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A Review of the Alteration of Polychlorinated Biphenyls in the Aquatic Environment with an Analysis of the PCB Contamination of the St. Lawrence River Fish

Introduction:

The presence of Polychlorinated Biphenyls (PCBs) within the environment and biota have been measured and studied in great amounts across the world as well as correlated to many adverse health affects in humans. The Environmental Protection Agency (EPA) relates PCBs to several cancerous and non-cancerous health affects[1]. Workers who have been exposed to PCBs have shown high incidence towards developing liver cancers and malignant melanomas. Exposure to PCBs have been linked to cause reproductive issues including low birth weights, decrease in gestation time, low sperm count, and low conception rates. PCBs have also been identified to cause immune system depression, effect neurological development, and disrupt hormone levels by suppressing the endocrine system[1].

It is apparent that PCBs bioaccumulate as they move upward in tropic levels and ultimately have adverse health affects on humans. However, it is not always clear which biological vector has the most toxic effect on humans. The immediate objectives of this study are to: a) investigate the alteration of PCB congener patterns as PCBs are transferred through various environmental media and biota, b) identify the original contaminating Aroclor mixture(s) within the St. Lawrence River, c) analyze PCB data sets of large predator fish in the St. Lawrence River to determine the fish's metabolism, and biomagnifications of PCBs, d) identify PCB congeners that can serve as possible indications of PCB exposure in humans from fish consumption, e) suggest improvements to New York State Fishing Advisories to increase awareness of advisories and decrease contaminated fish consumption.

Back Ground Information:

The presence of PCBs in the environment originates mainly from Industrial waste. PCBs were manufactured for a variety of industrial applications including use in transformers, capacitors, and hydraulic fluid[2]. The Mohawk Nation in Akwesasne, population 10,000+, is located on the banks of the St. Lawrence River. Akwesasne is also located directly downstream (East) of three US Federal Superfund Sites, formally factories owned by General Motors, Reynolds Aluminum and the Aluminum Company of America (ALCOA). All three factories used PCBs in production as well as contaminated the St. Lawrence River with PCB waste[3]. The primary Aroclor mixture used by these factories is Aroclor 1248, but studies have found patterns resembling Aroclor 1254 and 1260 within the Akwesasne environment and Mohawk serum samples[2, 3].

The release of PCBs into the St. Lawrence River proceeded to contaminate most environment media and biota, including the air, sediment, soil, fish, local meat and produce, and ultimately contaminating the Mohawk people[3]. The Mohawk culture stresses the relationship between humans and their environment, therefore is dependent on local fish as a source of food[4]. The Mohawks proximity to the Superfund Sites and their dependency on local fish increases their risk of PCB exposure.

The St. Lawrence River currently has fishing advisories posted for the entire river[5]. The US Food and Drug Administration do not recommend eating fish with PCB concentration levels above 2 ppm[6]. Women under 50 and children under the age of 15 are advised not to consume any fish from the St. Lawrence River[5]. The St. Lawrence fishing advisories state not to consume any fish from the Bay and Cove east of South Channel Bridge, the area of the river adjacent to the Akwesasne territory. By the NYS DOH and DEC standards, all local fish to Akwesasne are unsafe, making the Mohawk people especially susceptible to PCB contamination via fish consumption[5].

For the case of this review the chain of contamination will start with the release of a pure Aroclor mixture into the St. Lawrence River. The changes in PCB patterns as the congeners are adsorbed into the sediment, suspended in the water, and volatized at the surface of the water into air will be studied. Further alterations to the overall PCB pattern and individual congener structure will be discussed through routes of metabolism and accumulation in fish within the river.

Nature of PCBs

The Toxic Substances Control Act was passed in 1976 due to increasing evidence of adverse health affects of PCB exposure and bioaccumulation within the environment. By

1977 production of PCBs stopped [2]. Long-term bioaccumulation of PCBs is made clear by a study published in 2005[3]. Blood collected from Mohawk men and women whose age ranged from 18-95 years old found a PCB concentration ranged from 0.29 to 48.32 ng/g. The presence of 100 PCB congeners at detectable levels exemplifies the persistence of PCBs in the environment even after 30 years post production[3].

The persistent nature of PCBs is due to the stability of their chemical structures. Polychlorinated biphenyls are composed of 2 phenyl rings with numerous chlorines attached[2]. There are 10 different locations for chlorine bonds, positions 2, 3, 4, 5, 6 on the first phenyl ring and 2', 3', 4', 5', 6' on the second phenyl ring. The polychlorinated biphenyls are distinguished by chlorine positions and with an IUPAC numbering system. They are further categorized into homolog groups that separate biphenyls by the number of chlorines attached ranging from mono to deca. Positions around the biphenyl ring are numbered 2 through 6 and also have corresponding names. Chlorines in the 2, 2', 6, and 6' position are called ortho chlorines. Para positions are at 4 and 4' and 3, 3', 5, and 5' are the meta positions[2].

The structure of polychlorinated biphenyls results in chemicals that have a high boiling point, heavy molecular weight, low water solubility, and low flammability [2]. PCBs are also lipophilic, meaning the chemicals tend to settle within adipose tissue of animals and humans, decreasing the chance of metabolism. These qualities make PCBs ideal for industrial use and persistent within the environment and animals [2]. Two influencing factors on PCB persistence and bioaccumulation are the number of chlorines present and the specific positions of the chlorine atoms[7]. As the number of chlorines increase and homolog group increases, PCBs becomes less water soluble, less volatile, and more hydrophobic. PCBs congeners found in air and water samples are often less chlorinated homologs and tend to alter over time[2]. Serum samples taken from Mohawks were dominated by penta- to hepta- chlorination, showing that not only are higher chlorinated biphenyls are more persistent, but also tend to bioaccumulate over time[3].

Water is the main media in which PCBs were disposed of into the environment. It is estimated that 99% of the global mass of PCBs are found in soil and sediment, yet PCBs spread via networks of waterways and most drastically by the air[2]. Even though PCBs are hydrophobic, they still possess the ability to dissolve in water and evaporate into the air. Once PCBs volatize, PCBs can then spread via wind, rain, and snow, and once again obtain a solid state in sediment and soil[8]. It is the presence of gaseous PCBs in our environment that has caused PCBs to physically reach every corner of our earth, including traces found in the icebergs of Antarctica. As result of water being the source of most contamination, PCBs have reached every corner of the earth through water flow and volatilization of PCBs into the air [8].

Alterations of PCB Congener Patterns

Water and Air:

As threatening as PCB volatilization seems to be in regards to spreading contamination, the movement of PCBS through evaporation and water flow is an essential process to reduce PCB concentrations in those waterways that are highly contaminated. PCBs are removed from contaminated bodies of water 4 ways: river outflow, volatilization, sedimentation, and biological transformation within biota [8].

Most PCBs found within water samples are typically attached to suspended sediment and particles[2]. Even though PCBS are hydrophobic and have low vapor pressure, it is still possible for PBCs to dissolve in water. The Octanol Water Partition Coefficients (Kow) of PCB congeners are used to predict the dissolvability PCBs in water. The Kow increases as chlorinity and molecular weight increases, exemplifying that as PCBs become more chlorinated and heavier, the chance of dissolving decreases [2]. Typically, the PCB congeners found dissolved in water are less chlorinated congeners. For example, PCBs 1, 4 and 10 compose of 1/3 of the PCBs in Hudson River water samples[9].

The lipophilic nature of PCBs is the driving factor of PCB bioaccumulation[9]. The presence of dissolved and suspended PCB particles in water have lead scientists to debate if the main source of biouptake of PCBs in marine wildlife is from absorption in water or through the food chain. In the case of the marine food web, it is obvious that both sources contribute to PCB burden, but finding out which source is more harmful can be beneficial to remediation efforts. In an experiment to find out the role of PCBs exposure by water, chrionmids were kept in cages suspended in PCB contaminated water to determine the rate in which PCBs are absorbed and what specific congeners bioaccumulate[9]. They were fed non-contaminated food. Chironomids are insects found in aquatic habitats and are an essential part of the food chain leading up to larger marine species. In the hours 2-4 of suspension, equilibrium of transfer between PCB congeners 4, 10, and 19 from the water to the chironomids was reached. Higher chlorinated congeners, those greater than tetra chlorinated, did not reach equilibrium of transfer between chironimids and water until after 5 days of suspension. Even though the amount of time it took for higher chlorinated congeners to reach transfer equilibrium was significantly longer, those PCBs bioaccumulated at concentrations 10-50 times higher in the chironomids than the less chlorinated ones. This experiment exemplified the significance of biouptake solely through water exposure and the ability of highly chlorinated congeners to accumulate at much greater concentrations than lower chlorinated congeners[9].

It is important to study the biouptake of PCBs in smaller species at the lower end of the marine food chain because the PCBs small species accumulate will be magnified within their predators. A similar experiment done in the St. Lawrence River, caged rainbow trout in the river at an extremely contaminated water site, 900ng/g PCB concentration, and at a less concentrated PCB site, 40ng/g[9]. The rainbow trout were also fed uncontaminated food in order for PCB uptake to be solely from the water column. After 30 days, the fish in the highly contaminated water (900 ng/g) had an uptake rate of 60 $\pm$ 18 ng/g/day. At 40ng/g, the fish had an uptake rate of 13 $\pm$ 3 ng/g/day[9]. The rate is uptake is clearly related to the PCB concentration present. As PCB concentration in water decreases, the rate of uptake slows. The PCB patterns found in the fish closely resembled the patterns found in the water, with a slight shift towards higher chlorinated congeners over time.

This shift corresponds with the nature of higher chlorinated congeners to accumulate at greater rates than less chlorinated. This shift could also be due to the fish metabolizing less chlorinated PCBs. No equilibrium of PCB transfer between water and trout was reached after 30 days[9]. The lack of equilibrium in the rainbow trout indicates long-term accumulation through water. Fish are submerged in water their entire lives and constantly swimming and relocating through a body of water. Constant movement through areas of high PCB concentrations into lower concentrations can result in PCB uptake to never reach equilibrium. If this is the case, PCB uptake through water can continuously occur throughout the lifetime of a fish, resulting in high levels of accumulation.

Investigating the contribution of PCB burden from the water column is crucial component to understanding bioaccumulation in marine food webs. The results of PCBs patterns accumulated from water exposure matches the PCB patterns in the water column allows researchers to easily identify the influence of the food web in regards to PCB uptake[9]. PCB patterns in marine life that are found at the higher end of the food web do not have PCB patterns similar to water or sediment[10]. The difference in PCBs patterns indicates the role of food consumption to altering the PCB pattern and congener accumulation. Lake, McKinney, Lake, Osterman, and Heltshe further studied biouptake by water and the role of the food chain by comparing PCB patterns in water and sediment to mussels, mummichogs, eels and shrimps[10]. The PCB patterns in the water and sediment were very similar, except that the sediment was slightly more chlorinated. Mussels and mummichogs are benthic species and are at the bottom of the food web. The PCB patterns in both species closely matched the patterns found in the water and sediment. However, the patterns found in the shrimp and eels were extremely contrasting to the patterns found in sediment, water, mummichogs, and mussels. Shrimps and eels are also mainly benthic species, but sit higher in the food web than mummichogs and mussels; therefore accumulate congeners from their diet along with water absorption. The difference in patterns identifies pattern changes from the food web and the role of metabolism in larger species. Shrimp and eels are believed to have enzymes such as P4501A and P4502B that can metabolize congeners based on chlorine arrangement[10]. The combination of consuming lower marine species that have already bioaccumulated PCBs, metabolism of congeners, and biouptake through the water column, larger predator species will have different PCB patterns than water.

Understanding the influence of water and the food web to PCB patterns in fish is a factor within the main objectives of the study. In order to determine what Aroclor mixtures the fish have been contaminated with, back tracking exposure will be necessary. By eliminating changes in PCB patterns from the food web, factors such as bioaccumulation and metabolism are removed. The PCB patterns found in water and small benthic species should be a more accurate representation of the original Aroclor mixture. PCB patterns from the water and benthic species from the St. Lawrence River will be needed to investigate the original exposed Aroclor mixture.

Volatilization is an essential process to decreasing concentrations of PCBs and the presence of water is essential for volatilization to occur. Occurrences of volatilization among contaminated dry sediment and soils are significantly less than wet samples[2]. The importance of water for volatilization highlights the incidence of seasonal floods and tides to enhancing volatilization from dry surfaces. Based on congener analysis in air samples, it is found that ortho rich congeners are more easily volatized[2]. The ability for ortho rich congeners to volatilize is extremely important to note because processes of sedimentation and metabolism are unable to breakdown ortho rich congeners. Ortho rich congeners are also believed to be the most toxic of all the PCBs, making their ability to volatize a threat to the spread of PCB toxicity across the world[7]. Similar to predicting the dissolvability of PCBs, a low Octanol Water Partition will also determined the ability for PCBs to volatize. Low Kow is represented by PCBs with low molecular weight and low chlorinity, hence factors for volatilization[2]. Even though these factors are favored for volatilization and evaporation, almost all PCB congeners have been found in the gaseous phase.

An experiment conducted to test the volatilization potential of Aroclor 1242 in a sample of sand tracked changes in the Aroclor congener pattern over 24 hours[2]. Wide ranges of congeners, regardless of chlorinity and molecular weight, were able to achieve the vapor phase over the 24 hours. However, most congeners were temporarily vaporized and quickly returned to the sand sample. After the 24 hours past, the A1242 pattern had significant loss of less chlorinated congers, especially in PCB 1, 3, 4, 5, 8 and 10. The loss of less chlorinated congeners caused the A1242 pattern in the sand to shift right. The shift of A1242 to the right made it appear as it were a higher chlorinated mixture with a close resemblance to A1248[2]. Environmental alterations to PCBs often leaves patterns to be appear less chlorinated through degradation, metabolism, and dechlorination;

however, it is clear that volatilization removes less chlorinated congeners resulting in the appearance of a higher chlorinated pattern[2]. In reality, a pattern of volatilization is not anymore chlorinated than its original mixture. Volatilization causes a loss of concentration and PCB weight, but the pattern is misrepresented by the loss of lower chlorinated congeners. In a similar test of the volatilization of A1260, after 24 hours the contaminated sand almost identically resembled the original A1260 mixture. The lack of change in the pattern is intuitive since A1260 is a much higher chlorinated mixture, therefore has fewer lightly chlorinated congeners that can volatize[2]. These observations will be essential when studying PCB patterns within the St. Lawrence River. Sediment and water samples from the river will have the appearance of patterns of higher chlorinated congeners due to lighter ones dissolving and volatizing. Without knowledge of how PCB patterns change from volatilization, one can assume the wrong Aroclor mixture was the original exposing factor. This will be a difficult process for the St. Lawrence River due to the fact that the river has been exposed to several Aroclor mixtures, including 1245, 1248, and 1260[3]. A sample of volatized 1245 may resemble A1260, but it will be difficult to distinguish whether or not that sample is originally 1260 or has undergone volatilization.

Based on PCBs hydrophobic nature, it was long believed that decreases in PCB concentrations over time were a result of sediment accumulation and burial. However, a study done by Jeremiason, Hornbuckle & Elsenreich in Lake Superior disproved that sediment burial was the main loss of PCB concentrations[8]. The study took air, water, and sediment samples annually since 1977 in order to measure changes in PCB concentrations. The results found that from 1980-1992, 26,500kg of PCBs were lost through volatilization, where as sediment only accumulated 4,900kg[8]. The study further measured changes in PCB concentrations by measuring changes in individual congeners. As previously mentioned, PCBs with low molecular weights have a low octanol water partition, allowing them to dissolve more easily into water and volatilize[2]. Concentrations of congeners with lower weights decreased significantly over time within the Lake Superior water samples[8]. The presence of PCB congeners with higher molecular weights (chlorines $\geq$ 6) within water samples also decreased over time, but at a much faster pace. This may seem confusing at first, how is the concentration of heavier and more chlorinated PCBs decreasing at a faster rate within water than lighter ones? The decrease is not related to volatilization, but to sediment burial[8]. PCB congeners with higher molecular weight have higher octanol water partitions, making them more hydrophobic[2]. These extremely heavy and hydrophobic congeners do not stay suspended in water long; they end up sinking down to the river sediment[8]. From the study, it seems like PCBs within water are in a temporary state, either dissolved or suspended in the water because they are attached to sediment particles. Those lighter, less chlorinated congeners that are dissolved linger in the water before volatilization. Those that are suspended sink to the bottom of the river fast[8]. Based on these statements, it seems like the loss of PCB congeners in water splits up evenly by air and sediment, but the loss to air is much greater. Lighter chlorinated PCBs dominated the water column, hence why more PCBs are lost to volatilization than sediment burial[9].

A similar experiment to Jeremiason tested the rates of volatilization in Green Bay, Lake Michigan[8, 11]. Air samples detected over 85 congeners in the vapor phase, but similarly to Jeremiason, the air was dominated by lighter chlorinated congeners[8, 11]. By incorporating factors of wind speed, temperature, and the concentrations of PCBs in the water; Green Bay exemplified volatilization rates ranging from 13-1300ng/m^2/day[11]. The study found that an increase in wind speed increases volatilization. As temperature decreased, volatilization decreased. In the winter months, no volatilization was detected from Green Bay. The particular combination of air temperature greater than 10 C and wind speed greater than 4m/s, caused a significant increase in volatilization. After factoring in weather trends of temperature and wind speed, the loss of volatilization during the winter, and PCB concentrations found through Green Bay, it was concluded that volatilization accounts for 33%-43% of total PCB loss within lake[11]. This study further supports and enhances the significant role that volatilization plays in decreases PCB concentrations.

It was previously mentioned that the blood samples of Mohawks are dominated by penta-hepta chlorinated PCB congeners. Less chlorinated PCBs are not as persistent over time as higher chlorinated ones and can also be metabolized[3]. Since traces of lightly chlorinated congeners are not often detected in humans, the effects of exposure to volatilized PCBs were long debated. However, in the cases that lightly chlorinated PCBs are detected within the blood of humans, it has been concluded that their presence is due to PCB exposure via inhalation[2]. Further, since these lightly chlorinated congeners are not persistent, their presence in blood indicates that exposure is recent[2]. This knowledge can be useful in identifying areas of high PCB concentrations.

In conclusion, water serves as a media for the biouptake of PCBs into marine species, a contributing force to the volatilization of PCBs, and a way in which PCBs spread. The PCB patterns in water often resemble the original exposing Aroclor mixture but with a shift to the left. Less chlorinated congeners dissolve in water and are therefore found at higher concentrations within water than highly chlorinated congeners which sink into the sediment. As a result of lightly chlorinated congeners dissolving and volatizing, sediment patterns shift right, resembling higher chlorinated patterns. These pattern changes will help track changes to PCB congeners as they move up the food web, as well as assist in back tracking exposure to the original Aroclor mixture. The ability of PCBs to volatize is essential to the reduction of PCB concentrations within highly contaminated waterways, but it also the media in which PCB toxicity has reached every corner of the world. When lightly chlorinated congeners are detected in human blood serum, it is a result of recent inhalation exposure. The reoccurring exposure humans have to PCBs is a reminder of how persistent PCBs really are, even after 30 years of not being produced.

Sediment:

Polychlorinated biphenyls are persistent in the environment but also have the ability to naturally biodegrade over time. Once PCBs reach sediment, there is opportunity for dechlorination and degradation to occur.

Two types of bacteria found in sediment samples, aerobic and anaerobic, exemplify abilities to dechlorinate and biodegrade PCBs[12]. Aerobic bacteria are found on the surface of sediment in an oxygenated environment. Aerobic bacteria are able to break open the biphenyl ring by attacking the 2,3 position on a ring with 2 oxygen atoms. The ring is broken, producing a p-chlorobenzoic acid. Through this degradation process, aerobic bacteria virtually have the ability to fully eliminate PCBs[12].

As promising as aerobic bacteria degradation seems, there are restrictions that limit its ability to access and effectively destroy PCBs. The function of aerobic bacterial degradation is based upon breaking a phenyl ring at the 2,3 (ortho-meta) positions. Any PCB congeners with chlorines at the ortho-meta positions will be resistant to aerobic bacterial[12]. Aerobic bacteria tend to degrade only lower chlorinated PCBs. A figure by Abramowicz illustrates that as PCB congeners become more chlorinated, the percentage of degradation decreases significantly or stops all together[12]. Mono- through tetra-homologs generally exhibits aerobic degradation. More chlorinated PCBs can be broken down through aerobic bacteria, but is limited to specific bacteria strains. Abramowicz's study discusses the ability of LB400 bacteria strain (pseudomonas sp.) to breakdown 90% of Aroclor 1242 when incubated[12]. However, the effectiveness of LB400 is restricted to the limited concentrations of the bacteria found within sediment in the natural environment, unlike a laboratory setting. Even though LB400's effectiveness may not be as dominant in the natural environment, it is important to note its potential when considering methods of sediment remediation.

Other restrictions of aerobic degradation include access to PCB's. Aerobic bacteria are found at the surface of sediment and on the shores of riverbanks due to their need of oxygen for cellular respiration[12]. A study conducted on sediment in the Hudson River approximated that 90% of the remaining PCBs deposited on the surface of river sediment had become buried[13]. If this is true, then aerobic bacteria cannot access PCBs to perform degradation. The EPA acknowledges tendencies for PCBs to become buried, but clarifies that 90% of the top 2 inches of sediment contain PCBs[14]. That clarification is important because for years it was argued that when PCBs become buried, they are no longer an environmental threat. Although PCBs become buried, they remain towards the top of the sediment. With factors such as erosion and river flow, these sediments can easily become re-exposed to the surface of the sediment[14]. The chances of re-exposure allow for aerobic bacteria to continually degrade PCBs in sediment. If the same holds true for the St. Lawrence River, then aerobic bacterial degradation can play a significant role in the degradation of PCBs and lower concentrations found in sediment over time.

Anaerobic bacteria live in nonoxygenated environments such as aquatic sediments[12]. Anaerobic bacteria possess the ability to dechlorinate PCBs as opposed to aerobic bacteria's ability to break the phenyl ring. Anaerobic bacteria can make highly chlorinated PCBs less chlorinated. As PCBs become less chlorinated, their octanol water partition decreases, making PCBs less hydrophobic. Less chlorinated PCBs are favorable in the environment because they are more likely to undergo aerobic bacterial degradation and are less persistent over long periods of time[2]. Less chlorinated PCBs can also be adsorbed into water and volatize into the air and undergo further degradation through those media. The abilities of anaerobic bacteria to dechlorinate PCBs and aerobic bacteria to break the phenyl ring of less chlorinated PCBs are complementary[12]. By anaerobic bacteria's dechlorination of buried PCBs, and with the combined actions of river flow, weather, and dredging that can cause PCBs to resurface, aerobic bacteria can then break the phenyl rings and destroy the PCB[15]. Together both bacteria can significantly decrease the PCB concentration of a river over time.

Seven different processes of anaerobic dechlorination have been identified. Each process eliminates specifically positioned chlorines and establishes a mother-daughter relationship among PCBs and their dechlorinated product[15]. Bedard and Quensen commented on PCB dechlorination, "The most sensitive indicator of dechlorination is the appearance of typical PCB dechlorination products in proportions not found in the Aroclors. These will differ depending on the Aroclor(s) present and on the specificity of the dechlorination"[15, p.135]. These processes and patterns of dechlorinated products that Bedard and Quensen have established will be crucial when trying to decipher what Aroclor mixture was originally exposed to the marine food web of the St. Lawrence River. Bedard and Quensen also present figures that illustrate chains of dechlorination as chlorines are removed from PCBS that will be useful in following PCB patterns as the chemicals move up the trophic web[15].

The first process "M" is the removal of flanked and isolated chlorines in the meta positions 3 and 5[15]. Process M is less effective on highly chlorinated PCBs and is usually exhibited on mono to tetra homologs. Mother-daughter dechlorinated products (IUPAC) due to process M include: CBs 5,9,6 →CB 1; CBs 44, 52 → CBs 16, 18 → CB 4; CBs 56, 70 → CBs 33, 22, 31 → CB 8; CBs 49, 66, 71, 64 → CBs 17, 28, 32. As previously mentioned, Process M does not have a strong effect on highly chlorinated PCBs. The highest chlorinated PCB mentioned was only PCB 71. Process "Q" removes flanked and isolated para chlorines. Similar to process M, process Q is less effective on highly chlorinated PCBs. Dechlorination products from process Q include: PCBs 3, 4, 6, 16, 18, 19, 26, and 27. Together, process M and Q create pattern "C"[15]. It is not clear which process occurs first or if they occur simultaneously, but regardless, both processes create a pattern of less chlorinated products.

Processes H and H' remove flanked and double flanked para chlorines[15]. Neither process eliminates isolated para chlorines. Process H' has also been observed to remove meta chlorines from PCBs 5 (2,3), 21 (2,3,4), and 24 (2,3,6). Due to the similarities of both processes, Bedard and Quensen question whether or not process H' is a result of combined efforts of process H and meta dechlorination of congeners with a 2,3-, 2,3,6-, and 2,3,4- chlorines[15]. Dechlorination products from process H' include PCBs: 4, 6, 8, 17, 18, 19, 25*, 26*, 27*, 28, 31 and products of process H are PCBs: 6, 25, 26, 27, 28, 31, 47, 49, 52, 90, 91, 92, and 95. PCBs products 25*, 26*, and 27* are believed to have undergone meta dechlorination from process H'. Process 'P' also eliminates flanked and double flanked para chlorines, but on even higher chlorinated PCBs in homolog groups tetra through hexa. PCB products include 6, 25, 26, 27, 31, 40, 44, 49, 52, 83, 95[15]. Lastly, Process 'N' removes flanked and double flanked meta chlorines. Process N is found among highly chlorinated PCBs in homolog groups tetra through octa. Products of process N include PCBs: 28, 31, 32, 47, 49, 51, 53[15].

Processes H, H', P and N preferentially dechlorinate higher chlorinated PBCs and homologs. This is observable because their dechlorinated products have a higher chlorination than the products of processes M and Q[15]. Due to this quality, processes H, H', P and N have little effect on lower chlorinated Aroclor mixture such as 1242 and 1248, but instead will dechlorinate mixtures 1254 and 1260. Processes M and Q have been observed in the dechlorination of Aroclors 1242, 1248, and 1254[15]. Aroclor mixtures 1248, 1254, and 1260 are known contaminants of the St. Lawrence River, making all of the listed processes relevant to this study[3]. None of the discussed processes remove ortho chlorines. Failure to remove ortho chlorines and creation of open meta and para chlorines positions will play an essential role in PCB metabolism that is later discussed.

It is hypothesized that anaerobic bacteria contain enzymes that are attracted to chlorines at the meta and para positions around the phenyls rings[15]. It is further believed that these enzymes obtain thermodynamic energy by using the chlorines on PCBs as electron acceptors. A study conducted by Kim and Rhee tested changes within microorganisms' populations in PCB contaminated sediments[16]. The study used Aroclor 1248 as the contaminating factor. Aroclor 1248 under went a 28% decrease in chlorination over weeks 3 through 6. During this period of dechlorination, the growth rate of microorganisms within the sediment increased 98% from weeks 2 through 6. Once dechlorination reached a plateau, the microorganisms stopped growing and their population decreased[16]. Kim and Rhee hypothesized the spike in growth of microorganisms is fueled by the thermodynamic energy that is obtained during the dechlorination process. This study highlights nature's way of sediment remediation and the potential for anaerobic bacteria to dechlorinate PCBs[16].

The ability of anaerobic bacteria to remove meta and para chlorines from PCBs is essential for reducing the bioaccumulation of PCBs within the environment[15]. However, just as there were limitations to aerobic bacteria's ability to destroy PCBs, there are restrictions to the success of anaerobic bacteria to dechlorinate PCBs. The dechlorination on PCBs is hypothesized to be dependent on a few factors: 1) the anaerobic bacteria population present[17], 2) position of chlorine on the phenyl rings (ortho, meta and para) and the positions of surrounding chlorines (flanked, double flanked, isolated) [15], 3) Specific PCB congeners found within the sediment[18], and 4) the PCB concentration in sediment[19].

The first limitation mentioned, the presence of anaerobic bacteria in sediment, is essential for dechlorination to even be possible. A study, conducted in 1996, questioned whether bioavailability of dechlorinators within sediment or accumulation of dechlorination products was the main cause of dechlorination plateaus[17]. The experiment used Aroclor 1248 as the contaminating mixture. After 24 weeks of incubation, all dechlorination had stopped. To test if bioavailability of anaerobic bacteria had caused the plateau in dechlorination, additional dechlorinators were added to the 24-week-old sediment samples. The addition of new media to the old sediment samples resulted in no further dechlorination; therefore, proving that plateaus in dechlorination are not related to bioavailability of dechlorinators within sediment[17]. This conclusion is further supported by the results of Kim and Rhee's previously mentioned study[16]. Kim and Rhee showed that microorganism populations grow during dechlorination periods and shrink as dechlorination plateaus. Both studies eliminate bioavailability of anaerobic bacteria as a limit for dechlorination[16, 17].

Since availability of dechlorinators is not a cause of the dechlorination plateau; it was concluded that the accumulation of dechlorinated products must be responsible for the plateau[17]. Anaerobic bacteria target specific chlorines based on location and surrounding chlorines. Not all chlorines on the biphenyl rings are removable, including all ortho chlorines[15]. Once all removable chlorines have been detached, only dechlorinated products remain[17]. The plateau of dechlorination is a result of only daughter congeners remaining in the sediment that cannot receive further dechlorination. This conclusion also aligns with why additional media to the sediment did not cause further dechlorination to Aroclor 1248[17]. It was not the lack of bacteria within sediment, but instead the lack of accessible chlorines.

With the conclusion that the position of chlorines is a direct limitation to dechlorination, this leads to the limits of dechlorination of specific PCB congeners based upon chlorine atom arrangement. The daughter products of the seven processes of dechlorination suggest that dechlorination is congener specific, based upon chlorine positions and the overall chlorination of the PCB[15]. A study done Sokol, Bethoney and Rhee, investigated dechlorination rates of specific PCB congeners at designated concentrations over time[18]. PCBs 21 and 138 at concentrations 4, 9, 16, and 35 ppm were mixed with anaerobic bacteria sediment. These concentrations tested represent accurate proportions found within natural sediment after years of dispersion[18]. PCBs 21 and 138 serve as viable PCB congeners to test dechlorination because both have 2,3,4- configuration on the first phenyl ring, leaving opportunity for ortho, meta, or para dechlorination to occur (although ortho dechlorination is not reported as possible). PCB 21 configuration is 2,3,4 with the second phenyl ring empty, and PCB 138 is 2,3,4-2'4'5'[15]. Bedard and Quensen had previously demonstrated that higher chlorinated PCBs do not undergo dechlorination to the extent that lower chlorinated PCBs experience. The lower chlorination of the PCB, the more fully it will be dechlorinated by anaerobic bacteria[15]. By comparing the dechlorination rates of both PCB 21 and 139, conclusions on whether or not dechlorination is congener specific based upon the original chlorination of the PCB can be determined. Both PCB congeners were inoculated with anaerobic sediment for 7.5 months and tested frequency for dechlorination[18]. Dechlorination was first detected for PCB 21 after 2. 5 months of incubation in concentrations 9, 16, 35 ppm. It was not until 5 months into the experiment when concentration 4ppm exhibited dechlorination[18]. All samples produced PCB 7 (configuration 2,4) which reveals the loss of a double flanked meta chlorine that identifies with process H'[15]. At 7.5 months, 4, 9, and 16ppm were indistinguishable from their original concentrations of PCB 21. Concentration 35ppm still revealed PCB 21, and was still undergoing dechlorination[18]. The samples of PCB 138 had a significantly longer incubation period. It was not until 5 months when concentration

35ppm first showed signs of dechlorination. By the 7.5 month, no dechlorination occurred to concentrations 4, 9, 16 ppm. Concentration 35ppm had dechlorination products PCBs 47 (2,4-2'4') and PCB 49 (2,4-2'5')[18]. Both PCB 47 and 49 are a result of loss of double flanked meta chlorines and PCB 49 also shows the loss of a double flanked para chlorine which is a combined effort of processes H' and H[15]. The results of Sokol, Bethoney, & Rhee study not only exemplify the processes presented by Bedard and Quensen, they also correlate with the belief that lower concentrations of PCBs will not dechlorinate as fast or thoroughly[15, 18]. Further, the result that PCB 21 dechlorinated much faster at all concentrations, further supports that lower chlorinated PCBs are more susceptible to dechlorination that highly chlorinated PCBs such as PCB 138. Higher chlorinated PCBs incubate longer and experience less dechlorination than lower chlorinated ones[15, 18].

Sokol, Bethoney and Rhee's 1995 study highlights the importance of PCB concentration for effective dechlorination and how rates of dechlorination are congener specific. A follow up study by the same scientists in 1998, tested the rate and extent of dechlorination of Aroclor 1248 at 16 different concentrations[19]. The concentrations tested rang from 1ppm to 200ppm. It is important to study dechlorination rates of the Aroclor mixture and not just specific congeners since A1248 is the main contaminating agent in the St. Lawrence River. Within the St. Lawrence River sediment, there will not be one specific congener at a designated concentration found naturally; instead there are mixtures of PCB congeners, with different chlorination percentages, and different concentrations. Testing dechlorination of A1248 at 16 difference concentrations can give a realistic idea of the dechlorination that has happened in the St. Lawrence River sediment[19].

After 58 weeks of incubation, concentrations of A1248 at and lower than 35ppm yielded no signs dechlorination [19]. At the highest concentration tested, 200ppm, A1248 experienced a loss of chlorines atoms from 3.98 to 2.55 per biphenyl. Among the samples that experienced dechlorination, none underwent a loss of ortho chlorines atoms. The loss of meta chlorines was 5 times greater than the loss of para chlorines. Between weeks 25 and 35, dechlorination plateaus were reached for highest concentrations of between 100ppm and 180ppm. These plateaus were a result of accumulation of dechlorinated products[19].

Sokol, Bethoney and Rhee's study introduces the idea of concentrations thresholds[19]. There was no sign of dechlorination at concentrations lower then 35ppm. It is not certain whether or not those samples need longer than 58 weeks to incubate in order to dechlorinate or dechlorination is simply not possible. Other studies have suggested thresholds starting as high as 140ppm and as low as 10ppm and 20ppm. It makes sense that a clear threshold is not established due to Sokol, Bethoney and Rhee's previous study conducted in 1995[18]. The 1995 study proved that dechlorination was congener specific based upon chlorination and chlorine positions[18]. Aroclor 1248 is a combination of over 100 PCB congeners[19]. With the varying presence of congeners at different concentrations, dechlorination rates will be situation specific every time. Even though this study does not create a definite dechlorination threshold for A1248, it allows a general understanding of dechlorination patterns in the St. Lawrence River[19]. While examining sediment samples of the St. Lawrence River, there are expected daughter products, an overall loss in meta chlorines, and not a significant dechlorination at low concentrations of PCBs. To further solidify dechlorination patterns, a long term study based upon sediment dechlorination of the St. Lawrence River would be helpful in determining dechlorination rates after 58 weeks for lower concentrations samples.

In conclusion, degradation and dechlorination of PCBs occurs in sediment by the actions of aerobic and anaerobic bacteria. Limitations of both processes leave complete removal of PCBs from sediment unrealistic. Both aerobic and anaerobic bacteria effectiveness is dependent on chlorines positions and overall chlorination. Anaerobic bacteria fail to remove any ortho positioned chlorine atoms, but show great potential in the removal of meta and para chlorines. Dechlorination is congener and concentration dependent. Unfortunately, PCB's were released into waterways over 30 years ago; the chemicals have become dispersed at low concentrations due to tides, river flows, flooding, and weathering, hindering potential for dechlorination. It is also difficult to document exact dechlorination rates within nature, due to PCB concentrations decreasing in sediment because of absorption into other media. As PCBs have are dechlorinated, they are also dissolved into water, volatized into air, and are been ingested by marine life. Even with all of these restrictions to sediment study, the importance of PCB dechlorination should not be looked over while studying St. Lawrence River data. It is also important to study the composition of the bottom of the St. Lawrence River. There is potential for dechlorination among sediment; however, river bottoms that are composed of rock and gravel have shown to not exhibit and dechlorination[9]. It will be essential to map out the composition of the bottom of the St. Lawrence River near the factory sites and near the banks the Mohawks reside. The presence of daughter congeners in sediment samples will be a good indication of dechlorination and allow back tracking to determine to original Aroclor mixture. Knowing patterns of dechlorination will also allow congener patterns to be established for the marine food web. The fish data from the St. Lawrence River is taken in 2005 and 2007. PCB patterns in the fish will most likely resemble patterns of dechlorination since the sediment has been actively dechlorinating for over 30 years.

Persistence and Metabolism:

The specific positions of chlorines have also proven to be a determining factor in PCB persistence, clearance rates, and toxicity[2]. It is essential to understand patterns of PCB metabolism and biomagnification among organisms in the marine food web for this study. The first objective of this study was to analyze the alteration of PCB congener patterns as the PCBs are transferred through various environmental media and biota. Once patterns of metabolism and persistence among congeners are established, methods can be applied to the fish data. Determining how fish metabolize and/or bioaccumulate congeners can expose their role in human PCB exposure.

There are a various number of factors that determine the potential for PCBs to be excreted, metabolized, or bioaccumulated. One of the first predicting factors as to whether or not PCBs will be metabolized is their octanol water partition, Kow. PCBs with Kow< 5 will generally not bioaccumulate in fish and can be excreted, hence why fish do not exemplify lower chlorinated congeners such as mono, di and tri[20]. PCBs with Kow between 5 and 7 will bioaccumulate, but can be biotransformed to products that can be excreted, and lastly, those with a Kow greater than 7 will significantly bioaccumulate in fish. The octanol water partitions of PCBs increase as PCBs become more chlorinated, and the same applies for the half-lives of PCBs[20]. A long-term study of the elimination of PCBs among contaminated eels revealed that tetra to penta PCBs had half-lives ranging from 340-1450 days and PCBs with hexa or higher chlorination did not reveal any measurable half-lives. That study further exemplified the persistence of highly chlorinated chemicals since mono, di and tri PCBs were not even found in the eels[20].

Although PCBs with octanol water partitions betweens 5 and 7 bioaccumulate and cannot be readily excreted, some fish possess the ability to biotransform the PCBs into products that are more easily metabolized and excreted[20]. By the mechanisms of the enzyme cytocrome P-450, the structure and number of chlorines on the PCB can be altered. Two forms of CYP-450 have been related to fish metabolism, 450 1A and 450 2B[21]. A recent study in the Great Lakes found elevated levels of hydroxylated PCBs among fish sampled. It was hypothesized that the hydroxylated PCBs were a product of biotransformation due to CYP-450[21]. PCBs becomes a hydroxylated during a CYP450 Phase I biotransformation by inserting an OH- onto a phenyl ring. The addition of the OH increases the water solubility of the PCB, in which Phase II of biotransformation can eliminate the PCB. To test this hypothesis, 85 nonexposed juvenile rainbow trout were fed a contaminated diet composed of Aroclors 1242, 1254, and 1260 for thirty days. After

30 days of the contaminated diet, the PCBs levels in trout were monitored for 160 days. Hydroyxlated PCBs were found among all the fish, suggesting that the Kow was not lowered enough for Phase II excretion, however served as proof that fish can biotransform PCBs. The most commonly transformed PCBs among fish with those with vicinal hydrogen atoms in the meta-para position. CYP-4502B has been linked to be effective among PCBs with meta-para vicinal H atoms[21]. This study not only proved the formation of hydroxylated PCBs by fish, but scientists were also able to calculate depuration and biotransformation rates of specific PCBs in the trout which had never been done before.

Another study that explored biotransformation of PCBs looked into the ability of deepwater sculpin to form methylsulfonyl-PCBs, MeSO2-PCBs[22]. Deepwater sculpin are apart of a trout diet. The formation of MeSO2-PCBs has also been correlated to CYP2B, by insertion of the methylsulfonyl at a meta-para vicinal H location. PCBs levels and changes in PCBs congeners of the sculpin were compared against bloater chub, both are benthic species and have similar diets. The bloater chub had a significantly higher level of PCB burden than the sculpin, 12,800 $\pm$ 4040 ng/g vs. 4890 $\pm$ 1490 ng/g of lipid[22]. The PCB pattern in the sculpin was significantly depleted compared to the bloater chub, exemplifying MeSO2 PCBs and lower levels of most congeners. Congeners 101, 149, and 174 were significantly depleted via methysulfonyl biotransformation and it was estimated that sculpin could reduce their PCB burden up to 10%. MeSO2 PCBs were not detected in sediment samples, bloated chubs, or any of the food that the sculpin eat, proving that deepwater sculpin have advanced metabolic capabilities[22]. This study highlights that PCB metabolism potential is unique to most fish species, and once again is congener specific. It is a positive lead towards PCB accumulation of larger fish species such as trout since they eat sculpin and perhaps are accumulating less PCBs, however, trout have a vast diet.

As previously proven to be important in PCB accumulation and degradation in air, water and sediment, the number of chlorines in the meta, para, and ortho positions will also play a role in determining bioaccumulation rates and CYP450 metabolism within fish[7, 21, 22]. With the understandings of how PCBs can potentially be metabolized by CYP-450 and the octanol water partition as a strong predictor, it is important to question the role of the structure of the PCB in regards to fish metabolism. By being able to distinguish individual congeners that can and cannot be excreted, the PCB burden fish pass onto humans can be identified.

Many studies have suggested that PCBs without vicinal Hydrogen atoms at the meta-para position or ortho-meta position are most persistent in fish and among other organisms because they cannot be metabolized or excreted[7, 23]. Congener 153 is an example of a PCB without vicinal H atoms that is strongly biomagnified throughout[7]. A study conducted by Kannan, Reusch, Schulz-Bull, Petrick, & Duinker tested PCB metabolism among a marine food chain by separating PCBs into four groups based chlorine positions[23]. The study took place in the Baltic Sea in 1990 and sampled PCB concentrations in water, diatoms, copepods, mussels, shrimp, worms, flounders, herrings, and porpoises. The PCBs groups were established by the following criteria; Group 1:

congeners that did not have ortho-meta or meta-para vicinal H atoms; Group 2: congeners that have a meta-para vicinal H atoms; Group 3: congeners that have ortho-meta vicinal H atoms; Group 4: congeners that have both ortho-meta and meta-para vicinal H atoms[23]. Group 1 congeners were not metabolized and persistent among all the organisms sampled. Group 2 congeners were less persistent and suggested metabolism from cytochrome P-450 2B. Larger mammals such as the porpoise have the 2B enzyme that makes this metabolism possible. However, there was little metabolism of Group 2 congeners done by the fish species in the study. Group 3 congeners were also less persistent than group 1 and suggest metabolism from Cytchrome P-450 1A[23]. Kannan et al. and Metcalfe & Metcalfe believe that fish and other marine mammals efficiently use this enzyme for metabolism[23, 7]. With the knowledge that Group 3 congeners are successfully metabolized by fish species and other marine life, this information can be used to back track congener patterns in fish to an original Aroclor mixture. Group 3 congeners may also be considered to have under gone dechlorination while in sediment before reaching the fish. Although anaerobic bacterial dechlorination is unable to remove ortho chlorines, they could have removed the meta chlorine if the congener never possessed an ortho chlorine in its original form15. This exemplifies the importance of studying the trophodynamics of the marine food web. Lastly, Group 4 congeners were the least persistent PCBs due to the combined metabolic efforts of CYP1A and CYP2B[23]. These congeners will be the fastest to be removed from the St. Lawrence food web and can be factored back in when testing for an Aroclor mixture. Sediment dechlorination and metabolism work together for PCB elimination by anaerobic bacteria dechlorinating PCB congeners through removal of meta and para chlorines. The removal of the meta and para chlorines creates opportunities for fish to metabolize those PCBs[15, 23].

Kannan and et al.'s effort of establishing a pattern of metabolism along a food chain provides a good preliminary strategy to analyze the St. Lawrence food web data[23]. The data is further analyzed by factoring in the number of ortho positioned chlorines in congeners to metabolism rates. This classified PCBs into three groups, PCBs with >2 ortho chlorines and no vicinal H atoms, congeners with >2 ortho chlorines and vicinal H atoms at either meta-para or ortho-meta, and lastly, congeners with < 2 ortho chlorines and vicinal H atoms[23]. When the data was classified and plotted by this strategy, the results were similar to the original 4 groups' results. As expected, congeners without vicinal H atoms and 2+ ortho chlorines were not metabolized and persistent. The second group of PCBs with vicinal H atoms and 2+ ortho chlorines were moderately metabolized[23]. However, an interesting observation is that the second group congeners metabolized more efficiently with a decreasing number of ortho-chlorines. This observation indicates that even though the results did not change significantly by factoring in the number of ortho chlorines, it successfully can put congeners into order of efficient metabolism based upon its group (out of original 4) and the number of ortho-chlorines it has. This observation also correlates with anaerobic bacteria's inability to remove ortho chlorines[15]. Ortho chlorines play an essential role in establishing which PCB congeners tend to be persistent. When analyzing PCB congener data and seeking results that indicate metabolism, grouping congeners by vicinal H atom positions and then by number of ortho chlorines, may be the most effective way.

Metcalfe and Metcalfe's study separated PCBs into 5 groups when analyzing their data[7]. The 5 groups are similar to Kannan's 4 groups, only Metcalfe & Metcalfe factored in the number of ortho-chlorines present when considering meta-para or ortho-meta vicinal H atoms[7, 23]. Group 1 included congeners without any vicinal H atoms[7]. Group 2 is congeners with ortho-meta vicinal H atoms and 2+ ortho chlorines. Group 3 is also congeners with ortho-meta vicinal H atoms, but those with less than 1 ortho chlorine. Group 4 is congeners with meta-para vicinal H atoms and less than 2 ortho chlorines. Group 5 is also congeners with meta-para vicinal H atoms, but those with 3+ ortho chlorines[7]. The metabolism of PCBs by this classification aligned with Kannan's results[7, 23]. Group 1 and 2 were not metabolized and were the most persistent. Although group 2 had ortho-meta vicinal H atoms and the potential to be metabolized by CYP1A, the enzymes inhibited by the high number of ortho-chlorines, further enforcing the influence of ortho-chlorines on metabolism. Group 3 were not persistent among marine species due to the ortho-meta vicinal H atoms that fish can metabolize. The persistence of Group 4 and 5 congeners varied among mammals and birds sampled, which also correlated with fish inability and mammals' ability to metabolize through CYP2B[7]. Among the congeners tested for, there is also relative pattern that as the congeners octanol water partition increased, metabolism decreased. This pattern of hydrophobia relating to metabolism further increases the depth in understanding PCB metabolism.

A study by Niimi & Oliver, sampled 8 small rainbow trout, 10 small coho salmon, 10 lake trout, 12 large rainbow trout, and 9 large coho salmon for PCB exposure[24]. 92 congeners were detected ranging from mono to deca. The ten most common congeners among all of the fish sampled ranged from penta to hepta homologs, the congeners being 84, 87+97,101, 110, 118, 138, 149, 153, and 180[24]. When applying the same five groups that Metcalfe & Metcalfe used in their study to the ten most dominant PCBs, the results were parallel[7, 24]. Of the ten congeners, two were from Group 1, one was group 5, and all of the others were either group 2 or 4. That in group 2 had several ortho chlorines which inhibits metabolism and those from group 4 reflect the limited ability of fish to conduct CYP 2B metabolism[24]. The study provided further evidence that the number of ortho chlorines present, the higher chlorination of the congener, and the meta-para vicinal hydrogen atoms all inhibit metabolism. Further, the results were similar among all the fish tested; showing that metabolism among fish may not be specie specific.

The results of Niimi & Oliver[24], Metcalfe & Metcalfe[7], Kannan et al.[23], conflict with the results from Stapleton et al.[22] and Buckman et al[21]. The former have parallel findings that CYP1A is an active enzyme that eliminates PCBs in fish[24, 7, 23]. However, the latter, which are more recent studies, suggested that CYP2B is an active enzyme is fish and can biotransform PCBs[21, 22]. Regardless of which is the more dominant enzyme, both work among PCBs that have vicinal H atoms. The mechanisms and potential for CYP450 1A and 2B metabolism among fish is a subject that needs further research.

Best approach in identifying fish metabolism should not be by segregating specific cytochrome P 450 enzymes abilities, but by following Metcalfe & Metcalfe groups[7]. By using a combination of vicinal hydrogen atom positions, the number of ortho-chlorines present, and the octanol water partition; a pattern of metabolism and persistence among PCB congeners in fish can be established for the St. Lawrence River fish of interest. By comparing the dominant persistent PCBs found in the fish of the studies discussed above and identifying the ones in St. Lawrence River fish, it can be shown what specific PCBs fish pass onto humans.

Based on models of bioaccumulation, it is estimated that lake trout retain 80% of the PCBs they receive via ingestion, and 50% for Coho salmon[20]. When PCB uptake via water column is factored in, the potential for metabolism seems less hopeful. Through uptake via water column and consumption of contaminated benthic species, fish are constantly being exposed and accumulating more PCBs. Ultimately, the rate of elimination and metabolism will never exceed rate of bioaccumulation[21]. Overall, metabolism rates are slow compared to the rates in which fish are exposed. Fish will continue to bioaccumulate PCBs and spread them to humans in our future. The need for fish testing and fishing advisories is just as important now as it was 10, 20, and 30 years ago.

The concern that fish are a main source of PCB contamination to humans stems from the higher rates of PCB bioaccumulation among fish than those of other food sources to humans and the environmental media that pose a threat to humans. The Mohawk culture relies on their land for food, which includes local contaminated fish. A study that took place in Akwesasne in 2004 tested local fish, soil, meat, ducks, fruits and vegetables for PCB levels[4]. The soil, meat, ducks, and produce did not exemplify unusual levels of PCBs unlike the fish. It is not surprising that these listed vectors do not show elevated levels of PCB contamination because they are not submerged in the St. Lawrence River Fish are constantly accumulating more PCBs through uptake in the water column and eating contaminated food[9]. A multivariate model demonstrated the contributing role of each food vector to the overall PCB concentration in Mohawk serum, only fish showed statistical significance to contributing the PCB burden[4]. Of the eight dominant congeners that were identified among the Mohawk women sampled and none were relevant to PCB patterns in the soil, meat, duck and produce; however, three of the congeners were directly associated with PCB exposure via fish consumption. These eight congeners will be used in comparison to the dominant congeners found in St. Lawrence River fish.

Toxicity:

The position and number of ortho-chlorines is not only significant when determining metabolism patterns among food webs, but also serves as an indicator of toxicity. It is important to establish toxicity of PCBs along with metabolism patterns to be able to conclude if the persistent PCBs in the environment are also the most toxic. Metcalfe and Metcalfe, suggest that PCBs without any ortho positioned chlorines (non-ortho) or with only one ortho positioned chlorine (mono-ortho) are the most toxic PCBs because of their similarities to tetrachloro-dibenzodioxin (TCDD)[7]. TCDD is a by-product of industrial waste and is a known environmental contaminant that has toxic effects on humans such as embryo toxicity[2, 7]. The Toxic Equivalency Factors (TEF) calculates the toxicity of TCDD along with four mono-ortho and eight non-ortho chlorine substituted ‘dioxin like’ PCBs[25]. Of those PCBs on the TEF chart, PCB 126 is one-tenth the toxicity of TCDD, making it to most toxic PCB listed[25]. Metcalfe and Metcalfe included four out of the eight mono-ortho PCBs and all four of the non ortho PCBs listed with TEFs in their study as apart of Group 3, except CB 169 was in Group 1[7, 25]. Fortunately, for Metcalfe and Metcalfe’s study, most Group 3 congeners, including the 8 mono- and non- ortho toxic PCB congeners that were included were all detected at low levels within the environment, especially in higher trophic levels[7]. If these congeners mentioned are really the most toxic yet least persistent, the St. Lawrence River data sets should exemplify low levels of these congeners due to the inability of sediment dechlorination to remove ortho chlorines and the ability of fish to metabolize non- and mono- ortho chlorine congeners[7, 15]. The majority of persistent PCB congeners should consist of those that are highly chlorinated and contain 2 or more ortho chlorines.

Recent studies have looked into the toxicity and health effects of highly chlorinated and ortho rich congeners in comparison to dioxin like congeners. Ortho rich congeners appear to be the most persistent in the environment and among biota. A study conducted in Germany in 2006 compared the presence of dioxin like congeners to non-dioxin like congeners (those with 2 or more ortho substituted chlorines) in the formation of hepatic neoplasms and in tumors promotion[26]. An issue the study mentioned is that human exposure to PCBs always occurs in mixtures such as Aroclors. These mixtures contain both non-dioxin like and dioxin like and it is difficult to distinguish individual congeners or group of congeners responsible for an adverse health effect when all are present. The study found that it was not the concentration of a PCB mixture that increased the chances for developing neoplasms, but the concentration of total dioxin-like PCBs in the dose that increased development[26]. When non-dioxin like PCBs were increased and dioxin like PCBs were decreased, there were no significant changes in health and the exposed rates resembled the control. The study also tested the onset of liver tumors in exposed rodents. Once again, the conclusions aligned with dioxin-like PCBs being more potent in causing adverse health effects[26]. These findings agreed with Metcalfe and Metcalfe's statement that mono-ortho and non-ortho are the most toxic PCBs[7].

However, these results have proven to be inconsistent among other studies. Another study testing the onset of liver tumor formation in rats had extremely different results[27]. Groups of rats were exposed to Aroclor 1260 (a mixture of highly chlorinated congeners), congeners only with one or none ortho chlorines, congeners with two to four ortho chlorines, and a mixture of congeners with zero to four ortho chlorines[27]. All of the exposed groups of rats showed an increased in the promotion of liver tumors except the group exposed to congeners with only one or zero ortho chlorines. It was concluded that 80% of tumor promotion among rats exposed to A1260 is from non-dioxin like PCBs, those with 2 or more ortho chlorines[27].

Other studies with conflicting results to Knerr and Schrenk include two recent studies led by Alexey Goncharov[26, 28, 29]. The most recent study published in 2011, sought to establish the relationship between PCB exposure and elevated blood pressure[28]. PCB serum concentration was determined to be a determinant of high blood pressure. When a multiple linear regression model was applied, the most significant relationship between both elevated systolic and diastolic blood pressure was from PCBs with three and four ortho chlorines. There was also no evidence that dioxin like PCBs cause high blood pressure[28].

In 2009, Goncharov published a study testing the low testosterone levels among men who are exposed to PCBs[29]. The study identified four congeners that had a significant inverse relationship to testosterone levels, PCBs 74, 99, 153, and 206. Congener 74 is the only dioxin like congener with one ortho chlorine. Congeners 99 and 153 have two ortho chlorines and PCB 206 has three[29]. Even though 3 out of the 4 congeners identified are not dioxin-like, the roll of dioxin like congeners in affecting reproductive health should not be ruled out due to the presence of congeners 74[29]. This study supports that PCBs definitely cause adverse health effects and highlights that both dioxin like and non dioxin like congeners are responsible.

One other recent study analyzed which PCB congeners decrease cognitive function among adolescents[30]. The study grouped PCBs by dioxin like, non dioxin like (with multiple ortho substituted chlorines), persistent and non persistent. With a decrease in long term memory, all four groups were found to have a significant effect. In long term retrieval and delayed recall, once again all four groups had a significant effect, but PCBs with multiple ortho chlorines had a greater significant effect, dioxin like p= 0.04 vs. non dioxin like p= 0.01[30].

The conflicting results among studies in regards to which PCBs are the most toxic suggests that more research is necessary to identify specific groups of PCBs or specific congeners responsible for certain health effects. Regardless of conflicting results, all the studies showed that PCB exposure is a direct cause of multiple health problems including elevated blood pressure, decreased cognitive development, and the formation of tumors[28, 30, 27]. However, the congeners that will persist in the environment and among biota including humans are those that are not dioxin like, with two of more ortho chlorines and high chlorination. It is important to recognize that even tough toxic equivalency factors are not calculated for non-dioxin like PCBs, these PCBs still have proven toxic effects as mentioned in studies discussed above.

St. Lawrence River Fish Data Analysis:

In order to draw conclusions from the St. Lawrence River's fish PCB data, factors of vicinal hydrogen atom positions, the number of ortho-chlorines present, and the octanol water partitions of the dominant PCBs among the fish must be considered. By identifying dominant persistent congeners among the fish, metabolism and sedimentation of the congeners can be applied to establish an original Aroclor mixture. Understanding the St. Lawrence River environment's ability of altering PCBs allows scientists to predict PCB remediation timelines. Most importantly, identifying dominant persistent congeners in the fish can be applied to human PCB data to understand the PCB burden humans receive from eating fish.

There were three data sets available, each containing multiple fish samples from the St. Lawrence River[31, 32, 33]. Fish were tested for all 209 congeners and all detectable congeners were listed in data sets. The first data set taken in an unknown year tested twelve shiners, twenty one yellow perches, five bullheads, and six small mouth basses in various contaminated parts of the river[31]. Of the twelve shiners, the average PCB concentration was .46298ppm. Only one shiner was over the US FDA's recommended level of 2ppm for fish with a PCB concentration of 3.05278[31, 6]. In the most contaminated shiner, thirteen congeners dominated the fish, contributing 21% of the fish's overall contamination. Those thirteen congeners being 4+10, 52+69+73, 66+76+98+80+93+95+102+88. Of the twenty one perch tested, none exceeded 2ppm, the average PCB concentration was .39341[31]. The most contaminated perch had a PCB concentration of 1.34382 and was also dominated by thirteen congeners. Congeners 52+69+73, 66+76+98+80+93+95+102, 84+92+155 composed of 17% of the overall PCB burden of the fish, ten of which were also dominating congeners in the shiner. Among the bullhead samples, no fish was close to exceeding the FDA limit with an average PCB concentration of .16984. Of the dominating bullhead congeners, seven out of the twelve were dominant in the shiner and perch. Lastly, of the small mouth bass sampled, the average PCB concentration was 1.3583. One of the six fish exceeded recommended PCB levels with a concentration of 6.00189. All ten of the dominant congeners in the contaminated bass were dominant in the shiner and the bullhead[31].

From the first data set, eleven dominant congeners were identified from all the fish sampled[31]. The eleven congeners were uniformly present among all four species. The eleven dominant congeners are 52+69+73, 66+76+98+80+93+95+102+88. All but two, CB's 80 and 76, have more than two ortho chlorines. The eleven congeners have octanol water partitions ranging from 5.84 to 6.48, proving that PCBs without Kow below 5 will not accumulate[31, 34, 20]. The congeners were evenly distributed between Metcalfe & Metcalfe groups 2 to 5 [31, 7]. Only one congener was a group 1 PCB. The lack of group 1 congeners without any vicinal H atoms suggests that the dominant PCBs in the fish have undergone both CYP 2A and 1B metabolism in which they have been dechlorinated. There were a total of nine meta-para vinical H atoms and six ortho-meta vicinal H atoms among the dominant congeners[31]. The presence of both vicinal pairs does not allow a conclusion of which CYP450 is more efficient at metabolizing PCBs, instead suggests that both are occurring. When applying Bedard and Quensen's processes of sedimentation, several of the dominant PCBs have been identified as dechlorination products. Through processes H, H', N, and P, parent congeners 99, 101, 130, 149, 153, 171, and 180 were identified[15]. The Aroclor mixtures that those parent congeners typically dominate are A1254 and A1260[35].

The second data set was taken in 2005 [32]. The two species of fish were identified; perch and bullhead, exemplified significantly higher levels of PCB concentrations that greatly exceeded the FDA's recommended 2ppm. Of the three perch samples, the average PCB concentration was 7.779ppm, with the highest contamination of the three being 9.6914ppm. Fifteen congeners dominated the most contaminated perch, composing of 46% of the fish's overall PCB burden. Of those fifteen congeners, six congeners, 28, 47, 49, 52, 59, and 66, further dominated the PBC burden of the fish by composing of 23% of the concentration[32]. The three bullhead fish sampled averaged 10.196 ppm, with the most contaminated fish having a concentration of 16.529ppm. The most contaminated fish was dominated by the same 6 congeners in the perch, which comprised of 28% of the overall PCB concentration of the fish[32]. The dominant congeners were parallel among the two different species, suggesting that metabolism does not vary among species.

The six dominant congeners among the 2005 data had Kow ranging from 5.67 to 6.2, once again all in the range for accumulation[32, 34]. The congeners were in Metcalfe groups 2, 3, 4, half having meta-para vicinal H atoms and the other half with ortho-meta H atoms[7, 32]. The data did not lean towards CYP 450 1A or 2B metabolism, instead the presence of multiple ortho positions chlorines and high Kow seemed to be the dominating factors in the PCBs persistence. When Bedard and Quensen's sedimentation processes were applied, parent congeners 66, 99, 101, 105, 138, and 153 were identified from processes M, N, H, and H'[15, 32]. The parent congeners are all found in significant amounts of all three Aroclor mixtures: 1248, 1254, and 1260[35].

The data set from 2007 is the most recent sampling of fish from the St. Lawrence River near Akwesasne[33]. The fish are not identified by species, but of the fifteen sampled, the average PCB contamination was only .60024, significantly less than the 2005 data. Of the fifteen fish sampled, none exceeded the FDA PCB limit; the highest contaminated fish only had a PCB concentration of 1.736 [6, 33]. The dominating congeners in the most contaminated sampled were significantly more chlorinated than the previous two data sets. The ten dominant congeners were 90, 101, 110, 118, 138, 153, 163, 164, 180, and 187 [33]; all have multiple ortho-positioned chlorines and Kows ranging from 6.36 to 7.36 [34]. Six out of the ten 2007 dominant congeners were also identified in the Niimi and Oliver study to be dominant congeners in fish[24, 33].

Of the 2007 ten dominant fish congeners, five of the congeners are in group 1 and have no vicinal H pairs[33, 7]. There was only one congener with meta-para vicinal h atoms and four had ortho-meta pairs. This suggests that CYP450 2B had metabolized all congeners with meta-para vicinal h atoms, and the presence of multiple ortho positioned chlorine inhibited CYP 4501A. Four out of the ten dominant congeners in the 2007 data are predicted parent compounds of the dechlorinated dominant congeners in the 2005 data and undated data[31, 32, 33]. The 2007 data suggests the congeners that fish will ultimately accumulate the greatest as time goes by. The lighter chlorinated congeners in the other two data sets were not group 1 congeners, but instead congeners with vicinal H atoms that had further potential to be metabolized. The dominant 2007 congeners are all found in heavier Aroclor mixtures 1254 and mainly 1260[35].

Conclusions:

The three data sets are missing information, therefore this analysis has limitations. Information such as species, size of fish, and location where fish is caught is missing. The location is essential in determining where contamination is the greatest. It is proven to be difficult to identify the original Aroclor mixtures. Most identified dominant congeners and their parent congeners are present in all three Aroclor mixtures of interest, 1248, 1254, and 1260. There is also a great variation of PCB concentration levels among all data sets, making it difficult to assess to actual PCB burden in the fish of the St. Lawrence River. However, the conclusions that are able to be drawn can still prove to be useful in understanding PCB alterations over time and identifying congeners in humans. All of the dominant congeners identified among the three data sets aligned with previously discussed studies on PCB alterations from sedimentation and metabolism. The dominant congeners in the undated data and the 2005 data all exhibited sedimentation processes and metabolism via both CYP 450 1A and 2B.

The data sets exemplified that as time passes, ortho-dominated PCBs and those with a Kow greater than 5 will accumulate. Mono and non-ortho PCBs are believed to be the most toxic to humans, yet they are not accumulating in fish or humans. It is suggested that research should begin to study the health effects of highly chlorinated PCBs that are persistent, since those are the PCBs accumulating over time.

In 2004 study that tested Mohawk women's blood, eight dominant congeners were identified: 74, 99, 118, 138, 153, 180, 181, and 187 [4]. Five of the dominant congeners in the women, 118, 138, 153, 180, and 187, are all dominant congeners among the 2007 fish data[4, 33]. Further, congeners 138, 153, and 180 were identified as parent congeners to dominant congeners for in the 2005 data and undated data[31, 32]. The Mohawk study had also tested local duck, meat, and produce; none of which had unusual levels of PCB exposure or PCB patterns similar to the human blood serum[4]. The PCBS identified as dominant persistent congeners in fish correlates with dominant persistent PCBs in human serum, suggesting that humans are receiving an observable proportion of PCB exposure from fish consumption.

The dominant congeners in the 2007 data were parallel to previously identified dominant congeners in studies measuring the PCBs in fish. The dominant congeners in the 2005 and undated data sets can also be traced to parent congeners that are consistent with previous studies and the 2007 data. The collection of the twenty-seven identified dominant congeners among the fish in this study can serve as indications of PCB exposure from fish consumption in humans. These dominant PCBs in fish are not similar to the lightly chlorinated congeners that humans accumulate via air exposure, eliminating the possibility that humans who exhibit these congeners received them from sources other than fish. Other food sources to the Mohawks, meat, duck, fruits and vegetables, were eliminated as routes of exposure by the 2004 Mohawk study. Therefore, the presence of the 27 congeners suggested indicates PCB exposure by fish consumption in the Mohawks.

Discussion:

Although the 2007 data of St. Lawrence River fish does not show levels of PCB concentrations above the FDA's recommended amount, the alarming high levels of PCBs in the 2005 data calls for more testing of the fish in several locations adjacent to Akwesasne. In 2000, the EPA currently published "Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories [36]." The cancer risk level at 1 in 100,000 was calculated for consuming fish contaminated with PCBs. It is only recommended to eat an unrestricted amount of fish when the PCB concentration of the fish ranges from 0-0.0015ppm. No fish meals should be eaten if the PCB concentration is greater than .094ppm due to the increased risk of cancer. The EPA recommended PCB concentrations differ greatly from the FDA's which serves as a guideline to NYS fishing advisories[36].

In order to confirm the dominant congeners in fish are also the dominant congeners among the Mohawk people, another sampling of Mohawk serum is necessary along with surveying Mohawks on their fish consumption. By having current data on fish consumption, PCBs in fish, and PCBs in the Mohawks, a clear understanding of the PCB burden fish pass onto humans can be established. By testing fish in several locations of the river, areas of severe contamination can also be identified. The Mohawk culture relies on land and the water for their resources. Until current PCB levels in their local fish are established, education of the Mohawks to avoid local fish and turn to other local products such as meat and poultry should be continued.

Measures have been taken to provide the Mohawks along with New York State anglers with information about the safety on consuming fish from contaminated waterways, such as the St. Lawrence River. Every year, the New York State Department of Environmental Conservation (NYS DEC) collects over 2000 fish in NYS waters and tests them for various contaminants and heavy metals[5]. After the DEC finished testing the fish, the NYS Department of Health (DOH) decides if fishing advisories are necessary for certain bodies of water. The St. Lawrence fishing advisory states not to consume any fish from the Bay and Cove east of South Channel Bridge, exactly where the Akwesasne territory is located. By the NYS DOH and DEC, all local fish to Akwesasne are unsafe to date[5].

However, on the New York State Department of Environmental Conservation's website 'Fishing in the St. Lawrence River'[37], states under the section of 'Eating Your Fish' that:

> "Some fish species from the St. Lawrence River contain levels of chemical contaminants above the limits established by the U.S. Food and Drug Administration. While the effects on humans caused by eating these contaminated fish are not certain, as a safeguard, the NYS Department of Health has issued an advisory with recommendations for limiting fish consumption. This advisory is found in the regulations guide issued with your fishing license. We suggest you read the advisory and plan your fish consumption accordingly." [37]

Upsettingly, this is a current website. The DEC explicitly stating that the effects of the contaminants in the fish of the St. Lawrence River are not certain is misleading to the public. In regards to the effects of PCBs, the United States Environmental Protection Agency states on their site 'Polychlorinated Biphenyls: Basic Information' that[38]:

> "PCBs have been demonstrated to cause cancer, as well as a variety of other adverse health effects on the immune system, reproductive system, nervous system, and endocrine system." [38]

If fishing advisories are enacted to protect people from eating hazardous fish, how can anglers and the Mohawks protect themselves if NYS regards the effects of contaminants such as PCBs as uncertain, when the federal government recognizes them as carcinogens[37, 38]? This once again exemplifies the inconsistencies between governmental agencies. New York State fishing advisories are determined based upon the FDA's 2ppm and states the health effects of the chemicals are uncertain, where the EPA states PCBs are known carcinogens and fish with concentrations greater then .094ppm should not be eaten[36]. A consensus that puts the health of the people of New York and the United States needs to be made.

A new study testing the PCB contamination of the St. Lawrence River fish and of the Mohawk people will take years to complete. However, in the mean time, NYS fishing advisories should be clarified, stating the real harm that consuming fish contaminated with PCBs can impede on humans. The limits stated by the EPA should also be considered by NYS DEC and DOH. Programs educating the Mohawks on the details of their local fishing advisories should also be implemented so the Mohawks can make informed fishing decisions.

Unfortunately, due to the persistent nature of PCBs within the environment and biota, the St. Lawrence River will exhibit elevated levels of PCBs for decades, perhaps centuries to come. As grim as the contamination seems, there is a glimmer of hope for the fish in the St. Lawrence River. Current research in the Hudson River is finding that species of the Atlantic tomcod have evolved to resist PCB toxicity[39]. When comparing contaminated Atlantic tomcod to tomcods from clean water, the Hudson River fish have developed a genetic mutation that resists the binding of PCBs to specific transcription factor proteins; resulting in an overall reduction of the toxic effects of PCBs[39]. The evolution of the Hudson River fish offers hope that the St. Lawrence River fish can also evolve to resist PCBs. Without toxic PCBs accumulating in the St. Lawrence River fish, perhaps in the future the Mohawks can return to fishing their local waters to feed their people.

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