POLYCYCLIC AROMATIC HYDROCARBONS (PAHS), NITRO-PAHS AND PETROLEUM BIOMARKERS IN LAKE MICHIGAN

by

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Dedication

To my family and friends.

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Abstract

The overall objective of this research was to understand the distribution, sources and risks of PAHs and NPAHs in Lake Michigan, to characterize their emissions from a major source (diesel exhaust), and to model and predict their environmental fate in the Lake Michigan basin. The petroleum biomarkers hopanes and steranes were characterized along with PAHs and NPAHs to provide more information on hydrocarbon contamination.

Several types of samples were collected in Lake Michigan, including bottom sediments and fish. Homogenized composite samples were analyzed. Ecological and human health risks were evaluated by the toxic equivalency approach and sediment quality guidelines. Emissions of target compounds from diesel exhaust were characterized to examine the effects of fuel type, engine load and after-treatment. Finally, fugacity-based multimedia models were used to evaluate the overall behaviors of PAHs and NPAHs in Lake Michigan.

In summary, the results suggest the ubiquitous distribution of target compounds in sediments and fish from Lake Michigan. PAHs seem to display "biodilution" through the aquatic biota. Sediment concentrations show a spatial trend with high concentrations near-shore and low concentrations in the middle of the lake. Decrease of PAHs in Lake Michigan sediments from the 1990s is also suggested. Source apportionment identifies vehicle exhaust as the most important PAH source in this region, and bench tests demonstrate the effectiveness of alternative fuels and after-treatment in reducing the emissions. Finally, the modeling results suggest that the environmental concentrations and emission rates of PAHs and NPAHs can be estimated mutually by simple multimedia models.

This study provides new information regarding levels and risks of PAHs and NPAHs in the Great Lakes basin, especially the first report of NPAHs in sediment and biota, which can help target these chemicals for pollution prevention and reduction. This work obtains baseline data for trending progress towards elimination of toxic substances in the basin. It can also deepen our understanding to their behaviors in aquatic biota, and provide valuable information for fish advisories. Moreover, the modeling helps to improve understanding of the overall behavior of these compounds, and the estimated emission rates can complement and improve existing emission inventories.

Chapter 1 Introduction

1.1 Overview

The presence of persistent toxic substances in the Great Lakes has been of concern for many decades. These substances, which include polychlorinated biphenyls (PCBs), organochlorine pesticides, and polycyclic aromatic hydrocarbons (PAHs), enter the Great Lakes mainly from atmospheric deposition where they can persist in bottom sediments, be taken up by benthic organisms, and accumulated in aquatic wildlife (EPA 2012b). Many fish in the Great Lakes have high concentrations of persistent toxic contaminants, posing health risks to both humans and wildlife. In humans, exposure to persistent toxic substances has been linked to adverse health effects that include low birth weight, developmental problems in children, neurological problems, immune system disorders, and cancer (EPA 2012b). The United States Environmental Protection Agency (U.S. EPA) has been monitoring levels of persistent toxic substances in air and precipitation of the Great Lakes (EPA 2012b). In addition, the U.S. and Canada established the Great Lakes Binational Toxics Strategy in 2004 to control and eventually eliminate persistent toxic substances in the Great Lakes (EPA and EC 2004).

This dissertation focuses on Lake Michigan, the only Great Lake that is located entirely within the U.S. Lake Michigan has high concentrations of many contaminants, a result of historically large loadings from agricultural, municipal and industrial sources (Hickey et al. 2006). This dissertation focuses on four classes of chemicals, including polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs, hopanes and steranes. PAHs include a number of carcinogens (ATSDR 1995; Eisler 1987). Nitro-PAHs, the nitrated derivatives of PAHs, are potentially more toxic than PAHs (Tokiwa et al. 1987). Hopanes and steranes, known collectively as petroleum biomarkers, are potentially valuable tracers of hydrocarbon contaminations. Using new measurements, this research examines the distribution of PAHs, nitro-PAHs, hopanes and steranes in sediments and fish of Lake Michigan, identifies major sources contributing to sediment concentrations, characterizes their emissions from one important source - diesel engine exhaust, and evaluates their environmental fate using multimedia models.

This chapter is organized into five sections. Section 1.1 (this section) gives an overview of the study area, persistent toxic substances, and the research needs. Section 1.2 provides background information on the target compounds and reviews their physiochemical properties, sources, environmental fate, bioaccumulation and toxicity; a review of the contamination in Lake Michigan and data gaps follows; this section also discusses methods of source apportionment and multimedia modeling. Section 1.3 describes the overall objective and specific aims, and Section 1.4 presents the organization structure of this dissertation. Tables/figures and references are presented in Sections 1.5 and 1.6, respectively.

1.2 Background

1.2.1 Target compounds

The target contaminants examined in this research include four classes of semivolatile organic compounds (SVOCs). An SVOC is an organic compound that has a boiling point higher than water and which may vaporize when exposed to temperatures above room temperature (EPA 2013). The first class is PAHs, which are a group of persistent organic compounds that are ubiquitous in the environment (Eisler 1987). PAHs consist of hydrogen and carbon arranged in the form of two or more fused benzene rings (Eisler 1987). There are thousands of PAH compounds, each differing in the number and position of aromatic rings, but historically, analyses have focused on a relatively small subset of PAHs. U.S. EPA listed 16 PAHs as priority pollutants (CFR 1982), among which benzo[a]pyrene (BAP) is the most widely studied compound. This research includes these 16 priority PAHs as listed in Table 1.1.

Nitro-PAHs (NPAHs) are nitrated derivatives of PAHs. They can have stronger carcinogenic and mutagenic activity than the parent PAHs. Unlike PAHs, NPAHs have not been prioritized. This dissertation focuses on 11 NPAHs, listed in Table 1.1, that have been frequently detected in airborne particulate matter (PM) (Albinet et al. 2007; de Castro Vasconcellos et al. 2008), diesel exhaust PM (Liu et al. 2010; Khalek et al. 2011), and sediments (MDH 2011; Lübcke-von Varel et al. 2012; Sato et al. 1985).

Hopanes and steranes are two additional classes of SVOCs that are derived from cell membranes of prokaryotes (Ourisson and Rohmer 1992) and eukaryotes (Mackenzie et al. 1982), respectively. They are common constituents of crude oil (Manan et al. 2011) and derived products such as engine lubricating oil (Rogge et al. 1993). There is a wide variety of different

stereoisomers of these compounds. This research focuses on five hopanes and six steranes, listed in Table 1.1, that are most frequently found at relatively high concentrations in sediments and diesel exhaust PM samples (Boitsov et al. 2011; Liu et al. 2010; Khalek et al. 2011; Qu et al. 2007; Schauer et al. 2002, 1999).

1.2.1.1 Physical and chemical properties

Physical and chemical characteristics of PAHs can vary considerably. With increasing molecular weight, melting point, boiling point, and log *Kow* tend to increase, while water solubility and vapor pressure decrease. For example, considering the 16 target PAHs, the boiling point, water solubility, vapor pressure and log *Kow* range from 218 to 536 °C, from 0.00019 to 31 mg/L, from 1.33×10^{-8} to 11.3 Pa, and from 3.3 to 6.63, respectively (all at 25 °C and standard atmospheric pressure)(EPA 2012a). This large range of properties means that PAHs can vary substantially in their behavior and distribution in the environment, and in their biological effects (Eisler 1987). Generally, however, PAHs have low water solubility, high *Kow*, low volatility and Henry's Law constant, and moderate-to-high chemical stability. The physical and chemical properties of NPAHs are similar, but they generally have lower volatility and lower water solubility than their parent PAHs (EPA 2012a).

Hopanes and steranes generally have high boiling points (around 400 °C), very low water solubility (1×10^{-7} to 1×10^{-5} mg/L), low vapor pressure (1×10^{-5} to 1×10^{-3} Pa), and extremely high log *Kow* (9 to 11) (EPA 2012a). These compounds are highly stable in the environment, i.e., they are resistant to chemical, photochemical and microbial degradation (Manan et al. 2011; Neff and Durell 2012).

1.2.1.2 Sources, transport and fate

PAHs are released to the environment through natural and anthropogenic sources; the latter provides much higher emissions (Crane et al. 2010). There are three major types of PAH sources: pyrogenic PAHs are emitted during incomplete burning of coal, oil, gas, coke, wood, garbage, or other organic matters; petrogenic PAHs form in the earth by geological processes at low temperature, over long time periods and possibly at high pressures, and include crude oil, coal, coal tar pitch, asphalt or asphalt sealant, and tire particles; lastly, diagenetic PAHs found in recent sediments are derived from biogenic precursors like plant terpenes (Crane et al. 2010). Previous apportionment studies examining airborne and sediment PAHs in Chicago and Lake

Michigan have identified major sources as coke ovens, vehicle emissions and wood burning (Christensen et al. 1999; Li et al. 2003; Simcik et al. 1999). Most PAHs are pyrogenic and are subsequently released into the atmosphere, adsorbed on particulate matter, and then deposited on terrestrial surface or water bodies (Baek et al. 1991; Helfrich and Armstrong 1986; Neff 1979).

The distribution of PAHs differs by compounds. For example, BAP partitions mainly into soil (82%) and sediment (17%), while about 1% partitions into water and less than 1% into air, suspended sediment and biota (Hattemer-Frey and Travis 1991).

PAHs can undergo photo-oxidation and biodegradation in the environment (Suess 1976). However, PAHs in aquatic sediments degrade very slowly given the absence of radiation and oxygen (Suess 1976), and may persist indefinitely in oxygen-poor basins or in anoxic sediments (Neff 1979).

NPAHs arise mainly from two sources: (1) direct emissions from incomplete combustions of organic matter such as coal, oil, gas, coke and wood; and (2) gas-phase atmospheric reactions between PAHs and nitrogen oxides (Perrini et al. 2005). Atmospheric formation of NPAHs is initiated by OH radicals during daytime, and by NO_3 after sunset (Perrini et al. 2005; Albinet et al. 2007). Most airborne NPAHs are believed to be secondary pollutants formed by atmospheric reactions (Yaffe et al. 2001). Like PAHs, airborne NPAHs are mostly released or formed in the atmosphere, associated with particulate matter, and then deposited on land or water surfaces (Yaffe et al. 2001). There is also evidence of endogenous NPAH production in fish (tilapia) facilitated by nitrite (NO_2^-), which potentiates the mutagenicity of a noncarcinogenic PAH (phenanthrene) (Shailaja et al. 2006). The distribution of NPAHs depends on physical and chemical properties. For example, 1-nitropyrene mostly partitions into soil (99.2%), while 1-nitronaphthalene partitions mainly into air-gas phase (30.6%) and soil (68.9%) (Yaffe et al. 2001). Like PAHs, NPAHs also can undergo photo-degradation (Fan et al. 1996) and biodegradation (Heitkamp et al. 1991) in the environment.

Hopanes are pentacyclic triterpenoids derived from cell membranes of prokaryotes (bacteria) (Ourisson and Rohmer 1992), while steranes are derived from the sterols of cell membranes of eukaryotes, mainly algae and higher plants (Mackenzie et al. 1982). Hopanes and steranes arise from petrogenic or biogenic sources. Petrogenic hopanes and steranes are derived from bacteria, algae and higher plants in ancient times, undergo various transformation and

rearrangement during geological processes, and finally become constituents of crude oil (Peters et al. 2007). In contrast, biogenic hopanes and steranes arise from the decomposition of bacteria, algae and vascular plants during recent times (Qu et al. 2007; Xiong et al. 2010). Petrogenic and biogenic hopanes and steranes generally have distinct structures or configurations (Qu et al. 2007; Boitsov et al. 2011).

Hopanes and steranes are highly resistant to chemical, photochemical and microbial degradation (Manan et al. 2011; Neff and Durell 2012), so they have been used as signature or marker compounds to help identify sources of organic matters in lake sediments (Meyers and Ishiwatari 1993; Qu et al. 2007; Xiong et al. 2010) and the extent of biodegradation (Prince et al. 1994). They also have been used as tracers of vehicle exhaust in the atmosphere because they appear specific to the engine lubricating oil used in diesel and gasoline engines (Kleeman et al. 2008; Schauer et al. 2002, 1999).

1.2.1.3 Bioaccumulation in aquatic food web

PAHs in bottom sediments can be taken up by benthic organisms. Many aquatic invertebrates (clams, mollusks, crustaceans, etc.) cannot efficiently metabolize PAHs (Hahn et al. 1994; Varanasi et al. 1985) and sediment-associated PAHs can be accumulated in bottom-dwelling invertebrates and fish in the Great Lakes (Bruner et al. 1994; Eadie et al. 1982b; Eadie et al. 1982a; Levengood and Schaeffer 2011). Bioconcentration factors (BCFs) for aquatic biota, which represent the ratio of tissue to the water concentrations, range from roughly 10^1 to 10^4 for many PAHs (Eisler 1987). However, most PAHs are rapidly metabolized in fish because they possess the Ah receptor and sufficient cytochrome P450 (Livingstone 1998; Hahn et al. 1994), so PAHs generally show little tendency of biomagnifications in aquatic food webs (Eisler 1987).

Very little information is available regarding bioaccumulation of NPAHs in aquatic organisms. Only one recent study reported NPAHs at ppt to ppb levels in marine bivalves at Osaka Bay, Japan, which were much lower than PAH concentrations in bivalves (Uno et al. 2011). Like PAHs, NPAHs can induce cytochrome P450 activity in fish (Jung et al. 2001). NPAHs are metabolized in fish by nitro-reduction followed by acylation (Kitamura and Tatsumi 1996).

Hopanes and steranes have been measured in aquatic organisms to assess oil pollution (Manan et al. 2011; Neff and Durell 2012). Bioaccumulation has been reported in aquatic

invertebrates (amphipods and bivalves) (Neff and Durell 2012) and fish (Manan et al. 2011), with concentrations in the ppb levels, one or two orders of magnitude lower than PAH levels (Manan et al. 2011; Neff and Durell 2012).

1.2.1.4 Toxicity, health effects and exposure guidelines

Certain PAHs are potent carcinogens and systemic toxicants with wide-ranging effects in humans, non-human mammals, birds, invertebrates, plants, amphibians, and fish. Several PAHs and NPAHs are mutagens and carcinogens based on microbial mutagenicity bioassays and a forward mutation human assay (Fu et al. 1985; Sakai et al. 1985; Watanabe et al. 1995). The mechanism of toxic action is that PAHs and NPAHs form intercalation compounds with DNA molecules, which can affect DNA replication, recombination and repair, ultimately leading to DNA damage (Tokiwa et al. 1987). While PAHs are indirect mutagens which require metabolic activation, NPAHs are direct-acting mutagens. Many NPAHs have stronger carcinogenic and mutagenic activity than PAHs. For example, the mutagenic activity in *Salmonellaty phimurzum* TA98 of 1,8-dinitropyrene is three orders of magnitude higher than benzo[a]pyrene's, considered one of the most toxic PAHs (Tokiwa et al. 1987). Therefore, although concentrations are generally lower than PAHs, NPAHs may be toxicologically more important (Murakami et al. 2008). Toxicity information for hopanes and steranes is unavailable. Since these compounds are derived from cell membranes, however, it is likely that their toxicity is low.

PAHs can exert photo-induced toxicity in aquatic organisms when PAHs in biological tissues are exposed to UV radiation in sunlight (Arfsten et al. 1996). Phototoxic effects that have been observed in laboratory animals include acute skin reactions, enhancement of UV-induced carcinogenesis, and death (Arfsten et al. 1996). The mode of action is that a PAH molecule absorbs UV light, and then transfers the energy to an oxygen molecule, which creates an oxygen radical that can cause cell damage at respiratory surfaces (McDonald and Chapman 2002). Not all PAHs are phototoxic. Phototoxic compounds include anthracene, fluoranthene, pyrene and benzo[a]pyrene (Arfsten et al. 1996). Phototoxicity of one NPAH, 1-nitropyrene, has also been observed (Arfsten et al. 1996). Although phototoxicity of PAHs have been clearly demonstrated in laboratory studies, its ecological relevance remains uncertain (McDonald and Chapman 2002). Organisms have various protective mechanisms, e.g., behaviors and genetic adaptions, that can prevent exposure to sunlight or PAHs (McDonald and Chapman 2002).

Exposure limits have been proposed by U.S. EPA and other agencies for several PAHs, and limits or guidelines exist for domestic water, fish consumption, and air concentrations (ATSDR 1995). For the 16 EPA priority PAHs, consensus-based sediment quality guidelines (SQGs) are available for the protection of benthic-dwelling organisms (WDNR 2003). PAH concentrations in engine exhaust are unregulated (Khalek et al. 2011). No regulatory limits or guidelines exist for NPAHs.

Since PAHs and NPAHs almost always occur as mixtures in the environment, U.S. EPA has used the toxic equivalency (TEQ) approach for many years to account for mixture exposures and assess the carcinogenic risks to humans (Schoeny and Poirier 1993). In this approach, each PAH or NPAH is assigned a toxic equivalent factor (TEF, unitless) based on its relative carcinogenic potency compared to BAP (Table 1.1). B[a]P has a TEF of 1. Then the TEQ_{BAP} for the mixture is calculated as

$$TEQ_{BAP} = \sum_i (C_i \times TEF_i) \quad (1.1)$$

where Ci is the concentration of each PAH and NPAH in the mixture.

1.2.2 Status of Lake Michigan

The Great Lakes ecosystem is particularly vulnerable to contamination given the numerous urban and industrial discharges in the region, the Lakes' large surface area that increases loadings via atmospheric deposition (Simcik et al. 1999), and the long hydrologic retention times (De Vault et al. 1996). As noted, Lake Michigan has high levels of many contaminants given the historically large loadings from atmospheric deposition, urban runoff and municipal/industrial effluents due to the urban and industrial centers surrounding its southern portion (Hickey et al. 2006; Helfrich and Armstrong 1986). The Lake also receives inputs from petroleum spills, particularly since Indiana Harbor, Indiana and Chicago, Illinois are major distribution centers for petroleum products (Helfrich and Armstrong 1986). Coal-tar pavement sealant may be another important source of PAHs to Lake Michigan given its use in central and eastern U.S. cities (Van Metre and Mahler 2010).

Although PAH levels in air and precipitation of Lake Michigan have been monitored extensively by the Integrated Atmospheric Deposition Network (IADN) (Sun et al. 2006a; Sun et al. 2006b), studies on PAH levels in sediment and biota are limited. In addition, sediment and

biota in Lake Michigan have not been characterized for PAHs since the 1990s (Su et al. 1998; Zabik et al. 1996). Newer information is needed to assess the contaminant trends. No information is available regarding nitro-PAHs, hopanes and steranes in the sediment and biota of Lake Michigan; such data are extremely limited across the world.

1.2.3 Source identification and apportionment methods

Identification of the major sources of the target compounds can help understand the origin of these compounds and target those sources for emission reduction. Several approaches have been used for identifying and apportioning sources of PAHs, NPAHs and petroleum biomarkers in ambient air and sediments (Albinet et al. 2007; Boitsov et al. 2011; Bzdusek et al. 2004; Li et al. 2003; Qu et al. 2007; Tang et al. 2005; Yunker et al. 2002). These approaches can be grouped as diagnostic ratios and receptor models. Both approaches are based on uniqueness and stability of patterns of PAH, NPAH and petroleum biomarker emissions from different sources.

1.2.3.1 Diagnostic ratios

Ratios between concentrations of pairs of compounds that have unique values for each type of source can serve as a "fingerprint" to identify sources. Such diagnostic ratios are semi-quantitative, that is, they can identify the major sources, but they cannot apportion the contribution from each source. For example, an ANT/(ANT+PHE) ratio < 0.10 usually indicates petroleum-derived PAHs, while a ratio > 0.10 indicates a dominance of combustion-derived PAHs (Yunker et al. 2002) (Abbreviations of compounds are given in Table 1.1). For PAHs, the ratio of BAA/CHR has been used to identify urban influences (Gschwend and Hites 1981; Helfrich and Armstrong 1986); ratios of PHE/ANT, FLA/PYR, ANT / (ANT + PHE), FLA / (FLA + PYR), BAA / (BAA + CHR) and IcdP / (IcdP + BghiP) are commonly used to distinguish between high temperature combustion and low temperature petroleum sources (Gschwend and Hites 1981; Yunker et al. 2002; Budzinski et al. 1997). For NPAHs, the ratio of 2-NFLA/1-NPYR has been used to evaluate the contributions of primary (direct emissions) and secondary sources (formation in gas-phase atmospheric reactions) (Albinet et al. 2007). The 2-NFLA/2-NPYR concentration ratio has been used to distinguish between OH radical and NO_3 initiated oxidation pathways of NPAH formation (Albinet et al. 2007). The 1-NPYR/PYR ratio is an indicator of diesel-engine vehicles and coal combustion (Tang et al. 2005). For petroleum

biomarkers, several thermal maturity indicators can be used to distinguish between petrogenic and biogenic origins of hydrocarbons in sediments (Boitsov et al. 2011; Qu et al. 2007; Hostettler et al. 1992).

1.2.3.2 Receptor modeling

Receptor models utilize chemical measurements at a monitoring site (the receptor) and statistical techniques such as regression and factor analysis to calculate the relative contributions from major sources to the pollution at that site (EPA 2011). Receptor modeling is quantitative, because it can not only identify the major sources, but also apportion the relative contribution from each source. Receptor modeling has been applied using PAHs. Because source profiles (the pattern of emissions from each source) for NPAHs and petroleum biomarkers are mostly unavailable, receptor modeling has not been used for these compounds.

Receptor modeling is based on the assumption of mass conservation. The concentration of chemical species i at the receptor is assumed to be a linear combination of contributions from various sources (j = 1…m). The general equation is

$$\boldsymbol{C = AS + E} \tag{1.2}$$

where C is a n×1 vector of concentrations of chemical species i measured at a receptor site with $1 \leq i \leq n$; A is n×m source composition matrix of n compounds for each of the m sources modeled; S is a m×1 vector of the source contribution factor; and E is a n×1 error vector. (Li et al. 2003)

Eq. (1.2) may be solved using several techniques. This dissertation focuses on one of the most commonly used techniques, the chemical mass balance (CMB) approach, which is described below.

Chemical mass balance (CMB) methods

The CMB model requires source profiles (the mass fraction of a chemical in the emissions from each source type, i.e., matrix A) and ambient data (concentrations measured at the receptor) (EPA 2004). All source profiles must be known, as obtained using measurements or the literature. CMB methods also require that (1) the number of sources (m) must be less than or equal to the number of chemical species (n); (2) the composition of source emissions is consistent over the period of receptor and source sampling; (3) the chemical species do not react with each other, i.e., they add linearly; (4) all possible sources are identified and source profiles

are known; (5) the compositions of different sources are linearly independent of each other; and (6) measurement uncertainties are random, uncorrelated, and normally distributed (Li et al. 2003). CMB typically uses a least squares solution to the set of linear equations represented by eq. (1), that is, minimizing the sum of squares for error to obtain the source contribution factor Sj for each of the m sources (Li et al. 2003).

CMB models have been used extensively to apportion airborne pollutants including metals, SVOCs and VOCs. Applications in aquatic environments are not common, although CMB models have been used to apportion PAHs in Lake Michigan sediments (Christensen et al. 1999; Li et al. 2003) and sediments in other lakes (Van Metre and Mahler 2010).

Limitations of CMB methods include difficulties in identifying all possible sources and obtaining all source profiles. Also, source emissions and the composition of emissions can vary considerably over time (i.e., the source profile is not consistent). The emission mix has clearly changed, e.g., over the past century or two, emissions have shifted from wood to coal to petroleum (Christensen et al. 1999). Pertinent to this research are recent (2007) emission controls on diesel engines that significantly reduced PM and PAH emissions from newer vehicles (Liu et al. 2010). Moreover, PAHs and other compounds can undergo degradation and partitioning during environmental transport, which violates the assumption that profiles remain constant from source to receptor (Li et al. 2003; Galarneau 2008).

1.2.4 Multimedia fate models: the fugacity approach

Multimedia fate models use physical-chemical properties, reactivity, and transport characteristics to describe a comprehensive picture of a chemical's environmental behavior, based on the chemical's emission rates into the environment (Mackay and Paterson 1991). A widely used type of multimedia model is based on the concept of fugacity (Mackay 2010). Briefly, fugacity (*f*) describes an "escaping" tendency of a chemical, which has units of pressure (Pascal). It can be considered as the partial pressure of a chemical in a phase, and it is logarithmically related to chemical potential. Chemical potential, also known as partial molar free energy, is a form of potential energy that can be absorbed or released during a chemical reaction or a phase transition. Fugacity is an equilibrium criterion of chemical partitioning, that is, if a chemical partitions between two phases, it seeks to establish an equal fugacity in both phases (Mackay 2010).

Fugacity models are mass balance models in which the environment is modeled as several compartments. Each compartment is assumed to be homogeneous, or well-mixed (Mackay 2010). There are four levels of fugacity models. The most widely used are level III and level IV models. Level III is a steady-state model with emissions into the system, advective flows coming in and out of the system, reactions within compartments, and intermedia transport between compartments; the chemical is not in equilibrium between compartments. Level IV is a dynamic level III model, that is, the system is not in steady-state (Mackay 2010). These and other fugacity models can be used to estimate chemical concentrations in different environmental compartments under steady-and unsteady-state conditions (Mackay and Paterson 1991). They are particularly valuable in identifying key environmental processes and showing which environmental and chemical properties are the most important determinants of fate (Mackay and Hickie 2000). The fugacity-based models have been applied a wide variety of mostly persistent contaminants, including PCBs, PBDEs, pesticides and PAHs (Lim and Lastoskie 2011; Mackay and Hickie 2000; Mackay and Paterson 1991).

1.3 Research objectives

The overall objectives of this study are to understand the sources, distribution and risks of PAHs and NPAHs in Lake Michigan, to characterize emissions of these contaminants in diesel exhaust as a major potential source, and to model and predict the environmental fate of these contaminants. Petroleum biomarkers hopanes and steranes are also studied, but are not the main focus of this dissertation. The research has five specific objectives: the first two relate to PAHs and NPAHs in fish and sediment from Lake Michigan; the next two are concerned with diesel exhaust emissions; and the fifth is related to multimedia modeling. The following expands on these objectives and states its importance.

Objective 1: Characterize the distribution of PAHs and NPAHs (plus hopanes and steranes) **in predator fish from Lake Michigan, and estimate the carcinogenic risk from the consumption of these fish**. This information will help to understand the behavior of target SVOCs in top predator fish, including the effects of location, season and gender. The results can also be compared to PAH and NPAH levels in lower-trophic-level organisms (as reported in the literature) to assess biomagnification across the aquatic food web. The risk estimates can inform policy and risk communication regarding fish advisories and other health-related actions.

Objective 2: Characterize bottom sediments from Lake Michigan for PAH and NPAH (plus hopanes and steranes) **levels, identify the major sources, and apportion sources**. This objective is aimed at providing information that fills a substantial knowledge gap regarding the distribution of these contaminants, particularly for NPAHs, in Lake Michigan sediments. Since sediment represents the largest reservoir of SVOC contamination in the Great Lakes, sediment analyses are highly relevant to the restoration of the Great Lakes. The distribution of the target PAH and NPAH contaminants will provide information to help characterize governing transport and fate processes. PAH levels will be compared to literature data to evaluate temporal trends. In addition, the NPAH measurements can serve as baseline data for future studies examining trends, while the hopane and sterane data can help to identify sources of hydrocarbon contamination. The source apportionments will identify the major sources of PAHs and NPAHs, which could then be targeted for emission reduction.

Objective 3: Analyze PAH and NPAH (plus hopanes and steranes) **emissions from current and next generation diesel engines** (which differ in exhaust after-treatment technology and fuel). Diesel engine exhaust represents an important source of these contaminants, and vehicle emissions previously has been identified as one of the major sources of PAHs in Lake Michigan (Christensen and Arora 2007; Christensen et al. 1999; Li et al. 2003). Work in this objective will provide useful data on NPAH along with PAH emissions in diesel exhaust, and an understanding of the effects of new after-treatment technologies and fuels, in particularly, biodiesel fuels and particulate traps. Widespread use of these technologies and fuels will decrease emissions, reduce atmospheric deposition into Lake Michigan, and ultimately lower concentrations in the Great Lakes ecosystem.

Objective 4: Examine the integrity of PAHs and NPAHs (plus hopanes and steranes) **during filter processing and storage,** which is needed to analyze the particulate samples collected from diesel exhaust. This objective is a supplement to Objective 3. Filter processing and storage involve multiple, sequential and complex steps, during which target SVOCs can volatize, decompose, or transform. Thus, sample integrity must be characterized and maintained to obtain quantitative measurements. The results have important implications on SVOC sampling and analysis protocols, which should utilize stringent criteria and performance checks to limit possible biases occurring during filter and extract processing and storage.

Objective 5: Use multimedia models to predict the environmental fate of PAHs and NPAHs in Lake Michigan, and to estimate emission rates of these contaminants. Emission and process data is essential for understanding the overall behavior of PAHs and NPAHs in the Lake Michigan basin (including distribution, transport and fate in different environmental compartments) and in the Lake Michigan aquatic food web (incorporating bioaccumulation, trophic transfer, biomagnifciation, and other processes). A comparison between predicted and measured concentrations is used to illustrate model strengths and highlight potential issues, e.g., estimating model parameters. The model also is used in an inverse fashion to estimate emission rates of PAHs and NPAHs based on fitting concentrations measured in different environmental media (e.g., fish and sediment). This information can be used to evaluate and improve current and historical emission inventories of PAH and NPAH emissions.

1.4 Organization of this dissertation

This dissertation is organized into eight chapters. Chapter 1 (this chapter) has summarized the background, literature findings, objectives of this research and importance of each objective. Chapters 2 to 6 pertain to each specific objective described in Section 1.3. Chapter 2 presents the concentrations of target compounds in Lake Michigan lake trout, evaluates the effects of site, season, gender and trophic level, and estimates the associated human cancer risk. Chapter 3 presents the concentrations of target compounds in Lake Michigan sediments, evaluates their spatial distribution patterns, assesses the temporal trend of PAHs, and identifies/apportions major sources of these compounds. Chapter 4 investigates the emissions of target compounds from a major source – diesel engine exhaust -- and then examines the effects of fuel type, engine load and after-treatment. Chapter 5 investigates the effects of filter conditioning, filter storage and extract storage on the integrity of target compounds in diesel exhaust PM samples. Chapter 6 uses fugacity-based multimedia models to predict the environmental fate and to estimate the emission rates of PAHs and NPAHs in the Lake Michigan basin. The findings of several chapters have been published in peer-review journals, which are indicated in the footnote of those chapters. Finally, Chapter 7 integrates the major findings of the individual chapters, highlights the significance of this research, discusses the limitations, and provides suggestions for further studies.

1.5 Tables and figures

Table 1.1 List of target compounds

Group	Compound	Abbrev.	CAS #	MW g/mol	# of rings	TEF (new)	LogKow[e]
PAHs	Naphthalene	NAP	91-20-3	128	2	0.001 [b]	3.30 *
	Acenaphthylene	ACY	208-96-8	152	3	0.001 [b]	3.94 *
	Acenaphthene	ACE	83-32-9	154	3	0.001 [b]	3.92 *
	Fluorene	FLU	86-73-7	166	3	0.001 [b]	4.18 *
	Phenanthrene	PHE	85-01-8	178	3	0 [a]	4.46 *
	Anthracene	ANT	120-12-7	178	3	0 [a]	4.45 *
	Fluoranthene	FLA	206-44-0	202	4	0.08 [a]	5.16 *
	Pyrene	PYR	129-00-0	202	4	0 [a]	4.88 *
	Benzo[a]anthracene	BAA	56-55-3	228	4	0.2 [a]	5.76 *
	Chrysene	CHR	218-01-9	228	4	0.1 [a]	5.81 *
	Benzo[b]fluoranthene	BBF	205-99-2	252	5	0.8 [a]	5.78 *
	Benzo[k]fluoranthene	BKF	207-08-9	252	5	0.03 [a]	6.11 *
	Benzo[a]pyrene	BAP	50-32-8	252	5	1 [a]	6.13 *
	Dibenzo[a,h]anthracene	DBA	53-70-3	278	5	10 [a]	6.54 *
	Indeno[1,2,3-cd]pyrene	IcdP	193-39-5	276	6	0.07 [a]	6.70
	Benzo[g,h,i]perylene	BghiP	191-24-2	276	6	0.009 [a]	6.63 *
NPAHs	1-Nitronaphthalene	1-NNAP	86-57-7	173	2	n/a [d]	3.19 *
	2-Nitronaphthalene	2-NNAP	581-89-5	173	2	n/a [d]	3.24 *
	2-Nitrobiphenyl	2-NBPL	86-00-0	199	2	n/a [d]	3.57
	3-Nitrobiphenyl	3-NBPL	2113-58-8	199	2	n/a [d]	3.87 *
	4-Nitrobiphenyl	4-NBPL	92-93-3	199	2	n/a [d]	3.82 *
	5-Nitroacenaphthene	5-NACT	602-87-9	199	3	0.03 [c]	3.82 *
	2-Nitrofluorene	2-NFLU	607-57-8	211	3	0.01 [c]	3.37 *
	9-Nitroanthracene	9-NANT	602-60-8	223	3	n/a [d]	4.78 *
	9-Nitrophenanthrene	9-NPHE	954-46-1	223	3	n/a [d]	4.16
	1-Nitropyrene	1-NPYR	5522-43-0	247	4	0.1 [c]	5.06 *
	6-Nitrochrysene	6-NCHR	7496-02-8	273	4	10 [c]	5.34
Hopanes	17α(H),21β(H)-Hopane	Hop1	471-62-5	413	5	n/a [d]	10.78
	17α(H)-22,29,30-Trisnorhopane	Hop2	53584-59-1	371	5		9.45
	17α(H),21β(H)-30-Norhopane	Hop3	53584-60-4	399	5		10.36
	22R-17α(H),21β(H)-Homohopane	Hop4	60305-22-8	427	5		11.27
	22S-17α(h),21β(h)-Homohopane	Hop5	60305-23-9	427	5		11.27
Steranes	20S-5α(H), 14α(H), 17α(H)-Cholestane	Ste1	41083-75-4	373	4	n/a [d]	n/a
	20R-5α(H), 14α(H), 17α(H)-Cholestane	Ste2	481-21-0	373	4		10.36
	20R-5α(H), 14β(H), 17β(H)-Cholestane	Ste3	69483-47-2	373	4		n/a
	20R-5α(H), 14β(H), 17β(H)-24-Methylcholestane	Ste4	71117-90-3	387	4		n/a
	20R-5α(H), 14α(H), 17α(H)-24-Ethylcholestane	Ste5	62446-14-4	401	4		n/a
	20R-5α(H), 14β(H), 17β(H)-24-Ethylcholestane	Ste6	71117-92-5	401	4		n/a

[a] (Nisbet and LaGoy 1992); [b] (EPA 2010); [c] (RIDEM 2008)

[d] No information available.

[e] Estimated by (EPA 2012a).Values with an asterisk are from experimental database.

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Chapter 2 Characterization of target compounds in Lake Michigan fish[1]

2.1 Abstract

This study examines concentrations and risks of polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs (NPAHs), steranes and hopanes in lake trout collected in Lake Michigan. A total of 74 fish were collected in two seasons at three offshore sites. Σ_9PAH concentrations in whole fish ranged from 223 to 1,704 pg/g wet weight (ww), and PAH concentrations and profiles were similar across season, site and gender. Σ_9NPAH concentrations ranged from 0.2 to 31 pg/g ww, and carcinogenic compounds, including 1-nitropyrene and 6-nitrochrysene, were detected. In fall, NPAH concentrations were low at the Illinois site (0.2 – 0.5 pg/g ww), and site profiles differed considerably; in spring, concentrations and profiles were similar across sites, possibly reflecting changes in fish behavior. In fall, Σ_5Sterane and Σ_2Hopane levels reached 808 and 141 pg/g ww, respectively, but concentrations in spring were ten times lower. Concentrations in eggs (fall only) were in the same order of magnitude as those in whole fish. These results demonstrate the presence of target SVOCs in a top predator fish, and are consistent with PAH "biodilution" observed previously. Using the available toxicity information for PAHs and NPAHs, the expected cancer risk from consumption of lake trout sampled are low. However, it is notable that NPAHs contributed a significant portion of the TEQs in some samples. This study provides the first measurements of NPAHs in freshwater fish, and results suggest that additional assessment is warranted.

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2.2 Introduction

The presence of semivolatile organic compounds (SVOCs) in the Great Lakes has been a concern for decades. One class of SVOCs, polycyclic aromatic hydrocarbons (PAHs), includes a number of persistent and ubiquitous environment pollutants that are formed mainly through incomplete combustion and released into the atmosphere (Crane et al. 2010). Other PAH sources include petroleum and petroleum derived products, as well as diagenetic sources that are derived from biogenic precursors (Crane et al. 2010). These pollutants reach the aquatic environment through atmospheric deposition, urban runoff and municipal/industrial effluents, where they accumulate in bottom sediments and enter the aquatic food web. A number of PAH species are toxic to aquatic invertebrates and fish, potentially causing deformities, lesions, tumors, compromised immunity and death (Logan 2007). Accumulation of PAHs in sediments and bottom-dwelling invertebrates and fish has been documented in the Great Lakes (Eadie et al. 1982b; Levengood and Schaeffer 2011). Benthic fish in the Great Lakes region, such as brown bullhead (Yang and Baumann 2006), winter flounder (Koza et al. 1993), alewife and minnow (Levengood and Schaeffer 2011), have been studied for PAH contaminations, but few studies have examined top predator fish.

Nitrated PAHs, called nitro-PAHs (NPAHs), can have stronger carcinogenic and mutagenic activity than the parent PAHs. For example, the mutagenic activity in *Salmonella typhimurzum* TA98 of 1,8-dinitropyrene is three orders of magnitude higher than benzo[a]pyrene's, which is considered one of the most toxic PAHs (Tokiwa et al. 1987). NPAHs result from combustion-related emissions, as well as through transformations of atmospheric PAHs (Perrini et al. 2005). There is also evidence of endogenous production of mutagenic NPAHs in fish (tilapia) exposed to nitrite (NO_2-) and a noncarcinogenic PAH (phenanthrene) (Shailaja et al. 2006). As determined for other SVOCs like the PAHs (Simcik et al. 1996), atmospheric deposition is likely to be a major source of NPAHs in the Great Lakes. Information regarding bioaccumulation of NPAHs in aquatic organisms is very limited, although a recent study reported NPAHs at ppt to ppb levels in marine bivalves at Osaka Bay, Japan (Uno et al. 2011).

Hopanes and steranes are two additional classes of SVOCs. These materials are derived from the cell membranes of prokaryotes (Ourisson and Rohmer 1992) and eukaryotes (Ourisson

et al. 1987), respectively, and are constituents of crude oil (Manan et al. 2011). Both can enter the environment from petrogenic and pyrogenic sources (Manan et al. 2011). These compounds have been used as “markers” or "tracers" of vehicle exhaust because they are resistant to environmental degradation (Manan et al. 2011; Neff and Durell 2012), and because they appear specific to diesel and gasoline engine lubricating oils (Kleeman et al. 2008). Toxicity information for hopanes and steranes is unavailable. Bioaccumulation of these compounds has been reported in marine amphipods, bivalves (Neff and Durell 2012) and fish (Manan et al. 2011) and related to oil pollution, but concentrations in aquatic biota in the Great Lakes have not been reported.

The Great Lakes ecosystem is particularly vulnerable to contamination due to the numerous urban and industrial emission sources in the region, the Lakes’ large surface area that increases loadings via atmospheric deposition (Simcik et al. 1996), and the long hydrologic retention times. Lake Michigan has the highest contaminant levels of many contaminants, a result of historically large loadings from agricultural, municipal and industrial sources (Hickey et al. 2006; Chang et al. 2012). PAHs in the atmosphere and sediments of Lake Michigan have been extensively studied, and contributions from vehicle emissions and coal/coke oven emissions have been documented (Sun et al. 2006; Christensen and Arora 2007). However, little information exists regarding levels of PAHs in biota, especially fish. Lake trout (*Salvelinus namaycush*), a top predator fish, was extirpated from Lake Michigan in the 1950s, but stocked and rehabilitated since 1965 (Madenjian et al. 2002). Historically, this species has been used as an bioindicator species in the Great Lake Fish Monitoring and Surveillance Program (GLFMSP) for monitoring trends of PCBs, pesticides and mercury (Chang et al. 2012; Zananski et al. 2011), but PAHs, NPAHs, hopanes and steranes have not been monitored in this program.

This study characterizes concentrations and profiles of PAHs, NPAHs, hopanes and steranes in lake trout from Lake Michigan. We investigate possible differences among sampling sites, gender and season, and provide initial estimates of risks to human and fish health from these contaminants. To our knowledge, this is the first report of NPAHs in freshwater fish, and the first since the 1990s for PAHs in Lake Michigan lake trout (Zabik et al. 1996). The study is intended to provide new information regarding the concentration, distribution and risk of the target SVOCs in the aquatic biota of the Great Lakes.

2.3 Materials and methods

2.3.1 Fish collection and processing

Lake trout (*Salvelinus namaycush*) were collected at three offshore sites on Lake Michigan (Charlevoix, MI; Clay Banks, WI; Waukegan, IL; Figure 2.1) by personnel from the Michigan Department of Natural Resources (DNR), Illinois DNR and Wisconsin DNR. The fish collection was part of an annual Lake Michigan lakewide survey supported by the Great Lakes Fishery Commission to assess the progress toward rehabilitation of the lake trout population in Lake Michigan. The three sites were chosen for the present study to cover a north-south transect of Lake Michigan. Fish were collected by gillnet in fall (September – October) of 2011 and spring (April – May) of 2012 following well-defined protocols (De Vault et al. 1996). Individual fish were placed in plastic bags, frozen whole and shipped to the U.S. Geological Survey (USGS) Great Lakes Science Center (GLSC) in Ann Arbor, Michigan, where they were thawed, sexed, weighed, measured for total length, and homogenized individually in a Robot Coupe grinder. Eggs of female fish collected in the fall were removed prior to homogenization and analyzed separately. Subsamples of homogenized tissues were stored at -20 °C in solvent-washed glass jars with aluminum foil-lined screw caps until brought to the nearby University of Michigan School of Public Health for analysis. Whole fish (instead of fish fillets) were analyzed in order to be consistent with the protocol used by GLFMSP for PCBs and pesticides in Great Lakes lake trout (Chang et al. 2012; De Vault et al. 1996).

2.3.2 Materials

All solvents were HPLC grade and obtained from Fisher Scientific Inc.. Florisil (60-100 mesh) and sodium sulfate (anhydrous, certified ACS granular, 10-60 mesh) for column chromatography were supplied by the same vendor.

Calibration standards included a mixture of 16 PAHs (Sigma-Aldrich), a mixture of 8 NPAHs (Sigma-Aldrich), individual standards for 17α(H),21β(H)-hopane and 20S-5α(H),14α(H),17α(H)-cholestane (Chiron Laboratories), and SRM 2266 (National Institute of Standards and Technology). The SRM (standard reference material) 2266 is a solution of five hopanes and five steranes in iso-octane, which is intended primarily for use in the calibration of chromatographic instrumentation (NIST 2010). Fluoranthene-d10 (Cambridge Isotope

Laboratories Inc.) and an internal standard (IS) PAH mixture which includes anthracene-d10, benzo[a]pyrene-d12, chrysene-d12 and benzo[ghi]perylene-d12 (Wellington Laboratories), were used as ISs for PAH analyses. 1-Nitrofluoanthene-d9 (Cambridge Isotope Laboratories Inc.) was used as an IS for NPAH analyses. Lastly, n-tetracosane-d50 (Chiron Laboratories) was used as an IS for hopanes and steranes. Surrogate standards included C27-α,α,α-(20R)-cholestane-d2 (for hopanes and steranes), 1-nitropyrene-d9 (for NPAHs), chrysene-d12 and naphthalene-d8 (for PAHs) (Chiron Laboratories).

2.3.3 Sample preparation and chemical analysis

A 10-g subsample was taken from each homogenized sample, to which 15 µL of a surrogate standard (0.2 ng/µL of each compound) was added. The sample was dried with Na_2SO_4, extracted twice using dichloromethane/hexane (4:1, v/v), and sonicated for 30 min. Any fish tissue was separated from the extract by centrifugation and removed. The extract was passed through an activated Florisil column and fractionated into three portions: fraction A was eluted with 15 mL hexane; fraction B was eluted with 15 mL hexane/acetone (1:1, v/v); and fraction C was eluted with 30 mL methanol. Each fraction was then evaporated under a N_2 stream to 1mL. Fraction C was further cleaned to remove lipids by freezing at -79 °C for 5 h, and then separating and discarding the frozen lipid solids. Fractions A, B and C were analyzed for hopanes and steranes, PAHs, and NPAHs, respectively.

Target compounds were quantified using a gas chromatography-mass spectrometry (GC-MS; HP 6890/5973, Agilent Industries), an autosampler, and splitless 2 µL injections. Injector and detector temperatures were 275 ºC and 280 ºC, respectively. Separations used a capillary column (DB-5, 30 m x 0.25 mm id; film thickness 0.25 µm; J&W Scientific). The carrier gas was helium (1.5 mL/min, pressure of 37.4 kPa, average velocity of 31 cm/s), and the MS reagent gas was ultra high purity methane. The PAH analyses used a temperature program that started at 80 °C, then increased at 15 °C /min to 150 °C, then at 5 °C/min to 200 °C, and finally increased by 10 °C /min to 300 °C, which was held for 20 min, giving a total run time of 44.7 min. The MS detector was operated in electron impact (EI) mode. Scan mode was used to evaluate chromatography, and selective ion monitoring (SIM) mode was used for quantitative analysis with seven time windows (4, 5.8, 10, 14.25, 19, 22.2, and 25 min) and multiple ions. NPAH analyses used a temperature program that started at 40 °C held for 1.7 min, then increased by

25 °C/min to 150 °C and held for 10 min, increased by 10 °C/min to 220 °C and held for 10 min, and increased by 10 °C/min to 310 °C and maintained for 15 min giving a total run time of 57.1 min. The MSD was operated in negative chemical ionization (NCI) mode, again in scan mode to evaluate chromatography and in SIM mode for quantitative analysis (using ions 223, 247, and 297). Hopanes and steranes analyses used a third program: an initial oven temperature of 50 °C and no hold, ramping at 6 °C /min to 300 °C and holding for 10 min, giving a total run time of 41.7 min. Analyses used MSD-EI, again in scan mode to evaluate chromatography and in SIM mode for quantitative analysis (using ion 191 for hopanes and ions 217 and 218 for steranes). In each case, 15 µL of the IS (0.5 ng/µL of each compound) was added to each sample extract using a 25 µL syringe prior to GC-MS analysis.

2.3.4 Determination of lipid content

A 3-g subsample was taken from each homogenized sample, mixed with Na_2SO_4, and extracted twice by dichloromethane/hexane (4:1, v/v) using sonication. Fish tissue was separated from the extract by centrifugation and removed. Extract was dried under the fume hood and then weighed. The lipid content was calculated as the weight of the dried extract divided by the subsample weight.

2.3.5 Calibration and quality assurance (QA)

For calibration, each standard (mentioned in "Materials") was prepared at concentrations of 0.01, 0.05, 0.10, 0.50, and 1.00 ng/µL. All analytes were individually quantified against authentic standards.

QA measures included regular use of lab blanks, replicates, surrogate spike recovery tests and standard reference materials, specifically SRMs 1647e (Priority Pollutant PAHs in Acetonitrile), 2264 (Nitrated Aromatic Hydrocarbons in Methylene Chloride I) and 2266 (Hopanes and Steranes in, 2,2,4-Trimethylpentane) used for PAHs, NPAHs and hopanes/steranes, respectively. All SRMs were purchased from the National Institute of Standards and Technology (NIST). Replicates were performed for 10 whole fish samples and 2 egg samples. Measurement precisions, expressed as the average relative percent difference (RPD) across the 12 replicate measures and compounds in the group, were 25%, 31%, 19% and 12% for PAHs, NPAHs, steranes and hopanes, respectively. Target compounds were not detected in the blanks except for trace levels of naphthalene and phenanthrene. The spike

recovery of surrogate standards was 77-89% during the study, and the shift (abundance of target compounds in standard solutions before and after running a batch of samples) was within 20%.

2.3.6 Data analysis

Concentrations were calculated as pg per g wet weight (ww). Compounds with a detection frequency below 30% (Table A2.1) were excluded from the calculation of statistics (e.g., sums and relative abundances), following guidance for highly censored data (Antweiler and Taylor 2008). For compounds with a higher detection frequency, measurements below method detection limits (MDLs) were set to MDL/2. (MDLs are listed in Table A2.1). The relative abundance of each PAH compound was calculated as the concentration of each compound divided by the total concentration of PAHs with > 30% detection frequency (e.g., in whole fish, 9 PAHs were detected in over 30% of the samples, so the total concentration is denoted as Σ_9PAHs). The relative abundances of NPAHs, steranes and hopanes were similarly determined.

For statistical analyses, data were checked for normality using the Shapiro–Wilk test and for homogeneity of variances among groups using Levene's test. Group differences were tested using 1-way ANOVAs or t-tests if variables were normally distributed with equal variances. Non-parametric tests, including Kruskal-Wallis and Wilcoxon tests, were used to compare group means when data distributions were not normal or variances were not equal. Statistical analyses used SAS 9.3 (SAS Institute, Inc.).

For human health risks, the toxic equivalency (TEQ) for benzo[a]pyrene (BAP) was calculated for target compounds using toxic equivalent factors (TEFs, unitless) (Table A2.1) and the equation $TEQ_{BAP} = \Sigma_i (C_i \times TEF_i)$, where C_i is the concentration of the i^{th} PAH or NPAH in each sample (pg/g) (Levengood and Schaeffer 2011). The new TEF values (Table A2.1) provided in the 2010 EPA document (draft) (EPA 2010) were used for PAHs, and TEFs for 4 NPAHs were obtained from the Rhode Island Air Toxics guideline (RIDEM 2008). Two scenarios were considered: (1) the average scenario, including only the compounds with > 30% detection frequency, nondetect substituted by MDL/2; and (2) the worst-case scenario, including all target compounds, nondetect substituted by MDL/2. The excess lifetime cancer risk (dimensionless) was determined by multiplying the TEQ (BAP) by fish consumption rate (average 0.73 g/kg-day and high 2.2 g/kg-day) (EPA 2011) and the B[a]P oral cancer slope factor (7.3 per mg/kg-day) (EPA 2010). Since fish fillets were not analyzed, PAH concentrations

in fish muscles were estimated from the whole fish data using literature estimates of the tissue distribution of PAHs in fish and fish organ weights (Table A2.2).

2.4 Results and Discussion

2.4.1 Fish characteristics

Fish weights were similar across site and season, but females weighed more than males at sites 1 and 2 in fall 2011 (t-test; $p < 0.01$ at sites 1 and 2; $p = 0.60$ at site 3), and females remained slightly, but not significantly, heavier in spring 2012 (Table A2.3). The fall samples were collected just before spawning, and all but one female contained large amounts of eggs. Fish collected at site 3 were slightly but not significantly heavier than fish at other sites, and weights varied considerably (1,205 to 6,214 g), which likely masked differences due to gender.

The lipid content of the lake trout did not differ by gender, site or season (Table A2.3). In similarly sized lake trout (average of 639 mm in length) collected from Lake Ontario in 1986 (Madenjian et al. 2010), lipid content also did not vary by gender. However, lipid levels in lake trout collected in 1992 from Lake Michigan were considerably higher (17.9 ± 0.4%) (De Vault et al. 1996) than those in the present study (14.9 ± 3.1%). Fish in the present study may have had a low-lipid diet, e.g., abundant in relatively lean rainbow smelt and poor in fatty alewives (Madenjian et al. 2000). The decline in *Diporeia* abundance in Lake Michigan likely has lowered the lipid content of prey fish, leading to decreased lipid content of lake trout and other predatory salmonids (Madenjian et al. 2000). We did not observe the fall season decline in lipid content previously observed in Lake Michigan lake trout (Madenjian et al. 2000), possibly a result of dietary changes, the variation in the size of our fish (coefficient of variation in length = 8% versus 1.8% in the cited study), or sample size issues.

2.4.2 Whole fish: PAHs

Nine of the 16 target PAHs were found above MDLs in over 30% of the whole fish samples. Σ_9PAH concentrations averaged 546 ± 244 pg/g ww (n = 74) and varied over an 8-fold range among individual fish. Site means by season and gender ranged from 350 to 819 pg/g ww (Figure 2.2A), but differences between season, gender and site were insignificant or marginally significant, probably due to the large variation among individual fish and the relatively small sample size. Two- and three-ring compounds were most abundant, e.g., phenanthrene,

acenaphthylene, naphthalene and acenaphthene; abundances of four or five-ring compounds did not exceed 4% (Figure 2.3). PAH abundances did not vary by site, gender or season (Kruskal-Wallis and Wilcoxon tests). The fluoranthrene/pyrene ratio, an indicator of atmospheric transport distance that tends to increase at remote sites as pyrene undergoes more photo-oxidation (Zhang et al. 1993), was 6.3±10.3, 1.1±1.1, and 2.0±3.5 at the Charlevoix, Clay Banks and Waukegan sites, respectively. Differences between Charlevoix and Clay Banks sites (Mann-Whitney U test, $p = 0.001$) and between sites Charlevoix and Waukegan sites were significant (Mann-Whitney U test, $p = 0.004$), and reflect the proximity of the Clay Banks and Waukegan sites to major PAH sources near Green Bay and southwestern Lake Michigan, respectively (Figure 2.1).

A 1991 study of PAHs in Lake Michigan lake trout found Σ_{27}PAH levels of 1.52 ± 0.38 ng/g ww and slightly less, 1.47 ± 0.4 ng/g ww, for the 16 target PAHs in the present study (Zabik et al. 1996). The average PAH concentration in the present study is 63% lower. The lower concentrations can be explained by declining environmental levels, e.g., PAH concentrations and accumulation rates in Lake Michigan sediments have been falling since 1980 (Simcik et al. 1996; Huang et al. 2014), as have airborne concentrations in Chicago over the 1996 to 2004 period (Sun et al. 2006). It is also important to recognize that the Lake Michigan food web has changed considerably since 1990s, with the invasion of zebra and quagga mussels and the rapid decline of *Diporeia* (Nalepa et al. 2009; Nalepa et al. 2005). Zebra mussels can alter the contaminant cycling in Lake Michigan by bioconcentrating PAHs from the water column (Bruner et al. 1994); the mussels also cover large areas of the sediment surface which may reduce the amount of PAHs that reach the sediments. These factors may have decreased PAH concentrations in water and sediments, resulting in fewer PAHs entering the upper-trophic aquatic organisms. In addition, the fish in the earlier study were caught near-shore at Pentwater and close to two contaminated Areas of Concern (AOCs; White Lake and Muskegon Lake) that may have had higher PAH levels (Zabik et al. 1996). In contrast, fish in the present study were caught in open water areas distant from AOCs and other contaminant sources. ΣPAH concentrations in the Great Lakes food web generally decrease at higher trophic levels (Table 2.1). Roughly speaking, concentrations in aquatic invertebrates are about 100 to 1000 ng/g (Eadie et al. 1982a; Eadie et al. 1982b; Metcalfe et al. 1997), levels in bottom-feeding fish are 10 to 100 ng/g (Levengood and Schaeffer 2011; Baumann et al. 1991; Ridgway et al. 1999); and levels in top predator fish such as lake trout are 1 to 10 ng/g (Zabik et al. 1996). This trend is consistent with the “biodilution”

of PAHs observed in marine organisms (Takeuchi et al. 2009), and results from the rapid metabolism of PAHs by fish (Takeuchi et al. 2009) and the lack of effective oxidative enzyme systems in aquatic invertebrates (Hahn et al. 1994). While PAH concentrations in fish are low, PAH metabolites such as benzo[a]pyrene-7,8-dihydrodiol, 1-hydroxy benzo[a]pyrene, 3-hydroxy benzo[a]pyrene, 1-pyrenol, fluorenols, fluoranthenols, phenanthrols, and phenanthrene-9,10-diol, may persist in fish tissues (Varanasi and Stein 1991). The toxicological importance of these metabolites has been suggested by their association with hepatic lesions and liver neoplasms found in English sole from Puget Sound (Krahn et al. 1986).

The PAH concentrations in Lake Michigan lake trout in the present study (0.22 to 1.7 ng/g ww) are generally comparable to or lower than levels elsewhere. For example, concentrations of PAHs in western U.S. national park fish were very low and the values were not reported (Ackerman et al. 2008). The majority of fish samples collected in Mississippi Gulf Coast affected by the Deepwater Horizon oil spill did not exceed 10 ng/g ww (Xia et al. 2012). ΣPAH concentrations in commercial fish collected from the coastal waters of Madagascar following an oil spill ranged from 1.9 to 63 ng/g ww (Rumney et al. 2011). Pelagic (marbled flounder) and benthic fish (rockfish) in the west coast of Korea following an oil spill had Σ_{16}PAH concentration ranging from 9.6 to 22 ng/g dw (Jung et al. 2011). In constrast, ΣPAH concentrations in fish (brown ray, megrim and angler) from the Mediterranean Sea were higher, ranging from 210 to 227 ng/g ww (Storelli et al. 2013). The PAH concentrations in Lake Michigan lake trout were at the lower end of these observations, probably because there were few major oil spills in Lake Michigan, and since lake trout is at the very top of the aquatic food web. PAHs in sediments in open water regions of Lake Michigan are dominated by atmospheric deposition in the southern basin of the lake, which results in large part from combustion sources in the southwestern area (e.g., Chicago, Milwaukee and Gary); the waterborne contaminants are then transported and distributed throughout the lake (Simcik et al. 1996). As a result, sediments along a north-south transect of the open water showed fairly similar PAH concentrations and compositions (Simcik et al. 1996). Since tissue concentrations of PAHs in bottom prey fish are strongly related to sediment concentrations (Levengood and Schaeffer 2011), and lake trout feed mostly on bottom prey fish (Madenjian et al. 1998), similar PAH levels among the lake trout at the three sampling sites were expected. The similar PAH concentrations in fish collected in fall and spring seasons may reflect comparable atmospheric particle-phase PAH concentrations in

these seasons (Sun et al. 2006), or the significance of the PAH reservoir in the sediments, which changes concentration only very slowly.

Contaminant levels in fish reflect a balance between uptake from the water column, diet and sediments, and metabolism, partitioning and elimination. These processes can vary significantly by compound. For example, the predominance of phenanthrene reflects its high concentrations in water (Offenberg and Baker 2000), prey fish (Levengood and Schaeffer 2011) and sediments (Huang et al. 2014), as well as its relatively slow metabolism (or clearance) in fish (half-life of phenanthrene = 2.55 days; Table A2.1). The abundance of other 2- to 3- ring compounds, such as naphthalene and acenaphthylene, likely reflects uptake from the water via gills because of their greater concentration in water (Offenberg and Baker 2000) and the relatively slow metabolism (half-lives of 4.53 and 3.73 days, respectively). Although its concentration in water was also relatively high (Offenberg and Baker 2000), acenaphthene's fast metabolism in fish (half-life of 0.25 days) will lower levels in lake trout. The relatively low fluoranthene levels in lake trout compared to prey fish (Levengood and Schaeffer 2011) could result from its low bioavailability in the diet or low assimilation efficiency in lake trout. Despite pyrene's high concentrations in water (Offenberg and Baker 2000) and sediment (Huang et al. 2014), low levels in lake trout and other fish (Levengood and Schaeffer 2011) can be explained by rapid biotransformation (half-life of 0.56 days). The low levels of higher molecular weight (HMW) PAHs, e.g., benzo[a]pyrene and benzo[g,h,i]perylene, may reflect lower gill uptake and lower concentrations in water (Offenberg and Baker 2000). However, ingestion of sediments (bottom and /or suspended) can also contribute to PAH uptake. Although sediments were relatively abundant in HMW PAHs (Simcik et al. 1996), concentrations of these PAHs in fish may be low because of low gut assimilation efficiency and higher metabolism rates (e.g., short half-lives for benzo[a]pyrene and benzo[g,h,i]perylene, Table A2.1), which is also reflected in a small biota-sediment accumulation factor (BSAFs) at high *Kow* (Liang et al. 2007).

2.4.3 Whole fish: nitro-PAHs

Nine of the 11 target NPAHs were detected in at least 30% of the samples. Σ_9NPAH concentrations are presented in Figure 2.2B and Table A2.5. Σ_9NPAH concentrations in individual fish ranged from 0.2 to 31 pg/g ww, roughly 10 to 1000 times lower than Σ_{15}PAH concentrations. Like the PAHs, Σ_9NPAH concentrations did not differ by gender. Site differences

were seen only in fall 2011 when Σ_9NPAHs concentrations at Site Waukegan (IL) (0.39 ±0.10 pg/g ww) were lower than those at other sites. Seasonal differences were significant but inconsistent for all site-gender combinations excluding Charlevoix females and Clay Banks males. Σ_9NPAH and Σ_9PAH concentrations were not significantly correlated (Spearman r = -0.08, p = 0.499, N = 74).

The most abundant NPAH compounds were 3-nitrobiphenyl and 2-nitrobiphenyl, each contributing 14% and 23% (median) of the Σ_9NPAH, respectively (Figure A2.1). 1-Nitronaphthalene, 2-nitronaphthalene, 1-nitropyrene and 6-nitrochrysene had similar abundances. For most of the NPAH compounds, abundances varied by site in the fall, while abundances in the spring were similar (Kruskal-Wallis test) (Figure 2.4).

Several studies have reported NPAH concentrations in the low ng/g range in both freshwater and marine sediments around the world (Lübcke-von Varel et al. 2012; Ozaki et al. 2010), including our measurements in southern Lake Michigan sediments (Huang et al. 2014). However, information regarding NPAH concentrations in aquatic biota is very scarce. One study reported ΣNPAH concentrations from 380 to 4100 pg/g ww in mussels and from 430 to 4300 pg/g ww in oysters in Osaka Bay, Japan (Uno et al. 2011) (calculated from pg/g dry weight (dw) reported and a moisture content of 85% (He et al. 2002)), levels that were approximately 10 to 100 times lower than ΣPAH concentrations reported in those mussels and oysters (Uno et al. 2011). We found similar or greater ratios in lake trout. NPAH levels are anticipated to be low relative to PAH levels given the large differences in atmospheric levels (Albinet et al. 2007), engine exhaust (Liu et al. 2010), and sediments (Huang et al. 2014). However, NPAH concentrations can be altered due to reactions during atmospheric transport, abiotic loss in the water column, biodegradation in sediments, biotransformation in fish, fish behavior, and possibly endogenous formation. Thus, concentrations in fish will not be proportional to atmospheric concentrations or emission rates.

The low NPAH concentrations at the Waukegan site in the fall might have resulted from lake trout's homing behavior. Lake trout return to the same site during the fall spawning season, but occupy a larger area where fish from different spawning stocks will mix during the remainder of the year (Schmalz et al. 2002). In the fall samples, the lake trout collected at the Waukegan site likely belonged to a single local spawning stock given the proximity of Julian's

Reef, a major spawning site (Holey et al. 1995). In contrast, the spring sample probably reflected a mixed sample with individuals from other spawning stocks, resulting in similar average concentrations across the three sites. Homing behavior might also explain the similar NPAH profiles across sites in spring (Figure 2.4B) but not fall seasons (Figure 2.4A).

As noted earlier, the highly urbanized and industrial areas around Waukegan site near southwestern Lake Michigan contain many PAH and NPAH sources. While these emissions would be diluted during transport to sites Charlevoix and Clay Banks, additional NPAH formation is expected given the longer atmospheric transport time compared to site Waukegan. Unfortunately, atmospheric concentrations of NPAHs in the Great Lakes region have not been reported. At three sites in downtown and suburban Kanazawa, Japan, NPAH levels were higher in winter than summer; patterns were inconsistent in fall and spring (Hayakawa et al. 2002). In Los Angeles, California, atmospheric NPAH levels were higher in summer (Reisen and Arey 2005). These studies may have only limited relevance to the Great Lakes region. In addition, nearby sources and other factors may affect local water, sediment and food concentrations.

NPAH concentrations in lake trout result from atmospheric levels and deposition, levels accumulated in sediment, and uptake, metabolism and elimination processes. Information regarding NPAHs in sediments is limited, but includes a report for marine sediments in Japan (Ozaki et al. 2010) and our (yet unpublished) results for southern Lake Michigan (Huang et al. 2014). Abundances of individual NPAHs in lake trout are compared to those in biota, sediment, ambient air and diesel engine exhaust reported in the literature (Figure 2.5). Abundances of 1-nitronaphthalene and 2-nitronaphthalene in lake trout (around 10%) were low compared to abundances in both diesel exhaust (Liu et al. 2010) and the atmosphere (Albinet et al. 2007), likely reflecting the rapid clearance of these compounds relative to other NPAHs (Table A2.1). Much higher abundances of 1-nitronaphthalene and 2-nitronaphthalene (30-50%) were found in mussels and oysters from Osaka Bay, Japan (Uno et al. 2011), again highlighting effects of clearance rates. 2-Nitrobiphenyl and 3-nitrobiphenyl show a different pattern with high abundances (15 – 25%) in lake trout, probably reflecting slow metabolism (Table A2.1). Although these two compounds have not been detected in Lake Michigan sediments (Huang et al. 2014), they may be taken up by lake trout from the water column given their relatively low *Kow* (Table A2.1). Two other NPAHs, 1-nitropyrene and 6-nitrochrysene, had low abundances in lake trout (10-15%) and bivalves (Uno et al. 2011) compared to sediments (30-40%, (Huang et

al. 2014), Figure 2.5), probably a result of rapid clearance in fish and possibly other biota (Table A2.1). Sampling across major ecosystem compartments, e.g., water, sediments and across the food web, is needed to confirm the sources and mechanisms affecting levels of individual NPAH species.

2.4.4 Whole fish: steranes and hopanes

Five of the six target steranes were detected in more than 30% of the samples. Only two hopanes, 17α(H),21β(H)-hopane and 17α(H),21β(H)-30-norhopane, had a detection frequency above 30% in the fall, but they were not detected in any sample in the spring. Σ_2Hopane and Σ_5Sterane concentrations are presented in Figures 2.2C and 2.2D, respectively. In fall 2011, Σ_5Sterane concentrations averaged 269 ± 111 pg/g ww (range from 167 to 808 pg/g ww in individual fish), while $\Sigma_2$hopane concentrations averaged 37 ± 23 pg/g ww (11 to 141 pg/g ww). In spring 2012, sterane and hopane concentrations fell 10-fold. Like PAHs and NPAHs, no differences by gender were noted for either $\Sigma_5$sterane and $\Sigma_2$hopane levels. In fall 2011, both $\Sigma_5$sterane and $\Sigma_2$hopane showed the same spatial trend: Charlevoix < Clay Banks < Waukegan; and concentrations of both groups of compounds at Waukegan were significantly higher than those at Charlevoix (Mann-Whitney U test; $p < 0.001$). This trend disappeared in spring 2012. The relative abundances of the five detected steranes were similar across season, site and gender. 20S-5α(H), 14α(H), 17α(H)-cholestane and 20R-5α(H), 14β(H), 17β(H)-24-ethylcholestane were most abundant, averaging 36 ± 13 and 42 ± 18%, respectively. Abundances of the other 3 detected steranes were between 5% and 10%. In fall, the abundances of the two detected hopanes (17α(H),21β(H)-hopane and 17α(H),21β(H)-30-norhopane) were also consistent across site and gender, averaging 65 ± 10 and 35 ± 10%, respectively.

Two studies pertain to steranes and hopanes in aquatic biota. Concentrations of Σhopanes in aquaculture fish (red fish, grouper, tiger grouper, pomfret) in the Strait of Malacca were high, 17 to 250 ng/g ww (calculated from ng/g dw), possibly reflecting the extensive offshore oil and gas extraction and ocean shipping in this region (Manan et al. 2011). In the second study, concentrations of total biomarkers (10 hopanes and 6 steranes) ranged from undetected to 10 ng/g ww (calculated from ng/g dw) in arctic amphipods (*Anonyx nugax*) in the Alaskan Beaufort Sea (Neff and Durell 2012). These concentrations, which should not be

affected by offshore oil and gas activities (Neff and Durell 2012), are comparable to levels measured in Lake Michigan trout.

As noted above, steranes and hopanes enter the environment primarily from pyrogenic (e.g., traffic) and petrogenic (e.g., crude oil, asphalt and gasoline) sources. The Chicago/Gary area mentioned is a primary source area for steranes and hopanes for Lake Michigan, and southwesterly winds are common, especially in fall (Angel 2009). The decrease in sterane and hopane concentrations with distance from the Chicago/Gary area (sites Waukegan to Charlevoix) in fall 2011 suggests the significance of this source. Much lower sterane and hopane levels in spring 2012 might result from changes in SVOC loadings to Lake Michigan, fish uptake and diet, and possibly fish metabolism.

Hopane and sterane profiles measured in Lake Michigan sediments (Huang et al. 2014) show the predominance of 17α(H),21β(H)-hopane, as seen in lake trout. However, $\Sigma_6$sterane concentrations in sediments were lower than $\Sigma_5$hopane levels in sediment; the opposite was seen in lake trout. This pattern does not appear to result from metabolism in fish since the estimated half-life in fish of 20R-5α(H), 14α(H), 17α(H)-cholestane (only half-life available for steranes) is shorter than that for all five target hopanes (Table A2.1). Microbial biodegradation may play a role here given the long biodegradation half-life of 20R-5α(H), 14α(H), 17α(H)-cholestane (1662 days) relative to the five target hopanes (799-1249 days) (EPA 2012a). Levels in lake trout might also be driven by uptake via suspended particles and biota, in addition to sediment. The similar hopane and sterane compositions in the fish across season, site and gender suggest the same sources. Moreover, the same compounds (17α(H),21β(H)-hopane and 20S-5α(H), 14α(H), 17α(H)-cholestane) were most abundant in both fish and diesel engine exhaust (Liu et al. 2010). The presence of these petroleum biomarkers in Lake Michigan lake trout is consistent with traffic-related emissions that are deposited in the lake and accumulated by aquatic biota.

2.4.5 SVOCs in eggs

Female fish presented eggs in the fall, the spawning season. Fewer PAH compounds were detected in the eggs than in the whole fish. Of the 9 PAHs detected in over 30% of whole fish samples, anthracene and fluoranthene were not detected in over 30% of the egg samples (Table A2.1). Σ_7PAH concentration in the 15 egg samples ranged from 99 to 527 pg/g ww. (Averages by site are presented in Table A2.4.) Σ_7PAH concentrations in eggs were significantly lower than

the Σ_9PAH concentrations in the corresponding female fish (paired t-test, $t = 4.08$, $df = 14$, $p = 0.001$). Similar patterns have been observed for PCBs in lake trout from Lake Ontario, i.e., concentrations in gonads were 4-fold lower than those in female whole fish (Madenjian et al. 2010). PAH profiles in eggs also differed from those in whole fish where the abundance of phenanthrene was very low (Figure A2.2).

Compared to whole fish, two additional NPAH compounds were detected in eggs in over 30% of samples, i.e., all 11 target NPAHs were detected in eggs (Table A2.1). Σ_{11}NPAH concentration in the 15 egg samples ranged from 0.81 to 130 pg/g ww. (Site averages are presented in Table A2.5.) Σ_{11}NPAH concentrations in eggs were higher than the Σ_9NPAH concentrations in the corresponding female fish, and differences are marginally significant (paired t-test, $t = -1.94$, $df = 14$, $p = 0.072$). NPAH profiles in eggs resembled those in whole fish (Figure A2.3).

Eggs contained the same steranes and hopanes detected in whole fish. Σ_5Sterane and $\Sigma_2$hopane concentration in the eggs ranged from 204 to 493 pg/g ww and 10 to 119 pg/g ww, respectively. (Site averages are presented in Table A2.6.) Σ_5Sterane and $\Sigma_2$hopane concentrations in the eggs did not differ significantly from those in female fish, and abundances of individual compounds were also similar.

2.4.6 Screening level risk estimates

For the average scenario, using only the compounds detected in over 30% of whole fish samples and adjusting from whole fish to muscle tissue (see Data Analysis), the average TEQ from PAHs and NPAHs combined was 25 pg/g (19 pg/g from PAHs, 6 pg/g from NPAHs). Based on an oral cancer slope factor of 7.3 per mg/kg-day (EPA 1994), and average (0.73 g/kg-day) and high (2.2 g/kg-day) fish consumption rates (EPA 2011), consumption of lake trout from Lake Michigan gives lifetime cancer risks of 0.13 and 0.40 per million. For the worst case scenario, considering all target compounds (regardless of detection frequency), the average muscle TEQ from PAHs and NPAHs combined is 516 pg/g (510 pg/g from PAHs and 6 pg/g from NPAHs), and the resulting cancer risks are 2.7 and 8.3 per million. The difference is largely due to dibenzo[a,h]anthracene, which was only detected in 1.2% of the samples but which has a TEF = 10 (Table A2.1). Even in the worst case scenario, calculated risks fell within the upper range of protective risk guidelines, 1 to 10 per million; the total TEQ (averaged 516 pg/g) also

fell below the Canadian limit of 1 to 4 ng/g benzo[a]pyrene for fish and shellfish established to protect consumers from adverse health effects (MOE 1993). However, these calculations involve several uncertainties and caution is necessary in interpreting the results. First, several TEFs have been reported for certain PAH compounds, and some differ by several orders of magnitudes (EPA 2010). Using the highest TEF values (EPA 2010) and the worst case scenario, the average PAH TEQ was 1949 pg/g (range from 1763 to 3389 pg/g) in our sample (fish muscles), which exceeded the Canadian limits. Second, the oral cancer slope factor for BAP ranges from 4.5 to 11.7 per mg/kg-day (median of 7.3 per mg/kg-day) (EPA 1994). Combining these variables and using a high fish consumption rate (2.2 g/kg-day), the highest possible risk associated with lake trout consumption is 87 per million, well above guidelines. Moreover, since many NPAHs lack TEFs and not all NPAHs were measured, the current risk estimates may be underestimated. Even in the current calculation which only included the 4 NPAHs with available TEF values, NPAHs contributed a significant portion (40-80%) of the TEQs in some samples (under the average scenario).

Due to their rapid metabolism and resulting low concentrations, PAHs and NPAHs in lake trout may pose minimal human health risks, in contrast to risks resulting from the biomagnification of contaminants such as PCBs and mercury in Great Lakes fish (Chiang et al. 2012; Zananski et al. 2011). Still, the PAH and NPAH risk estimates presented may be underestimated, and additional toxicology studies and environmental measurements appear warranted.

In addition, PAH exposure has been associated with immunosuppression, decreased growth and DNA damage in juvenile salmon collected in Puget Sound at stomach content concentrations from 4,000 to 15,000 ng/g wet weight for total PAHs (16 EPA priority PAHs plus 5 alkylated PAHs) (Johnson et al. 2007). While we did not collect data regarding juvenile fish or analyze stomach contents separately, these concentrations are three to four orders of magnitude higher than levels (223 to 1,704 pg/g ww) we measured in whole adult fish from Lake Michigan (which included stomach contents). Phototoxicity of PAHs and NPAHs may also be a concern for fish health. Laboratory studies showed that toxic effects can occur when fish were exposed to PAHs at μg/L levels in water in the presence of sunlight (Arfsten et al. 1996). However, observed concentrations of ΣPAHs in southern Lake Michigan waters did not exceed 20 ng/L (Offenberg and Baker 2000), and the model-predicted water concentrations of phototoxic PAHs

and NPAHs (Chapter 6) based on measurements in lake trout (this chapter) and sediments (Chapter 3) did not exceed 10 ng/L. Moreover, coho salmon preferentially select habitats with shade to avoid exposure to sunlight (Kelly and Bothwell 2002), and lake trout may have similar behavior that can prevent them from harm UV radiation. Although direct evidence is lacking, phototoxicity may not be important for Lake Michigan lake trout, . While adverse effects seem unlikely, an analysis of fish health should focus on susceptible life stages and key target tissues.

2.4.7 Strengths and limitations

The present study has several strengths. First, a wide range of SVOCs was examined, and this appears to be the first reporting NPAH concentrations in fish. Second, fish were collected during two seasons, which allowed a comparison between spawning and non-spawning season. Third, because measurements used whole-fish homogenates, and eggs from female fish in the spawning season were separated and measured, whole-fish body burdens without the influence of eggs were determined. Finally, although the literature is sparse, many of our results are consistent with earlier reports.

The study has several limitations. First, only selected PAHs were measured. Investigation of PAH metabolites, due to possible persistence and toxicity, is warranted. Second, organ-specific analyses were not attempted. PAH concentrations are higher in fish liver and bile, and lower in muscles (Varanasi and Stein 1991). This also may be the case for NPAHs, steranes and hopanes. Since most fish mass was muscle, concentrations were low in whole-fish homogenates, which may have decreased detection rates of some compounds. Third, individual samples rather than composite samples were used, but this highlights fish-to-fish variation. Fourth, the small number of samples in each season-site-gender group did not allow some statistical analyses, e.g., repeated measures analysis of variance. Finally, tissue concentrations of SVOCs in lake trout were weakly correlated to the concentrations in sediments, which will be discussed in Chapter 3. It would be informative to sample bottom-feeding and small feeding range fish (e.g., catfish or carp) that reside near the sediment sampling sites.

2.5 Conclusions

The present study demonstrates the accumulation of PAHs, NPAHs, steranes and hopanes in lake trout from Lake Michigan, and provides the first report of the occurrence of NPAHs in freshwater fish. In whole fish, ΣPAH concentrations averaged 546 ± 244 pg/g ww,

and levels were similar across season and site. These low concentrations suggest “biodilution” of PAHs in lake trout. ΣNPAH concentrations averaged 7.2 ± 7.0 pg/g, and fish behavior and specifically spawning seems to affect the seasonal pattern of NPAH concentrations. Sterane and hopane concentrations were 2 to 20 times lower than PAH levels and differed by season, which might be attributed to substantial differences in uptake and/or clearance rates. Spatial differences also were seen steranes and hopanes in fall, indicating the impact of sources in the Chicago/Gary area. No difference by gender was observed for the target SVOCs.

All but one female fish collected in fall contained eggs. NPAH, sterane and hopane levels in eggs were similar to those in the corresponding female fish, but significantly lower for PAHs.

Upper bound worst-case estimates of lifetime human cancer risks due to lake trout consumption exceeded 10 per million, but generally PAHs and NPAHs in lake trout seem to pose a minimal threat to human health. However, NPAHs contributed a significant portion of the toxicity in some samples, and the risks from NPAHs were probably underestimated. Thus, further assessment of NPAH contamination in Great Lakes fish, and effects on fish and ecological health, is warranted.

2.6 Tables and figures

Table 2.1 ΣPAH concentrations reported for Great Lakes biota
(mean ± std unless otherwise specified)

	Species	Location	No. of PAHs measured	ΣPAH concentrations (ng/g wet weight)	Reference
Fish	Lake trout	Lake Michigan	16 EPA priority	Male: 0.56 ± 0.29 Female: 0.53 ± 0.18 Eggs: 0.30 ± 0.11	This study
	Lake trout	Lake Michigan Lake Superior	27	Lean: 1.52 ± 0.38 Fat/siscowet: 6.34 ± 0.94	Zabik et al., 1996
	Minnow Sunfish Alewife Round Goby Yellow Perch Crayfish	Calumet region of southwestern Lake Michigan	15 (16 EPA priority excluding NAP)	10 - 350 (range) 10 - 80 (range) 15 - 1064 (range) 55 (mean) 20 (mean) 10 - 130 (range)	Levengood et al., 2011
	White Sucker	Upstream and downstream of the Moses-Saunders power dam in St. Lawrence River near Cornwall, Ontario and Massena, New York	33 (including 17 methyl PAHs)	Upstream: 16 ± 6 Downstream: 11 ± 6	Ridgway et al., 1999
	Brown Bullhead	Lake Michigan tributaries St. Mary's River tributary Lake Erie tributary	5	20 - 24 (range) 24 (mean) 220 (mean)	Baumann et al., 1991
Invertebrates	nphipod: *Pontoporeia ho*	Lake Michigan	7	4000 - 7000 (range)	Eadie et al., 1982
	Oligochaete Worms Chironomid Midges	Lake Erie	8	300 - 400 (range) 400 - 800 (range)	Eadie et al., 1982
	Bivalves: Zebra mussel	Detroit River and western Lake Erie	16 EPA priority	12.6 - 98.7 (range)	Metcalfe et al., 1997

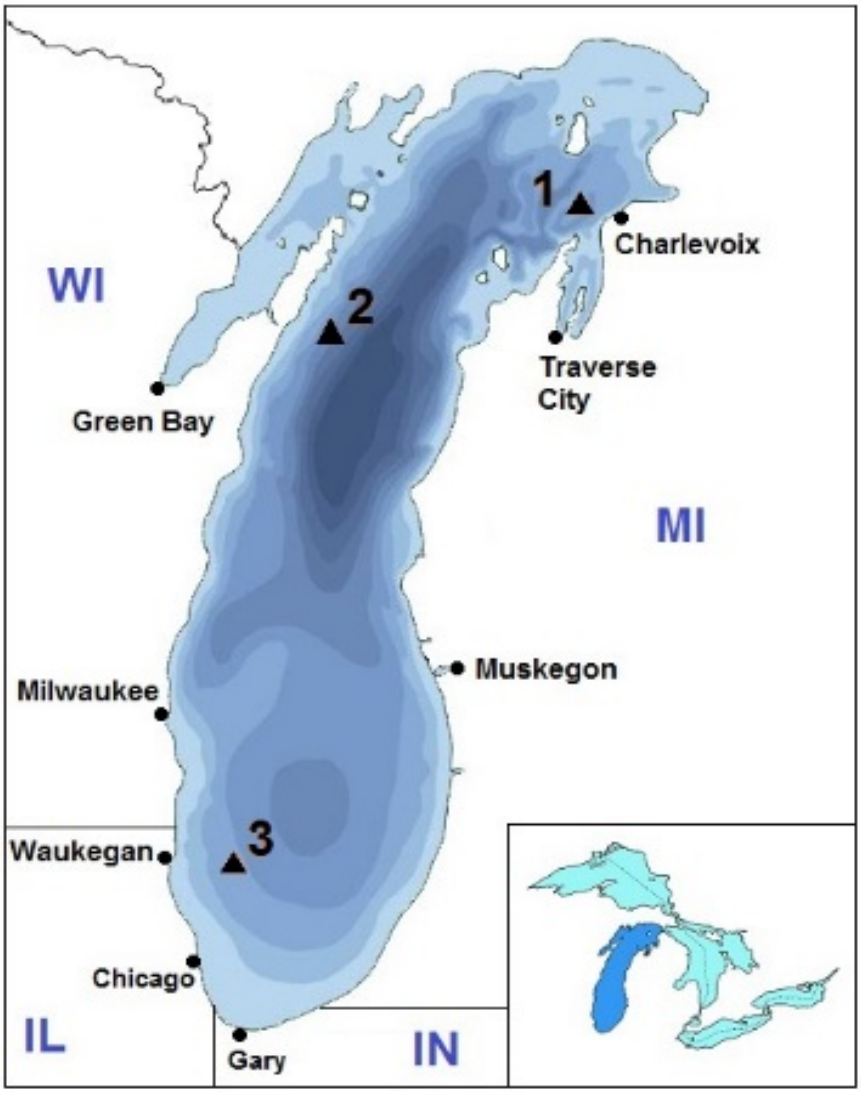

Figure 2.1 Map showing Lake Michigan and sampling locations.
Site 1: Charlevoix, MI; Site 2: Clay Banks, WI; Site 3: Waukegan, IL. The colors reflect water depth in increments of 100 ft (to >800 ft depth).

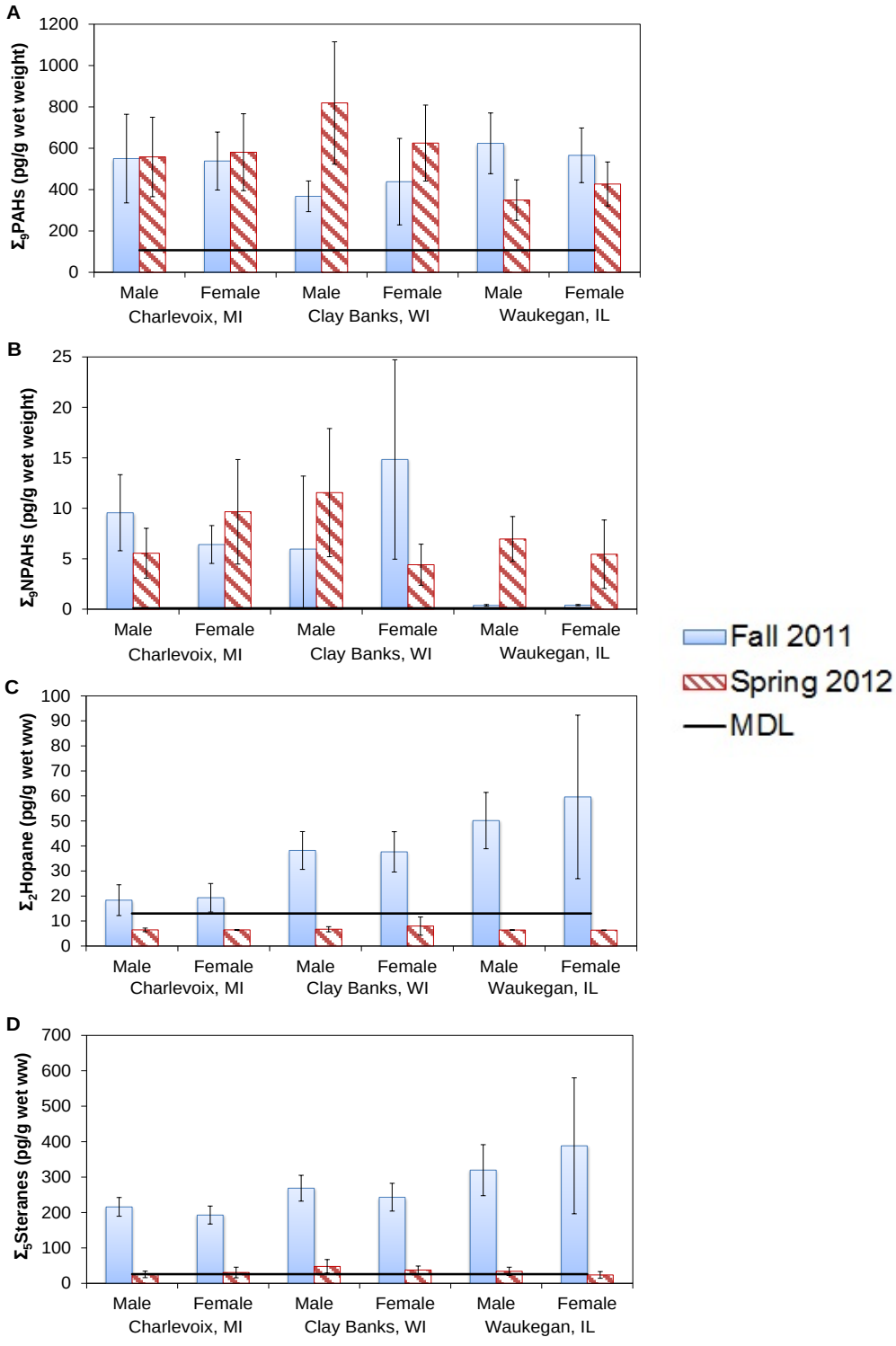

Figure 2.2 Concentrations of (A) Σ_9PAHs; (B) Σ_9NPAHs; (C) Σ_5Steranes; and (D) Σ_2Hopanes in whole fish (pg/g ww).
(Data presented are mean ± SD.)

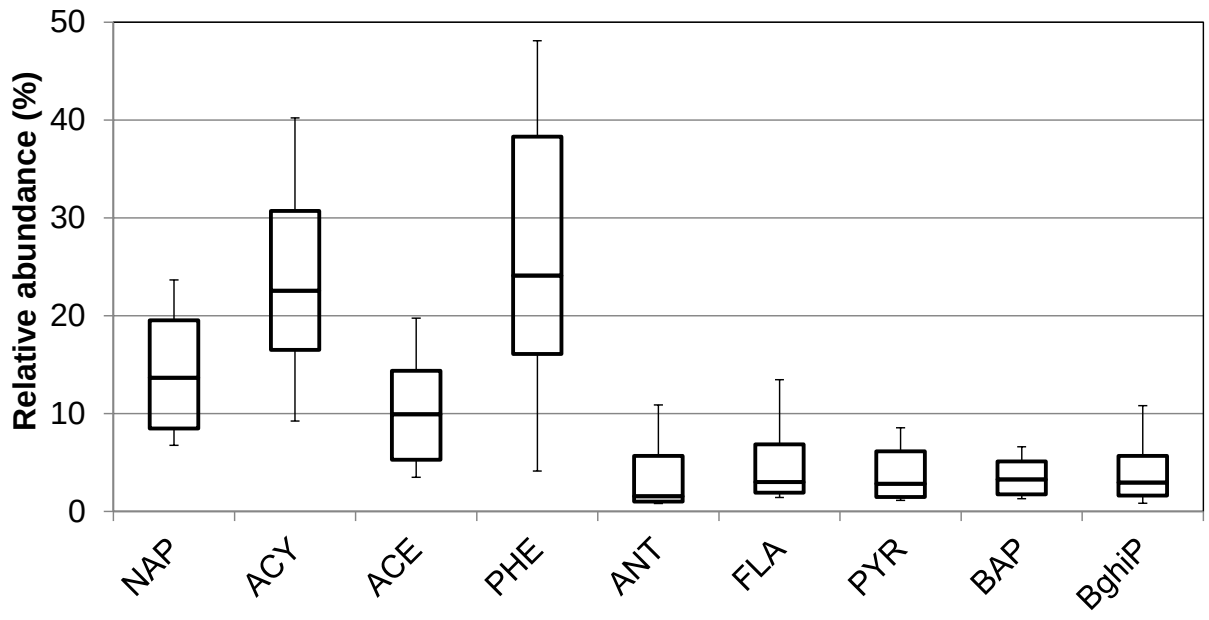

Figure 2.3 PAH profiles in whole fish. Boxplots show 10^{th,} 25th, 50th, 75th and 90th percentiles for pooled samples
N = 84 including replicates. Only compounds with >30% detection frequency are shown.
Acronyms of PAHs can be found in Table 1.1.

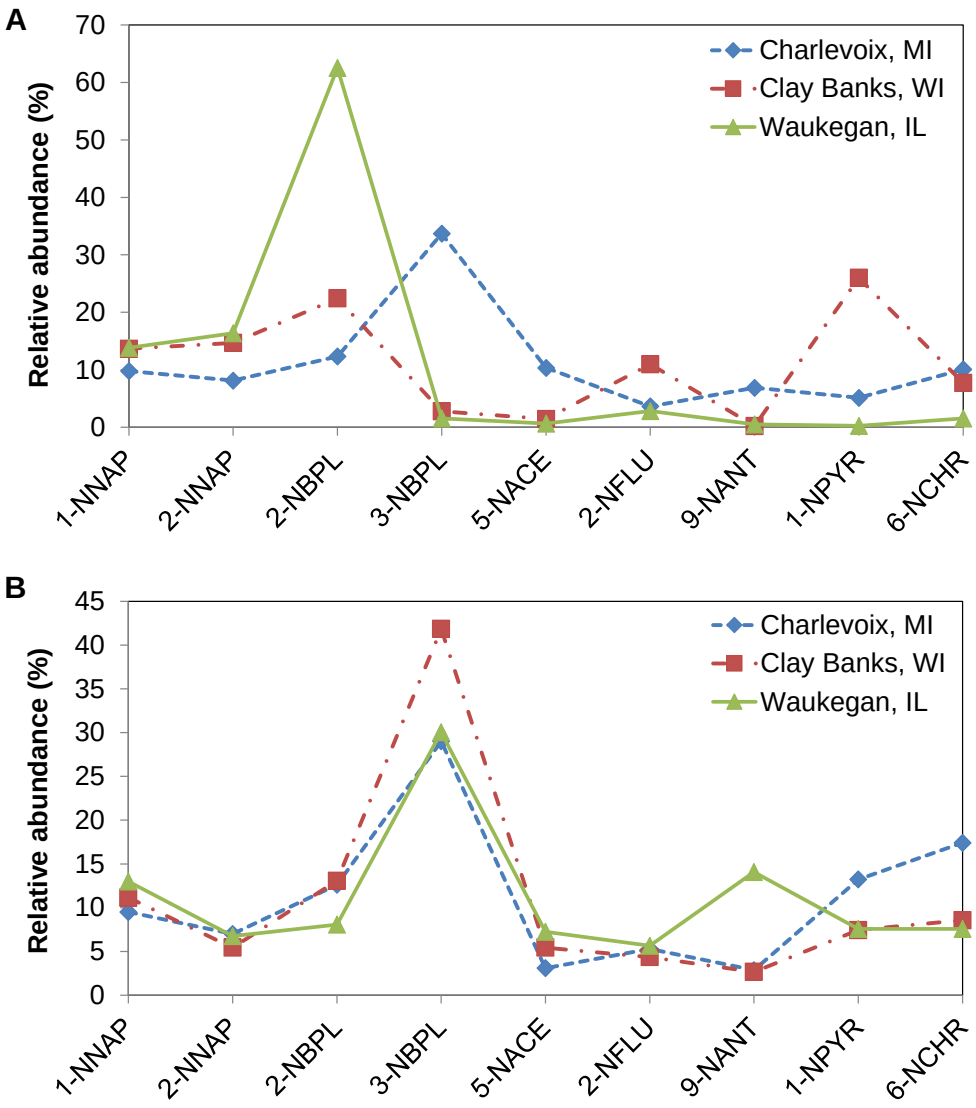

Figure 2.4 Mean relative abundance of each NPAH compound (A) in the fall; (B) in the spring for whole fish.
Only compounds with >30% detection frequency are shown. Acronyms of NPAHs can be found in Table 1.1.

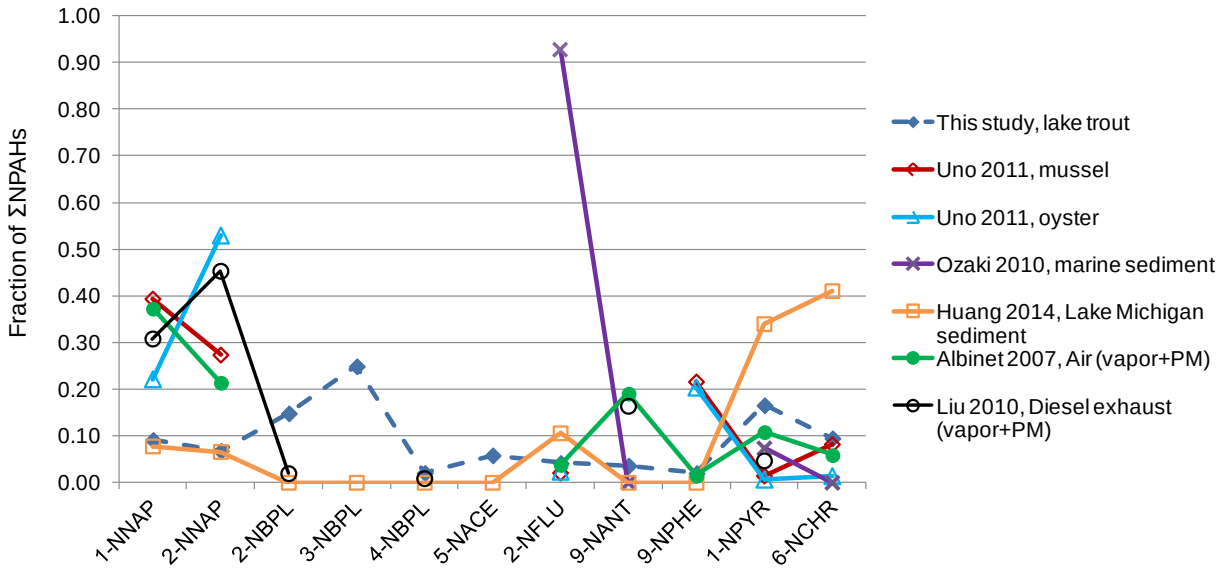

Figure 2.5 Comparison between this study's NPAH profile (dashed line) and profiles in literature.
Acronyms of NPAHs can be found in Table 1.1.

Table A2.1 MDLs and detection frequencies of target compounds

Group	Compound	TEF (new)	TEF (old)	LogBAF in fish[f]	Half-life in fish[f] (day)	MDL (pg/g ww)	Whole fish measurements (pg/g ww)					Egg measurements (pg/g ww)				
							Mean[g]	Std[g]	Min	Max	Detection freq. (%) (N = 84)	Mean[g]	Std[g]	Min	Max	Detection freq. (%) (N = 17)
PAHs	Naphthalene	0.001 [b]	0.001 [b]	2.25	4.53	6	75	37	< MDL	173	98.8	65	19	37	100	100
	Acenaphthylene	0.001 [b]	0.001 [b]	2.74	3.73	15	124	74	21	483	100	118	56	< MDL	213	94.1
	Acenaphthene	0.001 [b]	0.001 [b]	1.98	0.25	14	60	69	< MDL	478	94.0	34	21	< MDL	77	88.2
	Fluorene	0.001 [b]	0.001 [b]	2.96	1.38	15	7	1	< MDL	17	2.4	-	-	-	-	0
	Phenanthrene	0 [a]	0.001 [b]	3.09	2.55	14	171	156	14	850	100	23	21	< MDL	62	47.1
	Anthracene	0 [a]	0.01 [b]	3.06	2.54	10	21	30	< MDL	178	47.6	7	5	< MDL	23	11.8
	Fluoranthene	0.08 [a]	0.001 [b]	2.59	2.57	20	30	39	< MDL	285	41.7	-	-	-	-	0
	Pyrene	0 [a]	0.001 [b]	2.90	0.56	11	28	35	< MDL	193	61.9	24	35	< MDL	149	52.9
	Benz[a]anthracene	0.2 [a]	0.1 [c]	2.61	3.03	11	7	4	< MDL	24	19.0	6	3	< MDL	16	11.8
	Chrysene	0.1 [a]	0.001 [c]	3.30	3.14	16	9	4	< MDL	44	7.1	-	-	-	-	0
	Benzo[b]fluoranthene	0.8 [a]	0.1 [c]	3.07	2.67	27	23	16	< MDL	70	26.2	-	-	-	-	0
	Benzo[k]fluoranthene	0.03 [a]	0.01 [c]	2.48	3.37	29	18	12	< MDL	98	10.7	19	10	< MDL	44	23.5
	Benzo[a]pyrene	1 [a]	1 [c]	2.60	0.90	13	18	11	< MDL	43	66.7	27	19	< MDL	58	64.7
	Dibenz[a,h]anthracene	10 [a]	1 [c]	3.46	3.53	109	54	9	< MDL	131	1.2	-	-	-	-	0
	Indeno[1,2,3-cd]pyrene	0.07 [a]	0.1 [c]	2.50	1.17	77	45	24	< MDL	147	9.5	-	-	-	-	0
	Benzo[g,h,i]perylene	0.01 [a]	0.01 [b]	1.83	0.30	4	27	31	< MDL	154	92.9	13	11	< MDL	37	82.4
NPAHs	1-Nitronaphthalene	n/a [e]	n/a [e]	2.10	1.31	0.015	0.67	0.88	< MDL	4.34	95.2	0.68	0.75	0.03	2.19	100
	2-Nitronaphthalene	n/a [e]	n/a [e]	2.14	1.36	0.019	0.47	1.25	< MDL	11.35	91.7	0.74	0.59	0.10	2.38	100
	2-Nitrobiphenyl	n/a [e]	n/a [e]	2.43	1.96	0.015	1.00	2.09	< MDL	14.73	83.3	1.60	1.91	0.23	7.02	100
	3-Nitrobiphenyl	n/a [e]	n/a [e]	2.64	2.41	0.012	1.82	2.17	< MDL	13.40	72.6	5.55	8.09	0.22	25.37	100
	4-Nitrobiphenyl	n/a [e]	n/a [e]	2.61	2.33	0.055	0.14	0.38	< MDL	3.03	25.0	0.19	0.18	< MDL	0.56	52.9
	5-Nitroacenaphthene	0.03 [d]	0.03 [d]	2.61	2.33	0.005	0.50	1.14	< MDL	6.38	46.4	0.32	0.36	< MDL	1.17	64.7
	2-Nitrofluorene	0.01 [d]	0.01 [d]	1.93	0.31	0.022	0.29	0.52	< MDL	3.29	45.2	0.95	1.78	< MDL	7.36	58.8
	9-Nitroanthracene	n/a [e]	n/a [e]	2.60	1.00	0.004	0.25	0.45	< MDL	3.07	41.7	0.26	0.59	< MDL	2.45	58.8
	9-Nitrophenanthrene	n/a [e]	n/a [e]	2.37	0.65	0.003	0.14	0.41	< MDL	2.86	27.4	0.60	0.94	< MDL	3.06	58.8
	1-Nitropyrene	0.1 [d]	0.1 [d]	2.02	0.25	0.002	1.08	3.32	< MDL	20.69	69.0	10.32	22.15	< MDL	87.61	64.7
	6-Nitrochrysene	10 [d]	10 [d]	2.55	0.89	0.012	0.64	0.94	< MDL	6.12	75.0	1.00	1.61	< MDL	5.86	52.9
Hopanes	17α(H),21β(H)-Hopane	n/a [e]	n/a [e]	4.80	4237	5	12	13	< MDL	84	51.2	27	22	7	103	100
	17α(H)-22,29,30-Trisnorhopane			6.00	1294	8	4	1	< MDL	13	3.6	-	-	-	-	0
	17α(H),21β(H)-30-Norhopane			5.20	3199	8	9	8	< MDL	56	32.1	14	9	< MDL	33	64.7
	22R-17α(H),21β(H)-Homohopane			4.33	5838	5	3	1	< MDL	8	3.6	3	2	< MDL	8	17.6
	22S-17α(h),21β(h)-Homohopane			4.33	5838	5	2	1	< MDL	7	1.2	3	2	< MDL	7	11.8
Steranes	20S-5α(H), 14α(H), 17α(H)-Cholestane	n/a [e]	n/a [d]	n/a	n/a	4	48	41	< MDL	169	94.0	124	57	62	301	100
	20R-5α(H), 14α(H), 17α(H)-Cholestane			5.03	793	6	5	3	< MDL	15	50.0	11	6	7	29	100
	20R-5α(H), 14β(H), 17β(H)-Cholestane			n/a	n/a	4	4	3	< MDL	15	38.1	9	3	< MDL	17	94.1
	20R-5α(H), 14β(H), 17β(H)-24-Methylcholestane			n/a	n/a	6	10	9	< MDL	42	53.6	16	9	< MDL	37	94.1
	20R-5α(H), 14α(H), 17α(H)-24-Ethylcholestane			n/a	n/a	5	3	1	< MDL	11	6.0	-	-	-	-	0
	20R-5α(H), 14β(H), 17β(H)-24-Ethylcholestane			n/a	n/a	6	78	91	< MDL	576	79.8	145	63	53	256	100

[a] (EPA 2012b); [b] (Nisbet and LaGoy 1992); [c] (Schoeny and Poirier 1993); [d] (RIDEM 2008); [e] No information available.

[f] Estimated by (EPA 2012a). Values with an asterisk are from experimental database.

[g] Mean and standard deviations are calculated with all nondetect data substituted by MDL/2.

Table A2.2 PAH concentrations in fish organs

Fish organ weight[a] (as % of total body weight)				
Brain	Gill	Muscle	Bone	Liver
0.05	4	60	20	1

Concentrations of PAHs < 4 rings[b] (as a ratio of the concentration in muscle)				
Brain	Gill	Muscle	Bone	Liver
2	1.5	1	0.8	0.5

Concentrations of PAHs >= 4 ring[b] (as a ratio of the concentration in muscle)				
Brain	Gill	Muscle	Bone	Liver
4	1.5	1	1.5	0.8

[a] (Hoffman et al. 1999).

[b] (Deb et al. 2000).

Table A2.3 Mean weight, length and lipid content of lake trout samples (standard deviation in parenthesis)

Season	Site	Gender	Sample size	Weight[a] (g)	Length (mm)	Lipid Content[b] (%)
Fall 2011	Charlevoix, MI	M	6	2391 (344)	600 (36)	14.8 (1.2)
		F	6	3152 (204)	645 (9)	14.1 (1.5)
	Clay Banks, WI	M	6	2500 (191)	640 (25)	14.7 (1.4)
		F	6	3152 (318)	645 (17)	15.1 (0.7)
	Waukegan, IL	M	7	3197 (1124)	646 (90)	14.6 (2.0)
		F	5	3622 (1575)	641 (68)	15.0 (2.2)
Spring 2012	Charlevoix, MI	M	9	2717 (641)	614 (49)	16.0 (5.8)
		F	7	3070 (387)	628 (37)	15.8 (2.9)
	Clay Banks, WI	M	7	2352 (493)	613 (35)	14.0 (4.4)
		F	5	2840 (491)	649 (33)	14.3 (3.8)
	Waukegan, IL	M	5	3395 (435)	666 (29)	14.8 (4.3)
		F	5	3643 (809)	665 (57)	14.8 (3.5)
All combined			74	2962 (756)	639 (49)	14.9 (3.1)

[a] The weight for female fish includes eggs.

[b] The lipid content for female fish excludes eggs.

Table A2.4 Concentrations of ΣPAHs (pg/g ww)

Type	Season	Site	Gender	Sample size	Mean[a]	Std[a]
Whole fish	Fall 2011	Charlevoix, MI	Male	6	550.1	214.3
			Female	6	538.4	140.1
		Clay Banks, WI	Male	6	367.6	74.5
			Female	6	438.6	209.2
		Waukegan, IL	Male	7	624.0	146.7
			Female	5	565.9	132.1
	Spring 2012	Charlevoix, MI	Male	9	558.2	192.2
			Female	7	581.1	186.1
		Clay Banks, WI	Male	7	819.4	295.9
			Female	5	624.9	183.8
		Waukegan, IL	Male	3	349.7	97.9
			Female	2	427.2	106.7
Eggs	Fall 2011	Charlevoix, MI	-	6	242.8	81.2
		Clay Banks, WI		4	287.5	63.1
		Waukegan, IL		5	383.2	82.3

[a] Mean and std for whole fish include 9 compounds (with detection freq. >30%), and the mean and std for eggs include 7 compounds (with detection freq. >30%).

Table A2.5 Concentrations of ΣNPAHs (pg/g ww)

Type	Season	Site	Gender	Sample size	Mean[a]	Std[a]
Whole fish	Fall 2011	Charlevoix, MI	Male	6	11.69	4.06
			Female	6	7.13	1.92
		Clay Banks, WI	Male	6	6.02	7.23
			Female	6	15.08	9.88
		Waukegan, IL	Male	7	0.41	0.08
			Female	5	0.44	0.06
	Spring 2012	Charlevoix, MI	Male	9	5.67	2.48
			Female	7	9.68	5.17
		Clay Banks, WI	Male	7	11.61	6.35
			Female	5	4.48	2.04
		Waukegan, IL	Male	3	7.03	2.22
			Female	2	5.58	3.40
Eggs	Fall 2011	Charlevoix, MI	-	6	8.4	2.5
		Clay Banks, WI		4	68.1	35.3
		Waukegan, IL		5	1.1	0.2

[a] Mean and std for whole fish include 9 compounds (with detection freq. >30%), and the mean and std for eggs include 11 compounds (with detection freq. >30%).

Table A2.6 Concentrations of Petroleum biomarkers (pg/g ww)

					ΣHopanes		ΣSteranes	
Type	Season	Site	Gender	Sample size	Mean[a]	Std[a]	Mean[b]	Std[b]
Whole fish	Fall 2011	Charlevoix, MI	Male	6	18.3	6.2	215.7	26.4
			Female	6	19.3	5.7	192.9	25.4
		Clay Banks, WI	Male	6	38.2	7.6	268.6	36.4
			Female	6	37.7	8.1	243.2	39.4
		Waukegan, IL	Male	7	50.2	11.3	319.5	72.0
			Female	5	59.6	32.7	388.1	191.8
	Spring 2012	Charlevoix, MI	Male	9	< MDL		25.3	9.0
			Female	7			30.7	14.9
		Clay Banks, WI	Male	7			47.9	19.1
			Female	5			37.6	11.5
		Waukegan, IL	Male	3			34.0	11.7
			Female	2			23.7	9.4
Eggs	Fall 2011	Charlevoix, MI	-	6	15.7	3.8	292.1	75.8
		Clay Banks, WI		4	47.5	12.7	302.0	59.1
		Waukegan, IL		5	60.1	35.3	317.0	78.7

[a] Mean and std for both whole fish and eggs include 2 compounds (with detection freq. >30%).

[b] Mean and std for both whole fish and eggs include 5 compounds (with detection freq. >30%).

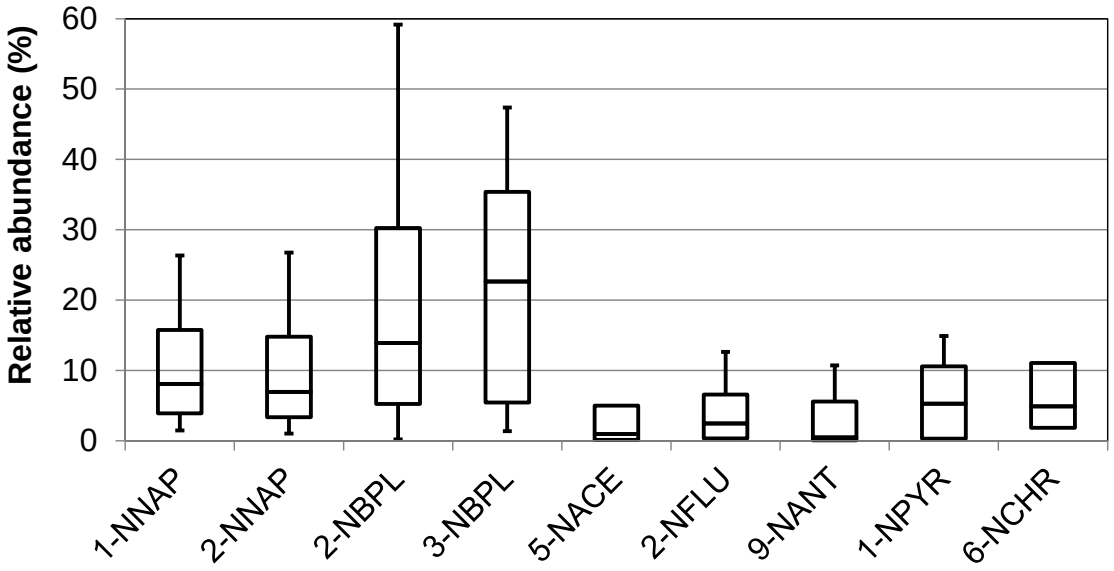

Figure A2.1 NPAH profiles in whole fish. Boxplots show 10^{th,} 25th, 50th, 75th and 90th percentiles for pooled samples.
Only compounds with >30% detection frequency are shown. Acronyms of NPAHs can be found in Table 1.1.

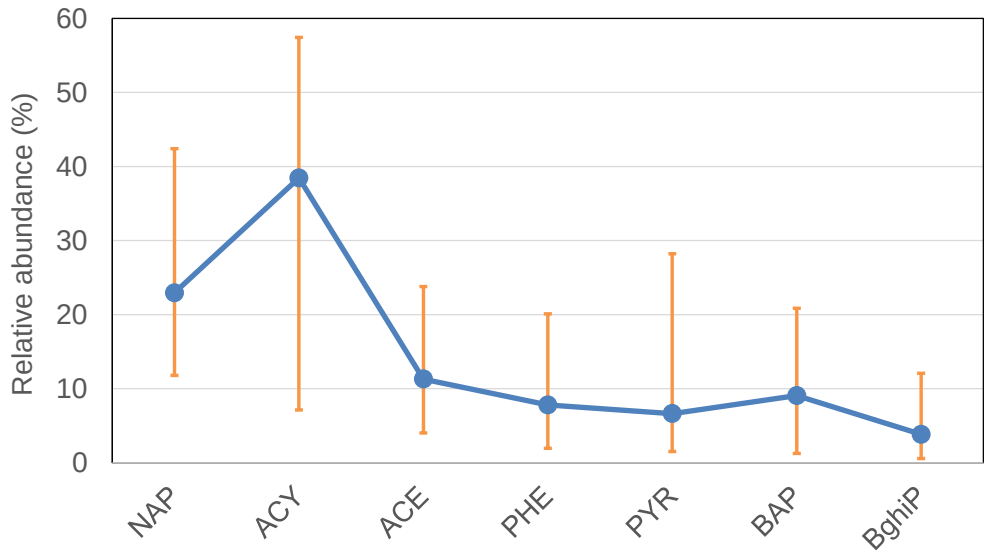

Figure A2.2 PAH profiles in eggs.
The points show averages, and the whiskers show maximum and minimum for pooled samples (N = 17 including replicates). Only compounds with >30% detection frequency are shown.

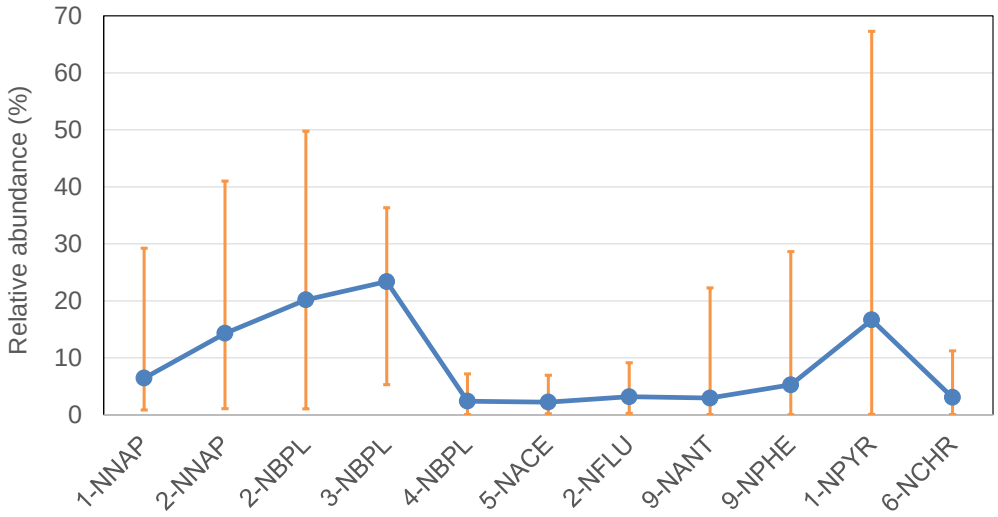

Figure A2.3 **N**PAH profiles in eggs.
The points show averages, and the whiskers show maximum and minimum for pooled samples (N = 17 including replicates). Only compounds with >30% detection frequency are shown.

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Chapter 3 Characterization and source apportionment of target compounds in sediments from Lake Michigan[2]

3.1 Abstract

PAHs in the Great Lakes basin are of concern due to their toxicity and persistence in bottom sediments. The nitro derivatives of PAHs, nitro-PAHs (NPAHs), which can have stronger carcinogenic and mutagenic activity than parent PAHs, may follow similar transport routes and also are accumulated in sediments. Limited information exists regarding the current distribution, trends and loadings of these compounds, especially NPAHs, in Lake Michigan sediments. This study characterizes PAHs, NPAHs, and biomarkers steranes and hopanes in surface sediments collected at 24 offshore sites in southern Lake Michigan. The total PAH concentration (ΣPAH_{15}, sum of 15 compounds) varied from 213 to 1291 ng/g dry weight (dw) across the sites, levels that are 2 to 10 times lower than those reported 20 to 30 years earlier. Compared to consensus-based sediment quality guidelines (SQGs), PAH concentrations suggest very low risk to benthic organisms. The total NPAH ($\Sigma NPAH_5$) concentration ranged from 2.9 to 18.6 ng/g dw, and included carcinogenic compounds 1-nitropyrene and 6-nitrochrysene. $\Sigma Hopane_5$ and $\Sigma Sterane_6$ concentrations ranged from 98 to 355 and 6.2 to 36 ng/g dw, respectively. Based on these concentrations and estimated sedimentation rates, Lake Michigan is currently receiving 11, 0.16, 0.25 and 3.6 metric tons per year (t/yr) of ΣPAH_{15}, $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$, respectively. Maps of OC-adjusted concentrations displayed declining concentrations with increasing off-shore distance in the study area. The relative abundances of the SVOCs were generally similar across sites. The major sources of PAHs and NPAHs are pyrogenic sources in nature, as based on diagnostic ratios; chemical mass balance (CMB) modeling apportioned these sources to emissions from diesel engines (56±18%), coal power plants (27±14%), coal-tar pavement sealants (16±11%), and coke ovens (7±12%). The biomarkers identify a combination of petrogenic and biogenic sources, with the southern end of

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the lake more impacted by petroleum. This first report of NPAH levels in sediments of Lake Michigan reveals several carcinogenic compounds at modest concentrations, and a need for further work to assess potential risks to aquatic organisms.

3.2 Introduction

PAH inputs to aquatic environments arise from atmospheric deposition, urban stormwater runoff and municipal/industrial effluents (Helfrich and Armstrong 1986). Petroleum spills may be a significant PAH source in certain locations (Helfrich and Armstrong 1986). Recently, coal-tar pavement sealants have been identified as an important source of PAHs in urban waterways, especially in central and eastern U.S. (P. C. Van Metre and Mahler 2010). Sealed pavements also emit PAHs by volatilization to urban air (P. C. Van Metre et al. 2012).

Lake Michigan receives large inputs of PAHs from atmospheric deposition, urban runoff and municipal/industrial effluents due to the large urban and industrial centers that surround its southern portion (Helfrich and Armstrong 1986). The Lake also receives inputs from petroleum spills given that Indiana Harbor, Indiana and Chicago, Illinois are major distribution centers for petroleum products (Helfrich and Armstrong 1986). In 1980, the cumulative loadings of PAH from these sources was estimated to be 50 to 55 metric tons/year (MT/yr), which included 40 MT/yr from atmospheric deposition (based on measured ΣPAH_{12} concentrations in air and dry and wet deposition rates) (Andren and Strand 1979), 0.8 to 8 MT/yr from urban runoff/municipal effluent (based on measured ΣPAH_{22} concentrations in sewage discharge and combined runoff/effluent flows) (Kveseth et al. 1982), and 5 to 7.5 MT/yr from commercial and private vessels and petroleum spills (Helfrich and Armstrong 1986). PAHs in Lake Michigan sediments have been characterized in several major studies conducted in the 1980s and 1990s (Eadie et al. 1982; Helfrich and Armstrong 1986; Simcik et al. 1996). Surface sediment accumulation rates for ΣPAH_{17} were estimated to be 50-70 ng/cm^2-yr in 1991-1993 (Simcik et al. 1996). While recent declines in PAH levels have been observed in sediment cores from Grand Traverse Bay, Lake Michigan (Schneider et al. 2001), only two studies (Helfrich and Armstrong 1986; Eadie et al. 1982) have reported PAH levels in sediments in the Lake's southern basin, the area closest to the source areas around Chicago, Illinois and Gary, Indiana where levels are likely to be highest. Several studies have examined sediments in Areas of Concern (AOC) of southern Lake Michigan, including Grand Calumet, Waukegan Harbor, Milwaukee Estuary, and Muskegon

Lake (Ghosh et al. 2003; MacDonald et al. 2002; A. Li et al. 2003; Kannan et al. 2001; Kemble et al. 2000). The National Coastal Condition Assessment (NCCA) project is planning to analyze near-shore sediments from the Great Lakes for 25 PAHs (EPA 2010). However, studies on open water sediments have not been carried out since the 1980s. Thus, assessment of current PAH levels in open lake sediments is needed to understand contaminant trends and the potential for environmental risks.

Airborne NPAHs also can enter aquatic environments through atmospheric deposition and urban runoff (Ozaki et al. 2010). Although the behavior of NPAHs in the atmosphere has been extensively studied (Albinet et al. 2007; de Castro Vasconcellos et al. 2008; Hayakawa et al. 2002; Librando and Fazzino 1993), few studies have investigated the occurrence of NPAHs in aquatic environments. The Minnesota Pollution Control Agency (MPCA) measured PAHs and NPAHs in urban stormwater pond sediments (Crane et al. 2010). While NPAHs were not detected in pond stations in the Twin Cities, MN, metropolitan area (Crane 2014), NPAHs including 2-nitrofluorene, 1-nitropyrene and 6-nitrochrysene were detected at ppb to ppm levels in Varney Pond, White Bear Lake, MN (MDH 2011). In Europe, NPAHs including nitronaphthalenes, 9-nitronaphthalene, 1-nitropyrene, 6-nitrochrysene and dinitropyrenes have been found at ppb levels in sediments from the Elbe River basin, Germany and Czech Republic (Lübcke-von Varel et al. 2012; Lübcke-von Varel et al. 2011). In sediments of the Suimon River, Japan, 1-nitropyrene and 2-nitrofulorene were measured at ppb levels (Sato et al. 1985). In marine (coastal) sediments collected off Barcelona, Spain, 1-nitropyrene and 6-nitrochrysene were found to contribute to the mutagenic activity (Fernandez et al. 1992), and NPAHs in the Hiroshima Bay Area, Japan, were found at concentrations up to 30 ng/g (2-nitrofluoranthene) (Ozaki et al. 2010). No information has been located regarding NPAHs in Great Lakes sediments.

Hopanes and steranes have been used as signature or marker compounds to help identify sources of organic matter in lake sediments (Meyers and Ishiwatari 1993; Xiong et al. 2010; Qu et al. 2007), the extent of biodegradation (Prince et al. 1994), and as tracers of vehicle exhaust in the atmosphere (given their specificity to lubricating oils used in diesel and gasoline engines) (Kleeman et al. 2008; Schauer et al. 2002, 1999). Information regarding hopanes and steranes in the Great Lakes is scarce. These compounds were detected in western Lake Ontario sediments where direct input of crude oil or petroleum products was indicated (Kruge et al. 1998). Hopanes

and steranes were also used to identify the source of the 2009 Sarnia oil spill in Lake Huron (Z. Wang et al. 2011). However, these compounds have not been reported for Lake Michigan.

The objective of this chapter is to characterize PAHs, NPAHs, hopanes and steranes in surficial sediments from the southern basin of Lake Michigan. The analysis includes an evaluation of compositional profiles, spatial patterns and loadings. Semi-quantitative forensic techniques (diagnostic ratios) are used to identify sources of target compounds, and quantitative CMB modeling is also used to apportion PAH sources. Temporal trends of PAHs are derived in conjunction with literature data. This first report regarding NPAHs and biomarkers in sediments of Lake Michigan provides information that can be used to monitor trends over time, identify sources of hydrocarbon contamination, and evaluate health and ecological consequences.

3.3 Materials and methods

3.3.1 Sample collection

Sediment samples were collected as "add-ons" to an ongoing program designed to assess long term trends in benthic communities in southern Lake Michigan, which has been conducted by the National Oceanic and Atmospheric Administration (NOAA) Great Lakes Environmental Research Laboratory (GLERL) at the same 40 sites since 1980. Of these, we selected 24 offshore sites (Figure 3.1) intended to sample various locations, depths and sediment types across the southern basin of Lake Michigan. Most of the sites (except S-2, S-3, S-4, V-1, H-8, B-7) were in depositional zones (Corcoran 2013). Sampling took place from August 16 to 19, 2011 on the R/V Laurentian (NOAA GLERL) using EPA methods (EPA 1999). The top 1 cm of sediment was collected by a Ponar Dredge, placed in solvent-washed brown glass jars with PTFE (polytetrafluoroethylene)-lined screw caps, and stored at 4 °C in a refrigerator on board. Immediately after the expedition, samples were transported in coolers to our Ann Arbor, Michigan laboratory and stored at -20 °C for two weeks before extraction.

3.3.2 Materials

All solvents were HPLC grade and obtained from Fisher Scientific Inc. (Pittsburgh, PA, USA). Florisil (60-100 mesh) and sodium sulfate (anhydrous, certified ACS granular, 10-60 mesh) for column chromatography were supplied by the same vendor. The method detection limits (MDLs) of the target compounds are presented in Table 3.1. Authentic standards of PAHs,

NPAHs and biomarkers were purchased from Cambridge Isotope Laboratories (Andover, MA), Sigma-Aldrich, (St. Louis, MO), and Chiron AS (Trondheim, Norway), respectively. Internal standards (ISs) for PAHs were fluoranthene-d10 (Cambridge Isotope Laboratories Inc., Andover, MA, USA) and an IS PAH mixture (Wellington Laboratories, Guelph, ON, Canada). 1-Nitrofluoanthene-d9 (Cambridge Isotope Laboratories Inc., Andover, MA, USA) was used as an IS for NPAH analyses, and n-tetracosane-d50 (Chiron Laboratories, Trondheim, Norway) as an IS for hopanes and steranes. Surrogate standards included C27-α,α,α-(20R)-cholestane-d2, 1-nitropyrene-d9, chrysene-d12 and naphthalene-d8 (Chiron Laboratories, Trondheim, Norway) .

3.3.3 Sample preparation and chemical analysis

After decanting the water layer on top of each sediment sample and homogenization, a 10-g subsample was taken to which 15 μL of the surrogate standard was added. The sample was dried with Na_2SO_4, extracted twice using dichloromethane/hexane (4:1, v/v), sonicated for 30 min twice, and any remaining solids were separated by centrifugation and removed. Extracts were passed through an activated Florisil column and fractionated into three portions. The fractionation and GC-MS analysis have been described in detail in Chapter 2 Section 2.3.3.

Quality assurance (QA) measures included the regular use of field and lab blanks, replicates, surrogate spike recovery tests and standard reference material (SRM 2266, NIST, USA). Replicates were performed for six samples (i.e., one replicate for every 4 samples). Measurement precisions, expressed as the average relative percent difference (RPD) across the six replicate measures and compounds in the group, were 22%, 19%, 26% and 23% for PAH, NPAH, sterane and hopane determinations above the reporting limits, respectively. Several target compounds were detected, but at trace levels, in blanks. Blank corrections were not used. The spike recovery was acceptable (between 70 to 100%), and the shift (abundance of target compounds in standard solutions before and after running a batch of samples) was below the 25% limit. QA data, including blanks, lab replicates and surrogate spike recoveries, are presented in Tables A3.1-A3.4.

The total organic carbon (OC) content was determined using the loss-on-ignition method (Q. Wang et al. 2011). Briefly, 2 to 4 g of sediment was placed in a borosilicate glass beaker, heated at 105 °C for 12 h to remove moisture, and then the sample dry weight was determined. The dried sediment was then placed in a muffle furnace (Neycraft Vulcan A-550), combusted at

500 °C for 12 h, and weighed. The weight loss was multiplied by 0.58 to calculate the OC mass (Q. Wang et al. 2011), and the OC content was calculated as the OC mass divided by the dry sample weight times 100%. Replicates performed for seven samples gave an average RPD of 7.1%. For method calibration and standard recovery tests, similar weights of L-glutamic acid (Sigma-Aldrich, St. Louis, MO) and mixture of glutamic acid and $CaCO_3$ (Sigma-Aldrich, St. Louis, MO) were prepared along with the samples. QA data for OC measurements, including replicates, blanks and recoveries, are presented in Table A3.5.

3.3.4 Data analysis

PAH, NPAH, hopane and sterane concentrations were calculated as ng/g wet weight (ww), and then divided by (1 – moisture content) to obtain ng/g dry weight (dw). (Measurements were not corrected for spike recoveries since QA bounds were acceptable.) Sums and abundances exclude the few compounds (acenaphthene, nitrobiphenyls, 5-nitroacenaphthene, 9-nitroanthracene and 9-nitrophenanthrene) that were undetected in all samples (Table 3.1). Compounds detected in over 30% of samples were included, and the nondetect were substituted by MDL/2 (MDLs presented in Table 3.1). For those compounds (with > 30% detection frequencies), concentrations within a compound class were summed, e.g., the 15 detected PAHs, 5 detected NPAHs, 6 detected steranes and 5 detected hopanes were designated as ΣPAH_{15}, $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$, respectively. The abundance of each PAH compound was calculated as the concentration of that compound divided by the ΣPAH_{15} concentration. Abundances of NPAHs, steranes and hopanes were calculated similarly.

Concentrations of individual PAHs (ng/g dw) at each site were divided by the corresponding %OC to yield a value normalized to 1% OC. To assess potential effects on benthic organisms, the normalized concentrations were compared to consensus-based sediment quality guidelines (SQGs) (WDNR 2003).

Concentrations of ΣPAH_{15}, $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$ (ng/g dw) were plotted against OC content (Figure A3.1) and regression analyses were performed. The regression analyses excluded sites B-6 and V-1 because the sample containers broke before OC measurements could be completed, and determinations using the available samples were not considered accurate due to lack of homogenization or contamination.

Concentrations (dry weight) were divided by their OC content to obtain OC-adjusted

concentrations (expressed as μg/g OC). (Data from sites B-6 and V-1 were excluded as noted above.)

OC-adjusted concentrations across southern Lake Michigan were estimated using 2-D ordinary Kriging and a power variogram $\gamma(h_{ij}) = \alpha h_{ij}^{1.5}$ where h_{ij} = distance between two points (Deglo De Besses 2013), and then plotted as concentration maps using surface charts (Microsoft Excel 2013, Microsoft, Redmond, CA).

The loading rate of ΣPAH_{14} into southern Lake Michigan, L (t/yr) was estimated as

$$L = \left[F \cdot (A \cdot 10^{10} cm^2/km^2) \cdot \frac{100-M}{100} \cdot \frac{OC}{100} \cdot C\right] \cdot 10^{-12} t/\mu g \quad (1)$$

where F = sedimentation rate (g/cm^2-yr), A = surface area of the southern portion (km^2), M = sediment moisture content (%), OC = sediment OC content (%), C = average OC-adjusted ΣPAH_{14} concentration (μg/g OC) across the study area derived from the Kriging map, and constants provide unit conversions. Loadings of $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$ were calculated similarly. Further details on these parameters are provided in Section 3.4.1.4. This approach assumed that the degradation of these compounds in sediments was slow and the losses in one year were negligible, which was probably true since long degradation half-lives in sediments were suggested in the literature (Mackay 2010; Mackay and Hickie 2000) or estimated by EPISuite (EPA 2012). Significant uncertainties can result from using a one-compartment model that assumes the average sedimentation rate, moisture content and OC content apply to all of southern Lake Michigan, as well as the Kriging-based estimates that use a limited data set that may incompletely account for localized and near-shore discharges. Still, the approach using eq. (1) provides insight regarding total loadings to open water lake sediments from all sources.

Nine diagnostic source ratios between individual compounds were calculated to help identify major sources of target SVOCs, and are listed and interpreted in Table 3.2. Maps for each ratio were also produced using 2-D Kriging and techniques described above.

3.3.5 Chemical mass balance (CMB) modeling

CMB modeling was used to apportion PAHs in southern Lake Michigan sediments, following applications performed previously (A. Li et al. 2003; Christensen et al. 1999; P. C. Van Metre and Mahler 2010). This approach assumes that the concentration of each chemical species measured at a receptor is linear combination of the contributions from various sources.

The EPA-CMB v8.2 software (EPA 2004a) with inputs including source profiles (described below) and experimentally measured PAH concentrations in Lake Michigan sediments, in ng/g dw (Table A3.9). The precision of each measurement, used in the model, was determined from duplicate laboratory analyses, and calculated as the average percent difference between duplicates (which ranged from 17% to 49% among the 16 PAHs) multiplied by the measured concentration.

Twelve PAH source profiles were considered (Table A3.10). They include eight coal- and traffic- related profiles based on a comprehensive compilation (A. Li et al. 2003), an industrial boiler profile that represents the average of four boiler types (heavy oil, diesel, heavy oil + natural gas and coke oven gas + blast furnace gas) (C.-T. Li et al. 1999), and a fireplace combustion profile for burning pine wood (Schauer et al. 2001). Two profiles for coal-tar sealed pavement dust were also included: the mean profile across six cities (Minneapolis, MN; Chicago, IL; Detroit, MI; Washington, D.C.; New Haven, CT and Austin, TX), and the Chicago profile (P. Van Metre et al. 2008). All of these profiles used PAH measurements in the particulate phase except the boiler profile, which included both vapor and particulate phases. The profiles combined BBF and BKF given the difficulties separating these two compounds (A. Li et al. 2003). An uncertainty of 40% was applied to each component of each profile (P. C. Van Metre and Mahler 2010; A. Li et al. 2003).

For the EPA-CMB v8.2 software, the maximum number of iterations was set at 20 (maximum allowable), and a maximum source uncertainty of 50% was used, exceeding the default (20%) given the higher uncertainties expected for PAHs in sediments due to complex transport and transformation processes (A. Li et al. 2003). The minimum source projection was 0.95 (default value), below which the source is considered as “inestimable,” and the option “source elimination” was selected, which eliminated physically impossible negative source contributions.

3.4 Results and discussion

The physical characteristics and SVOC concentrations at the 24 Lake Michigan sediment samples are presented in Table 3.3, and relative abundances of individual PAHs, NPAHs and biomarkers are shown in Figure 3.2. Maps of OC-adjusted ΣPAH_{15}, $\Sigma NPAH_{5}$, $\Sigma Sterane_{6}$ and $\Sigma Hopane_{5}$concentrations are displayed in Figure 3.3.

3.4.1 Concentrations and distributions

3.4.1.1 PAHs

Of the 16 target PAHs, 14 were detected at concentrations above MDLs in all samples. Acenaphthene was not detected in any sample, and fluorene was detected in only 9 of 24 samples above MDL (0.033 ng/g dw) with a maximum concentration of 1.1 ng/g dw (site B-5). Sites with detectable fluorene levels were dispersed and did not show any clear patterns. Three- and four-ring compounds were most abundant, e.g., phenanthrene, fluoranthene, pyrene and chrysene, and accounted for about 80% of ΣPAH_{15}, while five- and six-ring PAHs constituted the remainder. PAH profiles were similar across sites, as shown by Figure 3.2 and the correlation between concentrations at different sites (Table A3.6).

ΣPAH_{15} concentrations varied from 213 to 1291 ng/g dw among sites, and were significantly correlated with OC content ($p < 0.0001$, $R = 0.79$, $n = 22$; Figure A3.1a), as shown previously (Liang et al. 2007; X. Zhang et al. 1993). This correlation reflects the tendency for PAHs and other hydrophobic organic chemicals to sorb to organic matter in bottom sediments (Karickhoff 1981). The OC-adjusted concentrations better reflect spatial differences in the sediments. Samples collected closest to the AOCs, specifically sites H-8, S-2, X-1, H-28 and A-1 corresponding to Waukegan Harbor, Grand Calumet, Muskegon Lake, Kalamazoo River and the St. Joseph River estuary, respectively, had significantly higher ΣPAH_{15} concentrations (86 ± 43 μg/g OC) than off-shore sites (29 ± 16 μg/g OC; $t = 2.95$, $df = 4.3$, $p = 0.039$).

The concentration map (Figure 3.3a) shows a clear pattern of declining concentration with increasing distance from the shore. This map shows the mean values from the kriging interpolation and does not include the confidence intervals. In addition, concentrations in areas with few or no data points and near the coast can have large uncertainties. For example, the southwest near-shore regions near Chicago and Gary had the highest interpolated mean ΣPAH_{15} levels (160 to 180 μg/g OC). Gary is near an AOC, the Grand Calumet River, that is heavily contaminated with PAHs, as well as Indiana Harbor and the Ship Canal (EPA 2013; Nevers et al. 2013; MacDonald et al. 2002). Chicago is a large and highly industrialized city with potentially significant emissions from traffic, diesel engines, coke ovens, coal combustion, wood burning (Bzdusek et al. 2004; Simcik et al. 1999), and coal-tar pavement sealcoat (P. C. Van Metre and Mahler 2010). Measurements in sediments of the Grand Calumet region include ΣPAH_{17} levels

from 123 to 500 μg/g OC (4.9 to 20 μg/g dw, assumed 4% OC) in adjacent Lake Calumet (discharging into the Calumet River and then into Lake Michigan) (A. Li et al. 2003), mean ΣPAH_{16} concentrations of 1400 μg/g OC at Indiana Ridge Marsh, 2700 μg/g OC at the Indiana Ship Canal, 630 μg/g OC at the Little Calumet River, and 40 μg/g OC at Big Marsh (all converted from μg/g dw assuming 4% OC) (Levengood and Schaeffer 2011). These levels exceed the off-shore kriging estimates near Chicago and Gary by two- to ten-fold. Since the present study uses only 24 open-water sites with water depths greater than 10 m, and no samples were taken directly from areas near AOCs and urban and industrial discharges, the mapping results apply to only open water areas. While estimated concentrations appear reasonable, additional measurements are desirable for confirmation.

Time trends are examined using data from five earlier studies that measured PAH levels in Lake Michigan surficial sediments during the 1980s and 1990s. Three studies examined open water areas (Eadie et al. 1982; Helfrich and Armstrong 1986; Simcik et al. 1996), and two examined Green Bay (Su et al. 1998; X. Zhang et al. 1993), which is separated from the open water and near the industrial city of Green Bay and two AOCs. The six PAHs measured in common across the studies (phenanthrene, fluoranthene, pyrene, chrysene, benzo[a]pyrene (B[a]P), and benzo[ghi]perylene) were summed (designated ΣPAH_6) and are depicted in Figure 3.4. ΣPAH_6 concentrations in open water sediments declined from 1980 to 2011, and a linear regression using mean concentrations and five time points indicates an average decrease of 42 ± 5 ng/g per year (Figure 3.4; $R^2 = 0.96$). This trend likely reflects the decreasing PAH loadings into the lake, a result of lower airborne concentrations and atmosphere deposition rates, as observed in gaseous, particulate and precipitation sampling in Chicago (Sun et al. 2006a; Sun et al. 2006b), in vapor phase sampling at remote sites including Eagle Harbor and Sleeping Bear Dunes (Sun et al. 2006b), and as derived from sediment cores (Schneider et al. 2001; Simcik et al. 1996). The decline has been attributed to the transition from coal to oil and natural gas, controls on industrial emissions, reduced coke production, and changed coking technology (Simcik et al. 1996; Schneider et al. 2001); it may also reflect decreased emission rates from vehicles occurring since the 1970s (Beyea et al. 2008). The trend also might reflect analytical changes that have increased the resolution and sensitivity of measurements; sampling and analysis of sediment cores would be useful to confirm results.

Green Bay sediments have higher ΣPAH_6 concentrations that do not fit the trend line in

Figure 3.4. Green Bay is near two AOCs (Menominee River and Fox River Lower Green Bay) and an industrial city. High PAH concentrations due to coal tar wastes have been detected in Menominee River sediments, and leaking underground storage tanks have been a concern for the lower Fox River Basin (EPA 2013). The proximity to PAH sources likely explains the higher PAH concentrations seen in these sediments.

The PAH concentrations in open water sediments of southern Lake Michigan in the present study (213 to 1291 ng/g dw) are generally comparable to levels elsewhere, although lower than levels in highly polluted areas. For example, sediment ΣPAH_{19} concentrations in Isle Royale National Park, Lake Superior ranged from 17 to 346 ng/g dw at background sites, and 1516 to 3410 ng/g at marinas and docks (Cox and Clements 2013). Sediment ΣPAH_{13} concentrations reached 14 µg/g dw in Detroit River, but ranged from 0.25 to 2.0 µg/g in other parts of the Huron-Erie Corridor (Szalinska et al. 2011). In two headwater lakes of the Athabasca Oil Sands Region, Canada, ΣPAH_{16} concentrations ranged from 100 to 320 ng/g dw throughout the sediment cores (Jautzy et al. 2013). ΣPAH_{15} concentrations in Lake Taihu sediments in highly populated eastern China were 209 to 1003 ng/g dw (Y. Zhang et al. 2012). Surface sediments from Lake Koumoundourou, Greece had ΣPAH_{14} concentrations from 780 to 3600 ng/g dw (Hahladakis et al. 2013).

Normalized PAH concentrations were compared to consensus-based SQGs to assess potential effects on benthic organisms using recommendations by the Wisconsin Department of Natural Resources (WDNR 2003) (Table A3.8). For most individual PAHs and the PAH sum, concentrations were lower than threshold effect concentrations (TECs), indicating that the toxicity effect to benthic-dwelling organisms is unlikely. However, concentrations of pyrene, chrysene, and dibenzo[a,h]anthracene at site S-2 (259, 187, and 36 ng/g, respectively), and pyrene, benz[a]anthracene and chrysene at site H-28 (193, 134, and 183 ng/g) exceeded TECs, as did dibenzo[a,h]anthracene at sites A-1 and H-15 (47 and 38 ng/g). However, concentrations were much lower than midpoint effect concentrations (MECs) and probable effect concentrations (PECs), suggesting a low risk to the benthic organisms. The B[a]P toxic equivalents were not calculated for these samples given that the bottom sediments were too deep for human exposure and no other human exposure pathway was plausible.

3.4.1.2 NPAHs

Five of the 11 target NPAHs (1-nitronaphthlene, 2-nitronaphthalene, 2-nitrofluorene, 1-nitropyrene and 6-nitrochrysene) were detected above MDLs in all samples; the six other NPAHs were never detected. $\Sigma NPAH_5$ concentrations ranged from 2.9 to 18.6 ng/g dw (Table 3.3), roughly 10 to 100 times lower than the ΣPAH_{15} concentrations. (NPAH data at each site are presented in Table A3.13). The two most abundant compounds, 1-nitropyrene and 6-nitrochrysene, respectively comprised an average of 33% and 40% of the $\Sigma NPAH_5$ concentration (Figure 3.2b). The abundance of 6-nitrochrysene is noteworthy given its high carcinogenic potency, i.e., toxic equivalent factor or TEF = 10 relative to B[a]P (RIDEM 2008). Average abundances of the other NPAHs detected were below 10%.

Like the PAHs, $\Sigma NPAH_5$ levels were significantly correlated with OC content ($p < 0.0001$, $R^2 = 0.85$, n = 22, Figure A3.1b). OC-adjusted $\Sigma NPAH_5$ concentrations were higher at sites near AOCs and urban/industrial areas (1.33 ± 0.95 μg/g OC) compared to other sites (0.43 ± 0.28 μg/g OC; Mann-Whitney U = 10, p = 0.005). The concentration map again shows declining concentrations with increasing distance from the shore, and high concentrations near source areas (Chicago/Gary and Kalamazoo River) (Figure 3.3b), suggesting that urban and industrial sources are important in near-shore environments. Again, the map shows only mean values, and near shore areas are subject to large uncertainties. 2-Nitronaphthalene, 1-nitropyrene and 6-nitrochrysene were highly correlated with each other, while 1-nitronaphthalene was significantly correlated with 2-nitrofluorene (Table A3.6).

NPAH concentrations in the low ng/g range have been reported in several studies examining both freshwater and marine sediments. $\Sigma NPAH_5$ (1- and 2-nitronaphthalenes, 1-nitropyrene, 6-nitrochrysene and 9-nitrophenanthrene) in Elbe river, Germany and Czech Republic ranged from 5.4 to 14.9 ng/g (Lübcke-von Varel et al. 2011). In sediments of the Suimon River, Japan, 1-nitropyrene and 2-nitrofluorene averaged 25 and 1.5 ng/g, respectively (Sato et al. 1985). Marine coastal sediments near Barcelona, Spain had 1-nitropyrene and 6-nitrochrysene concentrations of 0.68 and 0.52 ng/g dw, respectively (Fernandez et al. 1992). $\Sigma NPAH_3$ (9-nitroanthracene, 1-nitropyrene and 6-nitrochrysene) concentrations in Hiroshima Bay, Japan averaged 2.2 ng/g dw (n=11; range: 0.25 to 7.34) (Ozaki et al. 2010). These levels are roughly comparable to those measured in southern Lake Michigan in the present study ($\Sigma NPAH_5$

average of 8.0 ± 3.9 ng/g, range from 2.9-18.6 ng/g dw, n = 24). Much higher levels were measured in sediments of a small municipal stormwater settling pond in Varney Pond, MN, e.g., 1-nitropyrene, 2-nitrofluorene and 6-nitrochrysene ranged from 19-120, 40-710, and 73-150 µg/g dw, respectively (MDH 2011), which suggests the significance of urban runoff sources.

3.4.1.3 Biomarkers

All target hopanes and steranes in all samples were detected above MDLs. $\Sigma Hopane_5$ concentrations ranged from 98 to 355 ng/g dw and $\Sigma Sterane_6$ levels from 6.2 to 36 ng/g dry weight. Like PAHs and NPAHs, levels were correlated with OC content ($p < 0.0001$, $R = 0.80$ and 0.83 for steranes and hopanes, respectively; $p < 0.0001$) (Figures A3.1c and d). OC-adjusted $\Sigma Sterane_6$ and $\Sigma Hopane_5$ concentrations displayed spatial patterns similar to the PAHs (Figures 3.3c and d), and concentrations were significantly higher near source areas (sites H-8, S-2, X-1, H-28 and A-1) than other sites (for $\Sigma Hopane_5$: Mann-Whitney $U = 12$, $p = 0.009$; for $\Sigma Sterane_6$: $t = 2.62$, $df = 4.41$, $p = 0.053$). Among steranes, 20R-5α(H),14β(H),17β(H)-24-methylcholestane and 20R-5α(H),14β(H),17β(H)-cholestane were most abundant, contributing to 36% and 23% of the total, respectively. Among hopanes, 17α(H),21β(H)-hopane was most abundant (35% of $\Sigma Hopane_5$); the four other hopanes had similar contributions (16-17%). The profiles of the biomarkers were similar across sites (Figure 3.4), and individual hopane and sterane compounds were significantly correlated to each other (Table A3.7).

3.4.1.4 Loading rates of SVOCs

The loading rate of ΣPAH_{15} to open water sediments of southern Lake Michigan, estimated using eq. (1), the spatial average OC-adjusted ΣPAH_{15} concentration (41 µg/g OC), a mass sedimentation rate of 0.0356 g/cm^2-yr for southern Lake Michigan (Corcoran 2013), the average moisture content of 41.5% and OC content of 2.2% (this study), and the estimated surface area of the southern portion (21600 km^2), is 4.1 t/yr. Although we did not measure concentrations in the northern portion of the lake, an approximate lake-wide loading rate is calculated by scaling up to the entire Lake surface area (58000 km^2), which gives a ΣPAH_{15} loading of 10.9 t/yr. Using the same approach, lake-wide loadings of $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$ are 0.16, 0.25 and 3.6 t/yr, respectively. The historical decline in PAH levels must be considered to compare these results to earlier loading estimates. A 1980 estimate of PAH loadings to Lake Michigan, obtained by summing the PAH inputs from various sources (some of

which had inconsistent PAH species) is 50 to 55 t/yr (Helfrich and Armstrong 1986). Using the regression line for PAH levels in Lake Michigan sediments shown in Figure 3.4, ΣPAH_6 concentration in 1980 (1679 ng/g dw) was 4.6 times the level in 2011 (368 ng/g dw). Assuming that ΣPAH_{14} concentrations follow the ΣPAH_6 trend, and that sedimentation rate, sediment moisture content, and OC content are unchanged, then the ΣPAH_{15} loading rate in 1980 would be 4.6 times the current (2011) loading or 50.6 t/yr, within the range reported for 1980. A second and independent estimate of PAH loadings, based on accumulation profiles in sediment cores sampled in 1991-1993 (and calculated using a ΣPAH_{17} deposition rate = 50-70 ng/cm^2-yr and area = 58000 km^2), is 29 to 41 t/yr (Simcik et al. 1996). For 1992, we estimate a ΣPAH_{15} loading rate of 35.2 t/yr, again within the reported range.

While the estimated ΣPAH_{15} loading rates agree well with the earlier estimates, uncertainties and possible errors in our estimates are recognized. Overall, the southern portion of the lake is more polluted than the northern, thus applying the average concentration in the southern portion to the whole lake may overestimate loadings. However, this may be offset since near-shore areas that tend to have higher concentrations were under-sampled. Second, sedimentation rate, sediment moisture content and OC content can vary across sites, and applying the average data from the southern portion to the whole lake may be inaccurate. Third, sampling and measurement variation can affect results, although open water sediment samples provide a high degree of spatial and temporal representativeness, particularly compared to airborne and deposition samples that can vary considerably. Given the difficulty in assessing these factors, especially the extrapolation to the whole lake, no quantitative estimates of uncertainty is provided. Despite these concerns, our loading estimates show remarkable agreement with earlier estimates that used independent methods. Moreover, the stability and representativeness of sediment samples suggests that this approach for estimating SVOC loadings is useful and applicable to other persistent and sediment-bound contaminants. However, the uncertainties of the approach, especially in near shore areas, should be recognized.

3.4.2 Source identification

3.4.2.1 PAHs

Diagnostic ratios

PAH sources contributing to southern Lake Michigan sediments are identified using

abundances and ratios of individual compounds, which serve as source indicators. Eight diagnostic ratios for identifying PAH sources (Table 3.4) are used to help identify sources. These ratios are semi-quantitative (i.e., numerical apportionments are not provided), and they are most useful when there is a single dominating source, which is unlikely in a large region like Lake Michigan. In addition, the ratios can be influenced by atmospheric reactions and selective loss processes, and thus may provide contradictory or inconsistent results (Katsoyiannis et al. 2011). Despite such limitations, the ratios can provide useful insights to contributing sources.

The BAA/CHR ratio is an indicator of urban influences by reflecting differences in the susceptibility to photo-oxidation (Table 3.2) (Gschwend and Hites 1981). The average BAA/CHR ratio (0.72 ± 0.23) is comparable with previous studies and within the range reported for sediments collected near urban areas (Gschwend and Hites 1981; Helfrich and Armstrong 1986). Site B4, most distant from urban sources (54 km from shore, 54 km from Benton Harbor and 175 km from Chicago), had the second lowest BAA/CHR ratio (0.57). However, this ratio was not significantly correlated with either distance from shore or to AOCs, industrial or urban areas, and there was no clear spatial pattern (see Figure A3.2c), as has been noted previously (Helfrich and Armstrong 1986). This lack of correlation can arise from several factors. First, the southern Lake Michigan contains several cities/harbors/AOCs, and the off-shore samples likely are affected by multiple sources. Second, this ratio does not apply to waterborne transport, e.g., materials discharged or deposited in near-shore areas and then transported and distributed to sediments (Simcik et al. 1999). Third, there is movement and redistribution of surface sediments in southern Lake Michigan (Corcoran 2013). Finally, as noted, this ratio could be influenced by other physiochemical processes (Katsoyiannis et al. 2011).

The remaining seven PAH ratios indicate petrogenic or pyrogenic sources (Table 3.2) (Yunker et al. 2002; Budzinski et al. 1997; Wang et al. 2006). PHE/ANT and FLA/PYR ratios are usually used simultaneously (Budzinski et al. 1997). A plot of PHE/ANT vs. FLA/PYR ratios suggests that pyrogenic (i.e., combustion) sources are the main contributor of PAHs at most (N=14) sites (Figure 3.5). Seven sites reflect a combination of petrogenic and pyrogenic sources. Three sites are in the petrogenic zone, but at the boundary, suggesting significant contributions from pyrogenic sources. The ANT/178 ratio averaged 0.12 ± 0.04 (N = 24) and several sites had ratios just slightly below 0.10. The FLA/202 ratio averaged 0.54 ± 0.13 (n=24); 17 sites had ratios >0.50; six sites had ratios between 0.40 and 0.50; and only one site had a ratio <0.4. All

sampling sites had BAA/228 ratios above 0.35, IcdP/276 ratios above 0.50, and ∑LPAHs/∑HPAHs ratios below 1. Together, these ratios suggest the dominance of pyrogenic (combustion) sources (Table 3.2). Moreover, the fact that all sites had IcdP/276 ratios above 0.50 and 17 sites had FLA/202 ratios above 0.50 indicated significant contribution from coal, wood or grass combustion; the FLA/202 ratio ranging between 0.40 and 0.50 at 6 sites suggested the contribution from liquid fossil fuel combustion, i.e., vehicle emissions (Table 3.2).

Although the PAH diagnostic ratios are qualitative and have limitations, they consistently identify pyrogenic sources as the major contributors of PAHs to southern Lake Michigan sediments. This is in accordance with previous apportionment studies, which identified traffic emissions, coal combustion and wood burning as major sources (Bzdusek et al. 2004; Christensen and Arora 2007; Simcik et al. 1996).

CMB results

Collinearity among the twelve PAH source profiles and the mean PAH profile in southern Lake Michigan sediments, initially evaluated using Pearson correlation coefficients (Table A3.11), showed several profiles were similar to the 24-site average profile, including those for coal-tar Chicago, pine-wood combustion, power plant, traffic average, and diesel engine exhaust (r = 0.93, 0.91, 0.89, 0.89 and 0.87, respectively). Several source profiles were significantly correlated, suggesting the possibility of multicollinearity issues, e.g., unstable source estimates and convergence issues. To investigate and help account for such concerns, ten CMB models using different fitting species and subsets of source profiles were tested. Each was run 24 times (one for each site), and the "preferred" model discussed here was selected as the model that converged at all 24 sites and showed good performance in terms of R^2, χ^2 and percentage of ΣPAH mass explained (Table A3.12). This model included four sources: coal-fired power plant, coke oven, diesel engine and coal-tar pavement dust Chicago. Since all four traffic-related profiles were highly correlated, the diesel engine profile is considered to represent traffic-related sources. Including industrial boiler and pine-wood combustion profiles caused convergence issues at many sites. This model included nine fitting species, specifically, PHE to BghiP with the exceptions of ANT and BAP, two compounds that are highly reactive in the atmosphere (A. Li et al. 2003; Gschwend and Hites 1981).

Using the selected four-source model, diesel engine exhaust was identified at all sites as the most significant source, contributing to 56 ± 18% of the ΣPAHs; coal power plant emissions were identified (source contribution larger than zero) at 22 sites, contributing 27 ± 14%; coal-tar sealed pavement dust was identified at 19 sites with a 16 ± 11% contribution; and coke oven was detected at 11 sites with a 7 ± 12% contribution (Table A3.12). These results are consistent with the diagnostic ratios as well as previous apportionment studies highlighting the importance of pyrogenic sources (e.g., traffic emissions and coal combustion) (Simcik et al. 1996; Bzdusek et al. 2004; Christensen and Arora 2007). While coal-tar sealed pavement dust was identified, its contribution was low compared to the 57% estimated for urban lakes in the eastern U.S. (P. C. Van Metre and Mahler 2010), possibly reflecting the significance of atmospheric deposition in Lake Michigan as compared to urban runoff, the principal pathway for coal-tar sealcoat dust.

Across the 24 sites, the CMB model performance was reasonable, e.g., R^2, χ^2 and percentage mass explained averaged 0.81, 3.1 and 106%, respectively (Table A3.12). However, CMB apportionments in urban lakes have attained better performance, e.g., $R^2 > 0.9$ and $\chi^2 < 2$ (P. C. Van Metre and Mahler 2010; A. Li et al. 2003). Given the potentially longer transport distances and times relevant to Lake Michigan, the assumption that profiles remain constant from source to receptor (sampled sediment) (EPA 2004b) may not reflect chemical, photochemical and biological degradation, as well as partitioning, that occurs during atmospheric transport, deposition and sediment burial (A. Li et al. 2003; Galarneau 2008). These concerns may be mitigated for urban lakes near sources. Target transformation and other techniques (Thurston and Spengler 1985; Pistikopoulos et al. 1990) might be used to help address such issues.

3.4.2.2 NPAHs

Few diagnostic source ratios are available for NPAHs. The 1-NPYR/PYR ratio has been used to distinguish contributions of diesel-engine vehicles and coal combustion emissions in both atmospheric particulate matter and marine sediments (Ozaki et al. 2010; Tang et al. 2005). This ratio is approximately 0.001 for lower temperature (900 °C) coal stove emissions , and 0.36 for higher temperature (2700 °C) diesel engine exhaust (Tang et al. 2005). Although the type of coal stove sampled was not stated, this ratio may apply to coal combustion in smaller furnaces that has a lower temperature than diesel engines. Coal and wood stoves/fireplaces are used for residential heating or aesthetics around Lake Michigan area, although wood stoves may be more

popular. In Lake Michigan sediments, the 1-NPYR/PYR ratio averaged 0.03 ± 0.01 (range from 0.01 to 0.08, N = 24, Table 3.4), which is comparable to levels reported in marine sediments (0.017 – 0.023) (Ozaki et al. 2010), suggesting a combination of diesel engine and coal combustion emissions, consistent with the sources identified for the parent PAHs. This ratio showed a complex spatial pattern (Figure A3.2h), possibly due to the instability of diagnostic ratios in the environment, as discussed above.

The NPAH profiles provide additional source information. In Lake Michigan sediments, 1-nitropyrene and 6-nitrochrysene were the predominant compounds, followed by 2-nitrofluorene. In the atmosphere, these compounds are primarily emitted by diesel vehicles (Albinet et al. 2007; Bamford and Baker 2003; Reisen and Arey 2005), and an airborne 2-nitrofluorene concentration equal to 15% of that of 1-nitropyrene is commonly observed (Albinet et al. 2007). Thus, levels of these compounds may suggest contribution from diesel engine exhaust. On the other hand, sediment concentrations of 6-nitrochrysene were slightly higher than those of 1-nitropyrene, while concentrations of 6-nitrochrysene generally are lower than those of 1-nitropyrene in diesel and gasoline engine emissions, tire debris and asphalt paste (Khalek et al. 2011; Ozaki et al. 2010). However, atmospheric formation of 6-nitrochrysene has been shown by exposing chrysene to 10 ppm of nitrogen dioxide (NO_2) (Tokiwa et al. 1981). Chrysene is a marker of coal combustion (Harrison et al. 1996), and the most abundant PAH in coal power plant emissions (Bzdusek et al. 2004). States surrounding Lake Michigan, including Illinois and Indiana, have numerous coal-fired power plants. Thus, we speculate that a portion of the chrysene emitted from coal combustion sources undergoes atmospheric transformation into 6-nitrochrysene, followed by deposited in the lake and incorporation into sediments. However, further studies are needed to confirm this pathway.

3.4.2.3 Biomarkers

Anthropogenic sources of hopanes and steranes in Lake Michigan include crude oil and derived products, e.g., engine lubricating oil. Although major oil spills have not been reported in southern Lake Michigan, it may be subject to some oil spillage since Indiana Harbor and Chicago are major distribution centers for petroleum products (Helfrich and Armstrong 1986). In addition, hopanes and steranes are present in lubricating oils of diesel and gasoline engines, and are components of vehicle exhaust (Liu et al. 2010; Khalek et al. 2011; Schauer et al. 2002,

1999). Similarly, outboard motor oil and exhaust from ships/boats are potential sources (Bieger et al. 1996). Sediments can also reflect natural sources of hopanes and steranes including decomposition of bacteria, algae and vascular plants (Qu et al. 2007; Xiong et al. 2010).

Unlike PAHs and NPAHs, diagnostic ratios of hopanes and steranes cannot distinguish between petrogenic and pyrogenic sources because these compounds are largely conserved during combustion and environmental transport (Manan et al. 2011; Neff and Durell 2012). However, these ratios can distinguish petrogenic and biogenic hydrocarbons (Hostettler et al. 1992; Neff and Durell 2012; Qu et al. 2007; van Dongen et al. 2008). One such ratio can be calculated considering the hopanes and steranes measured in the present study. This ratio, C_{31}-22S/(22S+22R), compares two diastereomers of C_{31}-homohopanes (17α(h), 21β(h)-homohopanes), and a value of 0.6 indicates equilibrium or full maturity, i.e., petroleum (Table 3.2). In Lake Michigan, this ratio ranged from 0.37 to 0.65; four sites had ratios >0.6; seven sites had ratios from 0.5 - 0.6; and 13 sites had ratios <0.5. The ratio map shows that the lower half of the study area tends to have ratios > 0.5, while the upper half generally displays ratios < 0.5 (Figure A3.2i). This suggests that the southern end of the lake was significantly impacted by petroleum (crude oil, vehicle emissions, etc.), while biogenic inputs (vascular plants, microbes) become more important in the central and northern parts. While consistent with earlier results, the ratio map does not represent sampling and analytical variability, and a single diagnostic ratio might not be robust, thus, measurement of additional compounds and calculation of several diagnostic ratios in future studies would help to confirm results.

3.5 Conclusions

Four groups of SVOCs were measured in sediments collected at 24 off-shore sites in southern Lake Michigan of varying water depths and sediment types. Compared to the 3-5 sampling sites used in previous studies, data from 24 sites better describe the spatial patterns and the influence of potential source areas, and provide more robust estimates of levels across the lake. ΣPAH_{15} concentrations, which ranged from 213 to 1291 ng/g dw, were highest at near-shore industrialized and contaminated areas. Overall, PAH levels in sediments have been declining for the past three decades, and only low risks to benthic organisms are indicated using consensus-based SQGs. We provide the first report of NPAHs in Lake Michigan sediments. $\Sigma NPAH_5$ concentrations ranged from 3 to 19 ng/g dw, and several highly toxic compounds were

detected, including 6-nitrochrysene. $\Sigma Hopane_5$ and $\Sigma Sterane_6$ concentrations ranged from 98 to 355 ng/g and 6 to 36 ng/g dw, respectively, and several petroleum-specific hopanes were detected. OC-adjusted SVOC concentrations increase at locations near AOCs and larger urban/industrial areas.

The estimated 2011 loading rates of ΣPAH_{14}, $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$ to open water sediments of Lake Michigan are 10.9, 0.16, 0.25 and 3.6 t/yr, respectively. The PAH loading estimate has excellent agreement with prior estimates obtained using different and independent methods; loading rates for the other three compound groups are the first presented in the literature. Relative abundances were similar across sites, indicating that common source types affected sediments across southern Lake Michigan. Based on diagnostic ratios and chemical mass balance modeling, PAHs were contributed by primarily pyrogenic sources, e.g., coal combustion and vehicle exhaust; coal-tar sealed pavement dust was also identified. Based on hopane biomarkers, both petroleum-derived and biogenic sources are important contributors of hydrocarbons in sediments. Finally, NPAH compounds with high carcinogenic potencies (e.g., 6-nitrochrysene) were measured at relatively high concentrations in sediments, suggesting that an assessment of ecological risks may be warranted.

3.6 Tables and Figures

Table 3.1 MDLs of target compounds, and summary of study measurements.

Group	Compound	MDL (ng/g dw)	Study measurements (ng/g dry sediment)				
			Mean	Std	Min	Max	Detection freq. (%) (N = 24)
PAHs	Naphthalene	0.03	6.2	2.8	2.5	12.7	100
	Acenaphthylene	0.07	1.4	0.7	0.7	3.9	100
	Acenaphthene	0.07	-	-	-	-	0
	Fluorene	0.10	0.22	0.30	ND	1.1	37
	Phenanthrene	0.07	70.4	36.3	29.1	179.1	100
	Anthracene	0.05	9.1	4.4	3.8	18.7	100
	Fluoranthene	0.10	134.1	84.2	47.3	385.2	100
	Pyrene	0.05	110.3	57.7	25.3	236.3	100
	Benz[a]anthracene	0.05	55.8	35.3	12.0	167.4	100
	Chrysene	0.08	75.9	40.4	19.3	175.3	100
	Benzo[b]fluoranthene	0.13	28.5	13.2	13.2	71.9	100
	Benzo[k]fluoranthene	0.14	22.5	9.5	10.4	44.2	100
	Benzo[a]pyrene	0.06	2.7	2.0	1.0	10.2	100
	Dibenzo[a,h]anthracene	0.53	24.9	17.9	7.8	95.4	100
	Indeno[1,2,3-cd]pyrene	0.38	18.2	13.5	5.9	74.1	100
	Benzo[g,h,i]perylene	0.02	6.7	5.2	2.3	27.5	100
NPAHs	1-Nitronaphthalene	0.07	0.61	0.39	0.19	1.69	100
	2-Nitronaphthalene	0.09	0.52	0.42	0.13	2.15	100
	2-Nitrobiphenyl	0.07	-	-	-	-	0
	3-Nitrobiphenyl	0.06	-	-	-	-	0
	4-Nitrobiphenyl	0.27	-	-	-	-	0
	5-Nitroacenaphthene	0.02	-	-	-	-	0
	2-Nitrofluorene	0.11	0.83	0.55	0.22	2.64	100
	9-Nitroanthracene	0.02	-	-	-	-	0
	9-Nitrophenanthrene	0.01	-	-	-	-	0
	1-Nitropyrene	0.01	2.67	1.41	0.85	6.44	100
	6-Nitrochrysene	0.06	3.22	1.67	1.15	8.09	100
Hopanes	17α(h),21β(h)-Hopane	0.02	64.5	34.3	28.6	160.8	100
	17α(h)-22,29,30-Trisnorhopane	0.04	30.9	15.9	11.7	70.9	100
	17α(h),21β(h)-30-Norhopane	0.04	33.3	18.9	12.5	92.4	100
	22R-17α(h),21β(h)-Homohopane	0.02	31.0	16.5	9.9	79.6	100
	22S-17α(h),21β(h)-Homohopane	0.02	31.5	17.1	15.9	76.3	100
Steranes	20S-5α(h), 14α(h), 17α(h)-Cholestane	0.02	0.7	0.5	0.2	1.7	100
	20R-5α(h), 14α(h), 17α(h)-Cholestane	0.03	1.1	0.5	0.4	2.8	100
	20R-5α(h), 14β(h), 17β(h)-Cholestane	0.02	3.4	1.9	1.3	8.4	100
	20R-5α(h), 14β(h), 17β(h)-24-Methylcholestane	0.03	5.5	3.6	1.7	15.2	100
	20R-5α(h), 14α(h), 17α(h)-24-Ethylcholestane	0.02	2.2	1.3	1.0	5.7	100
	20R-5α(h), 14β(h), 17β(h)-24-Ethylcholestane	0.03	1.9	1.2	0.7	5.2	100

MW: molecular weight. MDL: instrument detection limit. Std: standard deviation. ND: not detected.

Table 3.2 Diagnostic ratios used to identify possible sources of target SVOCs in Lake Michigan sediments

Ratio	Definition	Interpretation	Reason	Reference
PAHs				
PHE/ANT	PHE/ANT	<10: pyrogenic; >10: petrogenic.	Difference in thermodynamic stability.	Budzinski et al., 1997
FLA/PYR	FLA/PYR	<1: petrogenic; >1: pyrogenic.	Difference in thermodynamic stability.	Budzinski et al., 1997
BAA/CHR	BAA/CHR	Decreases from urban to remote sites.	BAA is more susceptible to photo-oxidation than CHR.	Gschwend et al., 1981
ANT/178	ANT/(ANT+PHE)	<0.1: petroleum; >0.1: combustion.	Difference in thermodynamic stability.	Yunker et al., 2002
FLA/202	FLA/(FLA+PYR)	<0.4: petroleum; 0.4-0.5: liquid fossil fuel combustion; >0.5: grass, wood or coal combustion.	Difference in thermodynamic stability.	Yunker et al., 2002
BAA/228	BAA/(BAA+CHR)	<0.2: petroleum; >0.35: combustion.	Difference in thermodynamic stability.	Yunker et al., 2002
IcdP/276	IcdP/(IcdP+BghiP)	<0.2: petroleum; 0.2-0.5: liquid fossil fuel combustion; >0.5: grass, wood or coal combustion.	Difference in thermodynamic stability.	Yunker et al., 2002
∑LPAHs/∑HPAHs	sum of ≤ 3-ring PAHs / sum of > 3-ring PAHs	<1: petrogenic; >1: pyrogenic.	More larger PAHs are formed during combustion.	Wang et al., 2006
NPAHs				
1-NPYR/PYR	1-nitropyrene/pyrene	0.36: diesel engines; 0.001: coal combustion.	Formation of NPAHs increase with increasing temperature.	Tang et al., 2005
Hopanes and steranes				
C_{31}-22S/(22S+22R)	22S-17α(h),21β(h)-homohopane/[22S-17α(h),21β(h)-homohopane+22R-17α(h),21β(h)-homohopane]	0.6 indicates full maturity (petroleum); >0.5: petrogenic dominates; <0.5: biogenic dominates.	Thermal maturity indicator.	Hostettler et al., 1992

Table 3.3 Lake Michigan sampling sites, physical data and SVOC concentrations

Site	Latitude (°N)	Longitude (°W)	Water depth (m)	Distance from shore (km)	Distance to nearest AOC/industrial/urban areas (km)	Sediment type	Moisture content (%)	OC (%)	Σ_{15}PAH (ng/g dry)	Σ_{11}NPAH (ng/g dry)	Σ_{6}Sterane (ng/g dry)	Σ_{5}Hopane (ng/g dry)
A-1	42.108	86.533	16	3.4	3.8	sand	8.4	0.6	496.9	5.13	9.7	174.4
B-2	42.400	86.450	52	13.3	34.2	silt	60.1	3.5	802.7	10.52	18.1	300.2
B-3	42.400	86.592	64	24.3	42.1	silt	59.2	2.3	831.6	8.20	20.6	256.1
B-4	42.392	87.017	124	53.9	53.5	silt over loam	60.0	3.6	512.9	9.60	17.7	196.2
B-5	42.375	87.350	108	38.0	38.9	silt over loam	49.3	2.7	508.6	9.13	21.3	207.4
B-6	42.375	87.500	84	25.6	26.6	silt over loam	40.6	4.7 *	327.3	6.59	11.4	138.9
B-7	42.367	87.667	45	12.0	12.8	silty sand	33.5	0.9	312.8	4.89	6.4	103.1
C-5	42.817	86.833	136	50.8	53.1	silt	64.0	4.6	860.1	17.02	20.8	323.0
C-6	42.795	87.447	105	25.4	44.6	silt over sandy loam	39.1	1.6	421.8	5.23	8.8	113.6
C-7	42.792	87.575	60	15.0	36.8	silt over sandy loam	47.8	2.0	609.5	8.50	15.6	221.2
EG-14	42.378	86.775	98	36.7	37.7	silt	62.0	4.4	1290.7	14.48	36.0	355.0
EG-18	42.293	86.643	60	22.2	23.6	silt	47.4	2.3	588.5	6.18	19.5	255.3
H-15	42.158	87.433	60	22.9	34.1	silty sand	28.4	0.5	332.6	2.91	7.8	102.3
H-22	42.139	86.664	48	14.6	14.8	silt	60.1	2.1	903.1	6.52	24.0	332.6
H-28	42.630	86.265	22	2.9	5.2	medium to coarse sand	18.2	0.3	347.4	5.64	10.0	98.7
H-29	42.630	86.306	32	6.3	8.3	silt	39.7	1.8	364.3	6.85	7.4	114.0
H-30	42.630	86.433	70	16.7	18.6	silt	66.1	4.0	1234.5	18.59	33.3	351.1
H-31	43.041	86.333	45	6.9	21.4	silt , some sand	55.6	3.0	588.9	8.90	15.1	186.0
H-8	42.399	87.771	19	2.6	6.0	silt with some clay	28.9	0.8	451.2	5.73	11.2	116.4
S-2	41.765	87.391	13	8.5	11.7	fine sand	20.6	0.3	398.1	7.81	8.8	125.8
S-3	41.850	87.320	25	19.4	22.7	silty sand	21.6	0.5	212.8	5.01	6.2	97.7
S-4	41.935	87.252	40	30.4	33.7	silty sand	23.8	0.5	315.8	6.85	9.5	127.8
V-1	41.697	87.013	17	1.6	35.9	clay, some sand	18.3	4.6 *	329.2	3.74	6.8	111.3
X-1	43.103	86.367	45	7.9	14.7	silt	42.2	1.7	564.1	6.76	10.4	181.1
Mean							41.5	2.2	566.9	8.0	14.8	191.2
Std							17.4	1.5	286.1	3.9	8.2	88.4
Min							8.4	0.3	212.8	2.9	6.2	97.7
Max							66.1	4.7	1290.7	18.6	36.0	355.0

OC: organic carbon. Std: standard deviation.
OC values marked with an asterisk were not used in regression analyses (see Section 2.4).

Table 3.4 SVOC diagnostic ratio results (organized based on distance from shore)

Site	Distance from shore (km)	Distance to nearest AOC/industrial/urban areas (km)	BAA/CHR	PHE/ANT	FLA/PYR	ANT/178	FLA/202	BAA/228	IND/276	∑LPAHs/ ∑HPAHs	1-NPYR/PYR	C_{31}-22S/ (22S+22R)
V-1	1.6	35.9	0.69	7.9	1.5	0.11	0.60	0.41	0.77	0.19	0.016	0.62
H-8	2.6	6	0.70	6.1	1.5	0.14	0.60	0.41	0.82	0.15	0.025	0.52
H-28	2.9	5.2	0.73	3.9	1.7	0.20	0.63	0.42	0.79	0.14	0.030	0.50
A-1	3.4	3.8	0.71	7.0	1.0	0.13	0.50	0.42	0.78	0.19	0.014	0.51
H-29	6.3	8.3	0.67	6.7	1.2	0.13	0.54	0.40	0.81	0.13	0.034	0.46
H-31	6.9	21.4	0.68	6.6	1.8	0.13	0.64	0.40	0.83	0.14	0.027	0.47
X-1	7.9	14.7	0.68	11.5	1.2	0.08	0.54	0.40	0.78	0.21	0.018	0.47
S-2	8.5	11.7	0.55	6.1	1.1	0.14	0.52	0.35	0.80	0.22	0.036	0.58
B-7	12.0	12.8	0.72	10.2	0.8	0.09	0.45	0.42	0.83	0.19	0.028	0.44
B-2	13.3	34.2	0.75	10.7	1.0	0.09	0.49	0.43	0.77	0.20	0.021	0.51
H-22	14.6	14.8	0.72	6.0	1.5	0.14	0.60	0.42	0.81	0.16	0.010	0.64
C-7	15.0	36.8	0.69	9.6	1.2	0.09	0.55	0.41	0.82	0.20	0.027	0.42
H-30	16.7	18.6	0.96	6.1	1.6	0.14	0.62	0.49	0.80	0.11	0.027	0.43
S-3	19.4	22.7	0.62	13.5	1.9	0.07	0.65	0.38	0.77	0.43	0.080	0.39
EG-18	22.2	23.6	0.85	6.8	1.1	0.13	0.51	0.46	0.81	0.24	0.017	0.65
H-15	22.9	34.1	0.73	9.4	1.6	0.10	0.61	0.42	0.81	0.24	0.018	0.64
B-3	24.3	42.1	0.75	10.3	0.7	0.09	0.43	0.43	0.77	0.21	0.011	0.37
C-6	25.4	44.6	0.75	7.8	0.6	0.11	0.36	0.43	0.80	0.11	0.017	0.47
B-6	25.6	26.6	0.73	7.2	0.9	0.12	0.47	0.42	0.77	0.14	0.034	0.47
S-4	30.4	33.7	0.95	5.0	1.4	0.17	0.59	0.49	0.78	0.23	0.044	0.54
EG-14	36.7	37.7	0.71	9.6	1.6	0.09	0.62	0.41	0.78	0.20	0.021	0.49
B-5	38.0	38.9	0.55	6.4	0.9	0.14	0.47	0.36	0.77	0.20	0.024	0.49
C-5	50.8	53.1	0.77	4.2	0.9	0.19	0.46	0.44	0.78	0.13	0.032	0.60
B-4	53.9	53.5	0.57	16.7	1.5	0.06	0.61	0.36	0.77	0.30	0.046	0.43
Mean			0.72	8.1	1.3	0.12	0.54	0.42	0.79	0.19	0.027	0.51
Std			0.1	3.0	0.4	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Min			0.55	3.9	0.6	0.06	0.36	0.35	0.77	0.11	0.010	0.37
Max			0.96	16.7	1.9	0.20	0.65	0.49	0.83	0.43	0.080	0.65

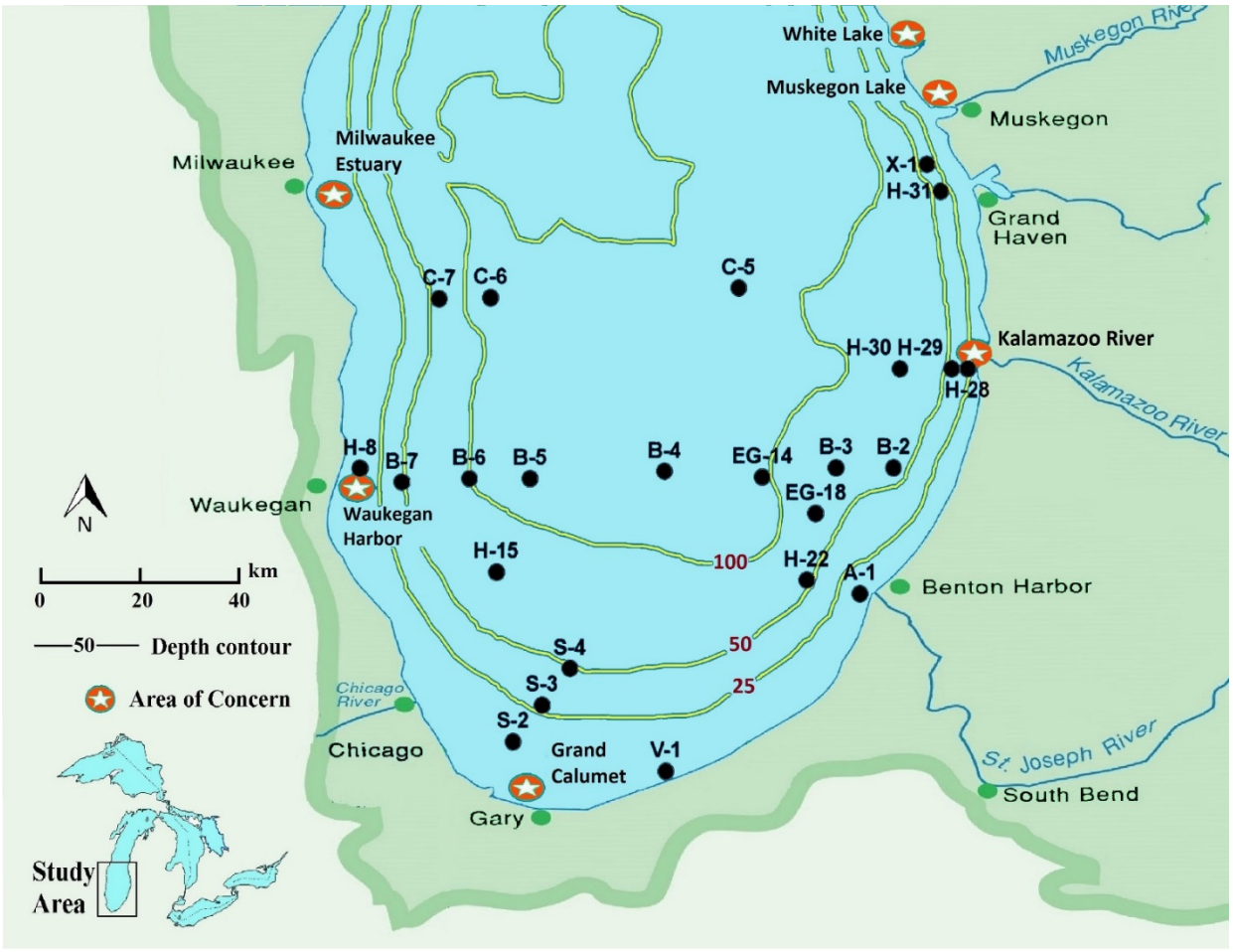

Figure 3.1 Lake Michigan sampling sites.
The green shaded area surrounding the lake indicates the drainage area.

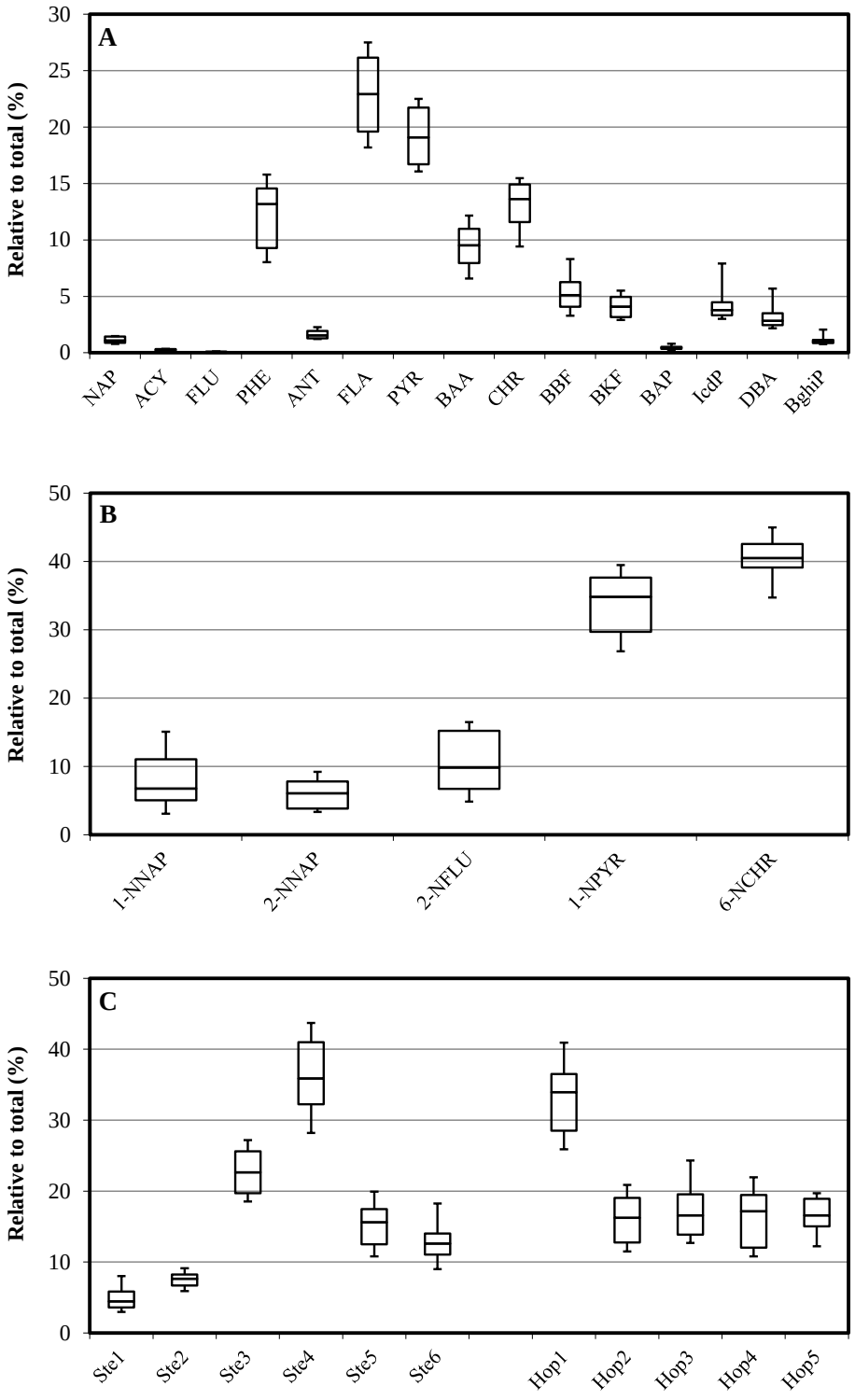

Figure 3.2 Boxplots showing the concentrations of individual (A) PAHs; (B) NPAHs; and (C) biomarkers that are normalized to the corresponding total concentrations.
Boxplots show 10^{th,} 25th, 50th, 75th and 90th percentiles.

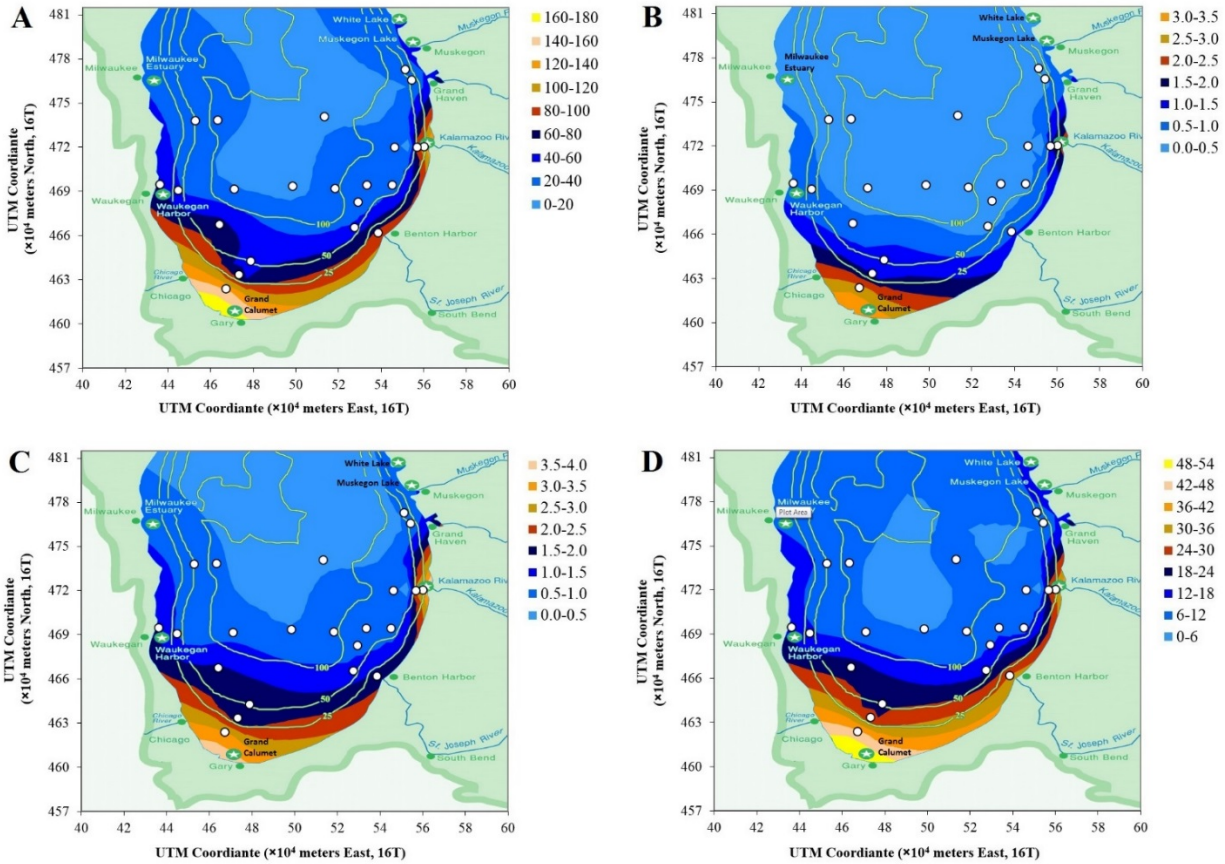

Figure 3.3 Concentration maps of OC-adjusted concentrations of (A) ΣPAH_{15}; (B) $\Sigma NPAH_5$; (C) $\Sigma Sterane_6$; and (D) $\Sigma Hopane_5$.
Units are µg/g OC. White dots indicate sediment sampling sites in this study. Graph axes show Universal Transverse Mercator coordinate system.

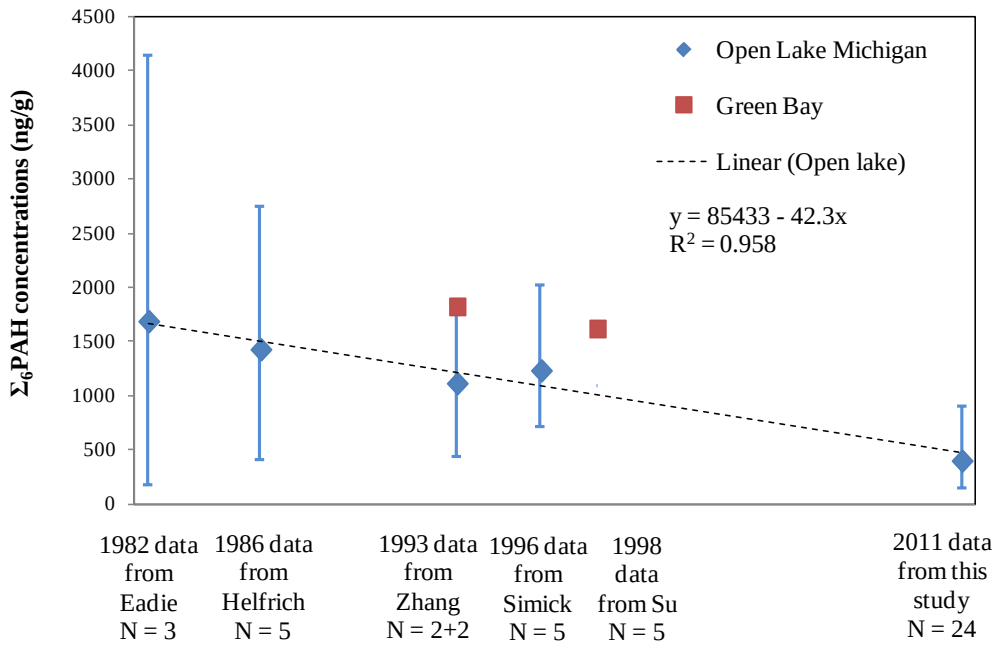

Figure 3.4 Trend of Σ_6PAH concentrations in Lake Michigan sediments. (showing mean, maximum and minimum of observations, plus regression line). $\Sigma PAH_6 = 85433 - 42.3x$, where x is time (year).

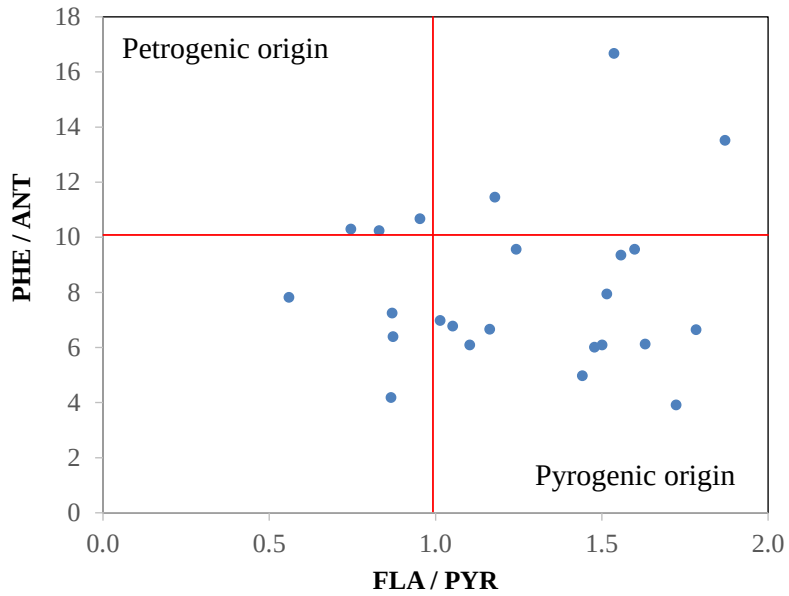

Figure 3.5 Plot of PHE / ANT ratios against FLA / PYR ratios for all samples

3.7 Appendix

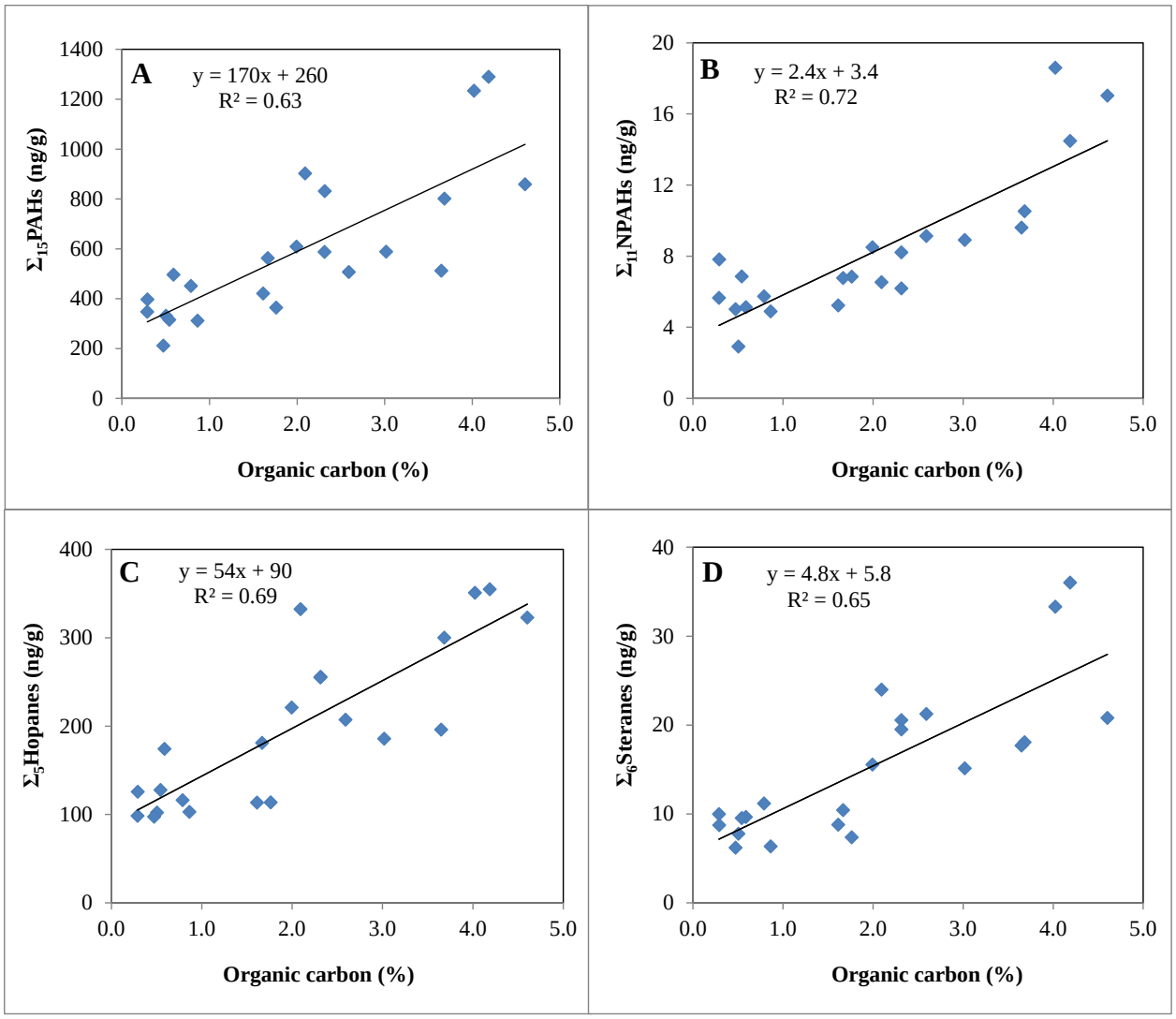

Figure A3.1 Correlation of organic carbon content with (A) ΣPAH_{15} concentrations; (B) $\Sigma NPAH_5$ concentrations; (C) $\Sigma Hopane_5$ concentrations; and (D) $\Sigma Sterane_6$ concentrations.

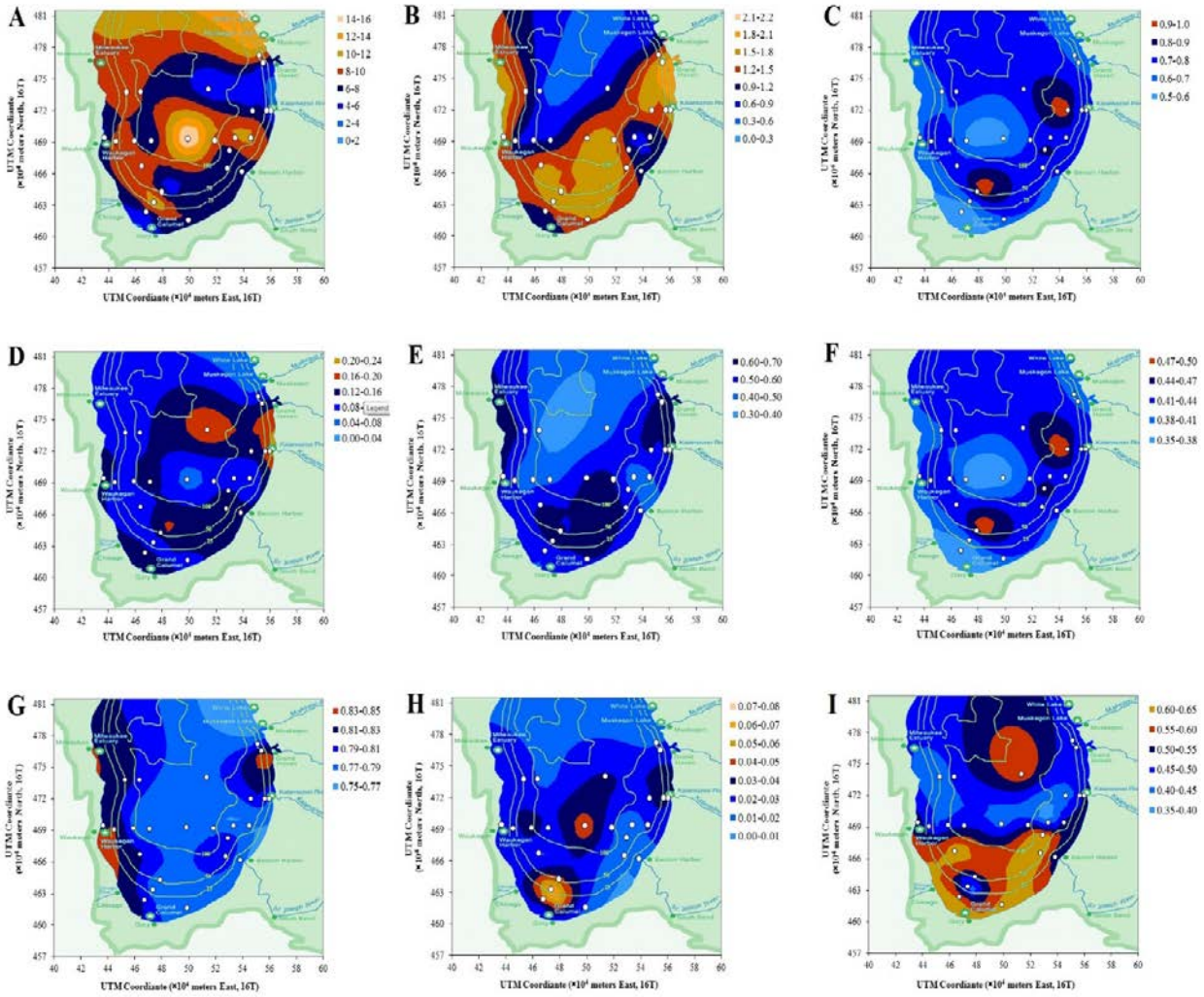

Figure A3.2 Maps of diagnostic ratios.
(A) PHE/ANT; (B) FLA/PYR; (C) BAA/CHR; (D) ANT/178; (E) FLA/202; (F) BAA/228; (G) IND/276; (H) 1-NPYR/PYR; and (I) C_{31}-22S/(22S+22R). Ratios are unitless. White dots indicate sediment sampling sites in this study. Graph axes show Universal Transverse Mercator coordinates.

Table A3.1 Analytical results of lab replicates and blanks for PAHs (ng/mL)

Sample	NAP	ACY	ACT	FLU	PHE	ANT	FLA	PYR	BAA	CHR	BBF	BKF	BAP	IcdP	DBA	BghiP
A-1	53.2	7.6	nd	nd	416	58.2	736	689	305	433	113	97	42.6	304	204	81
A-1 rep	47.5	8.5	nd	nd	453	66.2	673	699	284	396	116	90	35.2	327	188	101
B-2	52.5	8.4	nd	3.6	449	43.6	693	734	376	494	143	139	13.9	99	74	29
B-2 rep	44.4	8.0	nd	nd	452	40.3	677	701	352	482	138	127	9.2	95	74	30
B-5	24.4	5.9	nd	5.3	329	52.2	488	542	207	373	115	95	13.0	93	73	26
B-5 rep	22.3	5.2	nd	5.1	305	47.1	452	533	203	367	95	93	7.1	90	69	28
EG-14	29.2	6.4	nd	nd	450	43.1	827	518	246	356	104	90	26.7	232	183	63
EG-14 rep	35.6	7.2	nd	nd	443	52.6	845	528	272	372	112	95	23.5	248	189	77
H-28	34.5	7.3	nd	nd	222	56.1	725	419	285	384	167	104	9.4	89	65	19
H-28 rep	37.8	8.3	nd	nd	226	58.3	735	428	302	419	173	119	5.5	92	65	29
V-1	23.6	6.5	nd	4.1	427	53.9	815	534	224	313	196	99	43.0	294	201	84
V-1 rep	28.6	7.3	nd	4.2	442	55.3	834	555	194	301	188	113	29.3	295	197	92
Field blank	5.8	nd	nd	nd	nd	nd	nd	3.0	nd	10.5	nd	nd	nd	nd	nd	nd
Lab blank-1	3.2	nd	nd	nd	nd	nd	nd	2.5	nd	6.9	nd	nd	nd	nd	nd	nd
Lab blank-2	2.9	nd	nd	nd	nd	nd	nd	2.3	nd	8.8	nd	nd	nd	nd	nd	nd

nd: not detected.

Table A3.2 Analytical results of lab replicates and blanks for NPAHs (ng/mL)

Sample	1-NNAP	2-NNAP	2-NBPL	3-NBPL	4-NBPL	5-NACT	2-NFLU	9-NANT	9-NPHE	1-NPYR	6-NCHR
A-1	2.1	3.5	nd	nd	nd	nd	5.6	nd	nd	9.4	13.6
A-1 rep	2.3	4.0	nd	nd	nd	nd	5.6	nd	nd	10.0	15.4
B-2	1.6	3.0	nd	nd	nd	nd	4.9	nd	nd	15.0	17.3
B-2 rep	2.0	3.4	nd	nd	nd	nd	5.3	nd	nd	15.5	17.9
B-5	4.8	1.7	nd	nd	nd	nd	7.4	nd	nd	13.1	15.7
B-5 rep	4.4	1.6	nd	nd	nd	nd	7.4	nd	nd	12.9	15.4
EG-14	4.3	2.8	nd	nd	nd	nd	3.5	nd	nd	11.0	14.4
EG-14 rep	4.1	2.8	nd	nd	nd	nd	3.8	nd	nd	10.4	14.0
H-28	5.3	3.1	nd	nd	nd	nd	4.9	nd	nd	12.6	15.6
H-28 rep	5.7	3.5	nd	nd	nd	nd	5.3	nd	nd	13.0	15.9
V-1	5.6	2.3	nd	nd	nd	nd	4.8	nd	nd	8.3	15.3
V-1 rep	5.8	2.6	nd	nd	nd	nd	4.9	nd	nd	8.8	16.4
Field blank	0.5	nd	nd	nd	nd	nd	nd	nd	nd	0.9	1.0
Lab blank-1	0.3	nd	nd	nd	nd	nd	nd	nd	nd	0.7	0.7
Lab blank-2	0.2	nd	nd	nd	nd	nd	nd	nd	nd	0.6	0.7

nd: not detected.

Table A3.3 Analytical results of lab replicates and blanks for biomarkers (ng/mL)

Sample	Ste1	Ste2	Ste3	Ste4	Ste5	Ste6	Hop1	Hop2	Hop3	Hop4	Hop5
A-1	2.2	5.1	12.8	24.2	11.3	8.3	336	202	265	184	203
A-1 rep	2.8	5.5	13.5	25.3	16.2	9.4	366	228	288	195	196
B-2	4.9	5.2	21.4	31.6	8.4	7.5	514	303	164	143	156
B-2 rep	3.2	4.7	19.3	27.4	7.3	6.2	486	276	146	133	133
B-5	4.3	5.4	16.0	43.1	15.7	11.3	321	204	128	154	145
B-5 rep	4.5	5.5	16.6	45.1	17.4	13.5	336	210	131	157	151
EG-14	4.3	7.1	19.2	38.3	14.5	8.0	218	111	184	206	192
EG-14 rep	4.0	6.8	17.8	37.3	13.9	7.9	212	108	154	188	188
H-28	2.3	5.8	13.8	19.9	14.0	17.7	238	124	118	128	128
H-28 rep	2.7	6.6	14.3	21.8	14.9	18.5	252	125	119	133	136
V-1	1.5	6.4	14.8	20.5	14.6	8.2	278	205	274	127	201
V-1 rep	1.9	6.9	15.6	22.5	16.2	9.3	303	234	289	133	217
Field blank	nd	nd	nd	nd	nd	nd	6.9	nd	nd	nd	nd
Lab blank-1	nd	nd	nd	nd	nd	nd	4.9	nd	nd	nd	nd
Lab blank-2	nd	nd	nd	nd	nd	nd	5.3	nd	nd	nd	nd

nd: not detected.

Table A3.4. Surrogate standard recoveries (%)

Sample	PAH	NPAH	Biomarker
A-1	79.9	77.3	82.5
A-1 rep	82.0	81.1	84.4
B-2	85.2	83.3	86.9
B-2 rep	86.3	85.2	88.2
B-3	90.6	91.3	87.4
B-4	77.4	70.3	75.4
B-5	74.0	73.4	88.8
B-5 rep	72.5	77.3	92.3
B-6	92.5	87.3	83.4
B-7	85.5	83.5	85.4
C-5	95.3	92.6	86.4
C-6	75.5	70.4	87.5
C-7	74.2	75.8	85.3
EG-14	86.9	80.9	76.3
EG-14 rep	83.9	82.4	79.5
EG-18	87.3	84.8	90.3
H-15	74.7	71.6	77.4
H-22	77.0	75.3	77.2
H-28	86.5	83.5	86.4
H-28 rep	87.2	88.2	90.2
H-29	86.7	85.2	87.1
H-30	84.8	73.3	82.6
H-31	76.6	77.2	80.3
H-8	80.5	79.4	85.4
S-2	77.9	83.4	88.2
S-3	89.5	76.2	75.4
S-4	78.4	70.5	79.9
V-1	79.6	74.6	74.1
V-1 rep	78.0	72.1	77.1
X-1	81.4	88.0	72.4
Field blank	87.6	95.6	94.8
Lab blank-1	89.9	94.0	97.6
Lab blank-2	93.6	98.7	95.5

Table A3.5 Replicates, blanks and recoveries of OC analysis

Sample	OC (%)	RPD (%)
A-1	0.59	5.22
A-1 Re	0.56	
B-2	3.68	11.80
B-2 Re	3.27	
B-5	2.59	6.36
B-5 Re	2.76	
EG-14	4.19	8.02
EG-14 Re	4.54	
H-28	0.29	6.67
H-28 Re	0.31	
S-2	0.29	3.51
S-2 Re	0.28	
V-1	4.36	8.35
V-1 Re	4.74	

Sample	GA added (g)	$CaCO_3$ added (g)	Weight lost at 100 °C (g)	Weight loss at 500 °C (g)	OC recovery (%)
Blank-1			0	0.002	
Blank-2			0	0	
GA-1	3.401		0.003	3.398	99.91
GA-2	3.136		0.002	3.134	99.94
GA-3	3.328		0.002	3.325	99.91
GA+$CaCO_3$-1	2.704	1.722	0.003	2.672	98.82
GA+$CaCO_3$-2	1.847	2.057	0.003	1.810	98.00
GA+$CaCO_3$-3	1.717	2.518	0.002	1.697	98.84

RPD: relative percent difference.

GA: glutamic acid.

Table A3.6 Correlation coefficients (Spearman's r) between individual PAH and NPAH compounds (N = 24)

		NAP	ACY	PHE	ANT	FLA	PYR	BAA	CHR	BBF	BKF	BAP	IcdP	DBA	BghiP	1-NNAP	2-NNAP	2-NFLU
NAP	r	1.000	.766**	.602**	.587**	.810**	.732**	.748**	.749**	.381	.534**	.470*	.601**	.598**	.553**	.126	.732**	.582**
	Sig. (2-tailed)	.	.000	.002	.003	.000	.000	.000	.000	.066	.007	.021	.002	.002	.005	.557	.000	.003
ACY	r		1.000	.595**	.781**	.872**	.765**	.828**	.873**	.554**	.713**	.277	.477*	.470*	.359	.205	.716**	.526**
	Sig. (2-tailed)		.	.002	.000	.000	.000	.000	.000	.005	.000	.189	.018	.020	.085	.336	.000	.008
PHE	r			1.000	.729**	.743**	.743**	.563**	.623**	.625**	.678**	.666**	.659**	.660**	.650**	.423*	.518**	.624**
	Sig. (2-tailed)			.	.000	.000	.000	.004	.001	.001	.000	.000	.000	.000	.001	.040	.009	.001
ANT	r				1.000	.782**	.743**	.702**	.760**	.673**	.702**	.555**	.627**	.645**	.590**	.547**	.627**	.787**
	Sig. (2-tailed)				.	.000	.000	.000	.000	.000	.000	.005	.001	.001	.002	.006	.001	.000
FLA	r					1.000	.830**	.803**	.848**	.632**	.697**	.459*	.630**	.598**	.550**	.397	.734**	.603**
	Sig. (2-tailed)					.	.000	.000	.000	.001	.000	.024	.001	.002	.005	.054	.000	.002
PYR	r						1.000	.907**	.941**	.605**	.847**	.547**	.627**	.642**	.593**	.509*	.623**	.680**
	Sig. (2-tailed)						.	.000	.000	.002	.000	.006	.001	.001	.002	.011	.001	.000
BAA	r							1.000	.968**	.570**	.829**	.376	.536**	.560**	.445*	.431*	.626**	.602**
	Sig. (2-tailed)							.	.000	.004	.000	.070	.007	.004	.029	.035	.001	.002
CHR	r								1.000	.575**	.841**	.410*	.580**	.593**	.479*	.485*	.649**	.646**
	Sig. (2-tailed)								.	.003	.000	.046	.003	.002	.018	.016	.001	.001
BBF	r									1.000	.743**	.382	.373	.362	.380	.482*	.656**	.604**
	Sig. (2-tailed)									.	.000	.066	.073	.082	.067	.017	.001	.002
BKF	r										1.000	.455*	.511*	.542**	.417*	.404	.611**	.559**
	Sig. (2-tailed)										.	.026	.011	.006	.043	.050	.002	.005
BAP	r											1.000	.901**	.906**	.928**	.356	.294	.663**
	Sig. (2-tailed)											.	.000	.000	.000	.088	.163	.000
IcdP	r												1.000	.989**	.943**	.423*	.358	.682**
	Sig. (2-tailed)												.	.000	.000	.039	.086	.000
DBA	r													1.000	.933**	.435*	.321	.717**
	Sig. (2-tailed)													.	.000	.034	.126	.000
BghiP	r														1.000	.453*	.337	.733**
	Sig. (2-tailed)														.	.026	.108	.000
1-NNAP	r															1.000	.098	.647**
	Sig. (2-tailed)															.	.648	.001
2-NNAP	r																1.000	.425*
	Sig. (2-tailed)																.	.038
2-NFLU	r																	1.000
	Sig. (2-tailed)																	.
1-NPYR	r																	
	Sig. (2-tailed)																	
6-NCHR	r																	
	Sig. (2-tailed)																	

* Significant at 0.05 level.
** Significant at 0.01 level.

Table A3.7 Correlation coefficients (Spearman's r) between individual hopanes and steranes (N = 24)

		Ste1	Ste2	Ste3	Ste4	Ste5	Ste6	Hop1	Hop2	Hop3	Hop4	Hop5
Ste1	r	1.000	.833**	.723**	.769**	.756**	.510*	.818**	.589**	.500*	.702**	.761**
	Sig. (2-tailed)	.	.000	.000	.000	.000	.011	.000	.002	.013	.000	.000
Ste2	r		1.000	.855**	.854**	.788**	.705**	.787**	.770**	.681**	.820**	.881**
	Sig. (2-tailed)		.	.000	.000	.000	.000	.000	.000	.000	.000	.000
Ste3	r			1.000	.896**	.700**	.782**	.854**	.810**	.747**	.833**	.848**
	Sig. (2-tailed)			.	.000	.000	.000	.000	.000	.000	.000	.000
Ste4	r				1.000	.759**	.686**	.852**	.807**	.633**	.875**	.887**
	Sig. (2-tailed)				.	.000	.000	.000	.000	.001	.000	.000
Ste5	r					1.000	.753**	.753**	.646**	.666**	.637**	.837**
	Sig. (2-tailed)					.	.000	.000	.001	.000	.001	.000
Ste6	r						1.000	.663**	.681**	.717**	.619**	.691**
	Sig. (2-tailed)						.	.000	.000	.000	.001	.000
Hop1	r							1.000	.782**	.705**	.704**	.820**
	Sig. (2-tailed)							.	.000	.000	.000	.000
Hop2	r								1.000	.728**	.805**	.777**
	Sig. (2-tailed)								.	.000	.000	.000
Hop3	r									1.000	.610**	.768**
	Sig. (2-tailed)									.	.002	.000
Hop4	r										1.000	.814**
	Sig. (2-tailed)										.	.000
Hop5	r											1.000
	Sig. (2-tailed)											.

* Significant at 0.05 level.
** Significant at 0.01 level.

Table A3.8 Consensus-based sediment quality guidelines (ng/g dry weight at 1% TOC)

Chemical	TEC	MEC	PEC
Naphthalene	176	369	561
Acenaphthylene	5.9	67	128
Acenaphthene	6.7	48	89
Fluorene	77.4	307	536
Phenanthrene	204	687	1170
Anthracene	57.2	451	845
Fluoranthene	423	1327	2230
Pyrene	195	858	1520
Benz[a]anthracene	108	579	1050
Chrysene	166	728	1290
Benzo[b]fluoranthene	240	6820	13400
Benzo[k]fluoranthene	240	6820	13400
Benzo[a]pyrene	150	800	1450
Dibenzo[a,h]anthracene	33	84	135
Indeno[1,2,3-cd]pyrene	200	1700	3200
Benzo[g,h,i]perylene	170	1685	3200

TEC: threshold effect concentration.

MEC: midpoint effect concentrations.

PEC: probable effect concentrations.

Table A3.9 Measured PAH concentrations and uncertainties in southern Lake Michigan sediments (ng/g dw)

ID	TPAHC	TPAHU	NAPC	NAPU	ACYC	ACYU	ACEC	ACEU	FLUC	FLUU	PHEC	PHEU	ANTC	ANTU	FLAC	FLAU	PYRC	PYRU	BAAC	BAAU	CHRC	CHRU	BBKFC	BBKFU	BEPC	BEPU	BAPC	BAPU	IcdPC	IcdPU	DBAC	DBAU	BghiPC	BghiPU
A-1	496.91	99.38	7.12	1.21	1.14	0.20					61.48	12.30	0.01	0.00	99.62	17.93	98.24	19.65	41.68	9.17	58.55	12.29	29.44	5.89			5.50	2.69	44.68	8.94	27.70	7.20	12.96	2.46
B-2	802.25	160.45	11.91	2.02	2.01	0.36			0.50	0.23	109.55	21.91	0.02	0.00	166.92	30.05	175.16	35.03	88.95	19.57	118.89	24.97	66.86	13.37			2.89	1.41	23.75	4.75	17.99	4.08	7.10	1.35
B-3	831.56	166.31	6.62	1.12	1.41	0.25					124.14	24.83	0.02	0.00	154.91	27.88	207.90	41.58	91.20	20.06	120.88	25.39	53.31	10.66			3.25	1.59	27.41	5.48	20.23	5.26	8.23	1.56
B-4	512.93	102.59	6.59	1.12	1.16	0.21					105.58	21.12	0.02	0.00	131.51	23.67	85.58	17.12	29.64	6.52	51.65	10.85	51.18	10.24			2.76	1.35	20.30	4.06	14.68	3.82	5.95	1.13
B-5	507.48	101.50	4.99	0.85	1.19	0.21			1.11	0.51	67.00	13.52	0.02	0.00	100.25	18.05	114.97	22.99	43.86	9.65	79.09	16.61	42.34	8.47			2.11	1.03	19.61	3.92	15.13	3.93	5.75	1.09
B-6	327.31	65.46	4.69	0.80	0.94	0.17					29.78	5.96	0.01	0.00	62.76	11.30	72.17	14.43	30.83	6.78	42.19	8.86	56.31	11.26			1.35	0.66	10.92	2.18	8.02	2.09	3.23	0.61
B-7	312.44	62.49	5.21	0.88	0.78	0.14			0.34	0.16	39.15	7.83	0.01	0.00	56.31	10.14	67.84	13.57	32.46	7.14	45.17	9.49	33.24	6.65			1.37	0.67	13.69	2.74	10.64	2.77	2.75	0.52
C-5	859.31	171.86	9.35	1.59	2.27	0.41			0.86	0.39	69.89	13.98	0.02	0.00	165.72	29.83	191.31	38.26	101.30	22.29	131.39	27.59	107.50	21.50			3.90	1.91	29.69	5.94	22.10	5.74	8.17	1.55
C-6	421.81	84.36	3.26	0.55	0.95	0.17					33.19	6.64	0.01	0.00	60.39	10.87	108.07	21.61	54.20	11.92	72.22	15.17	46.58	9.32			1.96	0.96	17.48	3.50	14.91	3.88	4.34	0.83
C-7	609.49	121.90	6.36	1.08	1.46	0.26					84.04	16.81	0.01	0.00	157.37	28.33	126.69	25.34	56.67	12.47	81.86	17.19	56.87	11.37			1.50	0.74	14.33	2.87	10.33	2.69	3.21	0.61
EG-14	1290.72	258.14	12.69	2.16	2.70	0.49					179.14	35.83	0.03	0.01	333.87	60.10	208.90	41.78	102.59	22.57	144.99	30.45	79.91	15.98			10.20	5.00	95.37	19.07	74.15	19.28	27.46	5.22
EG-18	588.49	117.70	5.49	0.93	1.40	0.25					92.74	18.55	0.02	0.00	103.40	18.61	98.32	19.66	63.75	14.02	75.05	15.76	76.35	15.27			2.99	1.47	28.01	5.60	20.55	5.34	6.74	1.28
H-15	332.15	66.43	4.81	0.82	1.18	0.21			0.43	0.20	52.57	10.51	0.01	0.00	76.56	13.78	49.16	9.83	23.72	5.22	32.55	6.84	30.57	6.11			2.65	1.30	27.06	5.41	19.35	5.03	6.33	1.20
H-22	903.04	180.61	6.84	1.16	2.04	0.37					100.60	20.12	0.02	0.00	245.60	44.21	166.23	33.25	87.88	19.33	122.41	25.71	83.25	16.65			3.55	1.74	34.27	6.85	25.48	6.63	8.13	1.54
H-28	347.36	69.47	4.75	0.81	1.02	0.18					29.43	5.89	0.01	0.00	95.95	17.27	55.67	11.13	38.63	8.50	52.80	11.09	36.98	7.40			0.98	0.48	11.91	2.38	8.57	2.23	3.15	0.60
H-29	364.29	72.86	6.07	1.17	1.18	0.21					29.13	5.83	0.01	0.00	97.65	15.70	75.39	15.08	44.91	9.88	66.70	14.01	24.20	4.84			1.22	0.60	11.56	2.31	0.34	2.17	2.75	0.52
H-30	1234.50	246.90	11.09	1.89	3.91	0.70					94.35	18.87	0.03	0.01	385.22	69.34	236.28	47.26	167.40	36.83	175.28	36.81	70.62	14.12			2.63	1.29	38.14	7.63	24.52	6.37	9.62	1.83
H-31	588.87	117.77	7.56	1.28	2.07	0.37					54.95	10.99	0.02	0.00	184.98	33.30	103.71	20.74	56.71	12.48	83.63	17.56	40.28	8.06			1.35	0.66	23.82	4.76	16.66	4.33	4.86	0.92
H-0	451.20	90.24	3.59	0.61	1.46	0.26					46.54	9.31	0.01	0.00	121.77	21.92	01.13	16.23	48.16	10.60	60.00	14.45	46.39	9.20			1.22	0.60	12.21	2.44	9.66	2.51	2.61	0.50
S-2	397.61	79.52	3.40	0.58	1.09	0.20			0.50	0.23	57.51	11.50	0.01	0.00	83.61	15.05	75.80	15.16	29.81	6.56	54.57	11.46	52.94	10.59			1.77	0.87	13.70	2.74	10.61	2.76	3.36	0.64
S-3	212.46	42.49	2.53	0.43	0.70	0.13			0.35	0.16	56.04	11.21	0.01	0.00	47.29	8.51	25.28	5.06	11.96	2.63	19.27	4.05	28.26	5.65			0.96	0.47	7.79	1.56	5.95	1.55	2.30	0.44
S-4	315.02	63.16	4.52	0.77	1.00	0.19					43.96	0.79	0.01	0.00	05.91	15.46	59.59	11.92	26.59	5.05	20.01	5.00	31.59	6.32			1.50	0.74	11.90	2.30	0.99	2.34	3.34	0.63
V-1	328.75	65.75	2.55	0.43	0.68	0.12			0.41	0.19	42.89	8.58	0.01	0.00	81.50	14.67	53.79	10.76	20.87	4.59	30.46	6.40	29.44	5.89			3.67	1.80	29.14	5.83	19.71	5.13	8.66	1.65
X-1	563.58	112.72	6.11	1.04	0.83	0.15			0.51	0.23	84.85	16.97	0.01	0.00	128.73	23.17	109.25	21.85	44.75	9.85	66.06	13.87	49.06	9.81			4.33	2.12	30.99	6.20	22.53	5.86	8.67	1.65
Mean	566.90		6.20		1.44		-		0.56		70.38		9.12		134.08		110.27		55.77		75.94		50.98		-		2.73		24.91		18.20		6.65	

Table A3.10 PAH source profiles used in CMB modeling

Profile	Reference	NAPC	NAPU	ACYC	ACYU	ACEC	ACEU	FLUC	FLUU	PHEC	PHEU	ANTC	ANTU	FLAC	FLAU	PYRC	PYRU	BAAC	BAAU	CHRC	CHRU	BBKFC	BBKFU	BEPC	BEPU	BAPC	BAPU	IcdPC	IcdPU	DBAC	DBAU	BghiPC	BghiPU
Power plant	Li et al., 2003	-99	0	-99	0	-99	0	0.01	0.01	0.15	0.06	0.02	0.01	0.16	0.07	0.14	0.06	0.10	0.04	0.20	0.08	0.06	0.02	0.06	0.02	0.04	0.01	0.03	0.01	0.01	0.00	0.04	0.02
Residential coal burning	Li et al., 2003	-99	0	-99	0	-99	0	0.00	0.00	0.34	0.14	0.07	0.03	0.22	0.09	0.09	0.03	0.05	0.02	0.07	0.03	0.08	0.03	0.03	0.01	0.03	0.01	0.02	0.01	-99	0	0.02	0.01
Coke oven	Li et al., 2003	0.26	0.11	0.03	0.01	0.01	0.00	0.01	0.01	0.05	0.02	0.01	0.01	0.05	0.02	0.05	0.02	0.08	0.03	0.08	0.03	0.13	0.05	0.08	0.03	0.07	0.03	0.03	0.01	0.01	0.00	0.05	0.02
Coal average	Li et al., 2003	0.18	0.07	0.02	0.01	0.00	0.00	0.01	0.00	0.13	0.05	0.03	0.01	0.11	0.05	0.09	0.04	0.07	0.03	0.10	0.04	0.09	0.04	0.05	0.02	0.05	0.02	0.03	0.01	0.01	0.00	0.03	0.01
Gasoline engine	Li et al., 2003	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.03	0.01	0.01	0.00	0.14	0.06	0.24	0.10	0.06	0.03	0.09	0.04	0.11	0.04	0.08	0.03	0.06	0.02	0.03	0.01	0.01	0.01	0.13	0.05
Diesel engine	Li et al., 2003	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.24	0.09	0.03	0.01	0.25	0.10	0.17	0.07	0.03	0.01	0.09	0.03	0.05	0.02	0.03	0.01	0.03	0.01	0.02	0.01	0.04	0.02	0.03	0.01
Tunnel air	Li et al., 2003	0.03	0.01	0.01	0.00	0.02	0.01	0.01	0.00	0.09	0.04	0.02	0.01	0.09	0.04	0.11	0.04	0.06	0.02	0.09	0.04	0.12	0.05	0.06	0.02	0.07	0.03	0.09	0.03	0.04	0.02	0.10	0.04
Traffic average	Li et al., 2003	0.01	0.01	0.01	0.00	0.01	0.00	0.01	0.00	0.16	0.06	0.03	0.01	0.16	0.06	0.16	0.07	0.04	0.02	0.09	0.04	0.08	0.03	0.05	0.02	0.05	0.02	0.05	0.02	0.03	0.01	0.07	0.03
Industrial boiler	Li et al., 1999	0.71	0.28	0.01	0.00	0.01	0.00	0.02	0.01	0.05	0.02	0.01	0.00	0.09	0.04	0.03	0.01	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.01	0.00	0.02	0.01	0.02	0.01	0.01	0.01
Pine-Wood combustion	Schauer et al., 2001	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.05	0.02	0.02	0.01	0.27	0.11	0.26	0.10	0.08	0.03	0.08	0.03	0.10	0.04	0.03	0.01	0.05	0.02	0.04	0.01	-99	0	0.03	0.01
Coal-tar 6-city avg	Van Metre et al., 2008	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.08	0.03	0.01	0.00	0.20	0.08	0.15	0.06	0.06	0.02	0.10	0.04	0.16	0.06	0.06	0.02	0.07	0.03	0.05	0.02	0.01	0.00	0.05	0.02
Coal-tar Chicago	Van Metre et al., 2008	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.12	0.05	0.01	0.00	0.24	0.10	0.18	0.07	0.06	0.02	0.08	0.03	0.12	0.05	0.04	0.02	0.06	0.03	0.04	0.01	0.00	0.00	0.04	0.02

Two columns for each compound.
Left column: fraction of this compound relative to $\sum$PAH16.
Right column: uncertainty of the fraction.
"-99" indicates data is unavailable.

Table A3.11 Pearson correlations between PAH source profiles and mean measured profile.

	Mean of 24 sites	Power plant	Residential coal burning	Coke oven	Coal average	Gasoline engine	Diesel engine	Tunnel air	Traffic average	Industrial boiler	Pine-Wood combustion	Coal-tar 6-city avg	Coal-tar Chicago
Mean of 24 sites	1.00	**0.89**	**0.67**	0.03	0.53	**0.73**	**0.87**	**0.65**	**0.89**	-0.12	**0.91**	**0.87**	**0.93**
Power plant		1.00	**0.66**	0.48	**0.90**	0.61	**0.78**	0.64	**0.82**	0.45	**0.69**	**0.74**	**0.77**
Residential coal burning			1.00	0.20	**0.83**	0.24	**0.91**	0.44	**0.82**	**0.73**	0.44	0.54	**0.70**
Coke oven				1.00	**0.79**	0.08	-0.09	0.15	-0.04	**0.83**	-0.03	0.12	0.04
Coal average					1.00	0.30	0.52	0.40	0.50	**0.67**	0.37	0.45	0.50
Gasoline engine						1.00	0.54	**0.78**	**0.74**	-0.17	**0.84**	**0.82**	**0.77**
Diesel engine							1.00	0.56	**0.95**	-0.10	**0.75**	**0.73**	**0.88**
Tunnel air								1.00	**0.77**	-0.20	0.61	**0.83**	**0.73**
Traffic average									1.00	-0.17	**0.80**	**0.84**	**0.92**
Industrial boiler										1.00	-0.13	-0.19	-0.14
Pine-Wood combustion											1.00	**0.89**	**0.93**
Coal-tar 6-city avg												1.00	**0.95**
Coal-tar Chicago													1.00

Black type -- not significant; p-value>.05

Red type -- significant; $0.01 < p < 0.05$

Bold red type -- highly significant; $p < 0.01$

Table A3.12 CMB model performance and source contribution estimates

Fitting species (9): PHE, FLA, PYR, BAA, CHR, BBKF, IND, DBA, BGP								
					Source contribution (fraction)			
Site	R^2	χ^2	% mass explained	DF	Power plant	Coke oven	Diesel engine	Coal-tar Chicago
A-1	0.80	2.72	122.6	6	0.323	0.105	0.798	0
B-2	0.84	2.51	99.3	6	0.396	0	0.446	0.151
B-3	0.85	2.02	101.9	6	0.480	0	0.496	0.042
B-4	0.90	1.39	106.5	6	0.092	0	0.669	0.303
B-5	0.85	2.2	106.4	6	0.374	0	0.541	0.149
B-6	0.76	5.09	96.4	5	0.233	0.055	0.374	0.303
B-7	0.78	4.36	105	5	0.312	0.029	0.536	0.173
C-5	0.75	5.3	97.3	5	0.311	0.054	0.367	0.241
C-6	0.73	4.45	107.1	6	0.378	0.273	0.419	0
C-7	0.79	3.48	89.3	6	0.286	0	0.408	0.199
EG-14	0.83	2.23	121.6	6	0.255	0.087	0.875	0.000
EG-18	0.80	3.82	113.5	5	0.233	0.230	0.590	0.081
H-15	0.82	1.88	130.5	7	0	0.364	0.941	0
H-22	0.81	2.95	101.5	6	0.284	0	0.506	0.225
H-28	0.78	3.68	97.5	6	0.316	0	0.389	0.270
H-29	0.79	3.16	96.8	6	0.489	0	0.385	0.094
H-30	0.76	3.77	89.3	6	0.403	0	0.372	0.118
H-31	0.79	3.19	98.7	6	0.333	0	0.522	0.132
H-8	0.77	3.9	91.9	6	0.306	0	0.405	0.208
S-2	0.82	2.91	101.4	6	0.239	0	0.516	0.259
S-3	0.85	2.58	104.3	5	0.021	0.110	0.661	0.250
S-4	0.85	2.12	104.6	6	0.103	0	0.567	0.375
V-1	0.82	1.78	132.9	7	0	0.379	0.950	0
X-1	0.86	2.43	114.8	5	0.222	0.034	0.692	0.200
Mean	0.81	3.08	105.5		0.27	0.07	0.56	0.16
Std	0.04	1.05	11.8		0.14	0.12	0.18	0.11
Min	0.73	1.39	89.3		0.00	0.00	0.37	0.00
Max	0.90	5.30	132.9		0.49	0.38	0.95	0.38

A zero in source contribution indicates source was not detected by the model and was treated as zero source contribution.

Table A3.13 NPAH concentrations in southern Lake Michigan sediments (ng/g dw)

Site	1-NNAP	2-NNAP	2-NBPL	3-NBPL	4-NBPL	5-NACT	2-NFLU	9-NANT	9-NPHE	1-NPYR	6-NCHR
A-1	0.31	0.53	nd	nd	nd	nd	0.79	nd	nd	1.37	2.06
B-2	0.42	0.77	nd	nd	nd	nd	1.24	nd	nd	3.71	4.27
B-3	1.56	0.27	nd	nd	nd	nd	1.35	nd	nd	2.22	2.67
B-4	0.26	0.70	nd	nd	nd	nd	0.47	nd	nd	3.95	4.11
B-5	0.99	0.35	nd	nd	nd	nd	1.58	nd	nd	2.78	3.33
B-6	0.45	0.37	nd	nd	nd	nd	0.57	nd	nd	2.47	2.67
B-7	0.27	0.23	nd	nd	nd	nd	0.47	nd	nd	1.90	1.97
C-5	0.51	0.72	nd	nd	nd	nd	2.64	nd	nd	6.09	6.91
C-6	0.53	0.18	nd	nd	nd	nd	0.39	nd	nd	1.84	2.22
C-7	0.43	0.68	nd	nd	nd	nd	0.42	nd	nd	3.38	3.49
EG-14	1.69	1.12	nd	nd	nd	nd	1.45	nd	nd	4.32	5.70
EG-18	0.60	0.57	nd	nd	nd	nd	1.21	nd	nd	1.72	1.97
H-15	0.19	0.16	nd	nd	nd	nd	0.48	nd	nd	0.89	1.15
H-22	0.99	0.59	nd	nd	nd	nd	0.99	nd	nd	1.60	2.22
H-28	0.72	0.43	nd	nd	nd	nd	0.67	nd	nd	1.68	2.07
H-29	0.20	0.63	nd	nd	nd	nd	0.27	nd	nd	2.60	3.08
H-30	0.60	2.15	nd	nd	nd	nd	1.10	nd	nd	6.44	8.09
H-31	0.60	0.29	nd	nd	nd	nd	0.78	nd	nd	2.80	4.32
H-8	0.30	0.36	nd	nd	nd	nd	0.34	nd	nd	2.05	2.62
S-2	0.66	0.47	nd	nd	nd	nd	0.75	nd	nd	2.73	3.14
S-3	0.47	0.13	nd	nd	nd	nd	0.22	nd	nd	2.03	2.10
S-4	0.33	0.26	nd	nd	nd	nd	0.48	nd	nd	2.64	3.08
V-1	0.56	0.24	nd	nd	nd	nd	0.48	nd	nd	0.85	1.56
X-1	1.03	0.26	nd	nd	nd	nd	0.82	nd	nd	1.99	2.59

nd: not detected above MDL.

3.8 References

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Chapter 4 Characterization of exhaust emissions from diesel engines at various loads and speeds using different fuels and after-treatments

4.1 Abstract

Diesel exhaust emissions contain numerous semivolatile organic compounds (SVOCs) for which emission information is limited, especially for idling conditions, new fuels and after-treatment systems. This study investigates diesel exhaust emissions of particulate matter (PM), polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs (NPAHs), and sterane and hopane petroleum biomarkers from a heavy-duty (6.4 L) diesel engine at various loads (idle, 600 and 900 kPa BMEP), with three types of fuel (ultra low sulfur diesel or ULSD, Swedish low aromatic diesel, and neat soybean biodiesel), and with and without a diesel oxidation catalyst (DOC) and diesel particulate filter (DPF). Swedish diesel and biodiesel reduced emissions of $PM_{2.5}$, Σ_{16}PAHs, Σ_{11}NPAHs, Σ_2Hopanes and Σ_2Steranes, and biodiesel resulted in larger reductions. However, idling emissions increased for benzo[k]fluoranthene (Swedish diesel), 5-nitroacenaphthene (biodiesel) and $PM_{2.5}$ (biodiesel), a significant result given the attention to exposures from idling vehicles and the toxicity of high-molecular-weight PAHs and NPAHs. The DOC+DPF combination reduced emissions of $PM_{2.5}$ and the measured SVOCs during both DPF loading (>99% reduction) and DPF regeneration (83-99%). The toxicity of diesel exhaust, in terms of the estimated human carcinogenic risk, was greatly reduced using Swedish diesel and biodiesel fuels and the DOC+DPF. The PAH profiles showed high abundances of three and four ring compounds as well as naphthalene; NPAH profiles had high abundances of nitronaphthalenes, 1-nitropyrene and 9-nitroanthracene. The results demonstrate the effects of fuel type, engine load and after-treatment system on emissions. The emission data and chemical profiles presented can be used for emission inventories, exposure and risk assessments, and source apportionments.

4.2 Introduction

Vehicle exhaust emissions are one of the most important anthropogenic sources of air pollutants, and standards and regulations to reduce emissions of particulate matter (PM), carbon monoxide (CO), nitrogen oxides (NO_x), and nonmethane hydrocarbon (NMHC) emissions have been widely applied (Khalek et al. 2011; Karavalakis et al. 2010). Exhaust emissions include many other pollutants of public health concern, including semivolatile organic compounds (SVOCs) such as polycyclic aromatic hydrocarbons (PAHs) (Simcik et al. 1999; Li et al. 2003). SVOCs are not directly regulated, despite the toxicity of many compounds, except as part of PM mass or number concentration standards. Emissions of both regulated and unregulated pollutants depend on many factors, e.g., fuel type and quality, engine type, after-treatment technologies, engine operating (driving) conditions, and engine wear and maintenance (Karavalakis et al. 2010).

SVOCs in vehicle exhaust are found in both solid and vapor forms and, besides PAHs, include nitro-PAHs (NPAHs), hopanes, steranes, and many other classes of compounds (Khalek et al. 2011). PAHs can be emitted as unburned fuel and lubricating oil, or formed during the combustion process from other organic compounds (Karavalakis et al. 2010). NPAHs can be formed by reactions of PAHs with hydroxyl (OH) and nitrate (NO_3) radicals in the presence of NO_x, or through nitration during the combustion process (Karavalakis et al. 2010). Hopanes and steranes in vehicle exhaust mainly originate from engine lubricating oil (Schauer et al. 1999, 2002; Kleeman et al. 2008). Oils, fuels and after-treatment controls affect emissions, as summarized below.

Ultra-low sulfur conventional diesel (ULSD, sulfur content <15 ppm) has fueled most on-road diesel vehicles in the U.S. since 2006 (EPA), although the use of biodiesel and other alternative fuels has grown rapidly in recent years, and soy-based biodiesels are now widely available. In Sweden, a low sulfur (2 to 5 ppm) and low aromatic (<5% by volume) diesel is used (Westerholm et al. 2001). Such fuels, either neat or in blends with conventional diesel, generally reduce emissions of PM, CO and NMHC (Ratcliff et al. 2010; Chin et al. 2012). While biodiesel and low-aromatic fuels also are expected to decrease PAH and NPAH emissions due to the lower content of key PAH precursors (Ratcliff et al. 2010; Karavalakis et al. 2010), information regarding these (and other unregulated) emissions with alternative fuels is limited

and inconsistent. For example, substantially lower emissions of particle-associated PAHs and NPAHs have been found with soybean-based biodiesel and biodiesel blends as compared to reference diesel (Bagley et al. 1998; Westerholm et al. 2001; Correa and Arbilla 2006; Karavalakis et al. 2010; Ratcliff et al. 2010). However, emissions increased or were unchanged with canola oil-based biodiesel (Zou and Atkinson 2003), and while PAH emissions were reduced, NPAH and oxy-PAH emissions increased (Karavalakis et al. 2010). No study has reported on hopane and sterane emissions with alternative fuels, an important data gap given the use of these petroleum biomarker data in apportionment studies of ambient PM (Huang et al. 2006; Kleeman et al. 2009).

After-treatment technologies strongly affect engine emissions. Diesel particulate filters (DPF), used in conjunction with ULSD, can significantly reduce PM emissions (EPA 2013) and PM-associated PAH emissions (Heeb et al. 2010, 2008; Ratcliff et al. 2010). Effects reported for NPAHs, however, are inconsistent. For example, Ratcliff et al. measured >90% conversion of most NPAHs and 35% reduction of 1-nitropyrene (Ratcliff et al. 2010), while Heeb et al. found emissions of some NPAHs increased, possibly due to secondary nitration reactions in the DPF (Heeb et al. 2010, 2008). These studies did not measure 6-nitrochrysene, which has a high carcinogenic potency (RIDEM 2008). More information is needed to elucidate effects of DPFs on NPAH emissions, as well as on hopane and sterane emissions for which no information is available.

This chapter investigates exhaust emissions of PAHs, NPAHs, hopanes and steranes using well-controlled bench tests of a heavy-duty diesel engine. Emissions are tested at idle and two loaded conditions using three types of fuels, and with and without a DOC+DPF. The results can help elucidate effects of alternative fuels, engine load, and DPF on the emissions of toxic pollutants and petroleum marker compounds, and can be used to estimate exposures and risks.

4.3 Materials and methods

4.3.1 Engine, fuels and test conditions

The test engine is a Ford 2008 6.4 L “Power Stroke” engine manufactured by Navistar International Corporation (Lisle, IL) and used in pick-up trucks, SUVs, vans, school buses, and other vehicles. This 8 cylinder, 32 valve common rail direct-injection diesel engine is equipped with dual sequential turbocharging, cooled exhaust gas recirculation (EGR), and an EGR

oxidation catalyst. Bore, stroke and compression ratio are 98.0 mm, 104.9 mm, and 16.7:1, respectively. Maximum power and torque are 261 kW at 3000 rpm and 880 Nm at 2000 rpm.

Three fuels were tested: mid-cetane U.S. specification ultralow sulfur conventional diesel (ULSD, sulfur content <15 ppm); Swedish Environmental Class 1 (MK1) low sulfur and low aromatic (sulfur content <10 ppm, aromatics <5% by volume) diesel fuel (Haltermann Ltd., Hamburg, Germany); and neat soy-based biodiesel (B100, 100% soy methyl ester) (Peter Cremer North America, Cincinnati, OH). Fuel properties are listed in Table 4.1. The engine oil used in the study was Shell Rotella-T 15W-40 conventional petroleum lubricating oil.

The tested configuration approximates a 2004 engine calibration without exhaust gas after-treatment (Table 4.2). For tests with after-treatment, the engine was equipped with a DOC and a catalyzed silicon carbon DPF with 11±2 μm pores. The experiments used 11 test conditions described in Table 4.2. In tests 1-9, the engine was operated without after-treatment using each fuel at idle, low load (600 kPa BMEP at 1500 rpm), and medium load (900 kPa BMEP at 2500 rpm) conditions. Injection timing was set as at 3.5° after top dead center (ATDC). Test 10 sampled tailpipe PM during DPF loading, and test 11 sampled tailpipe PM during DPF regeneration. DPF regeneration used four injections, including two very late injections that elevate engine-out hydrocarbon concentration to boost temperature rise across the DOC. Except for condition 11, each test was run in triplicate, and three filter samples were collected sequentially.

4.3.2 Materials

PM samples were collected on 47 mm diameter Polytetrafluoroethylene (PTFE)-bonded glass fiber filters (Emfab™ TX40-HI20WW, borosilicate glass microfibers, reinforced with woven glass cloth and bonded with PTFE; Pall Corporation, Port Washington, NY, USA). PTFE-bonded glass fiber filters have been the filter media of choice for sampling diesel and gasoline engine particulate emissions to characterize PAHs and/or mutagenicity (Alsberg et al. 1985; Salmeen et al. 1984; Zinbo et al. 1995), and these filters are also used to sample PM emissions in engine compliance tests (CFR 2013).These filters can be more heavily loaded than PTFE membrane filters and the pressure drop across them rises more slowly. The mutagenicity and composition of diesel exhaust PMs collected on these and two other types of filters (PTFE membrane, quartz) are indistinguishable (Gorse Jr et al. 1982). Thus, PTFE-bonded glass fiber

filters should be a suitable filter media to collect particulate phase SVOCs in diesel exhaust and then study their composition and integrity during filter conditioning and storage (presented in Chapter 5).

Solvents were HPLC grade and obtained from Fisher Scientific Inc. (Pittsburgh, PA, USA). Florisil (60-100 mesh) and sodium sulfate (anhydrous, certified ACS granular, 10-60 mesh) for column chromatography were supplied by the same vendor.

4.3.3 Exhaust measurements

A heated AVL 415S smoke meter and heated sample line (AVL Inc., Plymouth, MI, USA) were used to measure filter smoke number (FSN), which was converted to mass of carbonaceous soot using a correlation proposed by Christian et al.(Christian et al. 1993) that also applies to low smoke levels and biodiesel (Northrop et al. 2011). The soluble organic fraction (SOF) was calculated as the difference between PM (described below) and carbonaceous soot, normalized by PM.

Exhaust PM was sampled using a partial flow dilution tunnel (BG-2, Sierra Instruments Inc., Monterey, CA, USA) and a flow rate of 10 L/min through a 0.95-cm diameter stainless steel sample probe inserted into the center of a straight section of exhaust pipe, facing upstream, and 2 m downstream of the engine's turbine (Figure A4.1). The probe was 40-cm long and heated to 191 °C. The dilution tunnel, at the end of the sample probe, mixed raw exhaust with filtered air with a dilution ratio of 6:1. The mixture then passed through a transfer tube (40 cm length×16 mm i.d.) to a 2.5-μm cyclone separator (Sierra Instruments Inc., Monterey, CA, USA), a second transfer tube (20 cm×16 mm i.d.), and to a Teflon filter cassette holder (Sierra Instruments Inc., Monterey, CA, USA) that supported a filter on a perforated stainless steel backing plate. The exposed area of the filter was 39 mm in diameter. The transfer tubes, cyclone separator, and filter were maintained at 47 ± 5 °C, and the filter flow rate was 60 L/min (face velocity of 91 cm/s).

Before sampling, dilution and total flows were stabilized at desired set points for 60 s, and additional dilution air was back flushed into the exhaust pipe. Sampling times were adjusted to sufficiently load filters with >280 μg of PM, at which point the pressure drop across the filter reached ~30 kPa. After sampling, the filter and cassette were immediately wrapped with Parafilm, placed in a sealed metal container, and transported to the filter conditioning and weighing laboratory. A 30-s high-flow purge cycle (100 L/min) was used to minimize PM

accumulation in the dilution tunnel and the transfer line prior to the next sample.

Filters were weighed before and after PM loading after conditioning for 24 h in a glove box (Series 100 twin plastic glove box isolator, Terra Universal, Inc.) held at 25 °C and 33% relative humidity (RH). Electrostatic charges on filters and instruments were neutralized using an ionizer (Terra Universal, Inc.) in the filter weighing chamber for 30 min before weighing. Filters were weighed twice to 1 μg precision using a microbalance (ME 5, Sartorius Inc., Edgewood, NY, USA). If weights agreed to within 5 μg, results were averaged; otherwise filters were reweighed.

To the extent possible, the PM sampling and analysis protocols were consistent with the US EPA engine testing procedures (CFR 2013), ISO-DIS 16183 (ISO 2009), verbal recommendations by Sierra Instruments, and internal standard operating protocols. To quantify the overall reproducibility (including engine, dilution tunnel, and filter conditioning, and filter weighing), nine PM samples were collected at specified conditions over a 2-day period. These tests showed good repeatability, i.e., the 95% confidence interval was ± 5.1% of the collected PM mass.

4.3.4 SVOC analysis

Filters were extracted by placing each in a 50 mL centrifuge tube, adding 25 mL of dichloromethane/hexane (4:1, v/v) to immerse the entire filter, and sonicating for 30 min (1510R-MTH, Branson Ultrasonics Corporation, Danbury, CT). The filter was then removed using a cotton stick and discarded. Extracts were passed through an activated Florisil column and fractionated into three portions. No additional cleaning of each fraction was necessary. The fractionation and GC-MS analysis have been described in detail in Chapter 2 Section 2.3.3.

4.3.5 Data analysis

Compounds that were not detected above IDLs (IDLs presented in Table A4.1) in all three replicate samples (using the same fuel type, after-treatment and engine load) were treated as zero. If compounds were detected above IDLs in at least one of the three replicates, the undetected values were substituted by IDL/2. PM mass (μg) was calculated as the difference in filter weights before and after loading. Emissions were expressed in terms of the brake-specific emissions (e.g., g/kWh or μg/kWh) for loaded conditions and as an emission rate per time (e.g.,

mg/s or ng/s) for idle conditions (since BMEP was near zero). Comparisons between idle and loaded conditions used emission rates per time. Reference or baseline emissions refer to emissions using ULSD and without the DPF. Baseline emissions were compared to literature data using ULSD as well as older fuels. For each test except test #11, SVOC measurements were averaged over three replicate filters, and differences between ULSD and Swedish fuels, between ULSD and B100 fuels, and between no after-treatment and DPF, were evaluated using 2-sample t tests (2-tailed, significance level $p = 0.05$).

For human health risks, the toxic equivalency of benzo[a]pyrene (BAP) (TEQ_{BAP}) was calculated for target compounds using toxic equivalency factors (TEFs, unitless) shown in Table 1.1 and the equation $TEQ_{BAP} = \Sigma_i C_i \times TEF_i$, where C_i is the emission rate of the i^{th} PAH or NPAH in each sample (ng/kWh). The TEFs use BAP as the baseline compound, whose TEF is assigned to be 1.

PAH and NPAH profiles were developed to represent on-road diesel sources for future use in receptor models that apportion sources of these compounds in ambient air and potentially other media based on the chemical composition of emissions. The profiles used emission rates (ng/s) for ULSD (required since 2007 in the U.S.) and weighted idle, low- and medium-load results by 24, 21 and 55%, respectively, reflecting activity data for medium and heavy duty diesel trucks (Huai et al. 2006; Lutsey et al. 2004). A composite $PM_{2.5}$ emission rate (ng/s) was also calculated using ULSD measurements and the same weightings. PAH and NPAH profiles were expressed as abundances, i.e., each compound was calculated as its fraction of total PAH emissions (ΣPAHs) or total NPAH emissions (ΣNPAHs). Profiles were plotted and compared to those in the literature, derived similarly, using Spearman correlation coefficients. All statistical analyses were performed using SPSS Statistics 21.0 (IBM Corporation).

4.4 Results and Discussion

4.4.1 Baseline emissions

Table 4.3 shows the emission rates of PM and target SVOCs using ULSD in the present study and previous studies. PM emissions under low- and high-load conditions were 0.033 ± 0.001 and 0.10 ± 0.002 g/kWh, respectively, comparable to measurements reported in other studies, most of which ranged from 0.03 to 0.3 g/kWh (Sharp et al. 2000a; Hori and Narusawa 2001; Lea-Langton et al. 2008; Tanaka et al. 1998; Ratcliff et al. 2010; Liu et al. 2010; Gambino

et al. 2001). For ΣPAHs, emissions at low load were slightly lower than earlier reports (Sharp et al. 2000b; Liu et al. 2010; Khalek et al. 2011), but emissions at high load (3.45 μg/kWh) were similar to a previous study (2.21 μg/kWh) (Sharp et al. 2000b). ΣNPAH emission rates (0.2 μg/kWh at high load) were similar to those in two previous studies (Sharp et al. 2000b; Gambino et al. 2001). ΣHopane and ΣSterane emission rates under high load (0.02 and 1 μg/kWh, respectively) were slightly lower than earlier reports (Khalek et al. 2011; Liu et al. 2010). For most SVOCs, emission rates increased considerably with load, e.g., ΣPAH emissions increased 6-fold from low to high load (0.55 to 3.45 μg/kWh) and ΣNPAH emissions also increased 6-fold (0.06 to 0.35 μg/kWh).

4.4.2 Effect of fuel

4.4.2.1 PM emissions, soot emissions and SOF

$PM_{2.5}$ emission rates strongly depended on fuel and engine load. Under load, Swedish diesel reduced $PM_{2.5}$ emissions by 7-27% compared to ULSD, and B100 reduced emissions by 68-81% (Table 4.4). With ULSD and Swedish fuels, idling emission rates were low compared to loaded conditions. However, with B100 the $PM_{2.5}$ emission rate during idling (0.60 ± 0.06 mg/s) and high load (0.65 ± 0.06 mg/s) were 5.5 times higher than under idling with ULSD (0.11 ± 0.01 mg/s).

Like $PM_{2.5}$, the emission rate of carbonaceous soot was reduced by Swedish diesel and B100 under the three conditions (Table 4.4). Notably, B100 increased $PM_{2.5}$ emissions during idling, but at the same time, reduced the emission rate of soot. The use of biodiesel also increased the SOF of the PM under all three conditions (Table 4.4). For the same fuel, the SOF decreased with increasing engine load.

A number of studies have found that biodiesel reduces $PM_{2.5}$ and soot emission rates as compared to ULSD (Sharp et al. 2000a; Chin et al. 2012; Lapuerta et al. 2008; EPA 2001). This has been attributable to the lower content of aromatic hydrocarbons and sulfur in biodiesel (and biodiesel blends) that serves as soot precursors (Sharp et al. 2000a; Lapuerta et al. 2008). Biodiesel also has higher oxygen content, which may lead to more complete combustion and the oxidation of already formed soot, further reducing $PM_{2.5}$ emissions (Lapuerta et al. 2008; Sharp et al. 2000a). The increased SOF using biodiesel has been attributed to an increased fraction of unburned fuel, which tends to condense on the filter used to collect particulate emissions (Sharp

et al. 2000a). Swedish diesel has been reported to reduce PM emissions by 11% compared to an European reference diesel (which is similar to ULSD) (Westerholm et al. 2001), again probably reflecting the lower sulfur as well as lower aromatic content in Swedish diesel.

B100 dramatically increased idle emission rates of $PM_{2.5}$. A similar increase was observed using B20 in a light-duty engine (Chin et al. 2012). Under idle conditions with low exhaust gas temperature (<150 °C), unburned fuel and oil may represent a significant fraction of $PM_{2.5}$ emissions. Thus, the increased $PM_{2.5}$ could result from unburned biodiesel fuel and oil emissions that exceed the reduction in soot and sulfate emissions (Sharp et al. 2000a). We also note that PM emission at idle may be underestimated due to the very low idle exhaust temperature (<150 °C), which could cause volatiles to be collected on exhaust pipe surfaces.

4.4.2.2 PAH emissions

The highest Σ_{16}PAH emission rates were obtained under high-load conditions when all 16 target PAHs were detected (Figure 4.1). Compared to ULSD, Swedish diesel reduced Σ_{16}PAH emissions by 46-69% and B100 reduced emissions by 80-98%; these reductions were statistically significant (Table 4.5). Individual compounds followed the same trend as Σ_{16}PAHs, with the exception that Swedish diesel slightly increased benzo[k]fluoranthene emissions under the idle condition. The lower emission rates obtained for PAHs are consistent with the low aromatic content in Swedish fuel and the absence of aromatics in biodiesel (Karavalakis et al. 2010; Ratcliff et al. 2010; Westerholm et al. 2001).

Σ_{16}PAH emission rates were positively correlated with $PM_{2.5}$ emission rates ($r = 0.93$, $p < 0.001$), but negatively correlated with the SOF ($r = -0.70$, $p < 0.001$), expected since biodiesel and Swedish diesel reduced Σ_{16}PAH emissions but increased SOF. Also, increased engine load was accompanied with increased Σ_{16}PAH emissions (Table 4.5) but decreased SOF (Table 4.4). Both biodiesel and Swedish diesel appear to increase the emissions of unburned fuel and oil in PM, which contribute to increased emissions of SOF, but these fuels contain few or no aromatics. Similarly, low load and idle conditions may also increase the fraction of unburned fuel and oil, leading to high SOF and low PAH emission rates.

4.4.2.3 NPAH emissions

Emission rates of total and speciated NPAHs are summarized in Table 4.5 and Figure 4.1,

respectively. All 11 target NPAHs were detected above MDLs while idling; 10 were detected under the low-load and high-load conditions. The most abundant compound was 1-nitropyrene, followed by 2-nitronaphthalene and 1-nitronaphthalene. Compared to ULSD, Swedish diesel reduced Σ_{11}NPAH emissions by 50-58% and B100 reduced emissions by 90-99%; these large reductions were statistically significant (Table 4.6). Individual compounds followed the same trend as Σ_{11}NPAHs, but B100 generated highest emissions of 5-nitroacenaphthene during idling. NPAH emission data in the literature are limited. Using a D12A 420 diesel engine (6-cylinder, heavy-duty), Swedish diesel reduced 1-nitropyrene emissions (both particulate and vapor) compared to a reference diesel (Westerholm et al. 2001), similar to our observations. Like Σ_{15}PAH, Σ_{11}NPAH emission rates were positively correlated with $PM_{2.5}$ emission rates ($r = 0.94$, $p < 0.001$) and negatively correlated with SOF ($r = -0.72$, $p < 0.001$).

5-Nitroacenaphthene was detected only with B100 in the no load (idling) condition. Higher NPAH emissions were measured during idling for a Euro 2 compliant VW Golf 1.9 TDi diesel engine with B100 and a low speed driving cycle (Karavalakis et al. 2010). Biodiesel generally increases emissions of NO_x (Chin et al. 2012; EPA 2001) and the SOF of PM, which consists of unburned fuel and oil (Sharp et al. 2000a; Karavalakis et al. 2010). Under low temperature idle conditions, biodiesel appears to lead to poorer combustion that facilitates the formation of products of incomplete combustion (PICs) such as PAHs, which then may react with hydroxyl (OH) and nitrate (NO_3) radicals in the presence of NO_x to form NPAHs, leading to increased emissions of selected NPAHs.

4.4.2.4 Hopane and sterane emissions

Emission rates of total and speciated hopanes and steranes are summarized in Table 4.5 and Figure 4.1. Only two hopanes and two steranes were detected above IDLs. Swedish diesel reduced Σ_2Hopane emissions (by 51-54%) compared to ULSD under idling and high-load conditions, but emissions increased (by 24%) under low-load conditions. Swedish diesel fuel reduced Σ_2Sterane emissions under all three conditions (by 36-77%). B100 reduced emissions of both Σ_2Hopane (by 65-92%) and Σ_2Sterane (by 82-96%). Emission rates of individual compounds followed the trend of the totals.

A number of studies have used hopanes and steranes as tracers of diesel and gasoline vehicles to apportion sources of ambient PM (Kleeman et al. 2009; Kleeman et al. 2008). These

compounds have been detected in vehicle exhaust but not in diesel/gasoline fuels (Schauer et al. 2002, 1999). It has been suggested that these compounds are found in the higher temperature fraction of crude oils, and thus are only found in lubricating oils (Rogge et al. 1993). Our results show that the fuel can significantly affect hopane and sterane emission rates. We speculate that fuel type may affect the amount of lubricating oil released into the exhaust through several possible mechanisms. First, with a constant injection time, the higher cetane number of B100 and Swedish diesel result in earlier combustion that causes fuel to burn closer to the injector and decreases the oil washed from the cylinder walls by the fuel spray. Second, higher cetane number fuels also may lead to less premixed combustion and more diffusion combustion, which may be better at oxidizing oil mist in the cylinder. Finally, less premixed combustion means lower rates of pressure rise, which might dislodge less oil from engine surfaces like piston rings. While further work is needed to understand the mechanisms, the variation in emission rates of these biomarker compounds imposes limits in their use as traffic-related tracers, as discussed later.

4.4.3 Effect of DPF

Effects of the DOC+DPF and DPF regeneration on PM and SVOC emissions are summarized in Table 4.6; effects on individual compounds are shown in Figure 4.2. The DPF caused large (>99%) reductions in PM, Σ_{16}PAH, Σ_{11}NPAH, Σ_{2}Hopane and Σ_{2}Sterane emission rates. While regeneration increased emissions compared to normal DPF use, emission rates remained much lower (83-99%) than those without DPF. This applied to individual compounds as well as the sums. During regeneration, exhaust temperatures are raised to burn off the PM accumulated on the DPF, and thus slightly increases in PM and SVOC emissions are expected. The DPF also increased the SOF of the PM since the carbonaceous soot was filtered out by the DPF. Consistent with a previous study (Ratcliff et al. 2010), our data confirmed that the DPF was highly effective in converting particle-bound PAHs and nitro-PAHs.

4.4.4 Toxicity of engine exhaust

Considering PAHs and NPAHs together, Swedish diesel reduced the TEQ_{BAP} by 42% to 59% for all three conditions, and B100 provided a 77% to 98% reduction (Table 4.5). These reductions were statistically significant. The DOC+DPF reduced the total TEQ_{BAP} of emissions by a factor of 2950 (factor of 590 while regenerating; Table 4.6. These data suggest that toxicity, measured as carcinogenic risk from PAHs and NPAHs, is greatly reduced by the use of DPF and

alternative fuels. This is consistent with previous studies that reported lower mutagenic potency of diesel exhaust with the use of biodiesel (Bünger et al. 1998) and Swedish diesel (Westerholm et al. 2001).

Although B100 reduced the total TEQ of diesel exhaust, it increased 5-nitroacenaphthene emissions during idling, as discussed above. This compound is classified as group 2B (possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC), although its TEF is only 0.03 relative to benzo[a]pyrene (RIDEM 2008). Still, given the recent attention to exposures from idling vehicles, the higher 5-nitroacenaphthene emissions found while idling with B100 may warrant further investigation.

4.4.5 PAH and NPAH profiles

PAH profiles have high abundances of naphthalene, phenanthrene, fluoranthene and pyrene, and low abundances of high molecular weight compounds (Table A4.2). Overall, the profiles resemble literature profiles (Khalek et al. 2011; Liu et al. 2010), e.g., Spearman correlation coefficients range from 0.69-0.75 ($p < 0.05$; Figure 4.3). NPAH profiles have high abundances of nitronaphthalenes, 9-nitroanthracene and 1-nitropyrene. While fewer data are available for comparison, this profile is highly correlated with one reported for year 2000 engines (Spearman $r = 0.90$, $p = 0.037$) (Khalek et al. 2011). While overall agreement is good, abundances of individual compounds can vary considerably among different studies, reflecting differences in engine configuration, operating conditions, sampling protocols, and other factors. The more volatile compounds, e.g., naphthalene, are particularly sensitive to partitioning between vapor and particulate phases (Singh et al. 1993), so studies combining both phases reported high abundances (70-80%) of this compound (Liu et al. 2010; Khalek et al. 2011).

The relative concentrations of engine exhaust emissions of PAHs, NPAHs and $PM_{2.5}$ vary considerably among studies. In five recent studies (Liu et al. 2010; Khalek et al. 2011; Chiang et al. 2012; Gambino et al. 2001; Sharp et al. 2000b, 2000a), $\Sigma PAH/PM_{2.5}$ ratios ranged from 2.2×10^{-5} to 0.23, and $\Sigma NPAH/PM_{2.5}$ ratios from 1.3×10^{-6} to 3.2×10^{-4}. In the present study, the $\Sigma PAH/PM_{2.5}$ and $\Sigma NPAH/PM_{2.5}$ ratios are 3.2×10^{-5} and 1.2×10^{-6}, respectively (Table A4.2). These results highlight considerable differences in the composition of vehicle exhaust PM, which depends on engine type and configurations, operating condition, fuel, control technology, etc. The variation also suggests that PAHs and NPAHs profiles measured using individual engines

may not be useful for quantitative apportionments of vehicle exhaust $PM_{2.5}$, although composite profiles, potentially derived from many vehicles and over multiple conditions, may be more representative and useful in this application.

As mentioned earlier, chemical compositions or profiles are used in chemical mass balance (CMB) and other receptor models to apportion pollutant sources. To apportion PAHs and NPAHs in sediments, a different application but using the same approach, profiles should use only the particulate phase (rather than combining vapor and particulate phases) since in sediments these contaminants arise mostly from deposition of airborne particulates (Li et al. 2003). In addition, the profiles should be expressed as fraction of ΣPAHs/ΣNPAHs, rather than PM mass. Given the variability of certain compounds, the profiles, specifically, the fitting species used, might focus on compounds that have similar abundances among studies, e.g., phenanthrene, fluoranthene, pyrene, and 1-nitropyrene, and exclude or down-weight compounds with large variability, e.g., naphthalene, benzo[a]pyrene and 2-nitrofluorene.

Hopanes and steranes have been used as tracers of vehicle exhaust PM (Kleeman et al. 2008; Kleeman et al. 2009). Our results suggest that fuel type significantly affects hopane and sterane emissions, as well as PAH and NPAH emissions. Thus it may be possible to establish a relationship between hopane/sterane and PAH/NPAH. Emission rates of Σ_{15}PAH, Σ_{11}NPAH, $PM_{2.5}$ and SOF were significantly correlated to 17α(H)21β(H)-hopane emissions ($r = 0.72 – 0.95$; Figure 4.4). Emissions of Σ_5Hopane, Σ_6Sterane and other individual hopane and sterane followed the same trend as 17α(H)21β(H)-hopane.

If the ratio of 17α(H)21β(H)-hopane to Σ_{15}PAH / Σ_{11}NPAH remains constant, then 17α(H)21β(H)-hopane can be used to apportion traffic-originated PAHs/NPAHs. This also assumes that 17α(H)21β(H)-hopane comes from only vehicle emissions, and that the ratio remains constant across different engines and engine types. However, six studies in the literature (Phuleria et al. 2006) show ratios of Σ_9PAH (FLA to IcdP)/17α(H)21β(H)-hopane and FLA/17α(H)21β(H)-hopane that range from 0.6 to 5400 and from 0.04 to 3000, respectively; the present study obtained ratios of 95 and 21. $PM_{2.5}$/17α(H)21β(H)-hopane ratios also vary, e.g., from 2000 to 227000 in three studies (Schauer et al. 1999, 2002; Rogge et al. 1993; Liu et al. 2010); the present study shows a ratio of 1.1×10^7. Such variation limits the value of hopanes and steranes as quantitative tracers of diesel exhaust emissions, although these compounds still have

diagnostic and qualitative value in detecting vehicle contributions to PAHs, NPAHs and ambient PM.

4.5 Conclusions

This study characterized exhaust emissions of $PM_{2.5}$, PAHs, NPAHs, steranes and hopanes from a heavy-duty diesel engine for three fuels, three engine conditions, and with and without a DOC+DPF. Swedish diesel, biodiesel and the DOC+DPF significantly reduced emissions of $PM_{2.5}$, PAHs, NPAHs, hopanes and steranes, although emissions of $PM_{2.5}$ and several compounds (benzo[k]fluoranthene and 5-nitroacenaphthene) increased during idling with biodiesel. Emissions of $PM_{2.5}$ and SVOCs increased with higher engine loads, with the important exception that $PM_{2.5}$ emissions increased during idling with B100. The toxicity of diesel exhaust, in terms of human carcinogenic risk, was reduced using the alternative fuels and the DOC+DPF. A PAH/NPAH profile for potential use in receptor models that apportion these compounds was developed by combining emission measurements during idle and load and accounting for variability. This profile was consistent with the literature, at least for certain compounds, which are suggested for use as fitting species. Emissions of petroleum biomarkers hopanes and steranes were significantly correlated with PAHs, NPAHs and $PM_{2.5}$, but abundances varied considerably, suggesting that these compounds can provide only qualitative or diagnostic results when used in apportionment studies.

4.6 Tables and figures

Table 4.1 Properties of ULSD, Swedish diesel and B100

Fuel parameters	ULSD	Swedish	Biodiesel	Test method
Product name	Amoco Ultra Low Sulfur #2 Premium Diesel Fuel	Halterman HF0860 Swedish Environmental Class I Diesel	Peter Cremer Nexol BD-99.9 Biodiesel	
Cetane number	46.7	55.9	55	ASTM D613
Kinematic viscosity (mm^2/s at 40 °C)	1.9-3.4	1.843	4.0	ASTM D445
Net heating value (MJ/kg)	42.699	43.535	37.348	ASTM D240
Carbon (wt%)	86.69	85.72	77.27	ASTM D5281
Hydrogen (wt%)	13.31	14.28	11.82	ASTM D5281
Oxygen (wt%)	<0.05		10.91	ASTM D5622
Sulfur content (wt ppm)	<15	<10	<1	ASTM D5453, D7039
C/H atomic ratio	1.829	1.985	1.823	ASTM D5281
Saturates/Olefins/Aromatics (vol%)	59.2 / 4.5 / 36.3	95.4 / 1.3 / 3.3	N/A	ASTM D1319

Table 4.2 Experimental design and test conditions

Test	Engine	Fuel	Calibration	BMEP (kPa)	Speed (rpm)	After-treatment	No. of samples	Power (kW)	EGR (%)	Start of injection (degree ATDC)*
1	6.4 L Ford	ULSD	2004	600	1500	none	3	48.2	14	3.5
2				900	2500	none	3	120.3	17	3.5
3				idle	650	none	3	0.1	8	3.5
4		Swedish		600	1500	none	3	48.1	14	3.5
5				900	2500	none	3	121	17	3.5
6				idle	650	none	3	0.2	8	3.5
7		B100		600	1500	none	3	47.8	14	3.5
8				900	2500	none	3	120.2	17	3.5
9				idle	650	none	3	0.1	8	3.5
10		ULSD		600	1500	DOC+DPF	3	48.4	14	-12/-3
11				500	1500	DOC+DPF regen	1	40.3	14	1/9/47/139

*ATDC: after top dead center. A negative number means degrees before top dead center (BTDC).

Table 4.3 Comparison of baseline emissions (with ULSD) in the present study and previous studies.

Engine	Displacement (L)	Power (kW)	Condition	PM (g/kWh)	ΣPAHs (μg/kWh)	?NPAHs (μg/kWh)	?Hopanes (μg/kWh)	?Steranes (μg/kWh)
2008 Ford Power Stroke	6.4	261	1500 rpm, 6 bar	0.033	0.55	0.06	0.003 ~ 0.3	0.24
			2500 rpm, 9 bar	0.10	3.45	0.35	0.02 ~ 2	0.90
1997 Cummins N14		276 (rated power)	EPA transient cycle	0.142				
1997 DDC Series 50		205 (rated power)	EPA transient cycle, no catalyst	0.137				
1997 DDC Series 50		205 (rated power)	EPA transient cycle, with catalyst	0.101				
1997 Cummins N14		276 (rated power)	EPA transient cycle		14.6	0.210		
1997 DDC Series 50		205 (rated power)	EPA transient cycle, no catalyst		6.36	0.184		
1997 DDC Series 50		205 (rated power)	EPA transient cycle, with catalyst		2.21	0.177		
6-cylinder DI	8.0	147 (maximum power, at 2900 rpm)	1000 rpm, 25% load	0.1-0.2				
			1000 rpm, 75% load	0.3-0.5				
Perkins Phaser 180Ti 6 cylinder DI	6.0	134 (maximum power, at 2600 rpm)	23 kW power output	0.060	544			
			47 kW power output	0.030	80			
6-cylinder DI	7.961	191 (rated power, at 2700 rpm)	Japanese 13 mode test cycle	0.181-0.241	9.6-20.9			
2002 Cummins ISB	5.9	224	AVL 8-mode test cycle		0.085	0.006		
2004 Heavy-duty	15		EPA transient cycle		1382	1.57	15.1	9.67
2007 Heavy-duty		Average 324	16-hr transient cycle	0.0014	992	0.94	0.80	0.54
6-cylinder EURO 2 DI	7.8	159 (maxium power, at 2100 rpm)	EURO 2 13 mode test cycle	0.26	9.661	0.351		

Table 4.4 PM and carbonaceous soot emission rates using different fuels. Change (%) is the percent change compared to ULSD for the same engine condition.

		PM		Soot		SOF	
Engine condition	Fuel	(mg/s)	Change (%)	(mg/s)	Change (%)	(unitless)	Change (%)
Idle	ULSD	0.11 ± 0.01		0.009 ± 0.003		0.92 ± 0.04	
	Swedish	0.02 ± 0.00 *	-82	0.003 ± 0.000 *	-67	0.85 ± 0.02 *	-8
	B100	0.60 ± 0.06 *	445	0.003 ± 0.000 *	-67	1.00 ± 0.00	9
Low-load	ULSD	0.44 ± 0.02		0.25 ± 0.02		0.43 ± 0.02	
	Swedish	0.41 ± 0.07	-7	0.27 ± 0.00	8	0.33 ± 0.12	-23
	B100	0.14 ± 0.01 *	-68	0.022 ± 0.001 *	-91	0.84 ± 0.01 *	95
High-load	ULSD	3.42 ± 0.08		2.9 ± 0.1		0.15 ± 0.04	
	Swedish	2.48 ± 0.03 *	-27	1.9 ± 0.1 *	-34	0.21 ± 0.03	40
	B100	0.65 ± 0.06 *	-81	0.14 ± 0.01 *	-95	0.78 ± 0.00 *	420

SOF: soluble organic fraction. SOF = (PM - soot) / PM

* Differed significantly from ULSD under the same engine condition ($p < 0.05$ using two-sample t-test).

Table 4.5 SVOC emission rates and TEQs using different fuels. Change (%) is the percent change compared to ULSD for the same engine condition.

		Σ_{16}PAH		Σ_{11}NPAH		Σ_5Hopane		Σ_6Sterane		TEQ_{BAP}	
Engine condition	Fuel	(ng/s)	Change (%)	(ng/s)	Change (%)	(ng/s)	Change (%)	(ng/s)	Change (%)	(ng/s)	Change (%)
Idle	ULSD	3.5 ± 0.5		0.23 ± 0.08		0.013 ± 0.002		1.21 ± 0.24		0.75 ± 0.04	
	Swedish	1.1 ± 0.2*	-69	0.116 ± 0.004	-50	0.006 ± 0.001*	-54	0.279 ± 0.025*	-77	0.31 ± 0.09 *	-59
	B100	0.084 ± 0.002*	-98	0.023 ± 0.004*	-90	0.001 ± 0.000*	-92	0.045 ± 0.003*	-96	0.017 ± 0.007 *	-98
Low-load	ULSD	7.4 ± 1.5		0.63 ± 0.12		0.037 ± 0.002		3.25 ± 0.10		1.6 ± 0.2	
	Swedish	4.0 ± 0.2*	-46	0.28 ± 0.05*	-56	0.046 ± 0.004*	24	2.09 ± 0.21*	-36	0.93 ± 0.05 *	-42
	B100	1.5 ± 0.4*	-80	0.016 ± 0.001*	-97	0.013 ± 0.003*	-65	0.570 ± 0.110*	-82	0.37 ± 0.12 *	-77
High-load	ULSD	115 ± 12		6.6 ± 0.9		0.657 ± 0.096		30.1 ± 7.5		23 ± 1	
	Swedish	38 ± 2*	-67	2.8 ± 1.0*	-58	0.323 ± 0.027*	-51	15.5 ± 2.7*	-49	10.2 ± 0.3 *	-56
	B100	4.9 ± 0.4*	-96	0.08 ± 0.01*	-99	0.057 ± 0.014*	-91	2.74 ± 0.43*	-91	1.11 ± 0.09 *	-95

ND: not detected.

* Differed significantly from ULSD under the same engine condition ($p < 0.05$ using two-sample t-test).

Table 4.6 PM and SVOC emission rates, SOF and TEQs with and without a DOC+DPF

After-treatment	Fuel	Engine condition	PM (mg/kWh)	SOF (unitless)	Σ_{16}PAH (ng/kWh)	Σ_{11}NPAH (ng/kWh)	Σ_5Hopane (ng/kWh)	Σ_6Sterane (ng/kWh)	TEQ_{BAP} (ng/kWh)
None		1500 rpm, 600 kPa	32.9 ± 1.3	0.43 ± 0.02	554 ± 114	47 ± 9	1.5 ± 0.3	243 ± 8	118 ± 13
w/ DOC+DPF	ULSD	1500 rpm, 600 kPa	0.1 ± 0.0	1.0 ± 0.0	1.5 ± 0.2	0.034 ± 0.004	ND	0.1 ± 0.0	0.04 ± 0.02
DOC+DPF regen		1500 rpm, 500 kPa	5.7	0.9	3.2	0.12	0.1	0.2	0.2

ND: not detected.

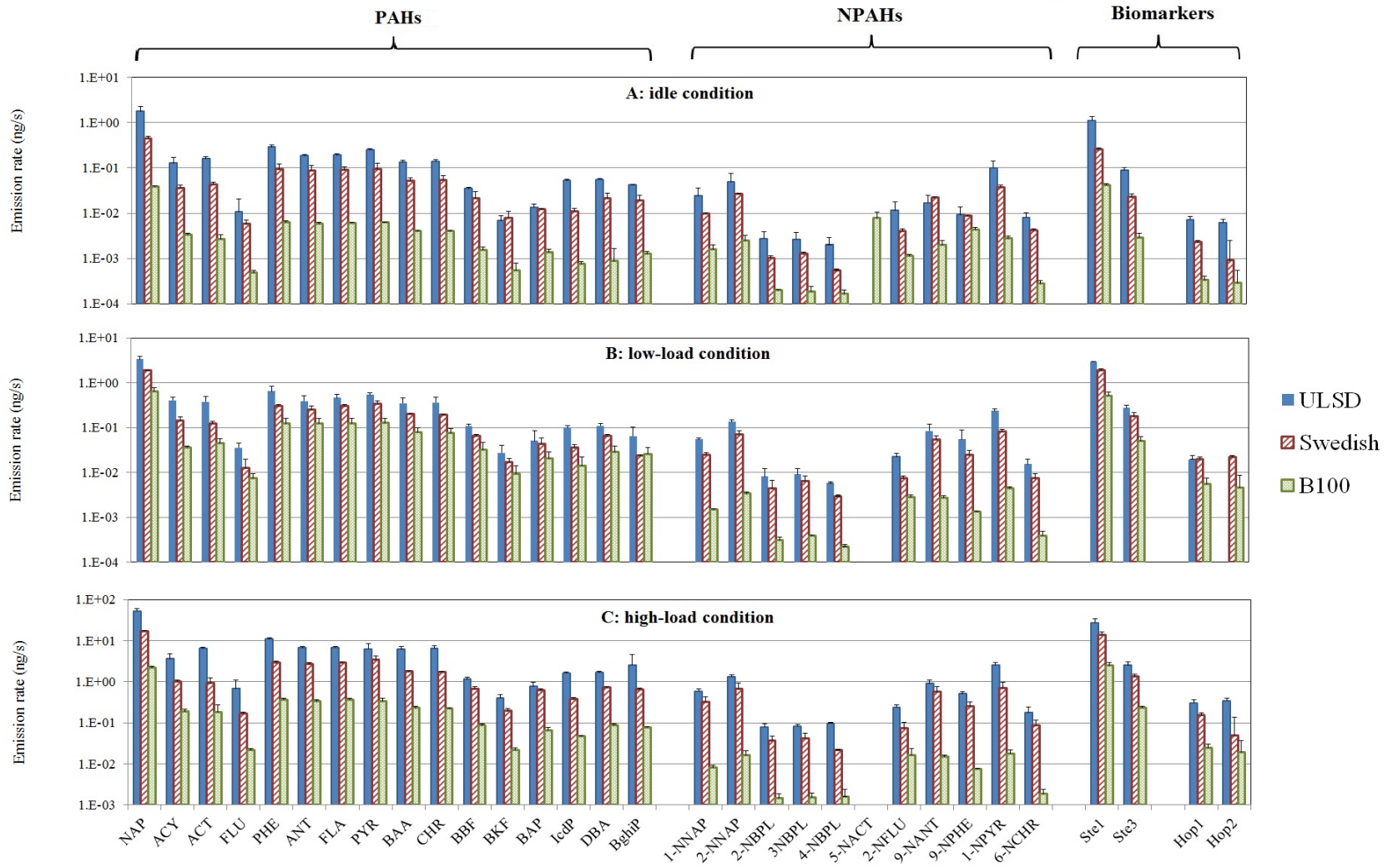

Figure 4.1 Effect of fuel on speciated SVOC emission rates (ng/s) for (A) idling, (B) low-load, and (C) high-load conditions. Error bars show one standard deviation.

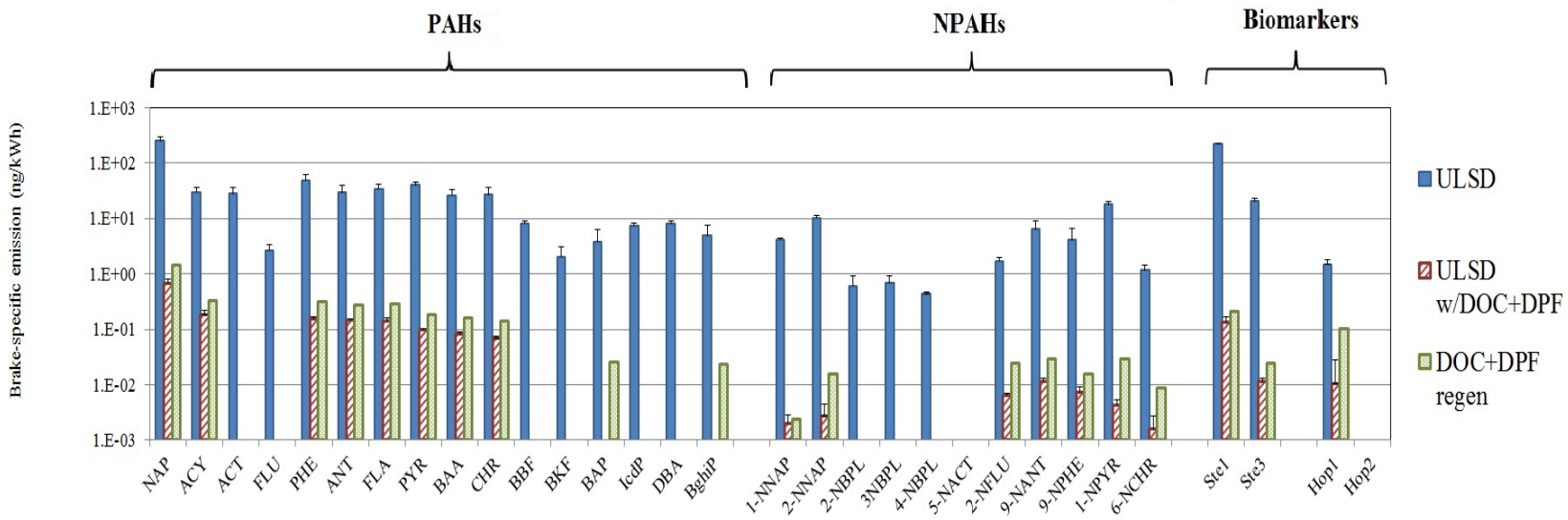

Figure 4.2 Effect of DOC+DPF on speciated SVOC emission

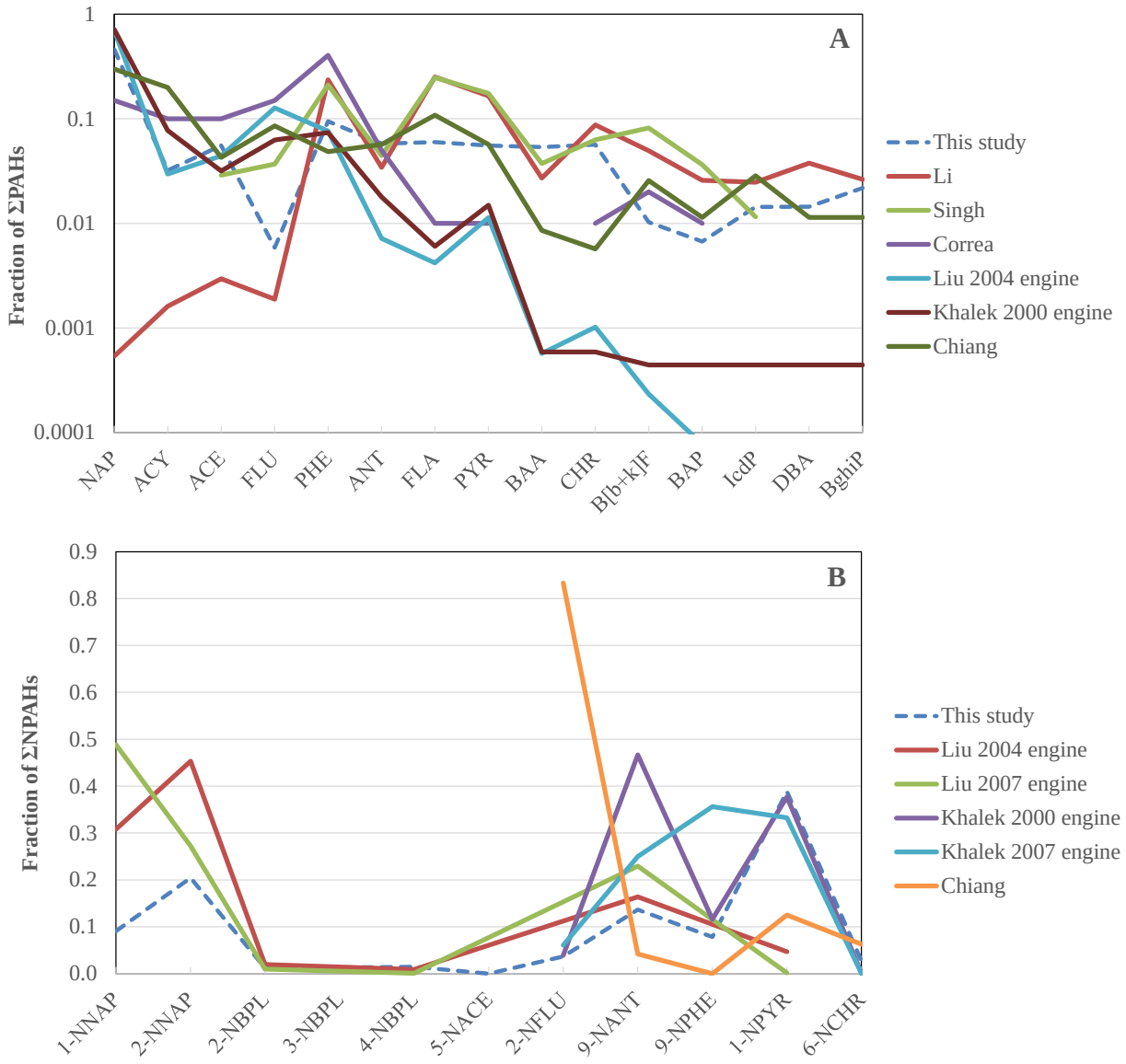

Figure 4.3 Comparison between this study's profile and profiles in literature. (A) PAHs; (B) NPAHs.

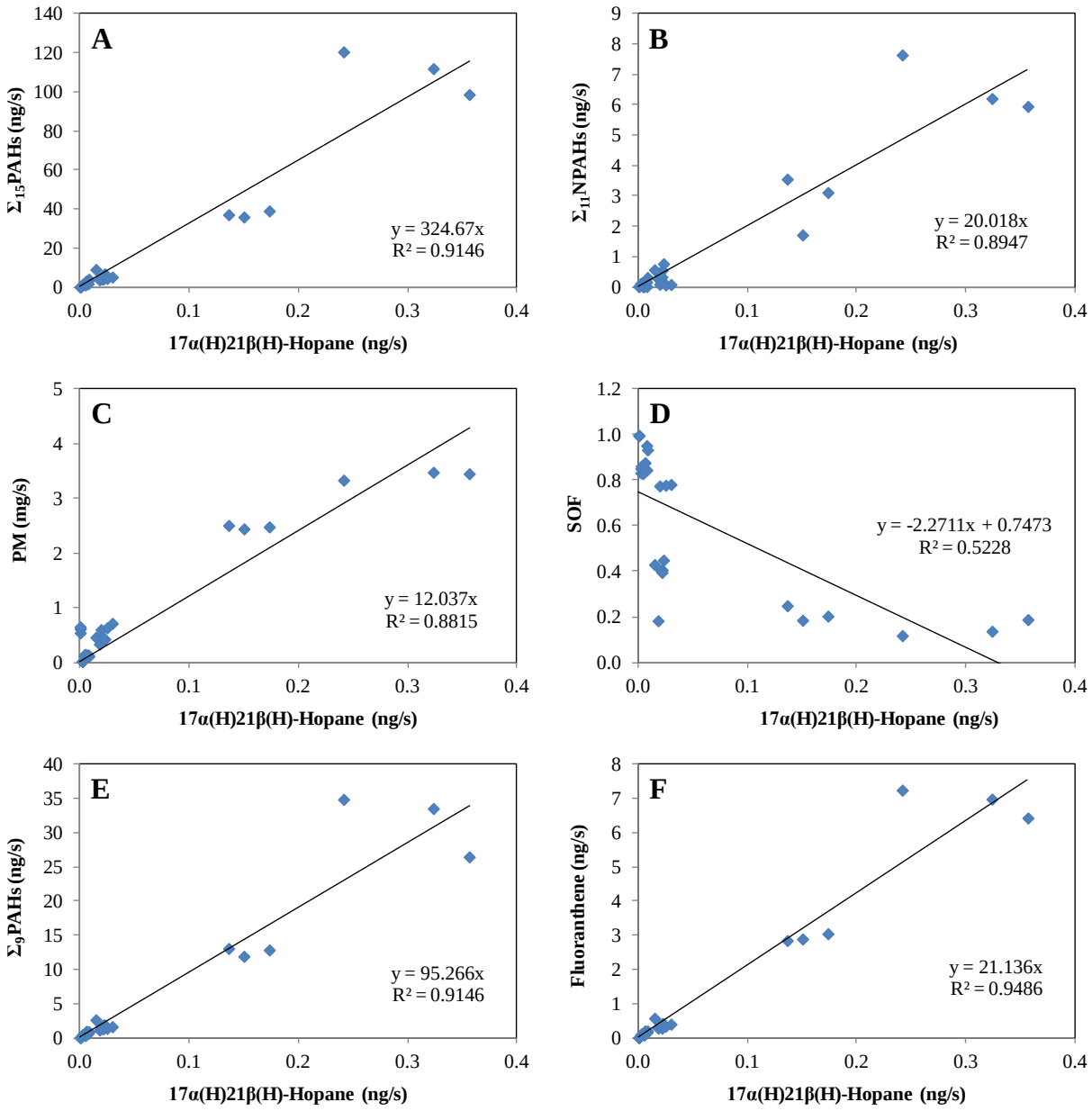

Figure 4.4 Emission rates of PAHs, NPAHs, PM and SOF versus 17α(H)21β(H)-hopane.

4.7 Appendix

Table A4.1 Instrument detection limits (IDLs) of target compounds

Group	Compound	IDL (ng/mL)
PAHs	Naphthalene	0.50
	Acenaphthylene	0.25
	Acenaphthene	0.22
	Fluorene	0.07
	Phenanthrene	0.22
	Anthracene	0.18
	Fluoranthene	0.32
	Pyrene	0.25
	Benzo[a]anthracene	0.25
	Chrysene	0.28
	Benzo[b]fluoranthene	0.08
	Benzo[k]fluoranthene	0.05
	Benzo[a]pyrene	0.05
	Dibenzo[a,h]anthracene	0.09
	Indeno[1,2,3-cd]pyrene	0.12
	Benzo[g,h,i]perylene	0.05
NPAHs	1-Nitronaphthalene	0.01
	2-Nitronaphthalene	0.01
	2-Nitrobiphenyl	0.01
	3-Nitrobiphenyl	0.01
	4-Nitrobiphenyl	0.01
	5-Nitroacenaphthene	0.01
	2-Nitrofluorene	0.01
	9-Nitroanthracene	0.01
	9-Nitrophenanthrene	0.01
	1-Nitropyrene	0.01
	6-Nitrochrysene	0.01
Hopanes	17α(H),21β(H)-Hopane	0.05
	17α(H)-22,29,30-Trisnorhopane	0.08
	17α(H),21β(H)-30-Norhopane	0.08
	22R-17α(H),21β(H)-Homohopane	0.05
	22S-17α(h),21β(h)-Homohopane	0.05
Steranes	20S-5α(H), 14α(H), 17α(H)-Cholestane	0.04
	20R-5α(H), 14α(H), 17α(H)-Cholestane	0.06
	20R-5α(H), 14β(H), 17β(H)-Cholestane	0.04
	20R-5α(H), 14β(H), 17β(H)-24-Methylcholestane	0.06
	20R-5α(H), 14α(H), 17α(H)-24-Ethylcholestane	0.05
	20R-5α(H), 14β(H), 17β(H)-24-Ethylcholestane	0.06

Table A4.2 Profiles of PAHs and NPAHs for diesel engine exhaust.

Group	Chemical	Emission rate (ng/s)	Fraction of the sum
$PM_{2.5}$		2000600	
PAHs	Naphthalene	30.2	0.461
	Acenaphthylene	2.10	0.032
	Acenaphthene	3.65	0.056
	Fluorene	0.39	0.006
	Phenanthrene	6.22	0.095
	Anthracene	3.81	0.058
	Fluoranthene	3.93	0.060
	Pyrene	3.64	0.055
	Benzo[a]anthracene	3.52	0.054
	Chrysene	3.72	0.057
	Benzo[b+k]fluoranthrene	0.67	0.010
	Benzo[a]pyrene	0.44	0.007
	Indeno[1,2,3-c,d]pyrene	0.94	0.014
	Dibenzo[a,h]anthracene	0.95	0.014
	Benzo[g,h,i]perylene	1.43	0.022
	Σ_{15}PAHs/PM	3.21E-05	
NPAHs	1-Nitronaphthalene	0.35	0.091
	2-Nitronaphthalene	0.78	0.204
	2-Nitrobiphenyl	0.05	0.012
	3-Nitrobiphenyl	0.05	0.013
	4-Nitrobiphenyl	0.06	0.015
	5-Nitroacenaphthene	0	0
	2-Nitrofluorene	0.14	0.037
	9-Nitroanthracene	0.52	0.136
	9-Nitrophenanthrene	0.30	0.078
	1-Nitropyrene	1.48	0.388
	6-Nitrochrysene	0.10	0.027
	Σ_{11}NPAHs/PM	1.91E-06	
Hopane	17α(H),21β(H)-Hopane	0.175	
	PM/17α(H),21β(H)-Hopane	1.1E+07	

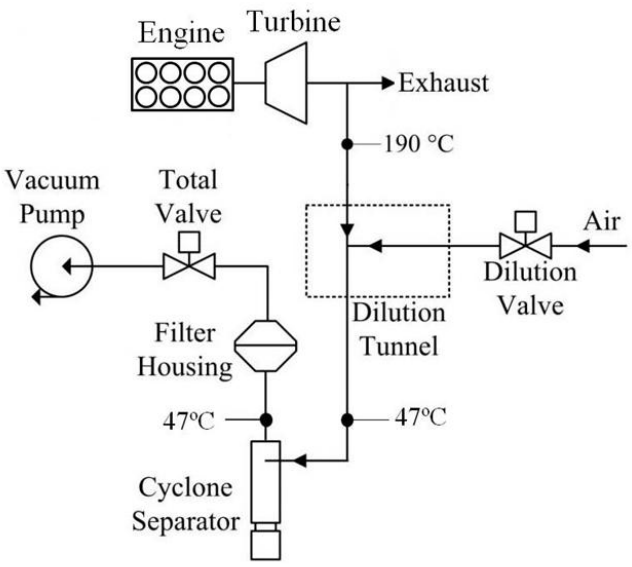

Figure A4.1 PM sampling system flow diagram

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Chapter 5 Integrity of target compounds in diesel exhaust particulate matter[3]

5.1 Abstract

Diesel exhaust particulate matter (PM) contains many semivolatile organic compounds (SVOCs) of environmental and health significance. This study investigates the integrity of 25 SVOCs, including polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs (NPAHs), and petroleum biomarkers hopanes and steranes, in diesel exhaust PM. Diesel engine PM, generated using an engine test bench, three engine conditions, and ultra-low sulfur diesel (ULSD), was collected on PTFE-bonded borosilicate glass fiber filters. Storage losses were evaluated for three cases: conditioning filters in clean air at 25 °C and 33% relative humidity (RH) for 24 h; storing filter samples (without extraction) wrapped in aluminum foil at 4 °C for up to one month; and storing filter extracts in glass vials capped with Teflon crimp seals at 4 °C for up to six months. After conditioning filters for 24 h, 30% of the more volatile PAHs were lost, but lower volatility NPAHs, hopanes and steranes showed negligible changes. Storing wrapped filters and extracts at 4 °C for up to one month did not lead to significant losses, but storing extracts for five months led to significant losses of PAHs and NPAHs; hopanes and steranes demonstrated greater integrity. These results suggest that even relatively brief filter conditioning periods, needed for gravimetric measurements of PM mass, and extended storage of filter extracts can lead to underestimates of SVOC concentrations. Thus, SVOC sampling and analysis protocols should utilize stringent criteria and performance checks to identify and limit possible biases occurring during filter and extract processing.

5.2 Introduction

Air sampling of target SVOCs involves, in brief, collection of a sufficient sample on an appropriate matrix, e.g., 10 to 150 m^3 of air on a Teflon or quartz filter for the particulate

[3] Results of this chapter have been published in *Water, Air & Soil Pollution* 224(8): 1-14.

fraction, and a polyurethane foam cartridge for the vapor fraction, followed by storage, extraction, purification and analysis.

Because PAHs and NPAHs can volatize, decompose or transform during sample handling and storage, an important consideration in measuring SVOCs is the integrity of collected samples, especially for air samples, which involve multiple, sequential and complex steps. Sample integrity must be characterized and maintained, otherwise the measurements cannot be used quantitatively, and even some qualitative uses may be limited. Several sampling and analysis protocols specify sample storage times. EPA Method SW-846 for solid samples (e.g., soil, sediment, sludge, ash, etc.) requires extraction within 14 days of sample collection, and analysis within 40 days following extraction (EPA 2008). EPA method 3542 for air sampling specifies the same extraction and analysis times, and also specifies storage of filters at 4 °C (EPA 1996).

A few studies have investigated the storage losses of SVOC samples. Mussel tissues stored at -80 °C and -120 °C showed PAH concentrations that were stable for up to 10 years (Schantz et al. 2001). Soils stored at 4 °C for two weeks showed significant losses of 2- to 5-ring PAHs due to biodegradation; storage at -20 °C or the addition of a biocide reduced losses (Rost et al. 2002). PM collected on Teflon filters wrapped in foil, packed in glass bottles, and stored at room temperature in a darkened room for up to 118 days gave consistent readings for PAHs ranging in volatility from fluoranthene/pyrene to coronene (Oda et al. 1998). PM collected on quartz fiber filters stored for one week at 4 °C were stable for 50 of 61 PAHs, although 11 or the more volatile PAHs showed losses of 50 to 80% (Oda et al. 1998).Tests using artificially generated particles and quartz filters indicated that PAHs were stable for up to 120 days when stored at -79 °C (Sverdrup et al. 1990). Only one NPAH study was identified, which tested the storage losses of a single NPAH compound (2-nitrofluorene) on quartz filters. Filter samples stored at both -79 °C and 20 °C showed losses of about 40% after 30 days of storage (Sverdrup et al. 1990).

The integrity of PAH samples following extraction, typically dissolved in organic solvents, has received some attention. Ampouled PAH solutions in acetonitrile and toluene were stable after 1-year storage at both -20 and 20 °C (Vaessen et al. 1988). 61 PAHs tested stored in acetonitrile were stable after 4 weeks of storage at 4 °C (Oda et al. 1998). Three PAHs (fluorene,

anthracene and benzo[a]pyrene) stored in six separate organic solvents (methanol, acetonitrile, DMSO, dichloromethane, hexane and cyclohexane) were stable for 20 days, with the exceptions of anthracene and benzo[a]pyrene in DMSO (Dabrowska et al. 2008). Post-extraction integrity of NPAH, hopane and sterane samples has not been demonstrated. Air sampling can involve storage and conditioning of PM filters if the gravimetric mass is determined from the same filter. No information was identified pertaining to losses of hopanes and steranes during filter storage and conditioning, or during extract storage. Similarly, no information was located regarding integrity of PAH and NPAH compounds during filter conditioning.

This chapter investigates the integrity of particulate phase PAHs, NPAHs, hopanes and steranes in diesel exhaust sampled on PTFE-bonded glass fiber filters. Samples obtained from well-controlled engine dynamometer laboratory tests are used to investigate effects of filter conditioning, storage of filters before extraction, and storage of extracts. We believe that this is the first study examining losses of NPAHs and diesel biomarkers in both filters and extracts. This study is also unique in its use of PM from a real engine, which is significant because sample integrity may be affected by the PM composition (or matrix). The study's recommendations have implications for the design, methods, and quality assurance activities of future studies.

5.3 Materials and Methods

5.3.1 Experimental design

Several series of laboratory experiments were used to investigate the integrity of SVOCs collected as PM samples of diesel engine exhaust. As target compounds, 14 PAHs, 7 NPAHs, 2 hopanes and 2 steranes were selected since they are frequently detected in airborne PM. Table 5.1 lists these compounds, CAS numbers, selected chemical properties, abbreviations used in this chapter, and instrumental detection limits (IDLs). Table 5.2 summarizes the test conditions for PM sampling. Diesel exhaust was generated by a 6.4 L 2008 Ford Power Stroke diesel engine using conventional ultra low sulfur diesel (ULSD). The engine was operated under three engine conditions: idle (650 rpm, 0 bar Brake Mean Effective Pressure or BMEP); low load (1500 rpm, 6 bar BMEP); and high load (2500 rpm, 9 bar BMEP).The engine configuration represented a 2004 engine calibration without exhaust gas after-treatment. This configuration and operating conditions were selected because they produce relatively high PM concentrations, which

facilitated sample collection, and because they are representative of many current in-use diesel engines.

A total of 24 filters were collected, including 1 exhaust blank and 5 field blanks, using the exhaust gas dilution sampling system (described below). Since samples were obtained consecutively, side-by-side replicate samples could not be collected. To obtain replicates and increase the number of samples available for the tests, nine filters were cut into quarters using a cleaned razor blade, and designated as sub-samples a, b, c and d. While these quarter filters had only one-fourth the mass of a full filter, loadings were designed to obtain sufficient mass for analytical purposes.

The first set of experiments tested the effect of filter conditioning, a standard practice to equilibrate air and exhaust sampling filters to a constant temperature and humidity needed to obtain repeatable weight measurements down to 1 or 2 µg (or better). These experiments used three loaded filters that had been sectioned into quarters. (Table A5.1 shows details pertaining to each set of experiments). Selected quarter filters were spiked with 1 µL spiking solution (described in Table 5.1) that contained known concentrations of the target compounds. These spiked filters served as a performance check to guarantee that the target SVOCs had large enough masses and can be measured quantitatively (i.e., well above MDLs). In addition, with the spiked and unspiked quarter filters, the possible difference in SVOC losses between high and low concentrations could be examined. For spiking, each quarter filter was supported by glass tubes placed on aluminum foil, and 1 µL of the spiking solution was slowly transferred from a 2 µL syringe throughout the filter. The spiking solution was not allowed to penetrate to the aluminum foil. Selected quarter filters were conditioned by placing them on clean aluminum foil in a glove box maintained at 25°C and 33% RH for 24 h. During conditioning, filters were unwrapped and exposed to air in the glove box.

A second set of experiments examined the effect of filter storage. These experiments used six loaded filters that were sectioned into quarters, and selected quarter filters were spiked as described above (Table A5.1). In this case, quarter filters were folded in half, individually wrapped in aluminum foil, packed in a zip-lock bag, and placed in a clean refrigerator at 4 °C for 0, 7 or 30 days.

The third experiment examined the integrity of extracts for extended storage periods (up to six months). A total of 12 filters were used. Full (uncut) and unspiked loaded filters were conditioned for 24 h after sampling, extracted, fractionated into three portions, and immediately analyzed, as described below. These extracts were then stored in 2 mL glass vials (Fisherbrand, Cat. No. 03-391-5) capped with Teflon crimp seals (National Scientific Company, Part No. C4011-1A), placed in a refrigerator at 4 °C, and reanalyzed after 1, 5 and 6 months.

5.3.2 Materials

PM samples were collected on 47 mm diameter PTFE-bonded glass fiber filters, which have been described in Chapter 4 Section 4.4.2.

Solvents were HPLC grade and obtained from Fisher Scientific Inc. (Pittsburgh, PA, USA). Florisil (60-100 mesh) and sodium sulfate (anhydrous, certified ACS granular, 10-60 mesh) for column chromatography were supplied by the same vendor. The spiking solution, which included 14 PAHs and 3 NPAHs at the concentrations listed in Table 5.1, was prepared from standard solutions of individual compounds (Cambridge Isotope Laboratories Inc., Andover, MA, USA).

5.3.3 Filter conditioning and weighing

Filters were conditioned and weighed before and after PM loading, following engine testing procedures (CFR 2013).Filters were conditioned in a glove box (Series 100 twin plastic glove box isolator, Terra Universal, Inc.) at 25°C and 33% RH for 24 h. Electrostatic charges on filters and instruments were neutralized using an ionizer (Terra Universal, Inc) placed in the filter weighing chamber for 30 min right before weighing. Then the filters were weighed twice to 1 µg precision using a microbalance (ME 5, Sartorius Inc., Edgewood, NY, USA). If the weights agreed within 5 µg, the results were averaged; otherwise filters were reweighed. After loading, selected filters were carefully cut into four equal sections.

5.3.4 Sample collection

The collection of PM samples from diesel engine exhaust has been described in detail in Chapter 4 Section 4.3.3.

5.3.5 Extraction, fractionation, and analysis

Filters were extracted by placing each in a 50 mL centrifuge tube, adding 25 mL of dichloromethane/hexane (4:1, v/v) to immerse the entire filter, and sonicating for 30 min (1510R-MTH, Branson Ultrasonics Corporation, Danbury, CT). The filter was then removed using a cotton stick and discarded. Extracts were passed through an activated Florisil column and fractionated into 3 portions. No additional cleaning of each fraction was necessary. The fractionation and GC-MS analysis have been described in detail in Chapter 2 Section 2.3.3.

5.3.6 Data analysis

Spiking reproducibility was quantified by calculating the absolute percent difference between the two quarters cut from the same filter that used the same conditioning and storage conditions. These statistics were calculated for each PAH and NPAH, as well as sum of the target PAHs and NPAHs (denoted as ΣPAHs and ΣNPAHs).

The effect of filter conditioning was evaluated as the relative change for each compound (using the difference between the averages of conditioned and unconditioned quarter filters, divided by the average of unconditioned filters). The uncertainty of the change was evaluated as its standard deviation, estimated by propagating the standard deviations of the average mass in conditioned and unconditioned quarter filters.

The effect of filter storage was evaluated as the relative change between quarter filters obtained from the same filter stored for different lengths of time. This helps to avoid variation due to engine emissions, spiking retention, and other factors. Specifically, unstored filter subsamples (unspiked: S5a and S5b; spiked: S2a and S2b) were compared to 7 day subsamples (unspiked: S5c and S5d; spiked: S2c and S2d). Similarly, subsamples stored for 7 days (unspiked: S6a and S6b; spiked: S3a and S3b) were compared to subsamples stored for 30 days (unspiked: S6c and S6d; spiked: S3c and S3d). Unspiked and spiked filters were compared separately.

The effect of extract storage was calculated as the relative change between stored and directly analyzed (unstored) extracts. Because losses for individual compounds across the three engine conditions were generally similar, results are averaged. Significant differences are discussed in the results.

5.3.7 Calibration and quality assurance

Calibration standards were prepared using three mixtures: 16 PAHs; 8 NPAHs (both Sigma-Aldrich, St. Louis, MO, USA); SRM 2266 and individual standards for one hopane (Hop19) and one sterane (Ster42) (Chiron Laboratories, Trondheim, Norway). Five concentrations were used: 0.01, 0.05, 0.10, 0.50, and 1.00ng/µL. Instrumental detection limits (IDLs) for each compound are reported in Table 5.1. All analytes were individually quantified against authentic standards when present as mixtures. Fluoranthene-d10 (Cambridge Isotope Laboratories Inc, Andover, MA, USA) and an internal standard (IS) PAH mixture (Wellington Laboratories, Guelph, ON, Canada) were used as ISs for PAH analyses. Nitrofluoanthene-d9 (Cambridge Isotope Laboratories Inc, Andover, MA, USA) was used as an IS for NPAH analyses. Lastly, n-tetracosane-d50 (Chiron Laboratories, Trondheim, Norway) was used as an IS for hopanes and steranes. Using a 25 µL syringe, 15 µL of the internal standard was added to each sample prior to GC-MS analysis.

Quality assurance (QA) measures included the regular use of blanks, replicates, spike recovery tests, and standard reference materials (SRM 2585, NIST, USA). To check for possible contamination, solvent blanks, lab blanks and field blanks were collected and analyzed using the procedures described above. No contamination of target compounds was found in the three types of blanks.

Spike recovery was 90-98% during the study, and the shift (abundance of target compounds in standard solutions before and after running a batch of samples) was within 20%. The reproducibility of spiked quarter filters for ΣPAH and ΣNPAH measurements, shown in Table A5.2, was within 10% for most filters. Reproducibility for individual compounds was similar (data not shown), indicating that spiking was reproducible. However, large differences were shown by one sample (S1, including subsamples S1a, S1b, S1c, and S1d). This was the first filter spiked, and the variability may be due to different spiking volumes among the quarter filters, penetration and loss of the spiking solution through the filter, or some other reason. (The reproducibility of spiking for hopanes and steraneswas not calculated as these compounds were not contained in the spiking solution.)

5.4 Results and Discussion

5.4.1 Filter conditioning

Unconditioned and conditioned filters (24 h at 25 °C and 33% RH) are compared in Table 5.3. PAH and NPAH results represent only unspiked filters collected at the low load condition (thus avoiding possible errors from spiking); hopane and sterane results include both spiked and unspiked filters (since spiking did not contain these compounds). Under the test conditions, five PAHs were detected on the quarter filters with masses from 0.02 ng for phenanthrene to 1.16 ng for naphthalene (Table A5.3). The average (± standard deviation) loss from conditioning was 27 ± 20% across the five PAHs. Acenaphthylene and phenanthrene had the highest losses, 39 ± 22% and 35 ± 36%, respectively. These PAHs had four or fewer rings and relatively high vapor pressure (Table 5.1), which may explain the losses. Considering that significant PAH levels have been detected downstream of PM filters in other studies (Schauer et al. 1999), it is not surprising that filter conditioning at 25 °C for 24 h can reduce PAHs in PM samples. Greater losses would be expected with longer conditioning periods, e.g., a 60% loss of fluorene has been reported for surrogate filter samples stored for 30 days at 20 °C (Sverdrup et al. 1990).

Six NPAHs were detected on the quarter filters (masses from 0.04 ng for 4-nitrobiphenyl to 0.72 ng for 6-nitrochrysene; Table A5.3). Conditioning produced an average loss of only 8 ± 38%. The replicates showed relatively large variation, probably because concentrations were low and sometimes near detection limits. The NPAHs have lower volatility than their parent PAHs (Dušek et al. 2002), and thus lower losses.

Hopane and sterane levels on the filters after conditioning increased by 23 ± 34% and 8 ± 21%, respectively, but these changes were not statistically significant (Table A5.3). Reproducibility was only fair for 17α(H)-22,29,30 trisnorhopane, probably because concentrations were low and close to detection limits. These compounds are known to be stable and non-volatile (Prince and Walters 2007), thus filter conditioning was not expected to produce significant losses.

In summary, filter conditioning produced significant losses of the most volatile PAHs, but other SVOCs were not affected. Experimental variation somewhat exceeded the normal criterion of 25%, a result of several factors. First, although samples S1 and S4 were collected

under the same engine operating condition, they were obtained sequentially (over a 1 h period), and thus reflect any variation in engine emissions and sampling conditions. Second, experimental variation reflects two sets of measurements (pre- and post-conditioning).Third, sectioning of filters can yield different amounts of PM and SVOCs. Fourth, GC-MS analyses on different days can vary due to instrumental fluctuations (Grimmer 1988; Kloster et al. 1992). Finally, as mentioned, concentrations of some compounds were low and near IDLs. Given these factors, the variation is reasonable.

5.4.2 Filter storage

Filter storage tests showed mostly small or negligible changes for PAHs, e.g., on average, PAHs gained 2 ± 32% after 7 days of storage, and lost 4 ± 15% after 30 days, as compared to 7 days (Table 5.3). These results are based on the seven PAHs found on the unspiked quarter filters, which were mostly four or fewer ring compounds as seen in the filter conditioning tests. Spiked filters also showed small changes for PAHs, with the exception of acenaphthene (ACT) due to an anomalously high concentration in sample S2d, a possible instrumental error. Excluding this compound, spiked PAHs gained 11 ± 46% after 7 days of storage, and lost 2 ± 16% after 30 days. Overall, PAHs in the stored filters were stable for 30 days, including the relatively volatile PAHs such as naphthalene, which had 30% losses after one day of conditioning.

NPAH changes due to filter storage were also small, e.g., average losses were 9 ±24% and 6 ± 17% after 7 and 30 days of storage, respectively, for combined spiked and unspiked quarter filters (Table 5.3). Several spiked and unspiked comparisons differed, e.g., 1-nitronaphthalene in spiked and unspiked quarter filters lost 30 ±9% and 8 ± 8%, respectively, after 7 days of storage, these results likely reflect measurement variation at the low concentrations seen (the spiking solution did not include 1-nitronaphthalene).

Changes in hopane and sterane levels during filter storage fell within the range expected for experimental variation, e.g., average changes below 12% (Table 5.3). Thus, filter storage for 30 days had little effect on these compounds.

Overall, SVOC changes over the 30 day period were modest, indicating many SVOCs will be retained for at least 30 days in filters that are wrapped in aluminum foil and stored at 4 °C. The variability in the experimental results is due to the same factors discussed in the

section on filter conditioning, e.g., low concentration samples, changes in the engine exhaust concentrations, and instrument fluctuations.

5.4.3 Extract storage

Extract storage tests are summarized in Figure 5.1. For PAHs dissolved in hexane/acetone, storage for one month had no effect, e.g., losses averaged 1 ± 44% across the three engine conditions. The high-load condition (2500 rpm - 9 bar) showed greater variation, however, individual filters were fairly consistent with those measured in the directly analyzed extract (data not shown). After five months, PAH losses became appreciable (56 ± 31%) and several compounds were not detected, e.g., anthracene and benzo[a]pyrene that were initially found at low concentrations, 0.08 and 0.45 ng/mL, respectively)(Figure A5.1c). After six months, no PAHs were detected. These results indicate that storage of PAH extracts in hexane/acetone solvents in glass vials with Teflon crimped seals at 4 °C should be limited to one month.

NPAHs dissolved in methanol and stored at 4 °C were stable for one month. Losses for the NPAHs detected (3, 5 and 6 compounds were detected under idle, low-load and high-load conditions, respectively) averaged 1 ± 66%. The large variation is due to the low concentrations of NPAHs in the extracts. After five months, losses averaged 23 ± 32% for the detected NPAHs (5 and 6 compounds in samples collected under low and high load, respectively) (Figure A5.2b & A5.2c). (The 5 month idle samples were considered invalid due to broken vial inserts. We attempted to restore these samples by re-dissolving the partially dried extract with additional solvent and transferring the mixture to new inserts and vials, but the NPAHs were not successfully recovered.) No NPAHs were detected in the extracts after six months of storage.

For the hopanes and steranes dissolved in hexane, storage for one month also showed negligible change (average loss of 2 ± 27%). After five months, losses for hopanes under the low-load condition (Figure A5.3b) were larger and variable (39 ± 28%), probably due to the low concentrations (0.04 – 0.07 ng/mL) of the extracts. Excluding the low-load condition, losses for hopanes and steranesaveraged 10 ± 28% at five months. After six months, hopanes and steranes were not detected. These results suggest that hopane and sterane extracts dissolved in hexane and stored at 4 °C are stable for at least one month, and five months may be acceptable with small (10%) losses.

After 6-month storage of extracts, most of the samples needed solvent additions (due to evaporation), which could introduce additional errors, e.g., incomplete mixing and transfer losses if vial inserts were replaced. This may explain the fact that no target SVOCs were detected in the extracts after six month.

In summary, storage of PAH, NPAH, hopane and sterane extracts in glass vials with Teflon crimped seals at 4 °C should be limited to about one month, although longer storage may not be detrimental for hopanes and steranes. Losses and variability will increase if solvents evaporate and solvent additions are used to re-dissolve extracts prior to analysis.

5.4.4 Strengths and limitations

This study was designed to investigate effects of processing and storage of SVOC samples collected as air samples. The study's strengths include the use of PM samples collected from diesel engine exhaust under controlled conditions, which allowed characterization using reproducible, real world and relevant samples. This is important since storage integrity may be affected by the matrix, i.e., the physical and chemical properties of the sample. Thus, results in the present paper should be more representative than those obtained using artificially generated PM. Other strengths include the use of environmentally relevant concentrations, consideration of a wide range of SVOCs, and the use of real extracts. On the last point, extracts may be affected by co-contaminants, impurities and other factors, and previous studies used known solutions of pure PAHs in solvents (Oda et al. 1998; Sverdrup et al. 1990; Vaessen et al. 1988). In addition, the use of sectioned filters provided replicates with filter-specific controls that helped to eliminate differences between filters, although it has the disadvantages of reducing sample mass and sensitivity, potentially increasing variability, and restricting PM mass measurements.

The study has several limitations. First, replicate filters collected under the same engine test condition were not ‘true’ replicates since samples were taken sequentially, not simultaneously. Although engine conditions were kept constant, variation in emissions and sampling conditions could alter the mass and composition of collected PM, as suggested by our results (Tables A5.4-A5.6). The temporal variation could be minimized using parallel sampling trains. The number of samples in each filter conditioning and storage test was small, and thus statistical hypothesis testing was not feasible. While the overall trends were clear, larger sample sizes would help to confirm the findings of the present study. Finally, we recognize that ambient

sampling networks, such as IMPROVE and Speciation Trends networks (Kleiman et al. 2003), use Teflon filters to collect PM2.5, not the borosilicate glass fiber filters used in the engine tests. We did not evaluate other filter types, but anticipate that our estimates of volatilization losses during filter conditioning will apply to other types of filters.

5.5 Conclusions

In this study, well controlled tests investigated the integrity of SVOCs samples collected from diesel engine exhaust. Conditioning filters for 24 h at 25 °C and 33% RH for weighing purposes did not significantly change concentrations of NPAHs, hopanes and steranes, however, approximately 30% of the more volatile PAHs were lost. Filters loaded with PM can be held for at least one month without appreciable losses of these four classes of compounds if the filter is wrapped in aluminum foil and held at 4 °C. Filter extracts (PAHs in hexane/acetone, NPAHs in methanol, hopanes and steranes in hexane) can be stored at 4 °C for at least one month without significant losses. Hopane and sterane extracts may be stored for five months or more with acceptable results.

Our findings show that even the relatively brief periods used to condition filters, which are needed for gravimetric measurements of PM mass, can lead to underestimates of PAH concentrations. Ideally, filter conditioning would not be used for SVOC measurements, and a separate parallel sampler would be used to determine gravimetric concentrations. Often, this is not feasible. To reduce potential biases, conditioning protocols might be altered by lowering temperatures (e.g., from 25 to 10 °C) and/or reducing conditioning times (e.g., from 24 to 12 h). Such temperatures will require refrigeration, and shorter times may not work if the filter or the collected PM is hydroscopic, e.g., PM containing a large fraction of sulfate aerosols. Additionally, SVOC sampling and analysis protocols might utilize performance checks and criteria aimed at identifying and limiting potential biases occurring during filter and extract processing, e.g., PM samples on glass fiber filters should be sealed appropriately, extracted within 30 days of collection, and analyzed within one month.

5.6 Tables and Figures

Table 5.1 List of target compounds

Group	Compound	Abbrev.	CAS #	# of rings	Vapor pressure (mmHg at 25 °C)	IDL (ng/mL)	Concentration in spiking solution (ng/µL)
PAHs	Naphthalene	NAP	91-20-3	2	8.50E-02	0.1	2.5
	Acenaphthylene	ACY	208-96-8	3	6.68E-03	0.2	2
	Acenaphthene	ACT	83-32-9	3	2.15E-03	0.1	5
	Phenanthrene	PHE	85-01-8	3	1.21E-04	0.1	10
	Anthracene	ANT	120-12-7	3	6.53E-06	0.1	1
	Fluoranthene	FLA	206-44-0	4	9.22E-06	0.2	7.5
	Pyrene	PYR	129-00-0	4	4.50E-06	0.1	2
	Benzo[a]anthracene	BAA	56-55-3	4	2.10E-07	0.1	10
	Chrysene	CHR	218-01-9	4	6.23E-09	0.2	10
	Benzo[b]fluoranthene	BBF	205-99-2	5	5.00E-07	0.3	10
	Benzo[k]fluoranthene	BKF	207-08-9	5	9.65E-10	0.3	0
	Benzo[a]pyrene	BAP	50-32-8	5	5.49E-09	0.1	1
	Indeno[1,2,3-c,d]pyrene	IND	193-39-5	6	1.25E-10 *	0.8	10
	Dibenzo[a,h]anthracene	DBA	53-70-3	5	9.55E-10	1.1	10
NPAHs	1-Nitronaphthalene	1-NNAP	86-57-7	2	4.80E-04	0.2	0
	2-Nitronaphthalene	2-NNAP	581-89-5	2	2.83E-04 *	0.2	1
	2-Nitrobiphenyl	2-NBPH	86-00-0	2	5.21E-04 *	0.2	0
	3-Nitrobiphenyl	3-NBPH	2113-58-8	2	1.01E-04 *	0.1	0.5
	4-Nitrobiphenyl	4-NBPH	92-93-3	2	3.01E-05 *	0.6	0
	2-Nitrofluorene	2-NFL	607-57-8	3	4.43E-06 *	0.2	0
	6-Nitrochrysene	6-NCHR	7496-02-8	4	7.61E-09 *	0.1	0.5
Hopanes	17α(h),21β(h)-Hopane	Hop19	471-62-5	5	3.91E-07 *	0.2	0
	17α(h)-22,29,30-Trisnorhopane	Hop15	53584-59-1	5	2.09E-06 *	0.6	0
Steranes	20s-5α(h),14α(h),17α(h)-Cholestane	Ster42	481-21-0	4	8.79E-06 *	0.3	0
	20r-5α(h),14β(h),17β(h)-Cholestane	Ster43	69483-47-2	4	n/a	0.3	0

*Predicted by (EPA 2012).

Table 5.2 Engine and PM sampling conditions for each filter.

Filter ID	Type	Speed (rpm)	BMEP[a] (bar)	Start of injection (degree ATDC)	EGR[b] (%)	Sampling time (s)	Sampling volume (L)	Cut into quarters
S1, S2, S3, S4, S5, S6	Sample	1500	6	3.5	14	330	55	Yes
B1, B2, B3	Field blank	n/a	n/a	n/a	n/a	n/a	n/a	Yes
S7, S8, S9	Sample	1500	6	3.5	14	330	55	No
S10, S11, S12	Sample	2500	9	3.5	17	90	15	No
S13, S14, S15	Sample	650	0	3.5	8	480	80	No
S16	Exhaust blank	n/a	n/a	n/a	n/a	330	55	No
S17, S18	Field blank	n/a	n/a	n/a	n/a	n/a	n/a	No

[a]BMEP: Brake Mean Effective Pressure.

[b]EGR: Exhaust Gas Recirculation.

Table 5.3 Effect of filter conditioning and storage, showing average percentage change and standard deviation (in parentheses).

		Filter conditioning	Filter storage			
		Change[a]	Change (Day7-1 vs. Day0)		Change (Day30 vs. Day7-2)	
Group	Compound	(conditioned vs. unconditioned) (%)	Unspiked (%)	Spiked (%)	Unspiked (%)	Spiked (%)
PAHs	Naphthalene	- 27 (3)	n/a	- 4 (8)	- 11 (12)	0 (3)
	Acenaphthylene	- 39 (22)	0 (31)	0 (86)	- 4 (5)	- 6 (46)
	Acenaphthene	- 3 (5)	- 6 (23)	123 (257)	- 1 (31)	0 (3)
	Phenanthrene	- 35 (36)	n/a	12 (48)	0 (1)	- 4 (6)
	Anthracene	n/a	n/a	2 (82)	n/a	- 3 (0)
	Fluoranthene	n/a	n/a	0 (21)	n/a	- 2 (12)
	Pyrene	- 29 (15)	- 2 (21)	- 10 (16)	- 7 (6)	- 9 (3)
	Benzo[a]anthracene	n/a	n/a	n/a	n/a	- 2 (7)
	Chrysene	n/a	n/a	n/a	n/a	- 2 (10)
	Benzo[b]fluoranthene	n/a	17 (44)	57 (13)	n/a	- 3 (8)
	Benzo[k]fluoranthene	n/a	n/a	n/a	n/a	7 (24)
	Benzo[a]pyrene	n/a	- 1 (32)	12 (43)	n/a	2 (6)
	Indeno[1,2,3-c,d]pyrene	n/a	n/a	26 (16)	n/a	- 5 (21)
	Dibenzo[a,h]anthracene	n/a	n/a	n/a	n/a	- 4 (6)
	Average	- 27 (20)	2 (32)	11 (46)[b]	- 4 (15)	- 2 (16)
NPAHs	1-Nitronaphthalene	n/a	- 8 (8)	- 30 (9)	- 11 (19)	- 25 (8)
	2-Nitronaphthalene	- 14 (4)	34 (8)	- 9 (37)	- 5 (40)	- 2 (29)
	2-Nitrobiphenyl	- 10 (61)	1 (9)	8 (8)	- 15 (7)	- 1 (2)
	3-Nitrobiphenyl	- 9 (45)	- 32 (15)	- 23 (4)	- 9 (8)	- 2 (7)
	4-Nitrobiphenyl	- 13 (31)	- 29 (8)	10 (49)	n/a	- 4 (4)
	6-Nitrochrysene	2 (21)	- 21 (11)	- 14 (46)	3 (18)	0 (2)
	Average	- 8 (38)	- 9 (10)	- 10 (32)	- 7 (20)	- 6 (13)
Hopanes[c]	17α(h),21β(h)-Hopane	7 (27)	- 2(14)		- 13 (16)	
	17α(h)-22,29,30-Trisnorhopane	39 (40)	- 22 (21)		25 (27)	
	Average	23 (34)	- 12 (18)		6 (22)	
Steranes[c]	20s-5α(h),14α(h),17α(h)-Cholestane	2 (14)	- 8 (11)		5 (11)	
	20r-5α(h),14β(h),17β(h)-Cholestane	13 (26)	- 9 (13)		1 (18)	
	Average	8 (21)	- 8 (12)		3 (15)	

[a]Results for PAHs and NPAHs include only unspiked filters to avoid possible errors due to spiking (see text).

[b]Excluded ACT.

[c]Results for hopanes and steranes combine both spiked and unspiked filters since the spiking solution did not contain these compounds.

n/a: Compound was not detected.

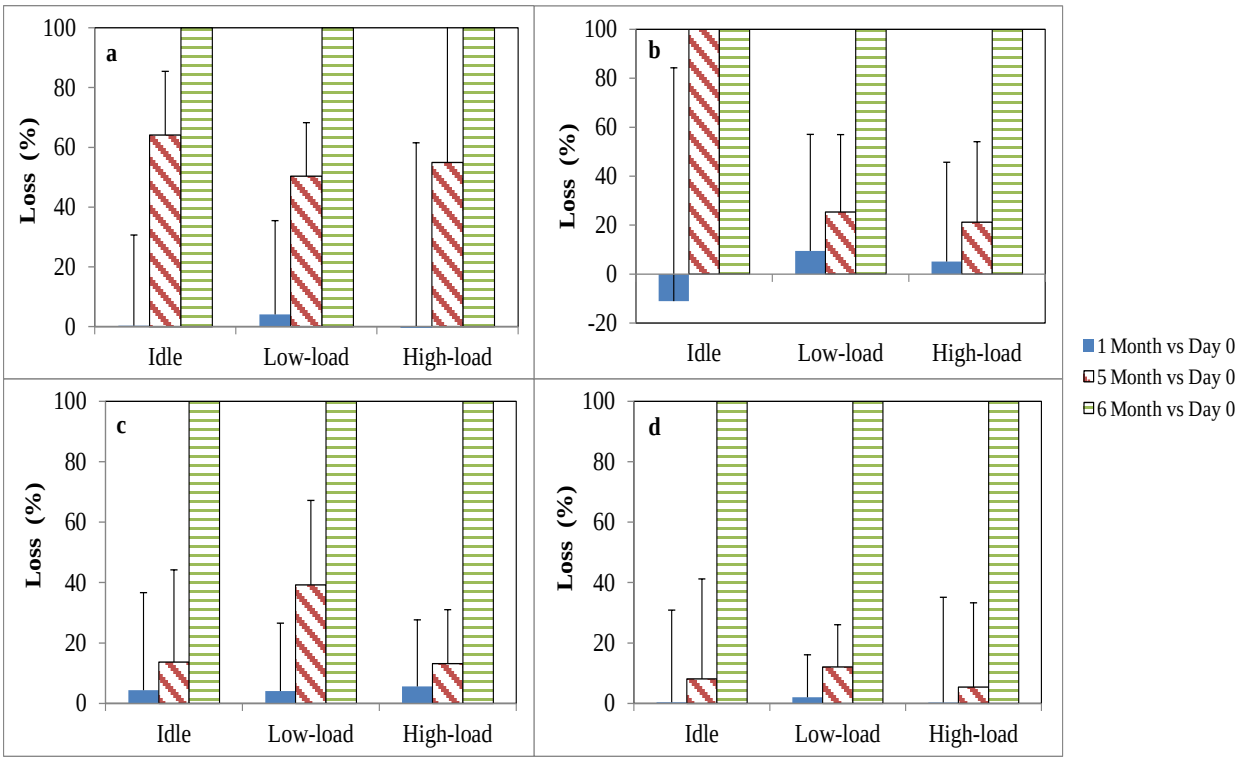

Figure 5.1 Effect of storage time for SVOCs in extracts.
Panels a through d show average percentage loss for sum of the detected PAHs, NPAHs, hopanes and steranes, respectively. Low-load is 1500 rpm – 6 bar. High-load is 2500 rpm – 9 bar. Error bars show one standard deviation.

5.7 Appendix

Table A5.1 Summary of experimental design.

Experiment 1: Stability during conditioning of filters

Filter ID[a]	Spiked[b]	Conditioning (h)
S1a, S1b	Yes	0
S1c, S1d	Yes	24
S4a, S4b	No	0
S4c, S4d	No	24
B1a, B1b	No	0
B1c, B1d	No	24

Experiment 2: Stability during storage of filters

Filter ID	Spiked[b]	Conditioning (h)	Storage of filter (d)
S2a, S2b	Yes	24	0
S2c, S2d, S3a, S3b	Yes	24	7
S3c, S3d	Yes	24	30
S5a, S5b	No	24	0
S5c, S5d, S6a, S6b	No	24	7
S6c, S6d	No	24	30
B2a, B2b	No	24	0
B2c, B2d, B3a, B3b	No	24	7
B3c, B3d	No	24	30

Experiment 3: Stability during storage of extracts

Filter ID	Spiked[b]	Conditioning	Storage of filter	Storage of extract
		h	d	m
S7-S18, Test #1	No	24	0	0
S7-S18, Test #2	No	24	0	1
S7-S18, Test #3	No	24	0	5
S7-S18, Test #4	No	24	0	6

[a]S1a, S1b, S1c and S1d are quarter filters from filter S1. See text for details.

[b]The composition of the spiking solution is shown in Table 5.1.

Table A5.2 Reproducibility of ΣPAH and ΣNPAH measurements among spiked quarter filters.

			ΣPAHs		ΣNPAHs	
Filter ID[a]	Conditioning (hr)	Storage of filter (day)	Average[b] (ng)	Absolute % difference[c] (%)	Average[b] (ng)	Absolute % difference[c] (%)
S1a, S1b	0	0	59.4	118	1.78	46.8
S1c, S1d	24	0	33.6	n/a[d]	0.56	84.1
S2a, S2b	24	0	42.5	7.2	0.67	1.0
S2c, S2d	24	7	53.2	20.3	0.67	1.3
S3a, S3b	24	7	43.5	7.8	1.62	1.3
S3c, S3d	24	30	41.8	10.7	1.60	1.9
Average[e]				11.5		1.4

[a] IDs are quarter filters.

[b]Average mass ΣPAHs or ΣNPAHs of the two quarter filters.

[c]Calculated as the absolute value of the difference between two quarter filters, divided by the average of these two quarter filters.

[d]Data for sample S1c is missing.

[e]Excludes data for samples S1a, S1b, S1c and S1d.

Table A5.3 Average mass of target SVOCs in conditioned and unconditioned quarter filters, and the change in conditioned quarter filters compared to unconditioned ones. Standard deviation in parentheses.

Group	Compound	Unconditioned (ng)	Conditioned (ng)	Change (%)
PAHs[a]	Sample size (N)	2	2	
	NAP	1.16 (0.04)	0.84 (0.01)	-27 (3)
	ACY	0.06 (0.02)	0.04 (0.01)	-39 (22)
	ACT	0.09 (0.00)	0.09 (0.00)	-3 (5)
	PHE	0.02 (0.01)	0.02 (0.00)	-35 (36)
	PYR	0.46 (0.02)	0.33 (0.07)	-29 (15)
	Average			-27 (20)
NPAHs[a]	Sample size (N)	2	2	
	2-NNAP	0.06 (0.00)	0.05 (0.00)	-14 (4)
	2-NBPH	0.09 (0.05)	0.08 (0.04)	-10 (61)
	3-NBPH	0.14 (0.05)	0.13 (0.04)	-9 (45)
	4-NBPH	0.04 (0.01)	0.04 (0.01)	-13 (31)
	6-NCHR	0.72 (0.10)	0.73 (0.11)	2 (21)
	Average			-9 (38)
Hopanes[b]	Sample size (N)	4	4	
	Hop19	0.02 (0.00)	0.02 (0.00)	7 (27)
	Hop15	0.02 (0.00)	0.03 (0.01)	39 (40)
	Average			23 (34)
Steranes[b]	Sample size (N)	4	4	
	Ster42	2.29 (0.20)	2.34 (0.25)	2 (14)
	Ster43	0.19 (0.03)	0.22 (0.04)	13 (26)
	Average			8 (21)

[a]Includes only unspiked quarter filters.

[b]Includes both spiked and unspiked quarter filters.

Table A5.4 Mass of PAHs in stored and unstored quarter filters, separated by filter. Data presented are mean (SD).

Treatment	Compound	Day 0 (ng)	Day 7-1 (ng)	Day 7-2 (ng)	Day 30 (ng)	Change (Day7-1 vs. Day0) (%)	Change (Day30 vs. Day7-2) (%)
Unspiked	Filters involved	S5a, S5b	S5c, S5d	S6a, S6b	S6c, S6d	S5a, S5b, S5c, S5d	S6a, S6b, S6c, S6d
	NAP	0.00 (0.00)	0.00 (0.00)	1.22 (0.00)	1.09 (0.15)	n/a	-11 (12)
	ACY	0.16 (0.04)	0.16 (0.03)	0.10 (0.00)	0.09 (0.00)	0 (31)	-4 (5)
	ACT	0.15 (0.02)	0.14 (0.03)	0.08 (0.02)	0.08 (0.02)	-6 (23)	-1 (31)
	PHE	0.00 (0.00)	0.00 (0.00)	0.02 (0.00)	0.02 (0.00)	n/a	0 (1)
	PYR	0.21 (0.03)	0.20 (0.03)	0.37 (0.02)	0.34 (0.01)	-2 (21)	-7 (6)
	BBF	0.47 (0.08)	0.55 (0.19)	0.00 (0.00)	0.00 (0.00)	17 (44)	n/a
	BAP	0.16 (0.04)	0.16 (0.03)	0.00 (0.00)	0.00 (0.00)	-1 (32)	n/a
	Average					2 (32)	-4 (15)
Spiked	Filters involved	S2a, S2b	S2c, S2d	S3a, S3b	S3c, S3d	S2a, S2b, S2c, S2d	S3a, S3b, S3c, S3d
	NAP	1.85 (0.09)	1.77 (0.13)	1.79 (0.06)	1.79 (0.00)	-4 (8)	0 (3)
	ACY	1.20 (0.16)	1.20 (1.02)	0.24 (0.08)	0.23 (0.09)	0 (86)	-6 (46)
	ACT	0.97 (0.23)	2.17 (2.45)	0.77 (0.01)	0.77 (0.02)	123 (257)	0 (3)
	PHE	2.97 (0.20)	3.35 (1.41)	2.60 (0.09)	2.50 (0.12)	12 (48)	-4 (6)
	ANT	1.91 (0.17)	1.95 (1.55)	0.30 (0.00)	0.30 (0.00)	2 (82)	-3 (0)
	FLA	3.56 (0.33)	3.55 (0.68)	1.26 (0.06)	1.24 (0.14)	0 (21)	-2 (12)
	PYR	3.70 (0.30)	3.55 (0.52)	3.44 (0.06)	3.14 (0.10)	-10 (16)	-9 (3)
	BAA	0.00 (0.00)	0.00 (0.00)	3.00 (0.15)	2.92 (0.15)	n/a	-2 (7)
	CHR	0.00 (0.00)	0.00 (0.00)	2.46 (0.18)	2.42 (0.18)	n/a	-2 (10)
	BBF	10.03 (0.44)	15.74 (1.09)	5.82 (0.39)	5.66 (0.26)	57 (13)	-3 (8)
	BKF	0.00 (0.00)	0.00 (0.00)	1.10 (0.21)	1.18 (0.14)	n/a	7 (24)
	BAP	2.57 (0.30)	2.87 (1.06)	0.20 (0.01)	0.21 (0.00)	12 (43)	2 (6)
	IND	13.71 (0.14)	17.30 (2.25)	17.93 (2.41)	16.95 (3.02)	26 (16)	-5 (21)
	DBA	0.00 (0.00)	0.00 (0.00)	2.53 (0.07)	2.44 (0.14)	n/a	-4 (6)
	Average					11 (46)[a]	-2 (16)

[a]Excludes ACT.

Table A5.5 Mass of NPAHs in stored and unstored quarter filters, separated by filter. Data presented are mean (SD).

Treatment	Compound	Day 0 (ng)	Day 7-1 (ng)	Day 7-2 (ng)	Day 30 (ng)	Change (Day7-1 vs. Day0) (%)	Change (Day30 vs. Day7-2) (%)
Unspiked	Filters involved	S5a, S5b	S5c, S5d	S6a, S6b	S6c, S6d	S5a, S5b, S5c, S5d	S6a, S6b, S6c, S6d
	1-NNAP	0.109 (0.005)	0.100 (0.007)	0.127 (0.022)	0.113 (0.014)	-8 (8)	-11 (19)
	2-NNAP	0.103 (0.002)	0.138 (0.007)	0.273 (0.084)	0.258 (0.076)	34 (8)	-5 (40)
	2-NBPH	0.138 (0.007)	0.140 (0.010)	0.128 (0.006)	0.109 (0.008)	1 (9)	-15 (7)
	3-NBPH	0.575 (0.014)	0.393 (0.084)	0.243 (0.012)	0.222 (0.015)	-32 (15)	-9 (8)
	4-NBPH	0.189 (0.013)	0.134 (0.013)	0.000 (0.000)	0.000 (0.000)	-29 (8)	n/a
	6-NCHR	0.808 (0.038)	0.640 (0.082)	0.753 (0.115)	0.777 (0.063)	-21 (11)	3 (18)
	Average					-9 (10)	-7 (20)
Spiked	Filters involved	S2a, S2b	S2c, S2d	S3a, S3b	S3c, S3d	S2a, S2b, S2c, S2d	S3a, S3b, S3c, S3d
	1-NNAP	0.017 (0.002)	0.012 (0.001)	0.018 (0.002)	0.014 (0.000)	-30 (9)	-25 (8)
	2-NNAP	0.076 (0.026)	0.070 (0.015)	0.071 (0.014)	0.069 (0.015)	-9 (37)	-2 (29)
	2-NBPH	0.273 (0.019)	0.294 (0.001)	0.284 (0.002)	0.283 (0.005)	8 (8)	-1 (2)
	3-NBPH	0.112 (0.001)	0.086 (0.004)	0.305 (0.015)	0.298 (0.014)	-23 (4)	-2 (7)
	4-NBPH	0.028 (0.012)	0.031 (0.003)	0.055 (0.002)	0.053 (0.001)	10 (49)	-4 (4)
	6-NCHR	0.072 (0.038)	0.062 (0.004)	0.804 (0.014)	0.806 (0.004)	-14 (46)	0 (2)
	Average					-10 (32)	-6 (13)

Table A5.6 Mass of hopanes and steranes in stored and unstored quarter filters, separated by filter. Data presented are mean (SD).

Group	Compound	Day 0 (ng)	Day 7-1 (ng)	Day 7-2 (ng)	Day 30 (ng)	Change (Day7-1 vs. Day0) (%)	Change (Day30 vs. Day7-2) (%)
	Filters involved	S2a, S2b, S5a, S5b	S2c, S2d, S5c, S5d	S3a, S3b, S6a, S6b	S3c, S3d, S6c, S6d		
Hopanes	Hop19	0.01 (0.00)	0.01 (0.00)	0.02 (0.00)	0.01 (0.00)	-2 (14)	-13 (16)
	Hop15	0.02 (0.00)	0.01 (0.00)	0.01 (0.00)	0.02 (0.00)	-22 (21)	25 (27)
	Average					-12 (18)	6 (22)
Steranes	Ster42	2.03 (0.07)	1.87 (0.21)	1.75 (0.17)	1.84 (0.06)	-8 (11)	5 (11)
	Ster43	0.18 (0.00)	0.17 (0.02)	0.16 (0.02)	0.16 (0.02)	-9 (13)	1 (18)
	Average					-8 (12)	3 (15)

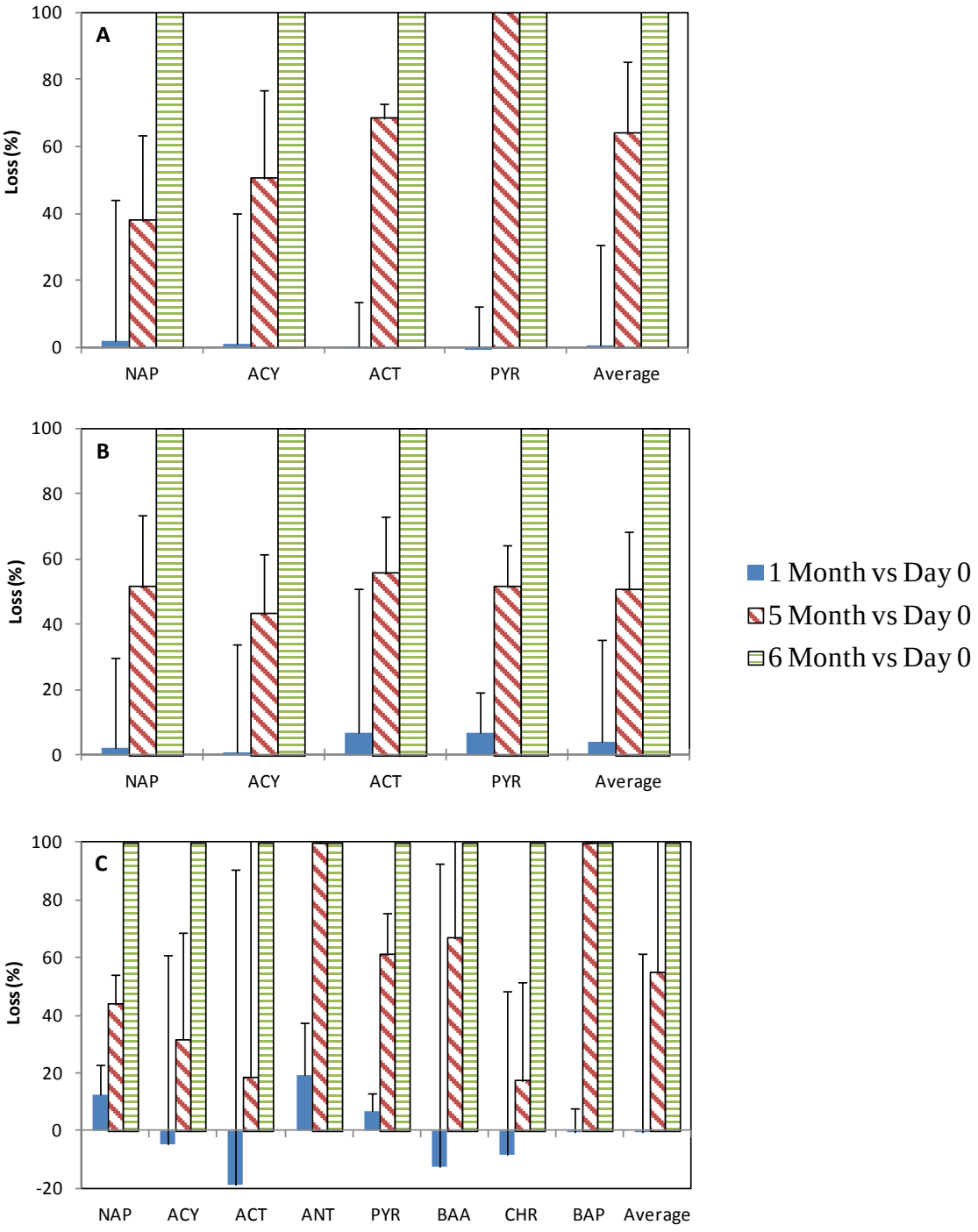

Figure A5.1 Losses of PAHs in stored extracts compared to directly analyzed extracts. (A) Idle; (B) 1500 rpm-6 bar; (C) 2500 rpm-9 bar.
Error bars show 1 SD.

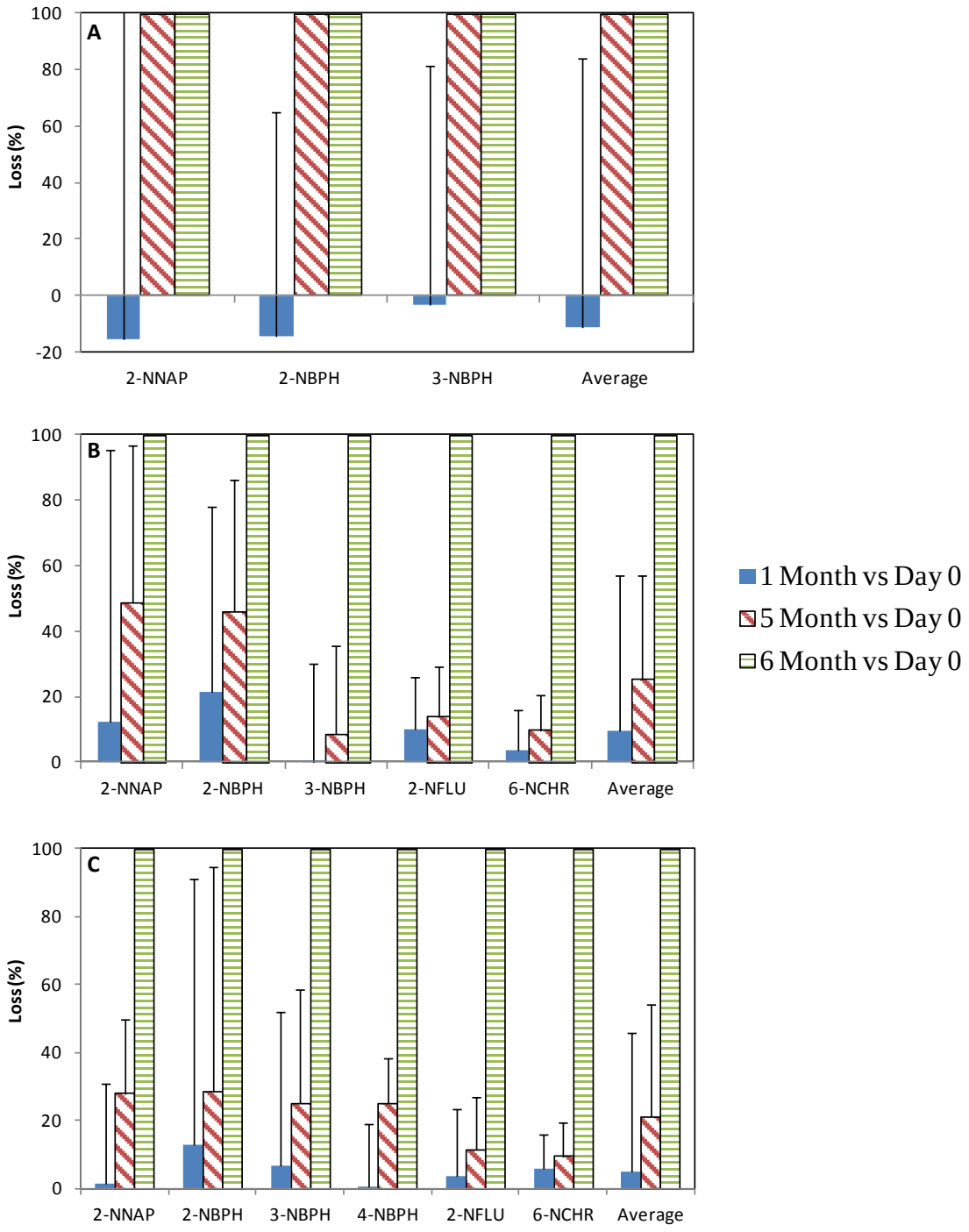

Figure A5.2 Losses of NPAHs in stored extracts compared to directly analyzed extracts. (A) Idle; (B) 1500 rpm-6 bar; (C) 2500 rpm-9 bar.
Error bars show 1 SD.

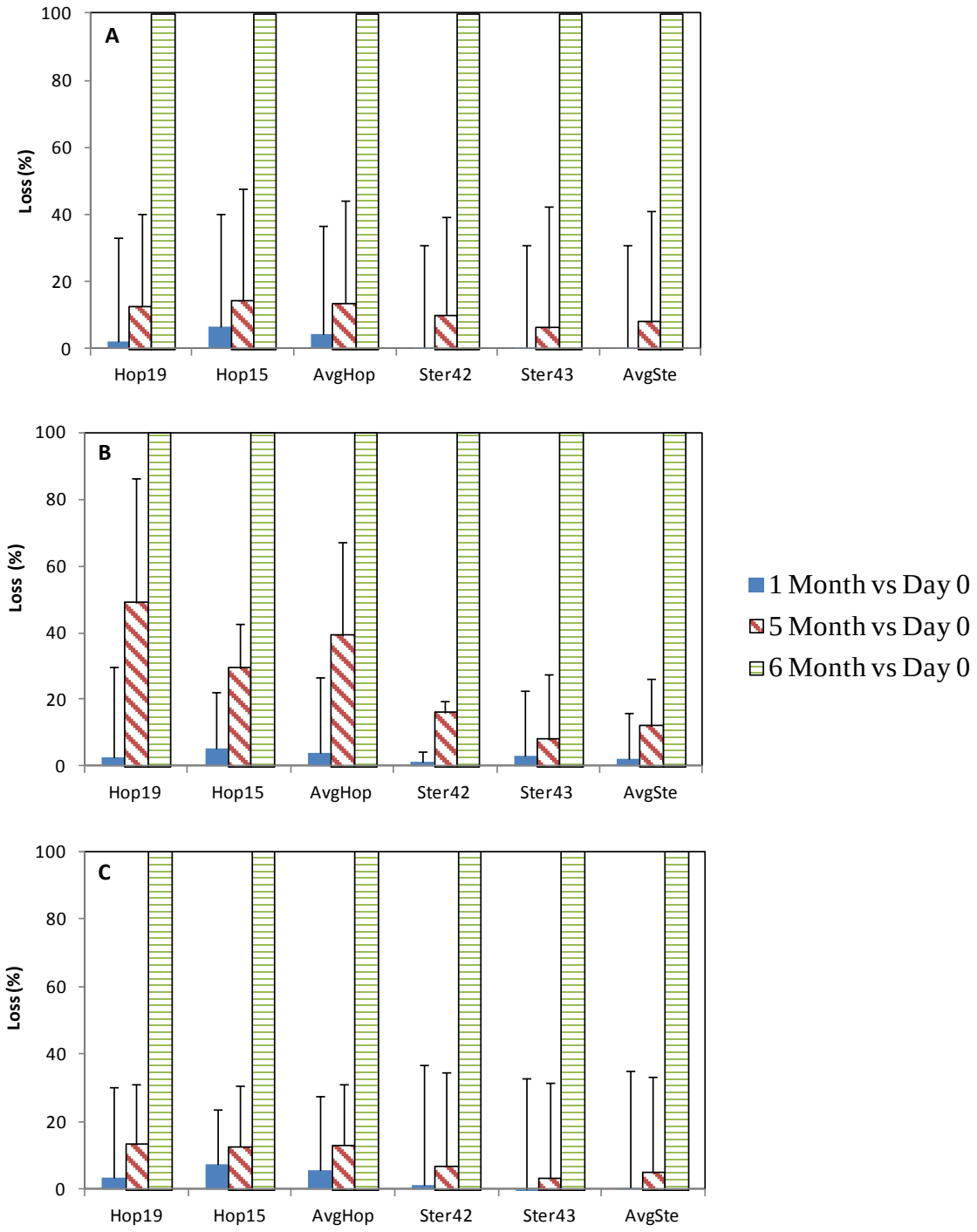

Figure A5.3 Losses of hopanes and steranes in stored extracts compared to directly analyzed extracts. (A) Idle; (B) 1500 rpm-6 bar; (C) 2500 rpm-9 bar.
Error bars show SD.

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Chapter 6 Multimedia fate modeling of PAHs and Nitro-PAHs in Lake Michigan

6.1 Abstract

While ambient levels of PAHs in the Great Lakes region have been monitored since 1990, and concentrations in water, sediment and biota have been occasionally reported, a comprehensive picture of their environmental distribution and fate has to be elucidated. In addition, the potentially more toxic nitro-derivatives of PAHs (NPAHs) have rarely been reported for this region. The present study uses fugacity-based models to predict the distribution and fate of PAHs and NPAHs in Lake Michigan, including air, water, soil, sediments and biota; results were compared to an extensive set of PAH and NPAH measurements in sediments and upper tropic level fish. The steady-state model used air emission data to predict concentrations of 16 priority PAHs. Due to the unavailability of NPAH emission data, emissions were estimated using the model in an inverse manner with the measured concentrations in sediments. Good agreement was found between predicted and measured PAH concentrations in air, but PAH concentrations in water and sediments were generally under-predicted, possibly caused by underestimating the degradation half-lives. The food web model accurately predicted concentrations in lake trout of heavier PAHs, but generally overestimated concentrations of lighter PAHs and NPAHs. This is attributed to an overestimate of metabolic half-lives and/or gut/gill absorption efficiencies. A dynamic model is used inversely to reconstruct historical emission rates of two representative PAHs using concentrations measurements in sediment cores. Results suggest that the PAH emission rates may be underestimated in the existing emission inventory for the Great Lakes region. Additional measurements and physiochemical data are needed to refine the models, and a more sophisticated model structure might be needed to better describe the environmental fate of NPAHs. The inverse modeling technique can help complement and improve current emission inventories.

6.2 Introduction

Polycyclic aromatic hydrocarbons (PAHs) are widely distributed and persistent environmental pollutants that are released into the atmosphere mainly through incomplete combustion (Neff 1979). In aquatic environments, PAHs arise from atmospheric deposition, urban runoff, municipal/industrial effluents and petroleum spills (Helfrich and Armstrong 1986). Some PAH compounds are carcinogenic to humans and fish (ATSDR 1995; Baumann et al. 1991; Logan 2007). Nitro-PAHs (NPAHs), which are nitro-derivatives of PAHs, also are widely distributed, a result of emissions from combustion sources as well as atmospheric transformations of PAHs (Yaffe et al. 2001; Perrini et al. 2005). Although environmental concentrations are far lower than the parent PAHs (Ozaki et al. 2010; Albinet et al. 2007), NPAHs can have stronger carcinogenic and mutagenic activity (Tokiwa et al. 1987).

The presence of PAHs in the U.S. Great Lakes has been of concern for decades (EPA and EC 2004) due to the many urban and industrial sources in this region, the large surface areas that increase atmospheric deposition (Simcik et al. 1999), the long hydrologic retention times and great depths (De Vault et al. 1996), and the presence of contaminated sites including the 26 Areas of Concern in the region. Historically, Lake Michigan has received large inputs of PAHs from the urban and industrial centers surrounding its southern portion (Helfrich and Armstrong 1986). Airborne PAHs levels have been monitored since 1990 by the Integrated Atmospheric Deposition Network (IADN) (Sun et al. 2006). Sediment measurements have been performed intermittently in 1982, 1986, 1993, 1996, 1998, 2001 and 2011 (Huang et al. 2014a; Eadie et al. 1982; Helfrich and Armstrong 1986; X. Zhang et al. 1993; Simcik et al. 1996; Su et al. 1998; Schneider et al. 2001). Surface water measurements are reported in a single study in 2000 (Offenberg and Baker 2000). PAH measurements in aquatic biota of Lake Michigan are scarce (Huang et al. 2014b; Zabik et al. 1996; Levengood and Schaeffer 2011; Eadie et al. 1982). Given the “biodilution” observed in marine organisms (Takeuchi et al. 2009; Baumard et al. 1998), PAH concentrations should be measured through the food web and not only in the top trophic level. With respect to NPAHs, our two recent reports (Huang et al. 2014a, 2014b) represent the only measurements in Lake Michigan. .

Significant anthropogenic PAH emissions date back to at least the Industrial Revolution. Efforts to estimate PAH emissions include the National Emissions Inventory (NEI), which

provides estimates of annual air emission at county, state and national level for 2002, 2005, 2008 and 2011 (EPA 2002-2011), and an earlier program of the Great Lakes Commission that assessed emissions from 1993 to 2008 (GLC 1993-2008). The accuracy and comprehensiveness of such inventories are always potential concerns since PAHs are emitted from numerous point, area, and mobile sources that are difficult to characterize, emission factors can be highly variable, and measurements are expensive and thus scarce. However, in large-scale system such as the Great Lakes, a historical record of airborne concentrations is provided indirectly via deposition and accumulation of PAHs in sediments. Thus, current levels and historical trends may be inferred using contaminant levels measured in surficial sediments and sediment cores, in conjunction with dynamic multimedia models. This makes it possible to estimate PAH emissions and levels in multiple compartments over time. Moreover, since sediment tends to be the ultimate sink of all releases, the approach can yield emission estimates that include all sources affecting the lake.

The present study estimates the fate and distribution of PAHs and NPAHs in Lake Michigan using fugacity-based models, which provide a relatively simple yet effective approach for multimedia analyses of chemical fate in the environment (Mackay 2010). Previously, level III fugacity models (steady-state, non-equilibrium) have been applied for several persistent organic contaminants (including several PAHs) in Southern Ontario and Quebec. Canada (Mackay and Hickie 2000; Mackay and Paterson 1991), and level IV models (nonsteady-state, non-equilibrium) have been applied for PCBs, DDT and brominated flame retardants (Lim and Lastoskie 2011; Li et al. 2006; Sweetman et al. 2002). These models also have been used to assess bioaccumulation of PCBs and PBBs in aquatic food webs (Campfens and Mackay 1997; Lim and Lastoskie 2011). Here, level III models are developed for PAHs and NPAHs in the Lake Michigan basin and compared to recent measurements in sediment and fish, and level IV models are developed to reconstruct the emission trends for selected PAHs. The models can help understand the overall behaviors of PAHs and NPAHs in the Lake Michigan drainage area (distribution, transport and fate in different environmental compartments) and aquatic food web (bioaccumulation, trophic transfer, biomagnifciation, etc.). Comparison between predicted and measured concentrations can help to validate the models. It can also examine the usefulness/appropriateness of these models for less persistent compounds such as NPAHs. Moreover, the model-estimated emission profiles can fill a knowledge gap regarding historical

emission rates of PAHs, and can help improve and complement existing emission inventories.

6.3 Modeling methods

6.3.1 Chemicals and modeling approach

Chemicals considered include the 16 EPA priority PAHs (CFR 1982) and 6 NPAHs that are commonly detected at relative high concentrations in diesel exhaust, fish and sediments (Huang et al. 2014a, 2014b; Liu et al. 2010; Khalek et al. 2011; Chiang et al. 2012), specifically, 1-nitronaphthalene, 2-nitronaphthalene, 2-nitrobiphenyl, 2-nitrofluorene, 1-nitropyrene and 6-nitrochrysene.

The level III fugacity-based model used is a steady-state, non-equilibrium model with environmental compartments for air, water, soil and sediment. Mass balance equations for each compartment are presented as Equations A6.1-A6.4, and detailed descriptions of the model formulation is presented elsewhere (Mackay 2010). This model (Level III version 2.80) is available from the Canadian Centre for Environmental Modelling and Chemistry (CEMC) (2004). We developed a comparable spreadsheet model in Excel (Microsoft, Redmond, CA) in order to facilitate uncertainty analysis using the @Risk software (Palisade Corporation, Ithaca, NY).

The dynamic (nonsteady-state) and non-equilibrium level IV model included the same four compartments described above, and the mass balance equations are presented in Equations A6.6-A6.9. The D values (transport parameters with units of mol/Pa-h (Mackay 2010)) in the level IV model were obtained from the level III model results. Emission and background concentration data are time-dependent functions (described later). The level IV model was used to estimate the historical emission profiles for two representative PAHs, benzo[a]pyrene and phenanthrene, which is discussed later.

The bioaccumulation model uses eight species to model the food web of Lake Michigan that are coupled by a feeding matrix (described later). For each species, chemical uptake comes from water intake and food consumption, and elimination occurs through metabolism, egestion, discharge through gills, and organism growth (growth dilution). The steady-state and dynamic mass balance equations for each species are presented in Equations A6.5 and A6.10.

6.3.2 Model inputs

Physiochemical and food web parameters

The study area encompasses Lake Michigan's surface water area and the drainage basin (Figure A6.1). Parameters for air, water, soil and sediment compartments, including transport velocities between compartments, are presented in Table A6.1. The transport velocities are estimated on data for Lake Huron (Lim and Lastoskie 2011), these lakes are linked and share similar climates.

Physiochemical properties of the chemicals, e.g., vapor pressure, melting point, water solubility and log *Kow* at 25 °C were obtained from EPISuite (EPA 2012) experimental database, if available, otherwise as estimated using EPIWIN. Vapor pressure, water solubility and log *Kow* at the Lake Michigan's mean temperature (11.7 °C) were recalculated using the van't Hoff equation and phase-change enthalpies (Lei et al. 2000; White 1986; Paasivirta et al. 1999; Chickos et al. 1999; Basařová and Svoboda 1995). Degradation half-lives in air, water, soil, sediment and top trophic-level fish (lake trout) were obtained from EPISuite. All physiochemical parameters are listed in Tables A6.2 and A6.3.

The Lake Michigan food web, depicted in Figure 6.1, included eight species (Charles P Madenjian et al. 2002; Charles P. Madenjian et al. 2012; 2012), representing both benthic (diporeia) and pelagic (plankton, mysid, fish) organisms. Dietary preferences were estimated using several Lake Michigan studies (Charles P. Madenjian et al. 1998; Wells and Beeton 1963; Davis et al. 1998; Hondorp et al. 2005). The species and their physiological properties are listed in Table A6.4, and the feeding matrix is presented in Table A6.5.

For many chemicals and aquatic organisms, experimental data regarding metabolic half-lives is limited. Unlike PCBs and PBDEs whose metabolism in aquatic organisms is negligible, metabolism is important for PAHs, and the metabolic rate differs by trophic level (Takeuchi et al. 2009; Hahn et al. 1994). Therefore, compared to the top tropic species (lake trout), we assumed that metabolic half-lives were three times longer for prey fish (alewife, bloater, sculpin, smelt), ten times longer in diporeia and mysid, and 100 times longer in plankton (food web base). These ratios were set arbitrarily, but reflect the presence of the Ah receptor and sufficient cytochrome P450 in fish (but not invertebrates) to metabolize PAHs (Livingstone 1998; Hahn et al. 1994), and the biodilution of PAHs observed in both marine and freshwater organisms

(Huang et al. 2014b; Takeuchi et al. 2009; Baumard et al. 1998). Metabolic half-lives are presented in Table A6.3.

Emissions and background concentrations

Atmospheric deposition accounts for an estimated 80% of PAH loadings into Lake Michigan's water (Helfrich and Armstrong 1986). Effluent discharges contribute much smaller fraction (2-16%) of the total loading, as do petroleum spills (10-15%) (Helfrich and Armstrong 1986). No information is available regarding to PAH loadings via run-off (water and sediment) or transfers via lake or river flows in the basin (water and sediment). We considered only air and water releases, and assumed that air emission comprised 90% of the total emissions since not all PAHs emitted into air would be deposited to the water body. For the level III model, the annual PAH emissions were obtained from the 2008 Great Lakes Regional Air Toxic Emissions Inventory (GLC 1993-2008), which include point, area and mobile sources in the overall Great Lakes region. Emissions in the Lake Michigan basin were assumed to be 24% of this total (Q. Zhang et al. 2003). Annual rates were converted to kg/h for the model, and water discharges were calculated as 1/9 of the air emission rate, as discussed above. Emission data is listed in Table A6.6.

Background concentrations for the level III model are presented in Table A6.6. Air concentrations of 13 target PAHs (excluding NAP, ACY and ACE) in the region have been monitored in the IADN at urban (Chicago) and rural (Sleeping Bear Dunes) sites near Lake Michigan since 1990. The most recent report (Sun et al. 2006) provides average concentrations in particulate and vapor phases from 1996 to 2003 (vapor phase since 1992 at the rural site). A lake-wide background concentration was estimated by summing the two phases and weighting the totals by 0.75 and 0.25 for the urban and rural sites, respectively, based on the prevailing southwestern winds . Concentrations of ACY and ACE were estimated using ratios of their airborne concentrations to B[a]P obtained from measurements in Chicago and off-shore sites in Lake Michigan (Simcik et al. 1999). For NAP, air monitoring data are scarce. The background level was estimated by determining the NAP/B[a]P ratio for U.S. air emissions (Y. Zhang and Tao 2009), which was multiplied by the B[a]P background concentration as calculated above.

No atmospheric measurements of NPAHs are available for the Great Lakes region. Using concentration data from Maryland, California, Europe, Japan and Brazil (Albinet et al. 2007;

Librando and Fazzino 1993; de Castro Vasconcellos et al. 2008; Reisen and Arey 2005; Bamford and Baker 2003; Murahashi et al. 1999), we determined the average ratio of each NPAH relative to B[a]P, which was then multiplied by the estimated B[a]P background concentration.

6.3.3 NPAH emissions and concentrations in Lake Trout

Year 2011 emission rates of NPAHs were estimated using the level III model and our 2011 measurements of NPAH concentrations in surficial sediments collected at 24 sites across southern Lake Michigan. NPAHs arise primarily as products of incomplete combustion and from atmospheric transformation of PAHs (Perrini et al. 2005). Thus, urban runoff and effluent discharge are anticipated to be negligible, and only airborne NPAH emissions were considered. (Background concentrations were estimated as discussed above.) NPAH concentrations in lake trout were calculated using the estimated emission rates, the level III multimedia model, and the food web model with the same parameters described for the PAH model. Predicted concentrations in the lake trout were compared to our recent measurements in Lake Michigan lake trout.

6.3.4 Reconstruction of historical PAH emissions

PAH emission trends were reconstructed using benzo[a]pyrene (B[a]P) and phenanthrene (PHE) measurements determined for 1850 to 1990 from a Lake Michigan sediment core study(Simcik et al. 1996). Accumulation rates (ng/cm^2-yr) were divided by the mass sedimentation rates (g/cm^2-yr) to estimate concentrations (ng/g). Concentrations were averaged across cores from four sites (18, 19, 47s and 68k) along a north-south transect of Lake Michigan. (Site 70m was excluded due to significant mixing.) B[a]P and PHE concentrations in 2011 were obtained as the average across 24 sites in southern Lake Michigan using our recent measurements (Huang et al. 2014a). The sediment data between 1990 and 2011 were interpolated, because available data has a large gap. The fugacity in sediment (f_4, Pa) was calculated as $f_4 = C_{sed} \cdot \rho_{sse}/(Z_{sse} \cdot MW \cdot 10^6)$ where C_{sed} = concentration in sediment (ng/g dry), ρ_{sse} = density of sediment solids (kg/m^3), Z_{sse} = fugacity capacity in sediment solids (mol/m^3-Pa), and MW = molecular weight (g/mol). The fugacity capacity (Z value), analogous to heat capacity, describes the capacity of a phase to absorb a chemical for a certain fugacity rise (Mackay 2010).

The level IV (dynamic) model was used to estimate B[a]P emissions from 1850 to 2011. Again, 90% of total emissions were to air (E_1) and 10% to water (E_2). Background concentrations in air were assumed to follow the same trend as air emissions (i.e., a constant ratio); background concentrations in water were assumed to be zero. For B[a]P and PHE, the background concentration in air (C_{b1}, ng/m^3) was assumed to be 0.75 E_1 and 3 E_2, respectively, based on ratios in 2008 (B[a]P: E_1 = 0.81 kg/h, C_{b1} = 0.61 ng/m^3; PHE: E_1 = 9.9 kg/h, C_{b1} = 30.2 ng/m^3). Air emissions (E_1) was further assumed to take a certain mathematical form which includes several unknown parameters. This E_1 function was used to solve the set of coupled ordinary differential equations (ODEs) (Supplemental Equations S6-S9) so the predicted f_4 includes the unknown parameters; finally the unknown parameters were determined by minimizing the sum of the squared difference between predicted and measured f_4 across all time points. These calculations were performed using Matlab R2013b (MathWorks Inc., Natick, MA): the stiff solver ode23tb solved the ODEs; and the function *fminsearch* (Nelder-Mead simplex direct search) estimated the best fits and parameters for the E_1 function.

6.3.5 Uncertainty analysis

While the modeling involves many possible sources of uncertainty in model predictions, sensitive analyses highlighted that the major uncertainties were associated with degradation half-lives, emission rates, and background concentrations. Degradation half-lives were taken mostly as EPISuite estimates, rather than experimental data, and uncertainties may exceed a factor of 10 (Lim and Lastoskie 2011; Gouin et al. 2004). For emission rates, the available inventory may be incomplete, or possibly overstate emissions, e.g., 2002 B[a]P emission estimates were reduced by 32.2% after revision (Soehl and Wu 2012). In addition, PAH emission rates for Lake Michigan were estimated using data from surrounding states. For background concentrations, PAH data were available only through 2003, and NPAH levels were estimated using measurements in other areas.

Monte Carlo (MC) analysis was used to address parameter uncertainty. For the level III PAH model, we used a uniform distribution for each degradation half-life with lower and upper bounds of 1/10 and 10 times the EPISuite estimate. For emission rates and background concentrations, log-normal distributions were assumed using confidence factors (CF) of 3 and 2,

respectively. (CF = 3, for example, means that 95% of the trial values will lie between one-third and three times the median.)

In modeling NPAH emission rates, the same uncertainties for degradation half-lives were applied. Considering the greater uncertainty of the background estimates, a log-normal distribution with a CF of 5 was applied.

For each modeled compound and each application, the MC analysis used 1000 simulations, and confidence intervals were expressed using the 5^{th} and 95^{th} percentiles of the model outputs. This analysis was carried out using @Risk 6.1 (Palisade Corporation, Ithaca, NY) and Excel (Microsoft, Redmond, WA).

6.4 Results and discussions

6.4.1 Level III model

6.4.1.1 Multimedia model for PAHs

Model results for the level III model are demonstrated in detail for B[a]P. Figure 6.2 depicts transfers and reservoirs for this compound over the modeled Lake Michigan domain using the parameters listed in Supplemental Tables S1-S2. The total atmospheric input, 5.92 kg/h, is dominated by advective flows into the airshed (86% of the total); local emissions make up the balance (14%). Airborne B[a]P is lost by advection (22%) and reaction (64%), and relatively little is deposited to the lake (6%) and the surrounding land (7%). The airborne concentration over the lake is 0.159 ng/m^3, which agrees well with the mean concentration reported in 1994-5 of ~0.2 ng/m^3 reported by Simcik et al. (Simcik et al. 1999), especially considering decreases that have occurred. (Newer data are unavailable.) In water, the major inputs are atmospheric deposition (80%) and direct emissions to water (20%), and losses are dominated by reaction and deposition to sediments. Advective losses are negligible due to the Lake's very long hydrologic retention time (99 years). Of the total 0.45 kg/h entering the lake, reactions in the water column consume the majority (76%); net transfers to sediment represent 24%; and evaporation is negligible. The predicted B[a]P concentration in the water column, 0.049 ng/L, can be compared to the mean of 0.4 ng/L measured in southern Lake Michigan near Chicago in 1994-1995 (Offenberg and Baker 2000). The model's lower value, representing a lake-wide average, appears reasonable given that the 1994-1995 measurements represent

impacted (potentially polluted) samples. In sediment, the predicted B[a]P concentration (2.94 ng/g) agrees remarkably well with our 2011 measurements (mean of 2.7 ng/g; range of 1.0-10.2 ng/g). Losses in the sediment include reaction (61%) and burial (39%).

Modeled results for the 16 PAHs are summarized and compared to measurements in air, water, soil and sediment in Table 6.1; results of the MC analysis, reflecting the range of concentrations in sediments attributable to uncertainty in degradation rates, emission rates, and background air concentrations, are shown in Figure 6.3. For the low molecular weight compounds, the largest reservoirs (containing most of the mass of the PAH) are the water column and the atmosphere. In contrast, the high molecular weight compounds accumulate mostly in soil and sediments. Modeled airborne concentrations closely match observed data (Simcik et al. 1999), reflecting the dominance of advective flows and rapid atmospheric reactions. Modeled concentrations in water and sediments show greater deviations and are generally underestimated compared to available observations. However, examining the MC analyses (Figure 6.3), medians of measured sediment concentrations for most PAHs are within 5^{th} and 95^{th} percentile predictions. Also, the predicted relative abundance between individual compounds agrees well with the observations, except for B[a]P and BghiP. Moreover, while the sediment concentrations for most compounds tend to be under-predicted by the model, the B[a]P concentration seems to be over-predicted. Since the point estimate of B[a]P concentration agrees well with the measurements (Table 6.1), it is possible that the deterministic parameters used for B[a]P are fairly accurate, and the Monte-Carlo analysis actually overestimate the uncertainties associated with B[a]P.

Under-predictions of water and sediment concentrations can be explained by several factors. First, as discussed previously, water sampling sites were very close to Chicago and sediment samples were collected in southern Lake Michigan; both sets of measurements may be significantly impacted by local sources and not reflect open water values. Second, EPISuite generally over-estimates the reactivity of persistent organic pollutants (Gouin et al. 2004), e.g., estimated half-lives in water, soil and sediments for PCBs are one to two orders of magnitude lower than other references (Gouin et al. 2004). If parameters for PAHs are similarly biased, then PAH concentrations will be under-estimated. Third, given the lack of data for effluent discharge and petroleum spills, emissions into water may be under-estimated. Similarly, the zero background concentration in water inflows assumed will contribute to the underestimate.

Finally, the level III model assumes equilibrium between compartments, but since some time is needed to achieve equilibrium, measured concentrations in sediments may reflect earlier and higher emission rates, e.g., as seen by the declining trend of airborne PAH concentrations (Sun et al. 2006). All of these factors might explain the low concentrations in sediments predicted using the 2008 emission rates.

Spatial and temporal variation in concentrations is not predicted by the model, which assumes that each model compartment is homogeneous, well-mixed, and time-invariant. Thus, seasonal effects, e.g., temperature changes that can affect a chemical's physiochemical properties and degradation rates, as well as site-to-site differences in degradation rates, sources, and other factors, are not modeled. Ideally, model predictions would incorporate both variation as well as uncertainty; the MC simulations help show the possible ranges but depend strongly on inputs, as discussed below.

6.4.1.2 Estimation of NPAH emission rates

As noted, NPAH emission rates are unavailable. NPAH emissions to the Lake Michigan airshed, as well as concentrations in air, water and soil, are estimated using "inverse modeling" approach, parameters derived in large part from EPISuite (EPA 2012), and our 2011 NPAH measurements in southern Lake Michigan sediments. MC analysis is used to incorporate uncertainties associated with degradation half-lives and background airborne concentrations. Emissions include both direct releases (e.g., from combustion sources), as well as secondary formation (e.g., from atmospheric reactions). The results, shown in Table 6.2, indicate that 1-nitronaphthalene has the highest emission rate (median of 69 kg/h), considerably exceeding that of other NPAHs, including those found at much higher concentrations in sediments (e.g., 1-nitropyrene, 6-nitrochrysene), a result governed by physiochemical properties, e.g., the lighter compounds partition mainly into air and water, and typically degrade much faster than heavier compounds. Estimated median emission rates of all NPAHs are lower than emission rates of the parent PAHs except for 2-nitrofluorene (46 kg/h) which considerably exceeds flourene (2.36 kg/h), which suggests large primary emissions of 2-nitrofluorene from combustion sources, e.g., diesel engines (Khalek et al. 2011; Chiang et al. 2012; Yang et al. 2010), or possibly production from other pathways. It should be noted that these estimates depend strongly on a number of

parameters and uncertainties are high. NPAH measurements in air, water and soil are needed to confirm these results.

6.4.1.3 Bioaccumulation of PAHs and NPAHs

Predicted water and sediment concentrations for PAHs are used in the food web model (Table 6.1) to evaluate the overall model performance based only on emission rates and physiochemical parameters (i.e., no corrections from real water and sediment measurements). For NPAHs, no predicted sediment concentrations are available since the emission rates are inversely estimated from measured sediment concentrations; thus, the average measured sediment concentrations and the median predicted water concentrations are used in the food web model (Table 6.2). Detailed results for B[a]P, presented in Table 6.3, show that bioaccumulation factors (BAF = concentration in organism divided by the concentration in water) generally decreases at higher trophic levels. For example, the highest concentration is found in diporeia (4738 pg/g ww) which resides in sediments where B[a]P concentrations are relatively high; diporeia also is unable to efficiently metabolize B[a]P (Livingstone 1998; Hahn et al. 1994). Slimpy sculpin had the highest concentrations among prey fish since most of its diet is diporeia. In contrast, the lowest levels are found in lake trout (15 pg/g ww) which efficiently metabolize these compounds. This predicted concentration agrees well with our measurements in Lake Michigan lake trout (5.6-43.4 pg/g, average 18.4 pg/g) (Huang et al. 2014b). Throughout the food web, B[a]P uptake is dominated by food consumption (except for plankton and diporeia which do not consume other organisms), a result of B[a]P's lipophilicity. B[a]P is lost primarily through respiration in invertebrates (plankton, mysid, diporeia), and through metabolism in fish.

The food web model results for all PAHs and NPAHs are summarized in Table A6.7. For PAHs with 5 or more rings (BBF to BghiP), predicted and observed concentrations in lake trout are within a factor of 5 with the exception of BghiP (predicted/observed ratio ≈ 20). This compound shows an especially short metabolic half-life in fish (7 h) estimated by EPISuite; also, predicted BghiP concentrations in water and sediments (used in the food web model) are low compared to observations (Table 6.1). Concentrations in lake trout for PAHs with 4 or fewer rings and for NPAHs are generally overestimated (especially for 1-NNAP, 2-NNAP and 2-NFLU). Considering that the predicted water and sediment PAH concentrations used in the food web model are lower than the real measurements (discussed above), this discrepancy would be

larger if real water and sediment measurements were used. This discrepancy may result from several factors: underestimating the clearance rates in aquatic organisms, e.g., a half-life of 17 h has been reported for anthracene in bluegill sunfish (Spacie et al. 1983), compared to the 61 h estimated by EPISuite; also, we made strong assumptions regarding half-lives at different trophic levels (due to the lack of information for fish and aquatic invertebrates); and applying the same gut absorption efficiencies (from water and food) to all compounds (for a particular species). For heavier compounds with high log *Kow*, predicted water concentrations are relatively low and the absorption efficiencies resulted in "reasonable" uptake rates from water and concentrations in fish. However, lighter compounds (and especially NAP, PHE, FLA, 1-NNAP, 2-NNAP and 2-FLU) have relatively high predicted water concentrations, which increases the predicted uptake from water and concentrations in fish to unlikely levels. While gut absorption efficiency is relatively constant for organic compounds with log *Kow* from 5 to 7 (Gobas et al. 1988), efficiency may change for compounds with log *Kow* less than 4. In addition, gut absorption may not be linear with water concentrations. For these and likely other reasons, uptake from water and thus concentrations in fish appear considerably overestimated. Moreover, there is a lack of measurements of NPAHs in water, and we suspect that NPAH concentrations (especially 1-NNAP, 2-NNAP and 2-FLU) are significantly over-predicted since these compounds are much more reactive than their parent PAHs. Clearly, NPAH measurements (especially in air and water) are needed to evaluate and refine the model. Finally, NPAHs in water may be photo-degraded in the surface microlayer, which has different physical and chemical properties from the water underneath (Daumus 1976), and such process cannot be handled by the current model which treats the water column as one homogeneous compartment. Thus, a more sophisticated model structure may be needed for highly reactive compounds, such as NPAHs.

It should be noted that the Lake Michigan food web has changed considerably over time. The native amphipod *Diporeia*, once the dominant benthic organism in Lake Michigan and served as an important prey of many forage fish, has been rapidly declining since 1990s, following the invasion of zebra and quagga mussels (Nalepa et al. 2009; Nalepa et al. 2005). The average population density of *Diporeia* in Lake Michigan was approximately 4000/m^2 in 1997, which dropped to 57-1409/m^2 in 2009 (Barbiero et al. 2011). While the density of invasive mussels has increased dramatically, diets of a few fish (e.g., lake whitefish) have shifted to include zebra and quagga mussels (Nalepa et al. 2009); diets of alewife, bloater, smelt and

sculpin still consist of mainly *Diporeia*, mysid and plankton, although the percentage of *Diporeia* is decreased (Hondorp et al. 2005). Therefore, *Diporiea* was kept in the food web used here, and mussels were not included. However, the mussels may play an important role in contaminant cycling in Lake Michigan. For example, the zebra mussels cover large areas of sediment surfaces and can bioconcentrate PAHs from the water column (Bruner et al. 1994), which may reduce the amount of PAHs that reach the sediments. In contrast, zebra mussels may increase the PAH concentrations in sediments through biodeposition of contaminated feces and pseudofeces (Bruner et al. 1994). Moreover, Lake Michigan has become more oligotrophic due to reduced phosphorus loadings (Charles P Madenjian et al. 2002), decline of energy-rich *Diporeia* and increase of energy-poor zebra/quagga mussels (Nalepa et al. 2009), so it is possible that there will be less organic carbon in surficial sediments that could increase PAH bioavailability. Thus, the inclusion of zebra/quagga mussels in the food web, as well as the associated environmental processes, should be considered in future modeling efforts.

6.4.2 Level IV model

6.4.2.1 Historical PAH emissions

As stated above, the level III model has a disadvantage that it assumes steady-state and cannot account for emissions in earlier years. Thus, to accurately predict the environmental concentrations of PAHs, a dynamic (level IV) model is needed, which requires continuous emission data from the onset of emissions. Significant anthropogenic PAH emissions began in the 19th century, but emission inventories are available only since 1993 (GLC 1993-2008). Thus, we seek to use PAH records in sediment cores and inverse modeling to reconstruct their historical emission profiles. Also, the approach here can account for all emission sources affecting the lake since sediment tends to be the ultimate sink, which can help complement the existing emission inventories. Inverse modeling is performed for two representative PAHs, B[a]P and PHE. For B[a]P, sediment concentrations predicted by the level III model agreed well with measurements (Table 6.1), thus the level IV model used same degradation half-lives. For PHE, the level III model considerably underestimated sediment concentrations (Table 6.1), probably because degradation rates were underestimated by EPISuite, as discussed above. Thus, we adjusted degradation half-lives for PHE in both water and sediment. (Model parameters, including degradation half-lives, are listed in Table A6.8.)

Estimated emission rates in air of B[a]P and PHE are shown in Figure 6.4, and concentrations in sediment predicted using these emissions and those derived from sediment cores are shown in Figure 6.5. Following the trend seen for sediment concentrations, emissions gradually increased from 1850 until the 1950s, which can be attributed to increased industrialization, coal use (Simcik et al. 1996; Christensen and Zhang 1993), and probably vehicle emissions. Both B[a]P and PHE emission sharply declined in the early 1970s, and current (2011) emission rates are back to 1850-1880 levels. This reduction likely results from the transition in heating fuels (from coal to oil and natural gas), reduced coke production, changes in coking technology, controls on emissions from industry (Christensen and Zhang 1993; Simcik et al. 1996; Schneider et al. 2001), and reduced vehicle emissions (including the impact of alternative fuels and exhaust after-treatment) (Ratcliff et al. 2010). For B[a]P, emission rates peaked in 1958 (74 kg/h) and sediment levels one year later (269 ng/g). For PHE, emissions peaked in 1951 (131.46 kg/h) and sediment levls in 1952 (389.55 ng/g). Both compounds show a one year lag between the emissions and sediment compartment, shorter than that reported (4 years) for hexabrominated biphenyl in Lake Huron (Lim and Lastoskie 2011), probably due to the shorter degradation half-lives of PAHs. Predicted sediment concentrations closely match measurements ($R^2 > 0.95$), although peaks are slightly displaced for PHE, in part because the 1955-1965 measurements showed rapid changes, and these values were probably treated as outliers and assigned a low weighting by the fitting algorithm (Figure 6.5). Ideally, additional measurements would be obtained to help confirm that these values are representative, since the sediment data between 1990 and 2011 were interpolated due to lack of data.

Emission rates estimated using the level IV model and sediment records exceed those reported in the Great Lakes Regional Air Toxic Emissions Inventory, especially for B[a]P where differences are a factor of 10 to 20; PHE estimates exceeded inventory values by 2 to 3 times. Much larger differences (factors of 100 – 1000) were recently reported for PAH emission rates derived using inverse modeling for the Athabasca oil sands region (AOSR), attributed to underestimates of indirect emission sources (Parajulee and Wania 2014). The Great Lakes region may be more complicated than the AOSR due to the numerous and diverse PAHs sources, which can be difficult to quantify and vary considerably over time and place. In 1997, the Great Lakes regional inventory reported that on- and non-road mobile sources contributed to 27% of total PAH emissions; apportionments based on Lake Michigan sediments were much higher, 45%.

The inventory also indicates that B[a]P emissions were unchanged from 1996 to 2008, while PHE emission rates declined by 8 folds. While it is not surprising that inventory estimates based mostly on emission factors and activity data diverge from estimates derived from receptor-based methods using monitoring and modeling, the magnitude of the differences suggest a need for further use of the receptor methods to help reconcile these differences. Given appropriate monitoring data, emission estimated using models and environmental monitoring data may provide a more reliable approach to estimate emissions.

6.4.2.2 PAH bioaccumulation trends

Food web concentrations were calculated using the dynamic food web model and the estimated B[a]P and PHE emission rates (Figure 6.6). For B[a]P, the same metabolic rates (for the 8 species) as in the steady-state model were used. For PHE, half-lives in each species were shortened by 5 times (Table A6.8), since the steady-state model greatly overestimated concentrations in lake trout (Table A6.7). With these parameters, predicted B[a]P and PHE concentrations in lake trout by the dynamic model for year 2011 were 56 and 524 pg/g, respectively, which agreed closely to our measurements (6 - 43 pg/g for B[a]P, 17 – 850 pg/g for PHE).

B[a]P and PHE predictions in aquatic invertebrates and fish followed the same trend as the air emission rates and sediment concentrations, e.g., levels peak in the 1950s. The highest B[a]P concentration in plankton occurred in 1958, which corresponds to the highest concentration in water, since plankton only respires in water and does not consume other organisms. Concentrations in other species, more affected by the sediments, peak one year later, 1959. This lag is short compared to that observed for more bioaccumative chemicals, as discussed earlier. For PHE, the highest concentrations in plankton, mysid and smelt occurred in 1951 (corresponds to peak water concentration) while the others occurred in 1952 (corresponds to peak sediment concentration). This is because the water:sediment concentration ratio of PHE is much higher than B[a]P's, so the influence of the water compartment dominates in more species.

The dynamic model gave concentration patterns through the food web that were similar to those discussed earlier for the steady-state model, e.g., B[a]P concentrations were highest in Diporeia, followed by mysid, plankton, prey fish, and lake trout. PHE trends were similar as

B[a]P; however, sculpin had higher concentrations than plankton because biotransformation (metabolism) of B[a]P is more efficient in sculpin (due to B[a]P's higher Kow), while biotransformation of both compounds are negligible in plankton. PHE concentrations in other prey fish were similar, reflecting the significance of uptake from water relative to diet.

6.5 Conclusions

The present study developed and applied fugacity-based models for 16 PAHs and 5 NPAHs in the Lake Michigan basin in order to characterize the distribution, fate and loadings of these potentially toxic compounds. Model parameters were derived from the literature or estimated using EPISuite, and Monte Carlo methods were used to address uncertainty and variability in key parameters, e.g., degradation half-lives, emission rates and background air concentrations. Model predictions were compared to recent measurements in sediment and fish.

Using the steady-state (level III) fugacity model and 2008 emission inventories, predicted PAH concentrations agree well with air measurements, but are generally underestimated in water and sediment. Air emission rates of NPAHs estimated using inverse level III model and 2011 sediment measurements show that 1-nitronaphthalene has the highest emission rate (median of 69 kg/h), and the emission rates of all 5 NPAHs are lower than those of the parent PAHs except for 2-nitrofluorene. The steady-state food web model produces concentrations in lake trout similar to our 2011 measurements for heavier PAHs (BBF to BghiP), but overestimates the concentrations for lighter PAHs and NPAHs. Historical emission rates over the past century, estimated using an inverse modeling technique, the level IV dynamic model and sediment records, suggest that PAH air emissions gradually increased from 1850 until the 1950s, peaked in 1960s, and sharply declined since the early 1970s. The results also suggest that PAH emission rates in the Great Lakes regional inventory are significantly underestimated.

This work is essential for understanding the overall behaviors of PAHs and NPAHs in the Lake Michigan drainage area aquatic food web. Results demonstrate that environmental concentrations of heavier PAHs can be accurately predicted by the model. The model can also be used inversely to estimate emission rates of PAHs from their environmental concentrations, which can provide information regarding historical emissions when there was no emission inventories available. Moreover, this information can complement and help improve existing emission inventories. On the other hand, the results suggest that certain model parameters are

inappropriate for lighter PAHs and NPAHs. Additional measurements and physiochemical data are needed to better estimate parameters and evaluate model predictions. Finally, more sophisticated model structures might be needed to better describe the environmental fate of reactive chemicals such as the NPAHs.

6.6 Tables and figures

Table 6.1 Comparison between predicted PAH concentrations from the level III model and observed concentrations

Compound	Air (ng/m^3)		Water (ng/L)		Soil (ng/g dry)		Sediment (ng/g dry)	
	Predicted[e]	Observed	Predicted[e]	Observed	Predicted[e]	Observed	Predicted[e]	Observed[f]
Naphthalene	76.4 (40.4%)	n/a	3.8 (56.8%)	n/a	2.20E-02 (2.4%)	n/a	0.30 (0.4%)	6.2 (2.5-12.7) [c]
Acenaphthylene	0.15 (3.3%)	0.3 [a]	0.16 (93.6%)	0.7 [b]	4.08E-05 (0.2%)	n/a	0.06 (3.0%)	1.4 (0.7-3.9) [c]
Acenaphthene	0.60 (18%)	0.8 [a]	0.09 (76.4%)	1.2 [b]	3.39E-04 (2.0%)	n/a	0.05 (3.6%)	0.01 (0.01-0.03) [c]
Fluorene	6.9 (26.7%)	5.4 [a]	0.64 (68.9%)	2.1 [b]	1.95E-03 (1.5%)	n/a	0.31 (2.9%)	17.3 (5.9-30.3) [d]
Phenanthrene	16.7 (5.7%)	11 [a]	7.8 (73.9%)	3.0 [b]	4.20E-02 (2.9%)	n/a	21.6 (17.5%)	70.4 (29.1-179.1) [c]
Anthracene	0.44 (5.8%)	0.3 [a]	0.20 (74.6%)	0.1 [b]	8.27E-04 (2.2%)	n/a	0.55 (17.3%)	9.1 (3.8-18.7) [c]
Fluoranthene	5.0 (2.6%)	3 [a]	2.9 (42.6%)	1.5 [b]	3.96E-02 (4.1%)	n/a	41.3 (50.6%)	134.1 (47.3-385.2) [c]
Pyrene	1.1 (3.2%)	1.6 [a]	0.7 (55.8%)	0.8 [b]	8.76E-03 (5.0%)	n/a	5.4 (36%)	110.3 (25.3-236.3) [c]
Benz[a]anthracene	0.19 (0.9%)	0.2 [a]	0.12 (16.7%)	0.2 [b]	2.53E-02 (24.6%)	n/a	5.07 (57.8%)	55.8 (12-167.4) [c]
Chrysene	0.24 (0.9%)	0.5 [a]	0.15 (16.6%)	0.4 [b]	2.89E-02 (22.6%)	n/a	6.5 (59.8%)	75.9 (19.3-175.3) [c]
Benzo[b]fluoranthene	0.57 (4.4%)	0.5 [a]	0.09 (19%)	0.4 [b]	6.20E-03 (9.6%)	n/a	3.68 (67%)	28.5 (13.2-71.9) [c]
Benzo[k]fluoranthene	0.09 (0.6%)	0 [a]	0.04 (8.1%)	0.3 [b]	3.76E-02 (50.1%)	n/a	2.64 (41.2%)	22.5 (10.4-44.2) [c]
Benzo[a]pyrene	0.16 (0.8%)	0.2 [a]	0.05 (7.2%)	0.4 [b]	5.19E-02 (55.2%)	n/a	2.94 (36.8%)	2.7 (1.0-10.2) [c]
Indeno[1,2,3-cd]pyrene	0.16 (0.6%)	0.2 [a]	0.04 (4.4%)	0.4 [b]	8.25E-02 (63.7%)	n/a	3.44 (31.2%)	24.9 (7.8-95.4) [c]
Dibenzo[a,h]anthracene	0.03 (0.6%)	0 [a]	0.01 (7.6%)	0.3 [b]	9.91E-03 (40.9%)	n/a	1.05 (50.9%)	18.2 (5.9-74.1) [c]
Benzo[g,h,i]perylene	0.11 (0.8%)	0.2 [a]	0.02 (2.9%)	0.3 [b]	5.39E-02 (76.5%)	n/a	1.19 (19.9%)	6.7 (2.3-27.5) [c]

[a] Simcik MF, 1999 (Simcik et al. 1999)

[b] Offenberg JH, 2000 (Offenberg and Baker 2000)

[c] Our data, 2011 (Huang et al. 2014a)

[d] Simcik MF, 1996 (Simcik et al. 1996)

[e] The value in parenthesis is the percent of total mass in that compartment.

[f] Data presented are mean (range).

Table 6.2 Estimation of NPAH emission rates and environmental concentrations by the level III model

Compound	Measurements[a]	Model predictions[b]			
	Sediment (ng/g dry)	Air (ng/m3)	Water (ng/L)	Soil (ng/g dry)	Emission to air (kg/h)
1-Nitronaphthalene	0.61	8.6 (6.3-41.2)	11.7 (11.6-14.2)	0.12 (0.02-0.55)	69 (11-444)
2-Nitronaphthalene	0.52	4.7 (3.2-25.7)	8.5 (8.3-10.4)	0.10 (0.01-0.51)	31 (0-304)
2-Nitrofluorene	0.83	4.5 (2.9-25.9)	8.7 (8.6-11.2)	0.15 (0.02-0.89)	46 (25-298)
1-Nitropyrene	2.67	0.09 (0.06-0.45)	0.17 (0.17-0.23)	0.16 (0.02-0.81)	0.3 (0-4.3)
6-Nitrochrysene	3.22	0.04 (0.03-0.16)	0.11 (0.11-0.15)	0.29 (0.03-1.24)	0.2 (0-2.0)

[a] Mean concentrations across 24 sampling sites in southern Lake Michigan.

[b] Data presented are median (5^{th} percentile – 95^{th} percentile).

Table 6.3 Bioaccumulation of B[a]P in Lake Michigan food web

	Plankton	Mysid	Diporeia	Slimy Sculpin	Rainbow Smelt	Bloater	Alewife	Lake trout
Fugacity (Pa)	5.27E-12	5.66E-12	2.76E-11	7.72E-13	4.58E-13	4.61E-13	4.70E-13	1.72E-14
Concentration (pg/g wet)	453	1295	4738	354	105	264	188	14.8
Equilibrium BCF	2.49E+04	6.64E+04	4.98E+04	1.33E+05	6.64E+04	1.66E+05	1.16E+05	2.49E+05
BAF (g/L)	9.2	26	97	7.2	2.1	5.4	3.8	0.30
Transfer rates ($\times 10^{-16}$mol/h)								
Uptake by respiration in water	0.45	11	0	118	227	512	344	5207
Uptake by respiration in sediment	0	0	6.0	0	0	0	0	0
Uptake from food	0	12	0	734	533	6622	2309	50673
Loss by respiration	0.44	11	5.3	17	19	44	30	17
Loss by egestion	0	1.8	0	13	18	65	35	175
Loss by metabolism	0.001	5.5	0.40	807	709	6917	2548	55543
Loss (dilution) by growth	0.009	4.3	0.31	16	14	108	40	145

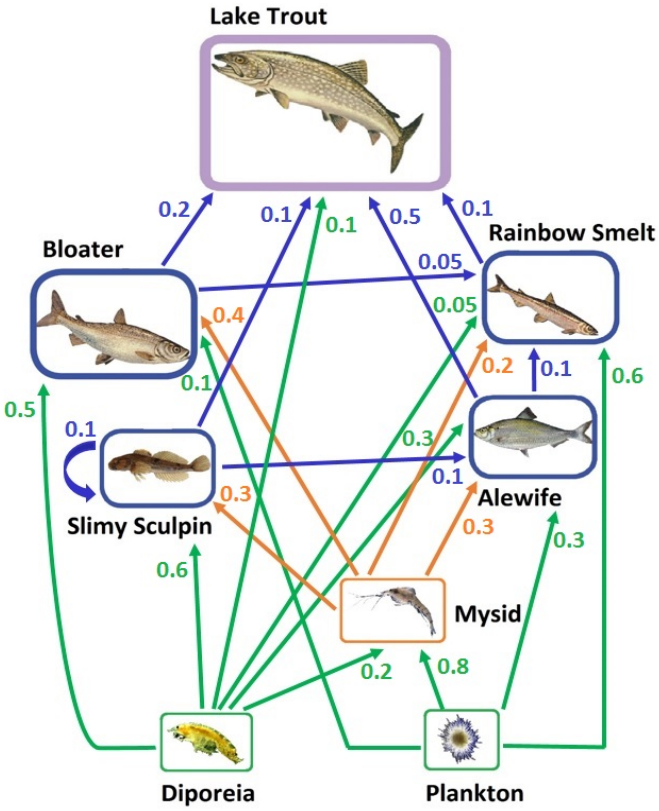

Figure 6.1 Schematic diagram of the Lake Michigan food web.
Colors indicate trophic levels. Dietary preferences are expressed as fractions of the total diet. For example, the diet of lake trout consists of 50% alewife, 20% bloater, 10% rainbow smelt, 10% slimy sculpin and 10% *Diporeia*.

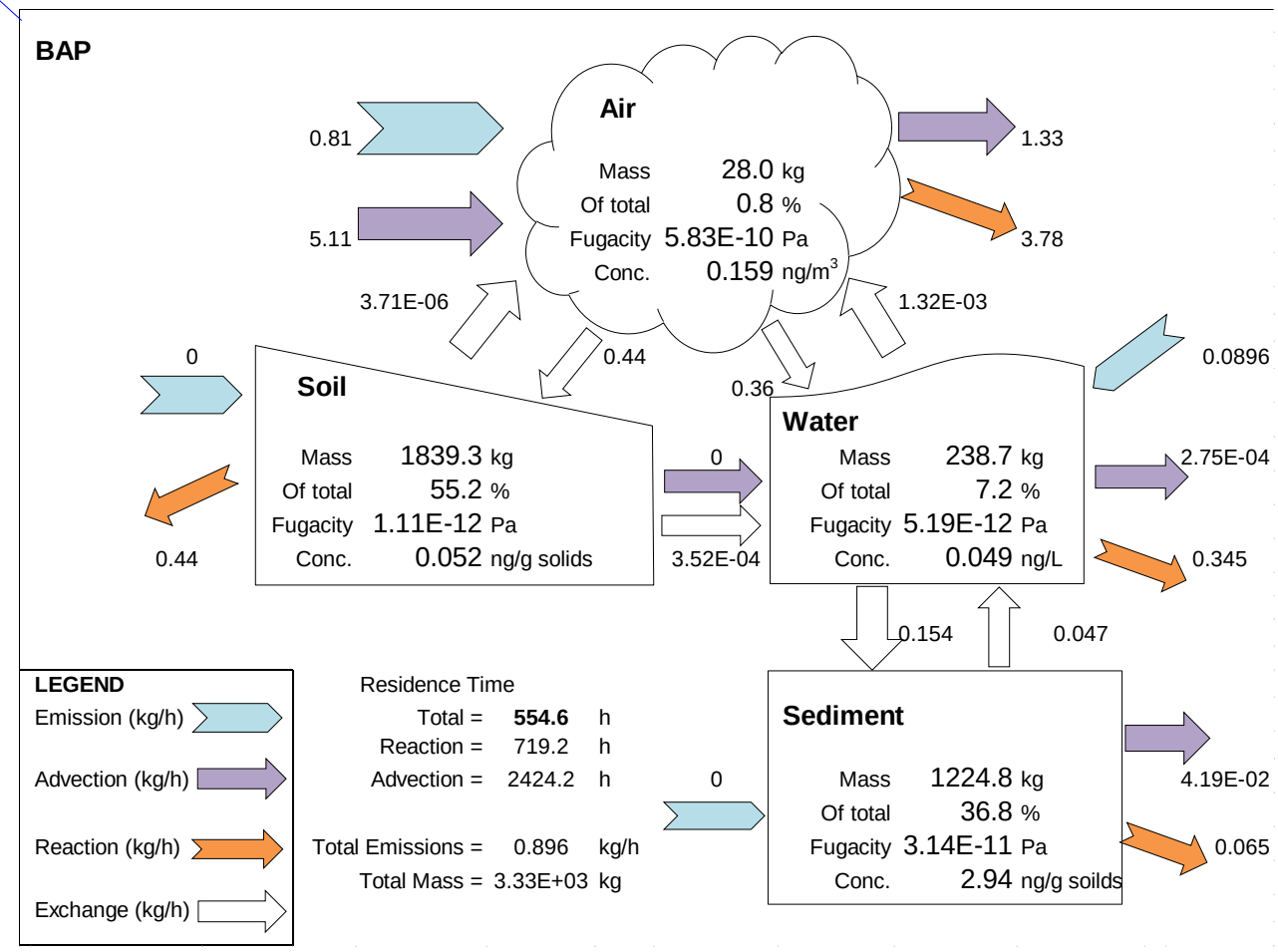

Figure 6.2 Level III mass balance diagram showing the fluxes (kg/h) of benzo[a]pyrene in Lake Michigan.
Adapted from (CEMC 2012).

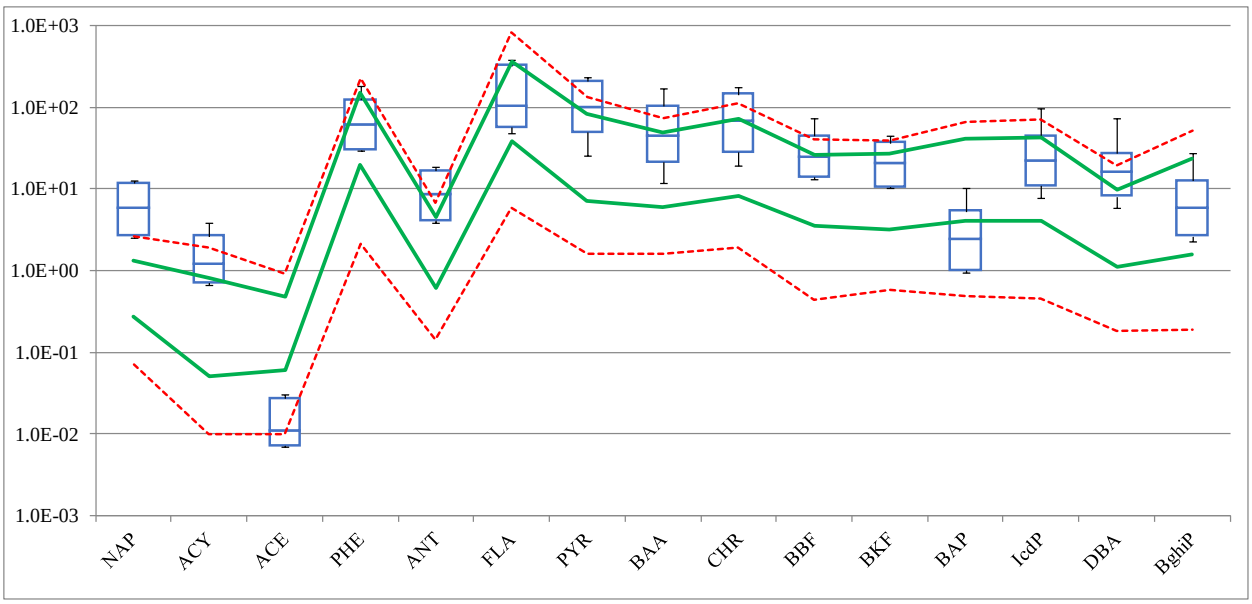

Figure 6.3 Comparison between measured and predicted PAH concentrations in Lake Michigan sediments.
Solid lines show 95th and 5th percentiles of Monte-Carlo results, while dashed lines show the maximum and minimum. The boxplots show maximum, 95th percentile, median, 5th percentile and minimum of the measured concentrations.

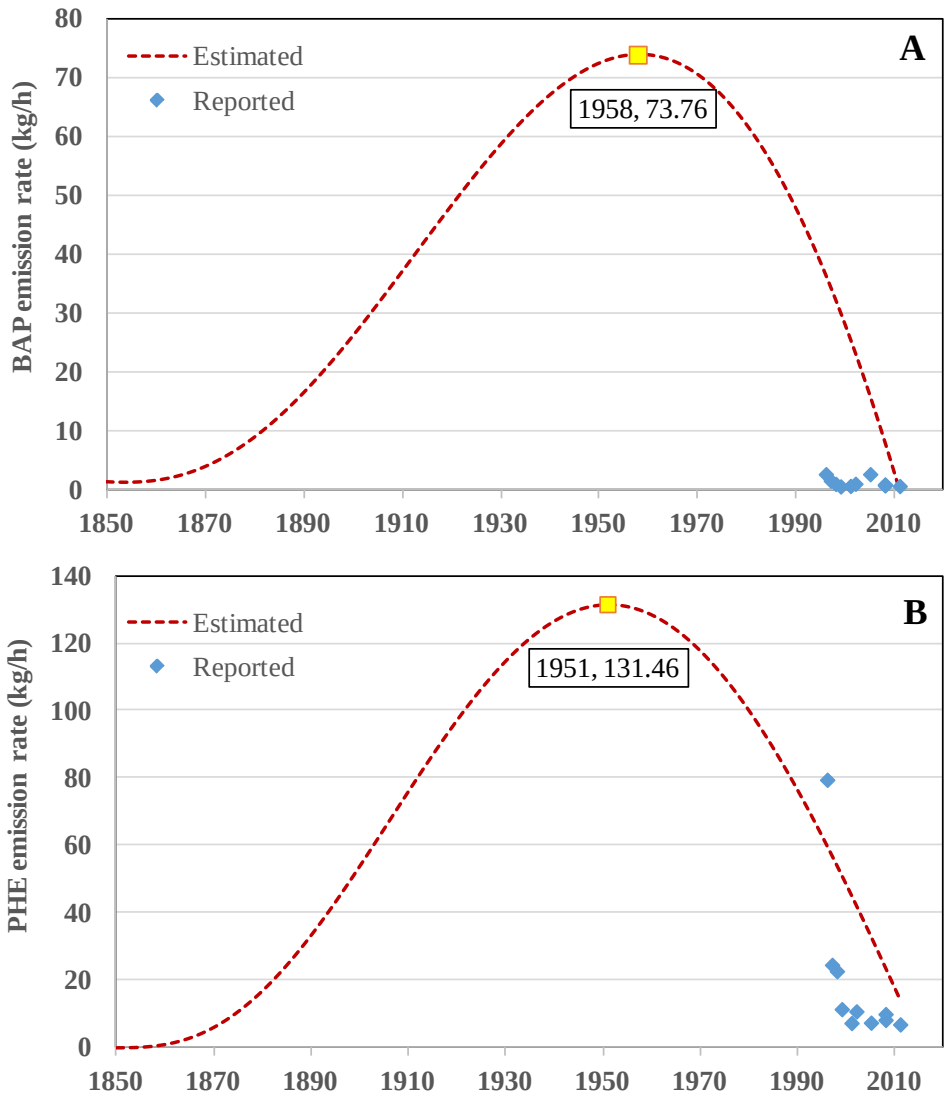

Figure 6.4 Estimated and reported air emission rates of (A) B[a]P and (B) PHE. Box indicates year and value of peak prediction.

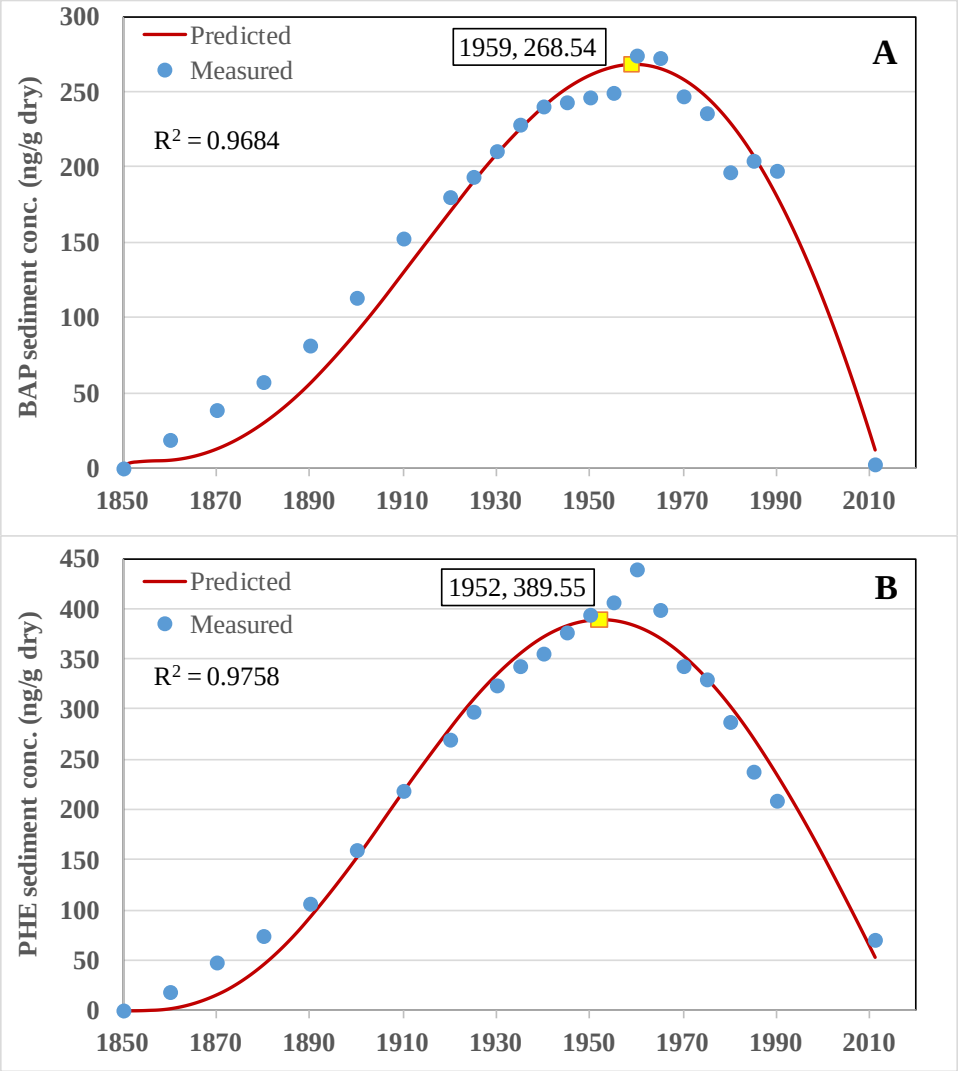

Figure 6.5 Predicted and measured sediment concentrations of (A) B[a]P and (B) PHE. Predicted represents the best fitted values based on the assumed emission scenario. Box indicates year and value of peak prediction.

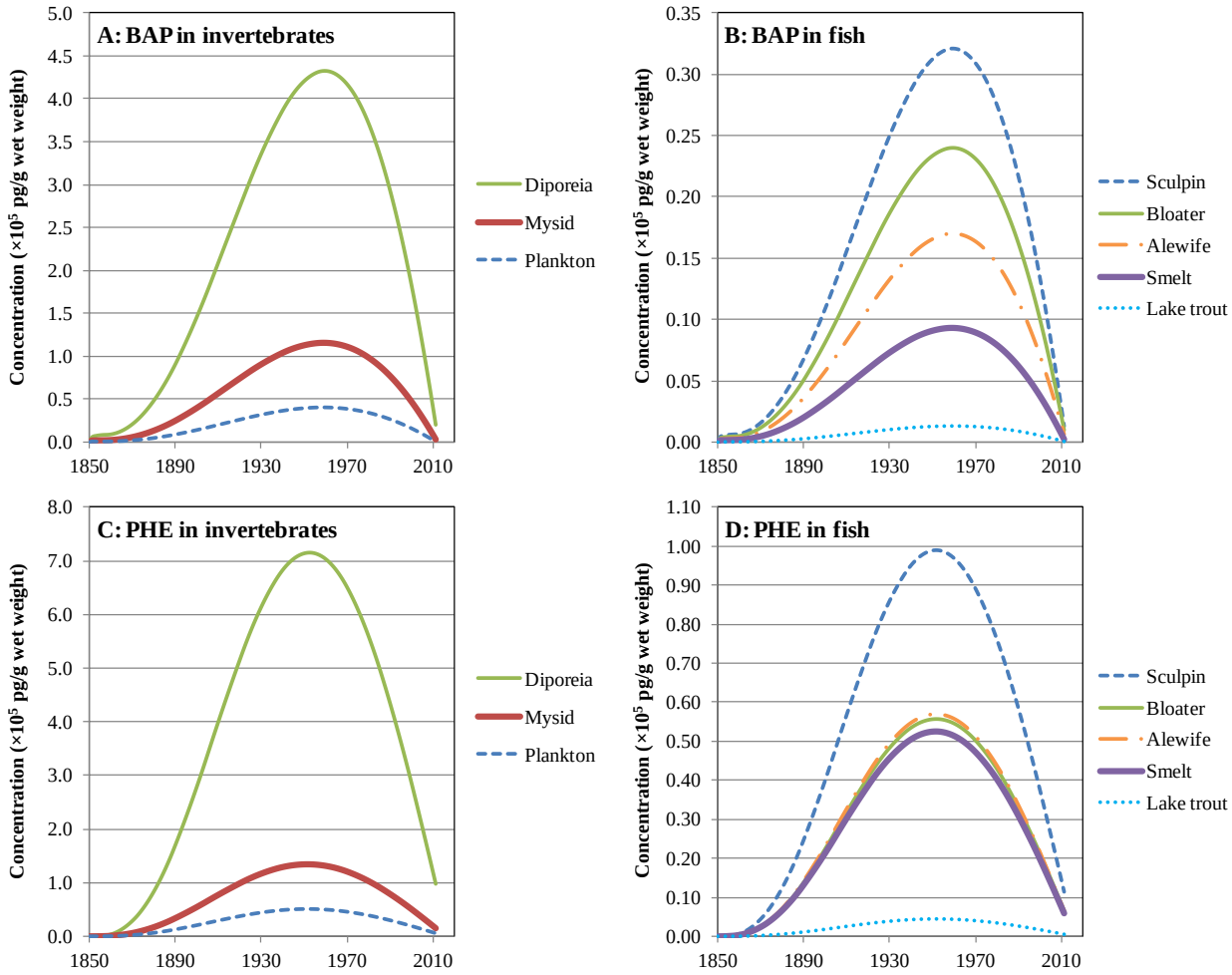

Figure 6.6 Predicted time-dependent concentrations of (A)(B) B[a]P and (C)(D) PHE in Lake Michigan food web.

6.7 Appendix

6.7.1 Model equations

6.7.1.1 The steady-state (level III) model

The mass balance equations for each compartment are as follows. They are the same for PAHs and NPAHs.

Air (subscript 1)

$$0 = (E_1 + G_{A1}C_{B1} + D_{21}f_2 + D_{31}f_3) - f_1 (D_{12} + D_{13} + D_{R1} + D_{A1}) \quad \text{(A6.1)}$$

Water (subscript 2)

$$0 = (E_2 + G_{A2}C_{B2} + D_{12}f_1 + D_{42}f_4) - f_2 (D_{21} + D_{24} + D_{R2} + D_{A2}) \quad \text{(A6.2)}$$

Soil (subscript 3)

$$0 = (E_3 + D_{13}f_1) - f_3 (D_{31} + D_{32} + D_{R3}) \quad \text{(A6.3)}$$

Sediment (subscript 4)

$$0 = (E_4 + D_{24}f_2) - f_4 (D_{42} + D_{R4} + D_{A4}) \quad \text{(A6.4)}$$

Biota (the i^{th} organism)

$$0 = \mathrm{D_W}(\mathrm{f_2} \cdot \mathrm{XW} + \mathrm{f_4} \cdot \mathrm{XS}) + \sum_{\mathrm{i=1}}^{8} \mathrm{Feed_{ij}} \cdot \mathrm{D_{ij}} - \mathrm{f_F}(\mathrm{D_W} + \mathrm{D_M} + \mathrm{D_E} + \mathrm{D_G}) \quad \text{(A6.5)}$$

f: fugacity (Pa)

E: emission rate (mol/h)

G_A: advection flow rate (m^3/h)

C_B: background concentration (mol/m^3)

D: D value (mol/Pa-h)

D_{12} represents the transport D value from compartment 1 to compartment 2, others in the same way.

Subscripts *R, F, W, M, E* and *G* represent reaction, the organism *i*, gill respiration, metabolism, egestion and growth, respectively.

XW: fraction of respiration from water

XS: fraction of respiration from sediment

Feedij: fraction of species i in the diet of species j

Dij: effective D value of food consumption when species *i* is consumed by species *j*

6.7.1.2 The dynamic (level IV) model

Air (subscript 1)

$$V_1Z_1(df_1/dt) = (E_1 + G_{A1}C_{B1} + D_{21}f_2 + D_{31}f_3) - f_1 (D_{12} + D_{13} + D_{R1} + D_{A1}) \quad \text{(A6.6)}$$

Water (subscript 2)

$$V_2Z_2(df_2/dt) = (E_2 + G_{A2}C_{B2} + D_{12}f_1 + D_{42}f_4) - f_2 (D_{21} + D_{24} + D_{R2} + D_{A2}) \quad \text{(A6.7)}$$

Soil (subscript 3)

$$V_3Z_3(df_3/dt) = (E_3 + D_{13}f_1) - f_3 (D_{31} + D_{32} + D_{R3}) \quad \text{(A6.8)}$$

Sediment (subscript 4)

$$V_4Z_4(df_4/dt) = (E_4 + D_{24}f_2) - f_4 (D_{42} + D_{R4} + D_{A4}) \quad \text{(A6.9)}$$

Biota (the i^{th} organism)

$$V_F Z_F\left(\frac{df_F}{dt}\right) = D_W(f_2 \cdot XW + f_4 \cdot XS) + \sum_{i=1}^{8} Feed_{ij} \cdot D_{ij} - f_F(D_W + D_M + D_E + D_G) \quad \text{(A6.10)}$$

V: compartment volume (m^3)

Z: fugacity capacity (mol/m^3-Pa)

6.7.2 Supplemental tables and figures

Table A6.1 Environmental properties and transport velocities.

Compartment	Parameter	Lake Michigan	Reference
	Mean temperature (°C)	11.6	*a*
Air	Area (m^2)	1.76E+11	*b*
	Height (m)	1000	*c*
	Aerosol volume fraction	5.22E-12	*c*
	Aerosol density (kg/m^3)	1800	*d*
	Wind speed at 5m (m/s)	5.35	*a*
	Residence time (h)	20.96	*d*
Water	Area (m^2)	5.78E+10	*b*
	Depth (m)	85	*b*
	Suspended particles fraction	5.00E-06	*d*
	Suspended density (kg/m^3)	2400	*d*
	Suspended OC fraction	0.2	*d*
	Biota fraction	1.00E-06	*d*
	Biota density (kg/m^3)	1000	*d*
	Biota lipid fraction	0.05	*d*
	Residence time (h)	8.67E+05	*b*
Soil	Depth (m)	0.25	*c*
	Air fraction	0.2	*d*
	Water fraction	0.3	*d*
	Solid density (kg/m^3)	2400	*d*
	Soil OC fraction	0.02	*d*
Sediment	Depth (m)	0.01	*c*
	Water fraction	0.7	*d*
	Solid fraction	0.3	*d*
	Solid density (kg/m^3)	2400	*d*
	Sediment OC fraction	0.04	*d*
	Residence time (h)	2.92E+04	*e*
Transfer rate (m/h)	Rain rate	9.30E-05	*d*
	Dry deposition	18.04	*d*
	Air side air-water MTC	41.85	*c*
	Water side air-water MTC	0.0801	*c*
	Soil-air phase diffusion MTC	0.04	*c*
	Soil-water phase diffusion MTC	1.00E-05	*c*
	Soil-air boundary layer MTC	1	*c*
	Sediment-water diffusion MTC	1.00E-04	*c*
	Sediment deposition	4.57E-07	*d*
	Sediment resuspension	1.14E-07	*d*
	Soil-water runoff rate	3.72E-05	*d*
	Soil-soid runoff rate	2.28E-08	*d*

a Reference ; *b* Reference (EPA); *c* Reference (Lim and Lastoskie 2011);*d* Reference (Q. Zhang et al. 2003); *e* Reference (Mackay 2010).

Table A6.2 Physiochemical properties and degradation half-lives of modeled compounds

Group	Chemical	Abbrev.	CAS#	MW	Melting point	Data temperature: 11.7 °C				Degradation half-lives				
						Water solubility	Vapor pressure	LogKow	Henry's law constant	Air	Water	Soil	Sediment	Fish (fat)
				(g/mol)	(°C)	(g/m^3)	(Pa)		($Pa \cdot m^3/mol$)	(h)	(h)	(h)	(h)	(h)
PAHs	Naphthalene	NAP	91-20-3	128.2	80	2.65E+01	7.94E+00	3.36	3.84E+01	11.9	900	1800	8100	108.7
	Acenaphthylene	ACY	208-96-8	152.2	93	1.47E+01	5.90E-01	4.01	6.10E+00	1	360	720	3240	89.5
	Acenaphthene	ACE	83-32-9	154.2	93	3.30E+00	1.89E-01	3.99	8.92E+00	4.4	900	1800	8100	6.0
	Fluorene	FLU	86-73-7	166.2	115	1.44E+00	4.97E-02	4.11	5.74E+00	19.7	360	720	3240	33.0
	Phenanthrene	PHE	85-01-8	178.2	99	1.00E+00	1.05E-02	4.53	1.86E+00	20	1440	2880	13000	61.3
	Anthracene	ANT	120-12-7	178.2	215	3.43E-02	5.68E-04	4.52	2.95E+00	6	1440	2880	13000	60.8
	Fluoranthene	FLA	206-44-0	202.3	108	2.23E-01	7.14E-04	5.23	6.48E-01	23.3	1440	2880	13000	61.6
	Pyrene	PYR	129-00-0	202.3	151	1.17E-01	3.50E-04	4.95	6.04E-01	5	1440	2880	13000	13.4
	Benz[a]anthracene	BAA	56-55-3	228.3	84	7.89E-03	1.64E-05	5.86	4.74E-01	5.1	1440	2880	13000	72.8
	Chrysene	CHR	218-01-9	228.3	258	1.61E-03	4.71E-07	5.89	6.65E-02	5	1440	2880	13000	75.4
	Benzo[b]fluoranthene	BBF	205-99-2	252.3	168	1.22E-03	3.77E-05	5.88	7.77E+00	14	1440	2880	13000	64.1
	Benzo[k]fluoranthene	BKF	207-08-9	252.3	217	6.38E-04	7.28E-08	6.21	2.88E-02	4.8	1440	2880	13000	80.9
	Benzo[a]pyrene	BAP	50-32-8	252.1	175	1.41E-03	4.09E-07	6.22	7.31E-02	5	1440	2880	13000	21.7
	Indeno[1,2,3-cd]pyrene	IcdP	193-39-5	276.3	164	1.47E-04	9.13E-09	6.80	1.71E-02	4.0	1440	2880	13000	28.1
	Dibenzo[a,h]anthracene	DBA	53-70-3	278.4	270	7.98E-04	6.87E-08	6.64	2.39E-02	5.1	1440	2880	13000	84.8
	Benzo[g,h,i]perylene	BghiP	191-24-2	276.3	278	2.26E-04	7.16E-09	6.73	8.77E-03	3.0	1440	2880	13000	7.1
NPAHs	1-Nitronaphthalene	1-NNAP	86-57-7	173.2	61	7.90E+00	3.70E-02	3.19	8.11E-01	47.5	900	1800	8100	31.5
	2-Nitronaphthalene	2-NNAP	581-89-5	173.2	79	8.07E+00	2.18E-02	3.24	4.67E-01	45.8	900	1800	8100	32.7
	2-Nitrofluorene	2-NFLU	607-57-8	211.2	157	1.74E-01	2.95E-04	3.37	3.57E-01	61.7	900	1800	8100	7.3
	1-Nitropyrene	1-NPYR	5522-43-0	247.3	155	9.80E-03	4.76E-06	5.06	1.20E-01	41.1	4320	8640	38900	6.0
	6-Nitrochrysene	6-NCHR	7496-02-8	273.3	187	1.25E-02	4.13E-07	5.34	9.06E-03	41.1	4320	8640	38900	21.3

All values are from EPISuite (EPA 2012)

Table A6.3 Metabolic half-lives in aquatic organisms used in the food web model

Group	Chemical	Metabolic half-lives (h)							
		Plankton	Mysid	Diporeia	Sculpin	Rainbow Smelt	Bloater	Alewife	Lake trout
PAHs	Naphthalene	32610	3261	3261	326	326	326	326	109
	Acenaphthylene	26850	2685	2685	269	269	269	269	90
	Acenaphthene	1800	180	180	18	18	18	18	6
	Fluorene	9900	990	990	99	99	99	99	33
	Phenanthrene	18390	1839	1839	184	184	184	184	61
	Anthracene	18252	1825	1825	183	183	183	183	61
	Fluoranthene	18480	1848	1848	185	185	185	185	62
	Pyrene	4020	402	402	40	40	40	40	13
	Benz[a]anthracene	21840	2184	2184	218	218	218	218	73
	Chrysene	22620	2262	2262	226	226	226	226	75
	Benzo[b]fluoranthene	19230	1923	1923	192	192	192	192	64
	Benzo[k]fluoranthene	24270	2427	2427	243	243	243	243	81
	Benzo[a]pyrene	6504	650	650	65	65	65	65	22
	Indeno[1,2,3-cd]pyrene	8430	843	843	84	84	84	84	28
	Dibenzo[a,h]anthracene	25440	2544	2544	254	254	254	254	85
	Benzo[g,h,i]perylene	2130	213	213	21	21	21	21	7
NPAHs	1-Nitronaphthalene	9461	946	946	95	95	95	95	32
	2-Nitronaphthalene	9799	980	980	98	98	98	98	33
	2-Nitrofluorene	2203	220	220	22	22	22	22	7
	1-Nitropyrene	1796	180	180	18	18	18	18	6
	6-Nitrochrysene	6398	640	640	64	64	64	64	21

Table A6.4 Aquatic organisms and their physiological properties in the food web of Lake Michigan

No.	Species	Volume (cm3)	LF	QD	GR (g/g-day)	FR (% per day)	XW	XS	GAW	GAO
1	Plankton	0.0005	0.015	3	0.025	0	1	0	5.3E-08	4
2	Mysid	0.1	0.04	3	0.02	20	1	0	5.3E-08	3.5
3	Diporeia	0.002	0.03	3	0.02	0	0	1	5.3E-08	4
4	Sculpin	5.4	0.08	3	0.005	4	1	0	5.3E-08	1.5
5	Rainbow Smelt	16	0.04	3	0.005	4	1	0	5.3E-08	1.5
6	Bloater	62[a]	0.1[b]	3	0.004	3.5	1	0	5.3E-08	1.5
7	Alewife	32	0.07	3	0.004	3.5	1	0	5.3E-08	1.5
8	Lake trout	2962[c]	0.15	3	0.002	2	1	0	5.3E-08	1.2

LF: lipid volume fraction.

QD: digestion factor.

GR: growth rate (fraction of volume/body mass per day).

FR: feeding rate (percent of body mass per day). XW: fraction of respiration from water.

XS: fraction of respiration from sediment.

GAO: gut absorption efficiency parameter (organic).

GAW: gut absorption efficiency parameter (water).

[a] Reference (Davis et al. 1998); [b] Reference (Charles P. Madenjian et al. 2000); [c] Our measurements in Lake Michigan lake trout (Huang et al. 2014b).

All other values were obtained from the FoodWeb model software (2006).

Table A6.5 Feeding matrix of the Lake Michigan food web

		Predator							
		Plankton	Mysid	Diporeia	Sculpin	Rainbow Smelt	Bloater	Alewife	Lake trout
Prey	Plankton	0	0.8	0	0	0.6	0.1	0.3	0
	Mysid	0	0	0	0.3	0.2	0.4	0.3	0
	Diporeia	0	0.2	0	0.6	0.05	0.5	0.3	0.1
	Sculpin	0	0	0	0.1	0	0	0.1	0.1
	Rainbow Smelt	0	0	0	0	0	0	0	0.1
	Bloater	0	0	0	0	0.05	0	0	0.2
	Alewife	0	0	0	0	0.1	0	0	0.5
	Lake trout	0	0	0	0	0	0	0	0

Table A6.6 Emission rates and background concentrations used in the level III model

Group	Chemical	Emission rate				Background concentration	
		To air (kg/h)	To water (kg/h)	To soil (kg/h)	To sediment (kg/h)	In air (ng/m^3)	In water (ng/L)
PAHs	Naphthalene	98.5	10.9	0	0	158.5	0
	Acenaphthylene	15.7	1.74			1.22	
	Acenaphthene	1.46	0.16			2.44	
	Fluorene	3.27	0.36			12.2	
	Phenanthrene	9.90	1.10			30.2	
	Anthracene	2.04	0.23			1.23	
	Fluoranthene	3.27	0.36			8.90	
	Pyrene	4.19	0.47			4.15	
	Benz[a]anthracene	1.91	0.21			0.56	
	Chrysene	1.36	0.15			0.85	
	Benzo[b]fluoranthene	0.55	0.06			1.16	
	Benzo[k]fluoranthene	0.41	0.05			0.38	
	Benzo[a]pyrene	0.81	0.09			0.61	
	Indeno[1,2,3-cd]pyrene	0.72	0.08			0.80	
	Dibenzo[a,h]anthracene	0.10	0.01			0.13	
	Benzo[g,h,i]perylene	0.98	0.11			0.62	
NPAHs	1-Nitronaphthalene	TBD	0	0	0	2.51	0
	2-Nitronaphthalene					2.51	
	2-Nitrofluorene					0.38	
	1-Nitropyrene					0.13	
	6-Nitrochrysene					0.063	

TBD: to be estimated by the model.

Table A6.7 Concentrations in aquatic organisms predicted by the steady-state food web model

		Model predictions (pg/g wet)								Measurement (pg/g wet)
Group	Compound	Plankton	Mysid	Diporeia	Slimy Sculpin	Rainbow Smelt	Bloater	Alewife	Lake trout	Lake trout
PAHs	Naphthalene	132	351	549	692	347	832	598	596	74.8 (3.0-172.6)
	Acenaphthylene	24	63	110	119	60	129	99	44	125.6 (21.2-483.1)
	Acenaphthene	13	35	91	35	19	20	21	2	64.4 (6.5-478.2)
	Phenanthrene	3813	10268	39386	16765	8280	14494	12274	2351	163.3 (16.8-849.9)
	Anthracene	98	263	1004	430	212	372	315	61	22.3 (4.5-178.4)
	Fluoranthene	6469	18203	74935	21073	8955	15398	12780	1711	28.7 (8.7-284.7)
	Pyrene	860	2285	9691	1210	607	683	662	51	25.1 (4.8-141.1)
	Benz[a]anthracene	779	2482	8955	2217	728	1687	1254	167	7.4 (4.9-23.8)
	Chrysene	1001	3222	11473	2901	943	2217	1642	222	8.8 (6.9-43.5)
	Benzo[b]fluoranthene	564	1795	6482	1440	470	1086	806	100	22.5 (12.1-70.1)
	Benzo[k]fluoranthene	402	1422	4484	1127	339	882	635	85	17.5 (12.6-97.7)
	Benzo[a]pyrene	453	1295	4738	354	105	264	188	15	18.4 (5.6-43.4)
	Indeno[1,2,3-cd]pyrene	495	1400	4356	343	99	269	188	14	45.6 (34.4-146.9)
	Dibenzo[a,h]anthracene	152	588	1594	379	110	303	214	27	53.4 (47.2-92.8)
	Benzo[g,h,i]perylene	176	227	1124	23	7	18	13	1	23.5 (1.9-149.4)
NPAHs	1-Nitronaphthalene	271	724	1119	1382	698	1559	1167	625	0.7 (0.01-4.3)
	2-Nitronaphthalene	221	590	949	1125	568	1265	948	497	0.5 (0.01-11.3)
	2-Nitrofluorene	305	812	1521	1275	670	1061	953	153	0.3 (0.01-3.3)
	1-Nitropyrene	263	680	4772	188	81	108	95	6.5	1.2 (0-20.7)
	6-Nitrochrysene	296	872	5792	531	184	359	279	24.7	0.7 (0.01-6.1)

Table A6.8 Degradation half-lives of phenanthrene (PHE) used in level IV models

	Half-life (h)	Reference
Air	19.7	*a*
Water	1440	*a*
Soil	17000	*b*
Sediment	55000	*b*
Plankton	3000	*c*
Mysid	300	*c*
Diporeia	300	*c*
Slimy Sculpin	30	*c*
Rainbow Smelt	30	*c*
Bloater	30	*c*
Alewife	30	*c*
Lake trout	10	*c*

a Reference (EPA 2012); *b* Reference (Mackay and Hickie 2000).

c Values are reduced by 5 times based on level III model results.

Figure A6.1 The study area, including Lake Michigan (blue area) and its drainage basin (green area).
The wind rose plot at the bottom left corner indicates the prevailing wind directions from 1961 to 1990 at Chicago (Angel 2009). This figure is adapted from the Michigan Sea Grant (MISC 2014).

6.8 References

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Chapter 7 Conclusions

The overall objective of this research was to understand the distribution, sources and risks of PAHs and NPAHs in Lake Michigan, to characterize their emissions from a major source (diesel exhaust), and to model and predict their environmental fate in the Lake Michigan basin. The petroleum biomarkers hopanes and steranes were characterized along with PAHs and NPAHs to provide more information on hydrocarbon contamination. The research had five specific aims: (1) Characterizing the distribution of target compounds in predator fish from Lake Michigan and estimating the carcinogenic risk from the consumption of these fish; (2) Characterizing bottom sediments from Lake Michigan for concentrations of the target compounds and identifying major sources; (3) Analyzing the emissions of target compounds from a major source - diesel engines; (4) Examining the integrity of target compounds during filter processing and storage needed for analysis of diesel exhaust emissions; and (5) Predicting the environmental fate and estimating the emission rates of PAHs and NPAHs in Lake Michigan using multimedia models.

This chapter summarizes the major findings, significance and limitations of this research. Section 7.1 describes the interrelationship between the specific aims. Section 7.2 discusses the major findings of each specific aim in turn. The implications of the findings and the limitations of the study are presented in Sections 7.3 and 7.4, respectively. The chapter is concluded with recommendations for future studies.

7.1 Integration of specific aims

The dissertation shows that the target compounds are widely distributed in the Lake Michigan basin. Specifically, NPAHs may be of health concern and may warrant further investigation since certain highly carcinogenic compounds were detected at moderate concentrations. In fish, PAHs display "biodilution," while in sediment, a spatial trend is seen with high concentrations near-shore and low concentrations in the middle of the lake. PAHs levels in Lake Michigan sediments have decreased from the 1990s. Currently, vehicle exhaust

was identified as the most important PAH source, while bench tests demonstrate the effectiveness of alternative fuels and after-treatment in reducing emissions of all target compounds. Finally, the fugacity modeling suggests that environmental concentrations and emission rates of PAHs and NPAHs can be estimated and inter-related using relatively simple multimedia models.

The five specific aims of this research are diverse in scope, but closely related and mutually supportive. Concentrations measured in lake trout (Chapter 2) served as empirical data for model validation (Chapter 6); the "biodilution" observed supported lower metabolic half-lives for the higher trophic levels of the model; and the similar concentrations across sites supported the use of a one-compartment model for Lake Michigan. Similarly, measured sediment concentrations (Chapter 3) were used for model validation and as input data in inverse-modeling for estimating emission rates of PAHs and NPAHs (Chapter 6). The source apportionment (Chapter 3) identified vehicle exhaust as the most important PAH source, which motivated the analyses of diesel exhaust emissions (Chapter 4), and the developed emission profiles helped to confirm apportionment. The investigation of filter processing and storage (Chapter 5) was needed to confirm sampling protocols from the engine bench tests (Chapter 4). Finally, the multimedia models (Chapter 6) helped to integrate the study's findings, and they presented an overall picture of the environmental behavior of PAHs and NPAHs.

7.2 Major findings of each specific aim

7.2.1 Distribution of target compounds in Lake Michigan fish

The concentrations of PAHs, NPAHs, hopanes and steranes in Lake Michigan lake trout were presented in Chapter 2. A total of 74 lake trout were collected in two seasons (spring and fall) at three offshore sites along a north-south transact of the lake. Whole-fish Σ_9PAH concentrations averaged 546 ± 244 pg/g wet weight (ww). The concentrations and profiles were similar across season, site and gender. The 3-ring compounds, such as phenanthrene, were the most abundant PAH compounds. PAH concentrations in lake trout were low compared to those in small prey fish (10 to 100 ng/g ww) (Levengood and Schaeffer 2011) and aquatic invertebrates (100-1000 ng/g ww) (Eadie et al. 1982b; Eadie et al. 1982a; Metcalfe et al. 1997) reported previously, suggesting "biodilution" of PAHs in aquatic food web, as observed in marine organisms (Baumard et al. 1998; Takeuchi et al. 2009). This is probably due to the rapid

metabolism of PAHs in higher trophic-level organisms (Hahn et al. 1994). However, metabolites of parent PAH compounds may persist in the fish tissue and have toxicological importance.

Σ_9NPAH concentrations averaged 7.2 ± 7.0 pg/g ww in whole fish. Literature data for Great Lakes fish is not available for NPAHs. However, mussels and oysters from Japan showed ΣNPAH concentrations ranging from 400 to 4000 pg/g ww, levels much higher than those in lake trout, so biodilution of NPAHs might also be possible. NPAH concentrations in fall samples were extremely low at the Waukegan site, and NPAH profiles differed among sites; in spring, concentrations and profiles were similar across sites. This could be in part attributed to lake trout's homing behavior in the fall; other factors may include local emission sources and localized water and sediment contamination.

Σ_2Hopane and Σ_5Sterane concentrations averaged 37 ± 23 pg/g ww and 269 ± 111 pg/g ww, respectively. Abundances of individual hopanes and steranes did not vary by season, site or gender, suggesting exposure to a similar and widespread source. However, spatial differences were seen for hopane and sterane concentrations in fall, indicating the impact of sources in the Chicago/Gary area. Hopane and sterane concentrations in fall were ten times higher than levels in spring, indicating substantial differences in uptake and/or clearance rates.

Fifteen female fish collected in fall contained eggs. SVOC concentrations in eggs were similar to those in the corresponding female fish for NPAHs, hopanes and steranes, but were significantly lower for PAHs.

The toxic equivalency (TEQ) approach was used to estimate the human health risks from PAHs and NPAHs associated with the consumption of Lake Michigan lake trout. Upper bound worst-case estimates of lifetime human cancer risks due exceeded 10 per million, but generally PAHs and NPAHs in lake trout seem to pose a minimum threat to human health. However, since many NPAHs lack toxic equivalent factors (TEFs), true risks may be underestimated. It is also notable that even using the existing TEFs, NPAHs contributed a significant portion (40-80%) of the toxicity in some samples. Thus, further assessment of NPAH contamination in Lake Michigan fish, and effects on fish and ecological health, is warranted.

7.2.2 Distribution and sources of target compounds in Lake Michigan sediments

Bottom sediments are the habitats of benthic-dwelling organisms. In Lake Michigan, a large reservoir of persistent organic pollutants, including PAHs, exists in bottom sediments, which is taken up by benthic organisms and enter the aquatic food web. The distribution and sources of PAHs, NPAHs, hopanes and steranes in southern Lake Michigan sediments were presented in Chapter 3.

Sediment sampling and analyses showed that 14 of 16 target PAHs, 5 of 11 target NPAHs, and all target hopanes and steranes were detected above MDLs at all of 24 sites sampled. Fluorene was detected in 37% of the samples, and the remaining compounds were undetected at all sites.

ΣPAH_{15} concentrations ranged from 213 to 1291 ng/g dry weight (dw), similar to concentrations in general areas and background sites in other Great Lakes and around the world, but slightly lower than those measured in highly polluted areas. PAH levels in Lake Michigan sediments have been declining for the past three decades. Currently, PAH levels fall below consensus-based sediment quality guidelines (SQGs) and thus pose very low risks to benthic organisms. $\Sigma NPAH_5$ concentrations ranged from 3 to 19 ng/g dw, similar to levels reported in other areas of the world. No SQGs are available for NPAHs, but several highly toxic compounds were detected, including 6-nitrochrysene. $\Sigma Hopane_5$ and $\Sigma Sterane_6$ concentrations ranged from 98 to 355 ng/g and 6 to 36 ng/g dw, respectively.

The estimated (2011) loading rates of ΣPAH_{15}, $\Sigma NPAH_5$, $\Sigma Sterane_6$ and $\Sigma Hopane_5$ to open water sediments of Lake Michigan are 10.9, 0.16, 0.25 and 3.6 MT/yr, respectively, which agree well with prior estimates obtained using different and independent methods (Helfrich and Armstrong 1986; Simcik et al. 1996). SVOC concentrations were significantly correlated with the sediment's organic carbon (OC) content. OC-adjusted SVOC concentrations were elevated at locations near AOCs and the larger urban/industrial areas. Abundances of individual compounds were similar across sites, indicating that common source types affected sediments across southern Lake Michigan.

Diagnostic molecular ratios indicated that PAHs in Lake Michigan sediment were dominated by pyrogenic (i.e., combustion) sources. Chemical mass balance (CMB) modeling identified diesel engine (representing traffic, 56%), coal-fired power plant (27%) and coal-tar

based pavement sealants (16%) as the major PAH sources in Lake Michigan sediments, while coke oven was identified as a minor source (7%). Diagnostic ratios and abundances of individual NPAH compounds also indicated contributions from diesel engine emissions and coal combustion. Based on hopane biomarkers, both petroleum-derived and biogenic sources are important contributors of hydrocarbons (including aliphatics and aromatics) in southern Lake Michigan sediments. Knowledge of major sources of these contaminants is useful for identifying the targets for pollution reduction efforts.

7.2.3 Emissions in diesel engine exhaust using different fuels and after-treatment systems

As indicated in Chapter 3, the dominant source of PAHs in surficial Lake Michigan sediments was diesel engine exhaust. Chapter 4 investigated the emissions of $PM_{2.5}$, PAHs, NPAHs, hopanes and steranes from a heavy-duty (6.4 L) diesel engine at various loads (idle, 600 and 900 kPa BMEP) with three types of fuel (ULSD, Swedish diesel and B100), and with and without exhaust after-treatment (DOC+DPF). Results demonstrated that Swedish diesel and B100 significantly reduced emissions of $PM_{2.5}$, PAHs, NPAHs, hopanes and steranes, and biodiesel resulted in larger reductions. However, during idling emissions of $PM_{2.5}$ and 5-nitroacenaphthene increased with B100, and emissions of benzo[k]fluoranthene increased with Swedish diesel. These results are important given the recent attention to exposures from idling vehicles and the toxicity of high-molecular-weight PAHs and NPAHs. The after-treatment system (DOC+DPF) caused large (>99%) reductions of $PM_{2.5}$ and Σ_{16}PAHs, Σ_{11}NPAHs, Σ_2Hopanes, Σ_2Steranes and individual compounds. Emissions of $PM_{2.5}$ and SVOCs increased with increasing engine load, with the notable exception of $PM_{2.5}$ emissions with B100 during idling. The toxicity of diesel exhaust, in terms of the estimated human carcinogenic risk, was greatly reduced by Swedish diesel, B100 and the DOC+DPF. These results suggest that the use of alternative fuels and exhaust after-treatment systems are effective ways to reduce $PM_{2.5}$, PAHs and NPAHs emissions and pollution.

A PAH/NPAH profile for diesel exhaust was developed by combining emissions measurements during idle and under load, and by accounting for variability. The PAH profile showed high abundances of three and four ring compounds, as well as naphthalene; the NPAH profile showed high abundances of nitronaphthalenes, 1-nitropyrene and 9-nitroanthracene.

These profiles can be used in receptor models to apportion PAHs and NPAHs. Based on a comparision of profiles in the literature, certain compounds are suggested for use as fitting species in source apportionments since abundances remained similar. In contrast, compounds with large variability among studies may not be suitable for source apportion purposes.

Emissions of biomarkers hopanes and steranes were significantly correlated with PAHs, NPAHs and $PM_{2.5}$, but abundances varied considerably among different studies. This suggests that the use of these compounds as PAH/NPAH/PM tracers should be limited to qualitative and diagnostic purposes.

7.2.4 Integrity of target compounds in diesel exhaust particulate matter

A number of filter and extract processing steps are needed to analyze target compounds in the PM samples collected from diesel engine exhaust, and the integrity of samples and the accuracy of measurements are non-trivial concerns. Chapter 5 investigated the effects of filter conditioning, filter storage and extract storage on the integrity of target compounds in diesel exhaust PM samples. Three cases were evaluated: conditioning filters in clean air at 25 °C and 33 % relative humidity for 24 h, storing filter samples (without extraction) wrapped in aluminum foil at 4 °C for up to 1 month, and storing filter extracts in glass vials capped with Teflon crimp seals at 4 °C for up to 6 months. All filters used were loaded filters using ULSD and no after-treatment.

Conditioning filters for 24 h did not significantly change the concentrations of NPAHs, hopanes and steranes; however, approximately 30% of the more volatile PAHs was lost. The tests showed that filters loaded with PM can be stored for at least 1 month without appreciable losses of target compounds if the filter is wrapped in aluminum foil and held at 4 °C. Filter extracts (PAHs in hexane/acetone, NPAHs in methanol, hopanes/steranes in hexane) can be stored at 4 °C for at least 1 month without significant losses. Storage for 5 or more months may be acceptable for hopane and sterane extracts, but losses of PAHs and NPAHs may be significant.

The laboratory tests show that even relatively brief periods used to condition filters (needed for gravimetric measurements of PM), can underestimate PAH concentrations. To reduce potential biases, conditioning protocols might be altered by lowering temperatures (e.g., from 25 to 10 °C) and/or reducing conditioning times (e.g., from 24 to 12 h). Additionally,

SVOC sampling and analysis protocols might utilize performance checks and criteria aimed at identifying and limiting potential biases occurring during filter and extract processing, e.g., PM samples on glass fiber filters should be sealed appropriately, extracted within 30 days of collection, and analyzed within 1 month.

7.2.5 Multimedia fate modeling

Emissions of PAHs/NPAHs (e.g., from diesel exhaust) were related to concentrations in environmental compartments, such as fish and sediment, using fugacity-based multimedia models for the Lake Michigan basin in Chapter 6. The study area was modeled as four compartments: air, water (including aquatic biota), soil and sediment; the aquatic biota was further modeled as an 8-species food web.

A steady-state model with air emission data was used to predict the concentrations of target PAHs in air, water, soil, sediments and aquatic biota. For NPAHs, emission rates were unavailable and were estimated using measured concentrations in sediments and inverse modeling; then their concentrations in biota were predicted using the estimated emission rates. For example, the model predicted that most benzo[a]pyrene (BAP) emitted into the atmosphere were lost by advection downwind and reaction, and only a small fraction is deposited to the lake and the surrounding land; most BAP was distributed in soil (55%) and sediment (37%). Overall, predicted and measured PAH concentrations in air agreed, but PAH concentrations in water and sediments were generally under-predicted, which may be caused by underestimate of the degradation half-lives. Concentrations in lake trout were accurately predicted for high-molecular-weight PAHs, but greatly overestimated for low-molecular-weight PAHs and NPAHs, possibly due to an overestimate of the metabolic half-life and/or gut/gill absorption efficiencies. Certain model parameters which work well for persistent organic pollutants (such as PCBs, PBDEs and high-molecular-weight PAHs), may not be appropriate for low-molecular-weight PAHs and NPAHs. Additional measurements, especially for NPAHs, are needed to evaluate model parameters. Moreover, for highly reactive chemicals like the NPAHs, a more sophisticated model structure (not just short half-lives) might be needed to more accurately describe environmental fate.

A dynamic model reconstructed historical emission rates of two representative PAHs (BAP and PHE) using their concentration records in Lake Michigan sediment cores. Time-

dependent bioaccumulation profiles were calculated using a dynamic food web model and the estimated emission rates. Predicted trends of BAP and PHE emission rates and concentrations in aquatic biota followed those of the sediment concentrations: a gradual increase beginning about 1850 to a peaked in the 1950s, followed by rapid decreased after the 1960s. The results suggest that PAH emission rates may be underestimated in the (official) Great Lakes emissions inventory. The modeling results provide information on historical PAH and NPAH emissions for a (long) period without an emission inventory. The results also can be used to improve the accuracy of current inventories.

7.3 Significance

This dissertation provided new information regarding the concentrations, distribution and risks of PAHs, NPAHs and petroleum biomarkers in the Great Lakes basin, and included the first reports of NPAHs in sediment and biota of Lake Michigan. The measurement data here can provide reference data for understanding PAH dynamics in other Great Lakes. This research improved our understanding of the behavior of these compounds in Lake Michigan, and identified several major sources that can be targeted for pollution prevention and reduction. These results are essential in moving towards the virtual elimination of these toxic substances in the basin. Moreover, the multimedia and food web modeling is easily transferable to understand the behavior of these compounds in other Great Lakes or large lake ecosystems.

Chapter 2 presented the first report of NPAHs in Lake Michigan fish. Since NPAHs are direct-acting mutagens and are potentially more toxic than PAHs (Tokiwa et al. 1987), their concentrations in fish are important in assessing the health risks to humans and wildlife. These data can also serve as baseline data for future monitoring. This study is also the first to report PAHs in top predator fish of Lake Michigan since 1990s (Zabik et al. 1996). The PAH and NPAH data, as well as the risk estimates, can inform policy and risk communication regarding fish advisories and other health-related actions. The study design facilitates a comprehensive understanding of the behavior of these compounds in top predator fish across Lake Michigan, including the effects of location, season and gender. The results can also be compared to PAH and NPAH levels in lower-trophic-level organisms to assess biomagnification.

Chapter 3 presented the first report of NPAHs, hopanes and steranes in Lake Michigan sediments, and the first data on PAH sediment concentrations since the 1990s (Su et al. 1998;

Zhang et al. 1993). The PAH levels can be compared to literature data to assess the temporal trend, and the NPAH data can serve as baseline data for future monitoring. The hopane and sterane data can be used to identify the sources of hydrocarbon contamination. PAH and NPAH concentrations in surficial sediments provide important information for evaluating potential health effects on benthic-dwelling organisms. This study sampled a relatively large number (N = 24) of sites across southern Lake Michigan, making it possible to assess the spatial distribution pattern of these contaminants, which highlighted the impact of AOCs and industrial/urban areas. Finally, this study proposed a way of estimating the loading of SVOCs into Lake Michigan based on their concentrations in surface sediments, which showed remarkable agreement with prior estimates (Helfrich and Armstrong 1986; Simcik et al. 1996). Compared to methods that compile loadings from various routes (Helfrich and Armstrong 1986; Melymuk et al. 2014), the method used is relatively simple and accounts for all possible routes of inputs. The source identification and apportionments in Chapter 4 identified the key PAH and NPAH sources as vehicle emissions, coal combustion and coal-tar based sealants. Again, these can be targeted for emission reduction.

Chapter 4 investigated PAH, NPAH, hopane and sterane emissions from a major source --- diesel engine exhaust. Vehicle emissions depended strongly on fuel types, engine operating conditions and exhaust after-treatment technologies. Information regarding NPAH emissions with alternative fuels and after-treatment systems is limited and inconsistent (Zou and Atkinson 2003; Karavalakis et al. 2010; Ratcliff et al. 2010; Heeb et al. 2010, 2008), and no information is available regarding hopane and sterane emissions with these new fuels and technologies. This study was unique in examining and comparing emissions with three types of fuels, three different engine operating conditions, and two after-treatment systems. The study also examined idle emissions, which have received considerable attention recently, but which remain poorly characterized. The significant reduction of target SVOC emissions observed suggest that the use of alternative fuels and after-treatment technologies should be promoted.

Filter and extract processing are necessary procedures in the analysis of target compounds in diesel exhaust PM samples. These procedures involve multiple, sequential and complex steps, during which target SVOCs can volatize, decompose, or transform. However, sample integrity for NPAHs, hopanes and steranes during filter and extract processing have not been characterized. This study is unique in examining the effects of filter conditioning, filter

storage and extract storage using real diesel exhaust PM samples. The results have important implications on SVOC sampling and analysis protocols, which should utilize stringent criteria and performance checks to limit possible biases during filter and extract processing and storage.

Chapter 6 used multimedia models and provided a comprehensive picture of the environmental fate of PAHs and NPAHs, essential information for understanding the behavior of these contaminants in Lake Michigan and its aquatic food web. While concentrations of high-molecular-weight PAHs were accurately predicted, improved parameters and possibly more sophisticated model structures are needed to accurately predict low-molecular-weight compounds. Using the models in an inverse fashion to estimate emission rates of PAHs and NPAHs from environmental concentrations provided information on historical emissions, and can help improve existing emission inventories, which are compiled only every several years, and which may not include all of the many sources of PAHs/NPAHs.

7.4 Limitations

The environmental behavior of PAHs, NPAHs, hopanes and steranes is complex, and the limitations of this research are recognized. In Chapter 2, only whole-fish homogenates were analyzed, as analyses were aimed at assessing the whole body burden of the fish and the health risks to other wildlife. However, organ-specific analysis, e.g., analysis of fillets, would be more appropriate for evaluating human health risks. In addition, only parent PAHs were measured, which may ignore the potential persistence and toxicity of PAH metabolites. Concentrations were measured in only a top predator fish (lake trout), and levels in prey fish and aquatic invertebrates would be useful to permit a direct analysis of trophic level effects.

In Chapter 3, the 24 sites sampled for sediments were all open-water sites with water depths greater than 10 m. No samples were taken at or very near potential source areas (e.g., AOCs, urban and industrial discharges), making it difficult to assess the impact of these sources. In addition, only surficial sediments were analyzed. Sampling and analysis of sediment cores can provide information regarding historical trends. Only sites in the southern portion of Lake Michigan were sampled. Ideally, sediment samples would be collected across the lake, and also at sites where fish collection occurred. Finally, only a subset of potential target compounds were measured. Sampling of additional compounds potentially would provide more information on sources.

Chapter 4 used bench tests of a heavy-duty diesel engine with three fuels, two after-treatment systems and three engine operating conditions. However, bench tests may not reflect real road conditions, and the limited sample cannot represent the full range of emissions resulting from different engines, model years, displacements, calibrations, after-treatment systems, fuel compositions, etc. Moreover, the tests used only steady-state conditions, rather than dynamic driving cycles.

For the integrity tests in Chapter 5, the number of samples in each filter conditioning and storage test was small, and thus statistical hypothesis testing was not feasible. Only borosilicate glass fiber filters were examined, and other filter types such Teflon filters were not evaluated.

The model in Chapter 6 applied the same (and often) default values of certain model parameters for all target PAHs and NPAHs. These included mass transfer coefficients and gut absorption efficiencies, sensitive variables for many model outputs. These defaults had been developed for persistent organic pollutants (e.g., PCBs, PBDEs and high-molecular-weight PAHs), and may not have been appropriate for less persistent NPAHs and low-molecular-weight PAHs. Only air emission data were available, and emissions to other compartments were estimated or ignored. Background concentrations in water were also ignored. Measurements in water and air were unavailable for NPAHs, so model evaluation was not attempted for these compounds. For the dynamic model application, there is a large data gap in PAH sediment concentrations (1990 to 2011), and concentrations were interpolated. For these reasons, the estimated emission rates may be inaccurate, and additional measurements would be helpful for confirming the results.

7.5 Recommendations for further study

7.5.1 Future studies on aquatic biota

Although the results in Chapter 2 showed that PAH and NPAH levels in Lake Michigan lake trout were low, the detection of highly carcinogenic NPAHs (e.g., 6-nitrochrysene) still highlighted the need for additional assessment or monitoring. Concentrations of PCBs and organochlorine pesticides have been monitored in Great Lakes predator fish (lake trout and walleye) since 1970s by the Great Lake Fish Monitoring and Surveillance Program (GLFMSP) (Chang et al. 2012; De Vault et al. 1996). This program could be extended to include PAHs and NPAHs due to the toxicity of these compounds. More continuous monitoring would also

provide trend information. The GLFMSP only samples two sites in each of the Great Lakes (Sturgeon Bay and Saugatuck in Lake Michigan). The number of sites could be increased to cover areas near AOCs, large cities and industrial centers, e.g., Green Bay, Milwaukee, Waukegan and Chicago/Gary for Lake Michigan. The monitoring program for sediments could also be extended, since sediments form the largest reservoir of contaminants and are a direct source of contaminants to benthic organisms.

The "biodilution" suggested in this research suggests the value of monitoring PAH and NPAH levels in prey fish and aquatic invertebrates. Future studies might sample predator fish, prey fish, invertebrates and sediments at the same sites, analyze them for PAH and NPAH concentrations, and determine the exact trophic level of the organisms using stable carbon and nitrogen isotope analysis. This would evaluate the direct impacts of sediments, and better estimate the relationship between trophic level and PAH/NPAH concentration.

7.5.2 Future studies on sediments

Chapter 3 investigated the target compounds in southern Lake Michigan sediments. Future studies might examine sediments from other areas of the lake (e.g., central and northern portion, Green Bay, Grand Traverse Bay), thus obtaining a more comprehensive picture across the whole Lake Michigan. Further, sampling and analysis of sediment cores would provide more information regarding historical trends. Additional compounds (e.g., alkylated PAHs, certain NPAHs and hopanes) might be measured to gain more insight regarding hydrocarbon sources.

7.5.3 Advanced source apportionment

Chapter 3 used CMB modeling to apportion PAH sources. This technique cannot be used for NPAHs due to the lack of source profiles. Thus, NPAH emissions and profiles from various sources (e.g., vehicle exhaust, power plants, coke ovens, wood burning, etc.) should be developed to facilitate CMB modeling. In addition to the diagnostic ratios and CMB modeling presented in Chapter 4, other techniques might be used to identify and apportion sources. These include factor analysis (FA) (Christensen and Arora 2007) and principal component analysis (PCA) (Stout and Graan 2010). Multiple techniques used together might help confirm results.

7.5.4 Future studies on vehicle emissions

This study demonstrated that different engine loads, fuel types and after-treatment systems produced large differences in diesel engine exhaust emissions. It is recommended to perform additional tests with various engine brands, model years, displacements, calibrations, after-treatment systems, and fuels. In addition to steady-state conditions, the EPA transient cycle and other driving cycles could be examined.

7.5.5 Improved multimedia modeling

For many NPAH compounds, experimentally-obtained physiochemical properties are unavailable, and these parameters were estimated (EPA 2012). Experimentally-determined values of NPAH parameters, such as vapor pressure, water solubility and log *Kow* , would be preferred. Other parameters, such as bioavailability and gut absorption efficiencies, could also be studied to better understand NPAH fate in the food web. The food web can be further extended to consider zebra and quagga mussels and their impacts in contaminant cycling. Measurements of NPAH concentrations in air, water and biota of Lake Michigan are needed to validate the model. Lastly, improved and more comprehensive emission inventories, especially for emissions to water and soil and including NPAHs, would be valuable for multimedia modeling.

7.6 References

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