shows that toxicants from industrial manufacturing, such as PCBs and dioxin, have entered the food web. Both PCBs and dioxin are lipophilic and can be tracked in eggs. PCBs and dioxin show declines but not nearly as great as seen for DDE. In fact dioxin, which is the most toxic of the compounds assessed, has declined the least, by only 35%. PCB levels in herring gull eggs have declined more than dioxin levels (~80%), but not nearly as much as DDT (Figure 4). Dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD) is formed as a by-product of the manufacture, molding, or burning of organic chemicals and plastics that contain chlorine, and by natural processes (forest fires or volcanic eruption). Dioxins are considered the most highly toxic pollutant produced and are omnipresent. PCBs are a heavier than water, oily substance that has had many applications where resistance to heat and environmental weathering are valued (Agency for Toxic Substances Disease Registry, 2000). Domestically manufactured since the late 1920's, companies ceased the production of PCBs in 1977, and the EPA banned their use in 1979.

Another closely related contaminant in the food web is polychlorinated naphthalene, which like PCBs exist in a variety of structures (congeners) depending on the pattern of carbon substitution. The structure determines both toxicity and persistence in tissues and the environment. PCNs, PCBs and dioxin all induce aryl hydrocarbon receptor mediated cytochrome P-450 induction.

A recent comparison of levels of PCNs in foods (Table 3) showed that fish have substantially higher levels than meat and meat products, poultry and dairy products (Fernandes et al., 2010). The PCN level in fish (average concentration ~ 20 ng/kg) was an order of magnitude higher than other food types tested. The PCN TEQ was about twice as high as in meats and meat products, the food with the next highest level, and was about 5

times higher than in poultry. Of the fish, farmed and organically produced salmon had the highest concentrations (37.3 ng/kg, 34.5 ng/kg, respectively) while cod fillets had the lowest (0.73 ng/kg).

Fish are also high in other industrial pollutants that are accidental ingredients. Polybromated diphenyl esthers (PBDEs) are flame-retardant chemicals that are added to plastics and foam products to make them difficult to burn. Because they are mixed into plastics and foams rather than bound to them, PBDEs can leave the products that contain them and enter the environment. Mean levels have been high in fish and considerably lower in meat and dairy, although analysis of recent samples shows that levels in fish have declined, though are still the higher than mean and dairy levels (Schecter et al., 2006; Shaw et al., 2008).

Fish are generally highly regarded for their healthful properties as a high protein, low fat food. Some fish are especially valued for their high levels of important omega-3 fatty acids. Salmon is sometimes considered a health food for these reasons. However, salmon also contains the accidental ingredients of pesticides and industrial contaminants. An important consideration in weighing its healthfulness is its origin. Just as domestic and imported foods differ in the number of samples with detectable levels of pesticides, salmon varies in its contaminants by origin. This probably reflects the salmon's own diet. Studies of wild versus farmed salmon show that farmed salmon have higher levels of mirex, DDT (total), endrin, dieldrin, Lindane and other pesticides as well as PCBs and dioxins (Hites et al., 2004; Huang et al., 2006). In fact, it was reported that if a fish sample was high in one contaminant it was in most tested (Huang et al., 2006).

Within the group of farmed salmon there are substantial differences as well. Farmed salmon from Washington State and Chile have the lowest levels of pesticides of all farmed salmon, whereas salmon farmed in Scotland, Norway, the Faroe Islands and western Canada had the highest levels (Huang et al., 2006). Levels within the group of wild salmon were generally lower but the distribution overlapped with levels of the farmed salmon from Chile and Washington State. PCB levels followed a similar pattern.

In terms of consumption, these levels mean that only a fairly small number of meals per month can be considered without risk. Risk evaluations differ in terms of how many additives are considered in evaluating the content. Most evaluation protocols consider just one additive, e.g., one pesticide or one POP. With that protocol the allowable number of salmon meals per month will be higher than an evaluation protocol that takes into account all the additives of concern. Using the EPA evaluation protocol, Carpenter and colleagues (Hardell et al., 2010; Shaw et al., 2008), calculated that no more than a quarter of a portion per month of salmon farm raised in Scotland was allowable, while four meals a month of wild caught salmon from Kodiac Alaska were allowed. None of these evaluation protocols take into account the benefits of salmon consumption and for now that calculation is the responsibility of the individual consumer to puzzle out.

The presence of additives to foods, intentional or accidental, raises two questions: how great are the risks to people (children, pregnant women, infants as well as adults), and is it better to opt out of the commercial food chain?

Traditionally risk assessment was based on an adult man of approximately 150–175 pounds, perhaps because most exposure to chemicals in the past was through occupations and these were largely populated by men. The realization that the placenta does not protect the fetus, learned in part from the terrible experience with thalidomide and diethystilbesterol, has led to risk assessment for children and pregnant women with their fetuses. Today, risk assessment from pesticides, herbicides and manufactured chemicals is difficult at best. The risk from any specific material will depend on dose (exposure), length of exposure, and the

life stage during exposure (pregnant woman, fetus, infant, subadult, adult (see Executive Order 13045, Protection of Children from Environmental Health Risks and Safety Risks), gender, other possible exposures, and underlying disease. Additionally, risk is dependent on the criterion outcome such as fertility, miscarriage, birth defects, birth size, postnatal growth, developmental progression, cognitive development, social development (e.g., asociality, attention deficit hyperactivity disorder), as well as numerous adult diseases from cardiovascular disease to diabetes, to autoimmune diseases to cancer. Thus, the risk associated with any exposure will vary by outcome; some exposures `bad' for cancer in adults may be `good' for fetal development. Finally, humans are exposed to a large variety of materials and a risk analysis that considers one toxicant at a time does not evaluate the effects of multiple, simultaneous exposures (mixtures) and their possible synergy. Evaluating all the risks for all listed outcomes from multiple exposures at different life stages is highly complex and requires numerous assumptions. Therefore, results of risk analysis are controversial among experts and confusing to consumers. Perhaps for this reason, many persons seek to avoid exposure entirely, especially dietary exposure. Controlling dietary exposure seems more within an individual's grasp compared to controlling exposure from air and water.

Today the organic food market is the fastest growing component of the grocery market itself. Indeed, organic production can make a difference in terms of the ingredients of the food at the point of consumption. For example, potatoes raised on organic farms had approximately half the levels of metals (zinc, copper, iron, manganese, chromium, nickel and cobalt), organochlorine and organophosphate pesticides compared to conventionally raised potatoes (Mansour et al., 2009a; Mansour et al., 2009b). Children placed on an organic diet for five days experienced an immediate and dramatic drop in the urinary levels of some pesticides and these levels then rose to baseline when the children resumed their normal, non-organic diets (Lu et al., 2006).

The packaging makes a difference as well. In one study of pregnant women, those who consumed no canned vegetables had significantly less bisphenol A levels compared to women who consumed 1 to 3 cans or women who consumed more than 3 cans per week. Bisphenol A is a common ingredient in plastics including those that have formed the inner lining of cans of food. Bisphenol A is an endocrine disrupting compound known to affect reproductive development (Maffini et al., 2006; Walker and Gore, 2011).

Going Local: A case study of the Akwesasne Mohawk Nation

Today there are many signs recommending that we consume locally produced foods, and many people try to raise as much food of their own as possible. There are a number of reasons to do so (to reduce food costs by obviating transportation costs; to diversify the local economy; to preserve a pastoral way of life in the face of suburbanization; etc.) and creating a clean food supply is certainly one of them.

In the US, many Native American peoples attempt to follow traditional food-ways and seek to consume locally produced foods. In fact, forty-three percent of the Indian population resides in rural areas and there are some 79,703 American Indian or Alaskan Native operators on farms and ranches across the US (U.S. Environmental Protection Agency, 2009). Diet and food preparation are singular markers of cultural identity and adhering to traditional food-ways are often a means of maintaining cultural identity in the face of a multitude of mainstream/western cultural influences. Furthermore, raising, gathering, or hunting traditional foods in traditional ways would seem to be an excellent strategy to reduce exposure to pesticides and pollutants generally, given the risks of consuming commercially produced foods as just described. However, the degree of contamination of a

food depends on the degree of contamination of the habitat, and particularly the food supply, in which that food is produced. As such, Native Americans and other consumers of locally raised food are at particular risk of exposure to contaminants if the local habitat is polluted.

A case in point is the Akwesasne Mohawk Nation. The Nation exists on the St. Regis Mohawk Reservation (known as a Reserve in Canada) and in many communities in close proximity to the reserve. It has a population of approximately 13,000 people although the nation is not censused by US or Canadian governments (Fitzgerald et al., 1998). The territory straddles the St. Lawrence River, and as a sovereign nation, it abuts New York State, Ontario and Quebec, Canada. The Nation is adjacent to and downstream from one federal superfund site and two New York state superfund sites. Contamination of the St. Lawrence River, and some of its tributaries that cross the reservation, has led to the contamination of local fish and wild life particularly with polychlorinated biphenyls (PCBs).

In industries neighboring the Akwesasne Mohawk Nation, PCBs were used in hydraulic fluid that was raised to high temperatures in the manufacturing process. Leakages and improper disposal of the PCB laden fluids introduced PCBs to the waterways and life within. In the mid 1980's contamination of local fish was reported (Lacetti, 1993; Sloan and Jock, 1990), and fish advisories were issued warning people not to consume, or to limit consumption, of local fish.

Numerous studies have established that PCBs are endocrine disrupting compounds (Gallo et al., 2011; Goncharov et al., 2009; Kraugerud et al., 2010; Schell and Gallo, 2010). Thus, there is widespread concern over exposure to PCBs, and deep concern among the Mohawk community as to how to avoid exposure.

Many non-occupational studies have found that diet is the primary route of exposure to PCBs. Other sources are breastfeeding and transplacental passage. Due to the persistence of many PCB congeners, a woman's PCB exposure while growing up influences her PCB level as an adult, and thus her transference of PCBs while pregnant and breastfeeding.

The fish advisories issued in the mid 1980's may be an influence on the levels of PCBs in the nation. Before the advisories, pregnant and lactating women were not aware that locally caught fish posed any risks to health and they consumed fish as a healthy food choice. Indeed, breast milk is considered one of the cornerstones of the traditional subsistence system (Cook, personal communication). Breast milk contributes to immunological, psychological and socializing functions in Mohawk society. Environmental health is inextricably linked through breastfeeding to the status of women in Mohawk society which traditionally is matrilineal. The importance of breast milk and its role in Mohawk society may be seen in the high rates of breast feeding initiation (84%) among women attending the WIC clinic at the St. Regis Mohawk Health Services. This is possibly the highest rate in the Nashville (provision) Area of the Indian Health Service (Cook, personal communication).

Akwesasne youth born before the advisories against eating local fish are likely to have had prenatal exposure from transplacental passage. In addition, if breastfed, they would have early postnatal exposure. Breastfeeding adds substantial exposure to all lipophilic compounds at a crucial stage of development. Youth "born-before" the advisories would also have postnatal exposure through their own diet. In contrast, youth "born-after" the advisories were issued would be expected to have less postnatal exposure through their own diet. "Born-after" youth may also have prenatal and lactational exposure depending on their mother's PCB levels and their breastfeeding history. Thus, we devolve three exposure components: prenatal (via transplacental passage), early postnatal (via lactation) and later postnatal (via offspring's own diet). The greatest contrast would be between born-before

youth who were breastfed and consumed locally raised food items versus born-after youth who were not breastfed and did not consume locally raised foods.

Our first study was conducted in partnership with the Akwesasne Mohawk Nation from 1995–2000 and focused on adolescents 10–16.9 years of age who resided in Mohawk households located on the St. Regis Reservation/Reserve (reserve is the Canadian equivalent of reservation) or in Mohawk households within 10 miles of the reserve/reservation. (More complete details are available from several publications (Gallo et al., 2005; Gallo et al., 2011; Schell et al., 2008; Schell et al., 2009b; Schell et al., 2003)). Mohawk data collectors identified the Mohawk households and collected all data without knowledge of participants' exposure status. Data collectors were trained by the principal investigator and co-investigators and underwent routine re-training. A fasting blood specimen was collected by venipuncture on first rising and was analyzed for PCBs by the University at Albany, School of Public Health, Exposure Assessment Laboratory. PCBs were measured in serum by high-resolution, congener-specific analysis that yielded the delineation of 101 PCB congeners, as well as *p,p'*-DDE, HCB, and mirex (DeCaprio et al., 2005; DeCaprio et al., 2000). This permitted the classification of congeners into groups of persistent and less persistent classes, the first representing all past exposure and the latter more current exposure. Sociodemographic information and knowledge of the consumption of local foods was obtained by questionnaire to the adolescent if over 13 years of age, or from the mother if 13 or younger. Information on breastfeeding history, and diet during pregnancy was obtained from the mother.

Between 2000 and 2005 these adolescents were invited to participate in a follow up study. Of the 271 participants in the first study, 152 young adults (61 males, 91 females) with an average age of 18.1 years participated. Again blood was collected and participants completed questionnaires concerning diet and behaviors that might have constituted exposure pathways, especially behaviors involving the consumption of locally caught or raised foods, as well as several other outcomes not related to the focus of this analysis.

Information on dietary intake over the past year was collected by interview using a number of questionnaires. In order to capture the most information about traditional food intake, questionnaires focused on the consumption and preparation of any locally grown consumables (eggs, meat, poultry, dairy products, etc.) and caught, hunted, or trapped fish (bass, bullhead, catfish, northern pike, perch, salmon, sturgeon), game or wildlife (deer, rabbit, frog, goose, muskrat, turtle, duck, goose, partridge, pheasant) in the past year, or more than a year ago.

Results

The adolescents' average PCB levels were between the 90th and 95th percentiles of the US reference sample (Schell and Gallo, 2010). When these adolescents were young adults, 4 years later on average, levels of the sum of 14 PCB congeners found in 50% or more of the sample (ΣPCB50) were more than twice the average for the US as described by the CDC (Gallo et al., 2011). Average levels of other pesticides such as DDE, HCB, as well as lead and mercury were not nearly as high, and not higher than the US average for a comparable sample.

Akwesasne adolescents who had been breast fed had significantly higher PCB levels than those who had not been (Schell et al., 2003), and as young adults breastfeeding history still made a significant difference in PCB levels (Gallo et al., 2011). This is evidence of the persistence of some PCB congeners. In addition, young adults who had been born after the fish advisories were issued (1985 or later) had significantly lower levels of PCBs (Gallo et

al., 2011). This suggests that pregnant women, mothers, and families in general limited consumption of locally caught fish after the advisories were issued.

Considering the likely dietary pathways of exposure in this community, we tested the relationship of different consumption patterns to the levels of PCBs (Figure 5). Young adults who said that they had consumed some locally caught fish, or wildlife or other locally produced food in the past 12 months had significantly higher levels of persistent PCBs, and of the 14 PCB congeners most commonly found in the sample (an equal combination of both persistent and less persistent ones ($n = 145$; $t= 2.81$, $p<0.001$; and $t=2.54$, $p=0.01$ respectively)

To investigate whether fish consumption was the critical dietary component we then compared those who ate local wildlife and locally produced foods ($n=45$) irrespective of fish consumption (figure 5). Consumers had significantly higher levels of the 14 common PCB congeners and the group of persistent congeners ($n= 145$; $t=2.01$ $p=0.05$; $t=2.1$, $p=0.04$ respectively).

Narrowing further, young adults who had consumed some deer, duck goose, muskrat, partridge, pheasant or rabbit, irrespective of fish or other game consumption, again had significantly higher levels of PCBs ($n=150$; Figure 5). Both of these narrower comparisons might suggest that fish consumption is not critical. However, when the consumers of locally caught fish were compared to those who had not consumed any fish, the PCB levels were significantly higher for the sum of 9 persistent PCBs found in 50% of the sample ($n=145$; $t= 2.27$; $p=0.02$).

These comparisons show that consumption of locally caught fish and game was associated with higher levels of PCBs. However, it is not possible to clearly delineate the additions from each specific food. It is likely that consumers of fish are also consumers of other local foods as consumption can be patterned by cultural factors, philosophical beliefs and household financial contingencies (Table 4).

Although it is difficult to delineate the contribution from recent consumption of specific local foods to PCB body burden, it is clear that as a group, these foods add to the individuals' pollutant burden. Other factors must also have a role since the size of that impact is not great. The absolute difference averages 12%. Thus, factors other than the diet in the past year must be contributing to exposure.

Discussion

Akwesasne Mohawk people have higher levels of PCBs due in part to a diet that includes traditional consumption of locally caught fish and wildlife. It is difficult to separate effects from different components of the diet since there is substantial correlation among dietary components most likely to convey PCBs. Nevertheless, avoiding locally caught, raised, or hunted foods should help reduce PCB levels in the population. Such avoidance places a burden on the nation because locally obtained foods are inexpensive and healthy, being low in fat and high in protein and micronutrients. Avoiding locally obtained foods means replacing them with packaged foods that are often high in fat and calories. At a time when overweight and obesity are significant health concerns, consumption of locally obtained foods could be highly beneficial were it not for the toxicant burden they contain. Perhaps more importantly, the avoidance of locally obtained foods means a loss of activities that express and reinforce Mohawk traditions, and that strengthen bonds between generations. For this reason it is difficult to know which is worse, consuming locally obtained foods, or not participating in traditional food-ways with possible losses in social integration.

The somewhat small difference in PCB levels between consumers and non-consumers we described is evidence that other sources of exposure pertain to current PCB levels. It also suggests that other means of reducing exposure may be discovered and this is a goal for future research.

Evaluating the different risks, e.g., those associated with consumption of foods containing contaminants, and those associated with cultural identity and or healthful eating is complex. Risk assessments by governmental agencies do not consider effects other than specific diseases or disabilities. Loss of cultural identity is not considered and seems to be assumed that there are healthful alternatives to any food considered at high risk due to contamination. For many persons, there are no such alternatives. This analysis was articulated by members of the the Akwesasne Mohawk Nation who promoted holistic risk-based environmental decision making (Arquette et al., 2002): “It is clear that to develop more holistic environmental health models, there is a need to identify and measure sociocultural impacts and integrate them with human health and ecologic effects. To incorporate these many different effects, a holistic model would need to examine and include aspects from many fields of study, integrating qualitative research findings with the sciences of toxicology, epidemiology, and ecology. Such an integrated model would need to be based on a very broad and flexible understanding of health, risk, and restoration, while acknowledging that these definitions are culturally based and community specific.” Although this approach makes sense, it has not yet been embraced by regulatory agencies and narrowly focused risk assessments continue using traditional biomedical definitions of health outcomes rather than seeking to insure well-being.

Human biologists have a long history of work with diet and nutrition, and with few exceptions, have concentrated on the nutritive value of foods. Today and arguably since urbanization, foods also have a non-nutritional component that has not been considered. Many POPs are known disruptors of endocrine signaling with important known or suspected effects on outcomes of interest to human biologists such as reproduction, growth, aging and menopause, cardiovascular disease, overweight, and diabetes (Everett et al., 2010; Everett and Matheson, 2010; Golub et al., 2008; Tan et al., 2009; Tilghman et al., 2010).

Further, the widespread presence of PCBs and other common toxicants such as DDT, phthalates, bisphenol-A, PBDEs, and polychlorinated napthalenes found in foods today, means that many populations may be affected by these non-nutritive elements. While we focus on urban populations and ones involved in commercial food consumption, high levels of POPs can be found in distinctively non-urban communities. Indeed, some of the highest levels of POPs are found among Inuit peoples living in very non-urban, remote areas (Donaldson et al., 2010; Wojtyniak et al., 2010). If possible, human biologists working in faraway locations may consider the effects of nonnutritional elements in their research.

Conclusion

Food adulteration has long history. Modern food typically contains numerous non-nutritional additives. Raising one's own food may help to avoid these additives but the food quality depends on the local environment in which the food is raised or caught. When the local environment is contaminated,' raising one's own' does not result in uncontaminated foods although it may satisfy by reinforcing values and beliefs characteristic of a particular culture.

Many of the non-nutritive additives are known to affect physiological systems Thus, food today can have a significant influence on human biology beyond its nutritive effects.

Contamination has a global reach and remote populations, often the subjects of study by human biologists, are not free of this contamination.

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Abbreviations

PCBs	Polychlorinated Biphenyls
POPs	Persistent organic pollutants
ppb	Parts per billion
ΣPCB50%	Sum of IUPAC#s 28,52,74,87,95,99,101[90],105,110,118, 138[+163+164],153,180,187
Σ9PersistentPCB	Sum of IUPAC#s 28,74,99,105,118,138[+163+164],153,180,187

References

Agency for Toxic Substances Disease Registry. Toxicological profile for polychlorinated biphenyls (PCBs). US Public Health Service, Agency for Toxic Substances and Disease Registry; Atalanta, GA: 2000.

Agency for Toxic Substances Disease Registry. Toxicological profile for DDT, DDE, DDD. U.S. Department of Health and Human Services, Public Health Service; Atlanta, GA: 2002.

Agency for Toxic Substances Disease Registry. ToxFAQs September 2003 Malathion. U.S. Department of Health and Human Services; 2003.

Arquette M, Cole M, Cook K, LaFrance B, Peters M, Ransom J, Sargent E, Smoke V, Stairs A. Holistic risk-based environmental decision making: a Native perspective. Environmental Health Perspectives. 2002; 110(Suppl 2):259–264. [PubMed: 11929736]

Coppin, CA.; High, JC. The Politics of Purity: Harvey Washington Wiley and the Origins of Federal Food Policy. University of Michigan Press; Ann Arbor: 1999.

Cupul-Uicab LA, Hernandez-Avila M, Terrazas-Medina EA, Pennell ML, Longnecker MP. Prenatal exposure to the major DDT metabolite 1,1-dichloro-2,2-bis(pchlorophenyl)ethylene (DDE) and growth in boys from Mexico. Environmental Research. 2010; 110(6):595–603. [PubMed: 20566194]

DeCaprio AP, Johnson GW, Tarbell AM, Carpenter DO, Chiarenzelli JR, Morse GS, Santiago-Rivera AL, Schymura MJ. Polychlorinated biphenyl (PCB) exposure assessment by multivariate statistical analysis of serum congener profiles in an adult Native American population. Environmental Research. 2005; 98(3):284–302. [PubMed: 15910784]

DeCaprio AP, Tarbell AM, Bott A, Wagemaker DL, Williams RL, O'Hehir CM. Routine analysis of 101 polychlorinated biphenyl congeners in human serum by parallel dual-column gas chromatography with electron capture detection. Journal of Analytical Toxicology. 2000; 24(6): 403–420. [PubMed: 10999346]

Denham, M.; Deane, G.; Gallo, MV.; Ravenscroft, J.; DeCaprio, A.; Akwesasne Task Force On The E. Schell, LM. Age at menarche among Akwesasne Mohawk girls exposed to lead, mercury, mirex, dichlorodiphenyldichloroethane (p,p'-DDE), hexachlorobenzene (HCB) and polychlorinated biphenyls (PCBs). "The Role of Environmental Factors on the Onset and Progression of Puberty -

Expert Panel Workshop": Sponsored by Serono Symposia International, Inc; Rosemont, IL. Nov5–8; 2003.

Donaldson SG, Van OJ, Tikhonov C, Feeley M, Armstrong B, Ayotte P, Boucher O, Bowers W, Chan L, Dallaire F, et al. Environmental contaminants and human health in the Canadian Arctic. The Science of the Total Environment. 2010; 408(22):5165–5234. [PubMed: 20728918]

Elobeid MA, Padilla MA, Brock DW, Ruden DM, Allison DB. Endocrine disruptors and obesity: an examination of selected persistent organic pollutants in the NHANES 1999–2002 data. IntJEnvironResPublic Health. 2010; 7(7):2988–3005.

Eskenazi B, Chevrier J, Rosas LG, Anderson HA, Bornman MS, Bouwman H, Chen A, Cohn BA, de JC, Henshel DS, et al. The Pine River statement: human health consequences of DDT use. Environmental Health Perspectives. 2009; 117(9):1359–1367. [PubMed: 19750098]

Eskenazi B, Marks AR, Bradman A, Harley K, Barr DB, Johnson C, Morga N, Jewell NP. Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. Environ Health Perspect. 2007; 115(5):792–798. [PubMed: 17520070]

Everett CJ, Frithsen I, Player M. Relationship of polychlorinated biphenyls with type 2 diabetes and hypertension. Journal of Environmental Monitoring. 2010

Everett CJ, Matheson EM. Biomarkers of pesticide exposure and diabetes in the 1999–2004 national health and nutrition examination survey. EnvironInt. 2010; 36(4):398–401.

Farahat FM, Fenske RA, Olson JR, Galvin K, Bonner MR, Rohlman DS, Farahat TM, Lein PJ, Anger WK. Chlorpyrifos exposures in Egyptian cotton field workers. Neurotoxicology. 2010; 31(3):297–304. [PubMed: 20193710]

Fernandes A, Mortimer D, Gem M, Smith F, Rose M, Panton S, Carr M. Polychlorinated Naphthalenes (PCNs): Congener Specific Analysis, Occurrence in Food, and Dietary Exposure in the UK. Environmental Science & Technology. 2010; 44(9):3533–3538. [PubMed: 20345094]

Food and Drug Administration. Total Diet Study. Center for Food Safety, U.S. Food and Drug Administration; College Park, MD: 2001.

Gallo MV, Schell LM, Akwesasne Task Force On The E. Height, weight and body mass index among Akwesasne Mohawk youth. American Journal of Human Biology. 2005; 17:269–279. [PubMed: 15849706]

Gallo MV, Schell LM, DeCaprio AP, Jacobs A. Levels of persistent organic pollutant and their predictors among young adults. Chemosphere. 2011; 83(10):1374–1382. [PubMed: 21458024]

Golub MS, Collman GW, Foster PM, Kimmel CA, Rajpert-De ME, Reiter EO, Sharpe RM, Skakkebaek NE, Toppari J. Public health implications of altered puberty timing. Pediatrics. 2008; 121(Suppl 3):S218–S230. [PubMed: 18245514]

Goncharov A, Rej R, Negoita S, Schymura M, Santiago-Rivera A, Morse G, Carpenter DO. Lower serum testosterone associated with elevated polychlorinated biphenyl concentrations in Native American men. Environmental Health Perspectives. 2009; 117(9):1454–1460. [PubMed: 19750113]

Hardell S, Tilander H, Welfinger-Smith G, Burger J, Carpenter DO. Levels of polychlorinated biphenyls (PCBs) and three organochlorine pesticides in fish from the Aleutian Islands of Alaska. PLoSOne. 2010; 5(8):e12396.

Hites RA, Foran JA, Schwager SJ, Knuth BA, Hamilton MC, Carpenter DO. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. Environmental Science and Technology. 2004; 38(19):4945–4949. [PubMed: 15506184]

Huang X, Hites RA, Foran JA, Hamilton C, Knuth BA, Schwager SJ, Carpenter DO. Consumption advisories for salmon based on risk of cancer and noncancer health effects. Environmental Research. 2006; 101(2):263–274. [PubMed: 16198332]

Kraugerud M, Zimmer KE, Dahl E, Berg V, Olsaker I, Farstad W, Ropstad E, Verhaegen S. Three structurally different polychlorinated biphenyl congeners (Pcb 118, 153, and 126) affect hormone production and gene expression in the human H295R in vitro model. J Toxicol Environ Health A. 2010; 73(16):1122–1132. [PubMed: 20574914]

Lacetti, G. Public health assessment. General Motors/Central Foundry Division. NYSDOH; Albany, NY: 1993.

Lopez-Espinosa MJ, Murcia M, Iniguez C, Vizcaino E, Llop S, Vioque J, Grimalt JO, Rebagliato M, Ballester F. Prenatal exposure to organochlorine compounds and birth size. Pediatrics. 2011; 128(1):e127–134. [PubMed: 21669889]

Lovasi GS, Quinn JW, Rauh VA, Perera FP, Andrews HF, Garfinkel R, Hoepner L, Whyatt R, Rundle A. Chlorpyrifos exposure and urban residential environment characteristics as determinants of early childhood neurodevelopment. American Journal of Public Health. 2011; 101(1):63–70. [PubMed: 20299657]

Lu C, Toepel K, Irish R, Fenske RA, Barr DB, Bravo R. Organic diets significantly lower children's dietary exposure to organophosphorus pesticides. Environ Health Perspect. 2006; 114(2):260–263. [PubMed: 16451864]

Lyche JL, Almaas C, Ropstad E, Nourizadeh-Lillabadi R, Alestrom P, Berg V, Skare JU, Stavik B. Natural mixtures of persistent organic pollutants (POP) increase weight gain, advance puberty, and induce changes in gene expression associated with steroid hormones and obesity in female zebrafish. JToxicolEnvironHealth Part A CurrIssJournal of Toxicology and Environmental Health - Part A: Current Issues. 2010; 73(15):1032–1057.

Lyche JL, Gutleb AC, Bergman A, Eriksen GS, Murk AJ, Ropstad E, Saunders M, Skaare JU. Reproductive and developmental toxicity of phthalates. JToxicolEnvironHealth B Crit Rev. 2009; 12(4):225–249.

Macionis, JJ.; Parrillo, VN. Cities and urban life. Prentice Hall; Upper Saddle River, N.J.: 2001.

Maffini MV, Rubin BS, Sonnenschein C, Soto AM. Endocrine disruptors and reproductive health: the case of bisphenol-A. MolCell Endocrinol. 2006; 254–255:179–186.

Mansour SA, Belal MH, Abou-Arab AA, Ashour HM, Gad MF. Evaluation of some pollutant levels in conventionally and organically farmed potato tubers and their risks to human health. Food Chem Toxicol. 2009a; 47(3):615–624. [PubMed: 19138717]

Mansour SA, Belal MH, Abou-Arab AA, Gad MF. Monitoring of pesticides and heavy metals in cucumber fruits produced from different farming systems. Chemosphere. 2009b; 75(5):601–609. [PubMed: 19237184]

Perera F, Hemminki K, Jedrychowski W, Whyatt R, Campbell U, Hsu Y, Santella R, Albertini R, O'Neill JP. In utero DNA damage from environmental pollution is associated with somatic gene mutation in newborns. Cancer Epidemiology, Biomarkers and Prevention. 2002; 11(10 Pt 1): 1134–1137.

Perera FP, Rauh V, Whyatt RM, Tang D, Tsai WY, Bernert JT, Tu YH, Andrews H, Barr DB, Camann DE, et al. A summary of recent findings on birth outcomes and developmental effects of prenatal ETS, PAH, and pesticide exposures. Neurotoxicology. 2005; 26(4):573–587. [PubMed: 16112323]

Pocock SJ, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. BMJ. 1994; 309(6963):1189–1197. [PubMed: 7987149]

Pollan, M. The omnivore's dilemma : a natural history of four meals. Penguin Press; New York: 2006.

Roy JR, Chakraborty S, Chakraborty TR. Estrogen-like endocrine disrupting chemicals affecting puberty in humans--a review. Medical science monitor : international medical journal of experimental and clinical research. 2009; 15(6):137–145.

Schecter A, Papke O, Harris TR, Tung KC, Musumba A, Olson J, Birnbaum L. Polybrominated diphenyl ether (PBDE) levels in an expanded market basket survey of U.S. food and estimated PBDE dietary intake by age and sex. Environmental Health Perspectives. 2006; 114(10):1515–1520. [PubMed: 17035135]

Schell LM, Burnitz KK, Lathrop PW. Pollution and human biology. Annals of Human Biology. 2010; 37(3):347–366. [PubMed: 20412024]

Schell LM, Gallo MV. Relationships of putative endocrine disruptors to human sexual maturation and thyroid activity in youth. Physiol Behav. 2010; 99(2):246–253. [PubMed: 19800354]

Schell LM, Gallo MV, Denham M, Ravenscroft J, DeCaprio AP, Carpenter DO. Relationship of thyroid hormone levels to levels of polychlorinated biphenyls, lead, p,p'- DDE, and other toxicants in Akwesasne Mohawk youth. Environ Health Perspect. 2008; 116(6):806–813. [PubMed: 18560538]

Schell LM, Gallo MV, Ravenscroft J. Environmental influences on human growth and development: historical review and case study of contemporary influences. Ann Hum Biol. 2009a; 36(5):459–477. [PubMed: 19626483]

Schell LM, Gallo MV, Ravenscroft J, DeCaprio AP. Persistent organic pollutants and anti-thyroid peroxidase levels in Akwesasne Mohawk young adults. Environmental Research. 2009b; 109(1): 86–92. [PubMed: 18995849]

Schell LM, Hubicki LA, DeCaprio AP, Gallo MV, Ravenscroft J, Tarbell A, Jacobs A, David D, Worswick P. Organochlorines, lead, and mercury in Akwesasne Mohawk youth. Environmental Health Perspectives. 2003; 111(7):954–961. [PubMed: 12782498]

Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. Environmental Research. 1994; 65(1):42–55. [PubMed: 8162884]

Shaw SD, Berger ML, Brenner D, Carpenter DO, Tao L, Hong CS, Kannan K. Polybrominated diphenyl ethers (PBDEs) in farmed and wild salmon marketed in the Northeastern United States. Chemosphere. 2008; 71(8):1422–1431. [PubMed: 18313722]

Sloan, RJ.; Jock, K. Chemical contaminants in fish from the St. Lawrence River drainage on lands of the Mohawk nation at Akwesasne and near the General Motors Corporation/Central Foundry Divisioin, Massena, NY Plant. New York State Department of Environmental Conservation; Albany, NY: 1990.

Slotkin TA. Guidelines for developmental neurotoxicity and their impact on organophosphate pesticides: a personal view from an academic perspective. Neurotoxicology. 2004; 25(4):631–640. [PubMed: 15183016]

Slotkin TA. Does early-life exposure to organophosphate insecticides lead to prediabetes and obesity? Reprod Toxicol. 2011; 31(3):297–301. [PubMed: 20850519]

Smollett, T.; Machen, A. The expedition of Humphry Clinker. Modern library; New York: 1929.

Tan J, Loganath A, Obbard JP. Exposure to persistent organic pollutants in utero and related maternal characteristics on birth outcomes: a multivariate data analysis approach. Chemosphere. 2009; 74(3):428–433. [PubMed: 18986677]

Tilghman SL, Nierth-Simpson EN, Wallace R, Burow ME, McLachlan JA. Environmental hormones: Multiple pathways for response may lead to multiple disease outcomes. Steroids. 2010; 75(8–9): 520–523. [PubMed: 20466011]

U.S. Environmental Protection Agency. The National Pesticide Tribal Program. Washington, DC: 2009. Report nr EPA-735-F-09-007

United States Department of Agriculture. Pesticide Monitoring Program - FY 2008. U.S. Department of Health and Human Services; 2009. p. 2-29.

United States Department of Agriculture. Pesticide Data Program Annual Summary, Calendar Year 2009. U.S. Dept. of Agriculture, Agricultural Marketing Service, Science & Technology; 2011.

Viswanath G, Chatterjee S, Dabral S, Nanguneri SR, Divya G, Roy P. Anti-androgenic endocrine disrupting activities of chlorpyrifos and piperophos. Journal of Steroid Biochemistry and Molecular Biology. 2010; 120(1):22–29. [PubMed: 20211256]

Walker DM, Gore AC. Transgenerational neuroendocrine disruption of reproduction. NatRevEndocrinol. 2011; 7(4):197–207.

Wallis, JJ.; North, D. Measuring the Transaction Section in the American Economy, 1870–1970. In: Engerman, SL.; Gallman, RE., editors. Long-Term Factors in American Economic Growth. University of Chicago Press; Chicago: 1986. p. 95-162.

Whyatt RM, Rauh V, Barr DB, Camann DE, Andrews HF, Garfinkel R, Hoepner LA, Diaz D, Dietrich J, Reyes A, et al. Prenatal insecticide exposures and birth weight and length among an urban minority cohort. Environmental Health Perspectives. 2004; 112(10):1125–1132. [PubMed: 15238288]

Wilson, B. Swindled : the dark history of food fraud, from poisoned candy to counterfeit coffee. Princeton University Press; Princeton: 2008.

Wohl, AS. Public Health in Victorian Britain. Harvard University Press; Cambridge, MA: 1983. Endangered Lives.

Wojtyniak BJ, Rabczenko D, Jonsson BA, Zvezday V, Pedersen HS, Rylander L, Toft G, Ludwicki JK, Goralczyk K, Lesovaya A, et al. Association of maternal serum concentrations of 2,2', 4,4'5,5'-

hexachlorobiphenyl (CB-153) and 1,1-dichloro-2,2-bis (p-chlorophenyl)-ethylene (p,p'-DDE) levels with birth weight, gestational age and preterm births in Inuit and European populations. Environmental Health. 2010; 9:56. [PubMed: 20819217]

Zhao Q, Gadagbui B, Dourson M. Lower birth weight as a critical effect of chlorpyrifos: a comparison of human and animal data. Regulatory toxicology and pharmacology : RTP. 2005; 42(1):55–63. [PubMed: 15896443]

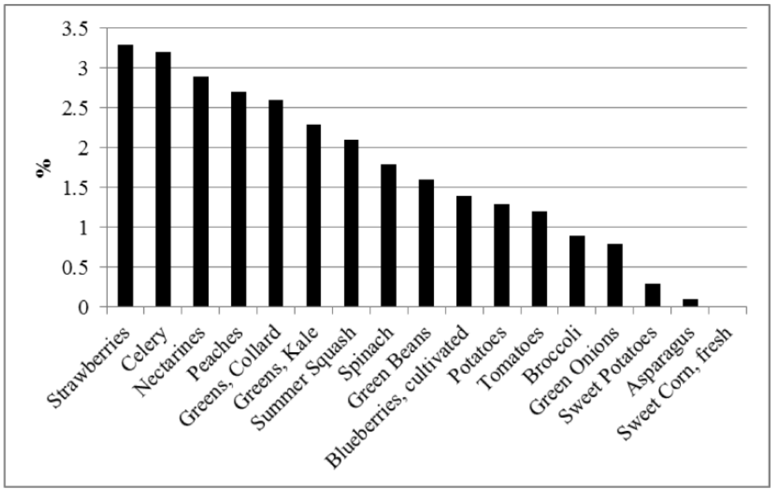

Figure 1.

Percentage of fruit and vegetable samples with detectable pesticide residues.

Source: USDA Pesticide Data Program – Annual Summary, Calendar Year 2008. Release date 12/2009.

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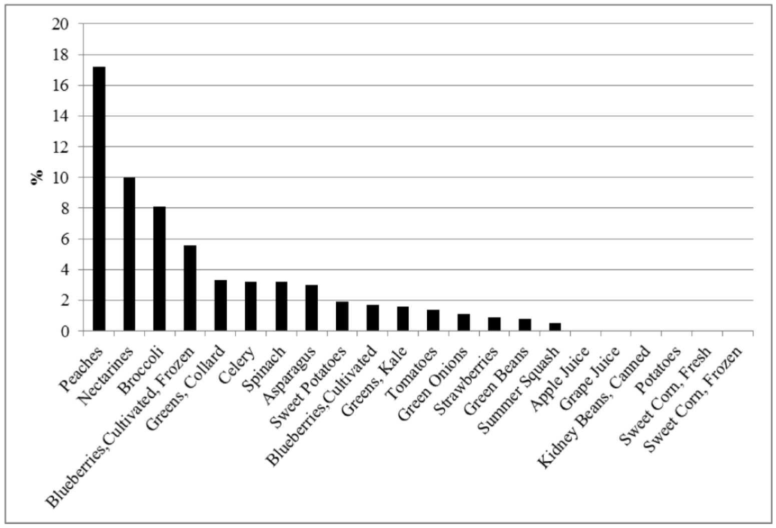

Figure 2.

Fruits and vegetables with detectable levels of pesticide: Chloropyrifos.

Source: USDA Pesticide Data Program – Annual Summary, Calendar Year 2008. Release date 12/2009.

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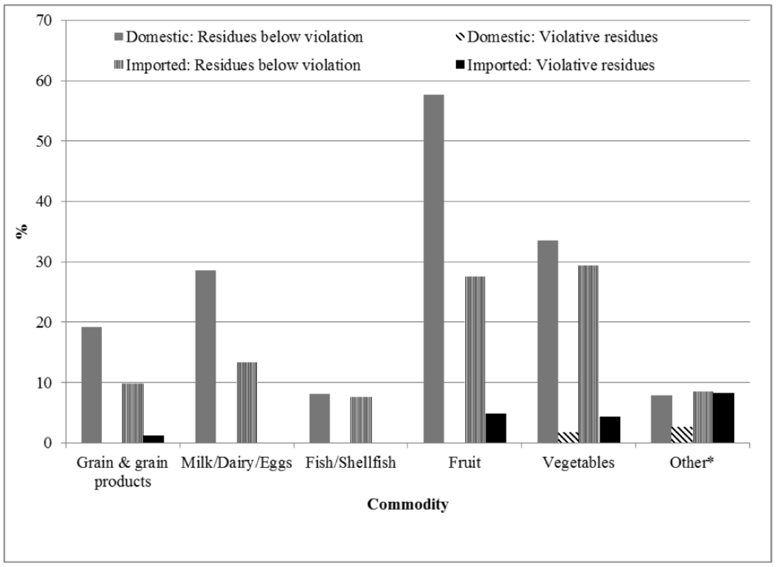

Figure 3.
Pesticide residues in domestic and imported samples by commodity group (2008, n=1,398).
*Other= nuts, seeds, oils, honey, candy, spices, multiple food products and dietary supplements.
Source: USDA Pesticide Data Program – Annual Summary, Calendar Year 2008. Release date 12/2009.

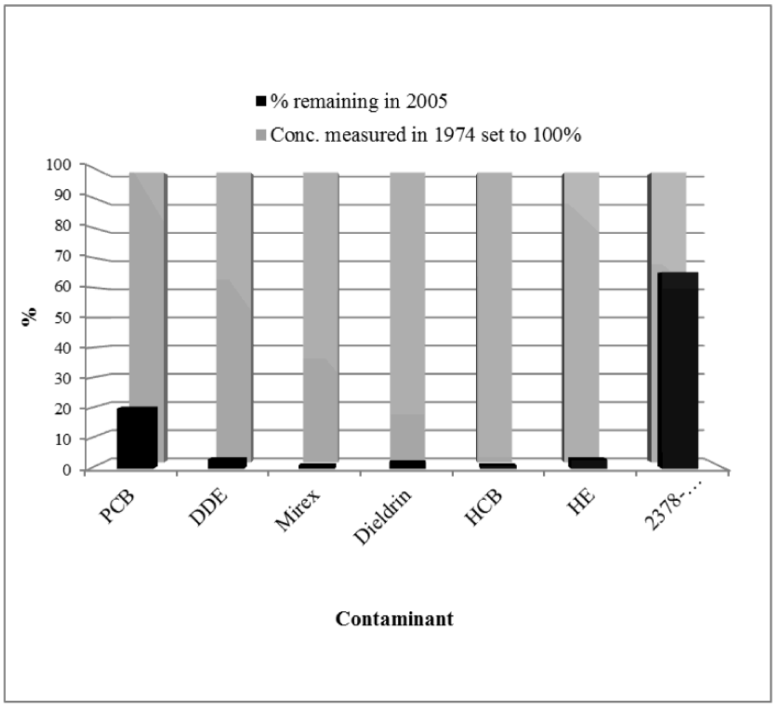

Figure 4.
Percent decline of 7 contaminants in Herring Gull eggs from 1974 to 2005.
Source: Environment Canada, Herring Gull Monitoring Program. 2009 State of the Great Lakes.
*Dioxin first measured in 1984 and last measured in 2003.

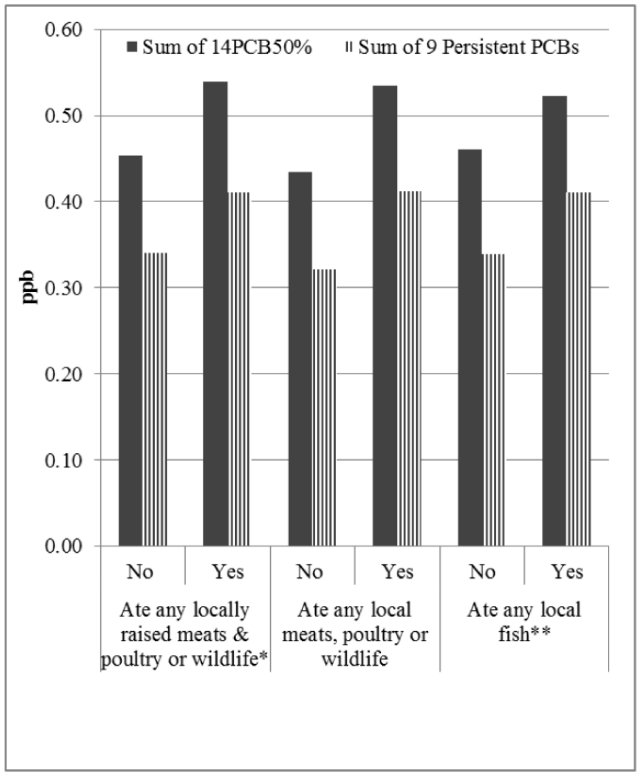

Figure 5.

Different consumption patterns of Akwesasne Youth.

Σ14PCB50%: Sum of IUPAC#s 28,52,74,87,95,99,101[+90],105,110,118,138[+163+164], 153,180,187

Σ9Persistent PCBs: Sum of IUPAC#s 28,74,99,105,118,138[+163+164],153,180,187

*Any local wild life: deer, rabbit, frog, muskrat, turtle, duck, goose, partridge, pheasant

**Any local fish: bass, bullhead, catfish, northern pike, perch, salmon, sturgeon, trout

Table 1

Common non-nutrients in foods.

Class	Examples
Pesticides	DDT, dieldrin, endosulfan, quintozene, chlorpyrifos methyl, hexachlobenzene
Persistent Organic Pollutants	Polychlorinated biphenyls, polybromated biphenyls, polychlorinated napthalenes, dibenzofurans, dibenzodioxins
Metals	Lead, mercury
Plasticizers	Bisphenol-A, phthalates

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Table 2

Pesticides in prepared foods

Pesticide	% Occurrence
DDT	22
Malathion	12
Dieldrin	11
Endosulfan	11
Quintozene	10
Chlorpyrifos methyl	9
Hexachlorobenzene (HCB)	8

Source: (United States Department of Agriculture, 2011)

Table 3

Polychlorinated naphthalene in food (ng/kg).

Food	ΣPCN	ΣPCN TEQ
Meat and meat products	1.64	0.005
Fish	19.9	0.010
Poultry	2.28	0.002
Dairy products	1.15	0.003

Source: (Fernandes et al., 2010).

TEQ: Toxic equivalents

Table 4

Correlation among behaviors reflecting consumption of local foods.

	Adolescents consumed any local fish in the past year[a]		Adolescents consumed any local wildlife in the past year[b]	
	r	**p**	**r**	**p**
Adolescents consumed any locally raised, caught, hunted, or trapped fish, meat, or wildlife in the past year[c]	.746	<0.001	.695	<0.001
Adolescents consumed any local fish in the past year	-	-	.336	<0.001

[a]Any local fish: bass, bullhead, catfish, northern pike, perch, salmon, sturgeon, trout

[b]Any local wild life: deer, rabbit, frog, muskrat, turtle, duck, goose, partridge, pheasant

[c]Any locally raised, caught, hunted, or trapped or fish, meat, or wildlife during the past year: locally produced dairy products, eggs, beef pork, poultry; wildlife (deer, rabbit, frog, muskrat, turtle, duck, goose, partridge, pheasant); fish (bass, bullhead, catfish, northern pike, perch, salmon, sturgeon, trout)