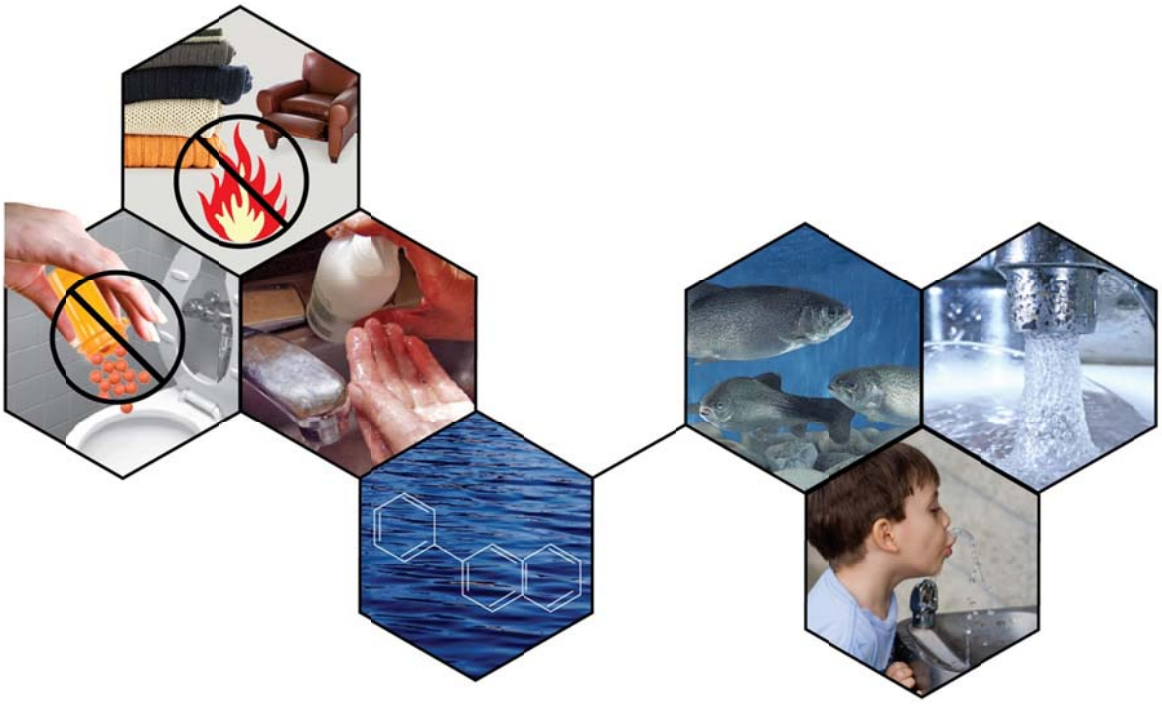
Contaminants of Emerging Concern
In the
Tidal Delaware River

Pilot Monitoring Survey
2007 -2009



Delaware River Basin Commission

final report prepared by

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1.0 Abstract

Contaminants of emerging concern are unregulated substances that have entered the environment through human activities. Current regulatory approaches are inadequate to address these contaminants and the increasing public concern over their environmental and human health implications. A pilot multi-year survey of contaminants of emerging concern in the main stem of the tidal Delaware River sampled and analyzed ambient waters in 2007, 2008 and 2009 for pharmaceuticals and personal care products, as well as perfluoroalkyl and polyfluoroalkyl substances (PFASs) by liquid chromatography/tandem mass spectrometry (LC/MSMS); hormones, sterols and nonylphenols by gas chromatography/mass spectrometry (GC/MS); and polybrominated diphenyl ethers (PBDE) by high resolution gas chromatography/mass spectrometry (HRGC/MS). Pharmaceuticals and personal care products (PPCP) detected at concentrations of ng/L in the river were comparable to compounds and concentrations measured in other studies of ambient water with the exception of codeine and metformin. Fifteen PPCP were identified for focused study in surface waters (acetaminophen, carbamazepine, clarithromycin, codeine, dehydronifedipine, erythromycin-hydrate, fluoxetine, gemfibrozil, 2-hydroxy-ibuprofen, ibuprofen, lincomycin, metformin, sulfamethoxazole, thiabendazole, and triclocarban) based on the criteria of environmental occurrence, aquatic ecotoxicity, potential human health effects to sensitive populations, and analytical feasibility. In addition, PPCP reported in fish tissue in other locations should be assessed in the Delaware Estuary. Natural and synthetic hormones were detected in ng/L levels. Hormones detected at low concentrations and at limited locations included estrone, norethindrone, 17-alpha-ethynyl-estradiol, desogestrel and testosterone. Hormones have been ranked in the top of chemicals in U.S. surface waters for ecological effects and warrant further study. PFASs were measured in ng/L concentrations with perfluorononanoate (C9) measured at the highest concentration. Although concentrations of PFASs in water appear to be trending downward each year, additional ecotoxicology and bioaccumulation information is needed for these compounds especially on longer chain and sulfonated PFASs. Nonylphenol levels did not exceed current United States Environmental Protection Agency national water quality criteria. PBDE were measured in pg/L to ng/L concentrations with homolog distributions similar to those observed in other North American locations. Because of the low levels found in water, additional monitoring of PBDE by the DRBC will focus on bioaccumulation in fish tissue. The effects of PPCP in estuarine and coastal waters are not well studied. Future work in the Delaware River should evaluate the sources as well as the fate and effects of PPCP in the water column, sediments and biota.

2.0 Introduction

The goal of the survey was to collect ambient water data for use in compiling basin data on contaminants of emerging concern (CEC) identified in the DRBC report titled Emerging Contaminants of Concern in the Delaware River Basin (http://www.state.nj.us/drbc/EmergingContaminantsFeb2007.pdf). Participants included the Delaware River Basin Commission (DRBC), Axys Analytical Services Ltd, and Delaware Department of Natural Resources Environmental Control and Environmental Laboratory Section (DNREC-ELS). More than 84,000 chemicals are listed on the USEPA Toxic Substance Control Act (TSCA) chemical substance inventory. New chemicals are introduced each year and released to the environment while improved analytical methods are available to detect many of these compounds. In addition, there is a growing body of information on adverse effects from some contaminants. Scientists, the public, and regulators have an increased interest in substances and toxic effects not historically monitored or assessed. The compounds included in the multi-year survey are unregulated compounds (pharmaceuticals, hormones and sterols, perfluoroalkyl and polyfluoroalkyl substances, and polybrominated diphenyl ethers) or recently regulated compounds (nonylphenols). The survey was conducted in the tidal Delaware River, the part of the river that has tidal flux from Trenton to the head of the Bay. This is an urbanized and industrialized area as shown in Figure 1 (river segment predominantly surrounded by developed (red) land use). Over 6 million residents live in contributing watersheds to the tidal Delaware River creating an area of concentrated consumer product usage. Two sites (E12 at RM105.4 and E16 at RM 131.1) in the survey are within segments of the river designated for use as public water supplies after reasonable treatment (Table3 and Figure 3). All sites in the survey are within segments of the river designated for fish ingestion as well as water uses such as maintenance of resident fish and other aquatic life and wildlife protection.

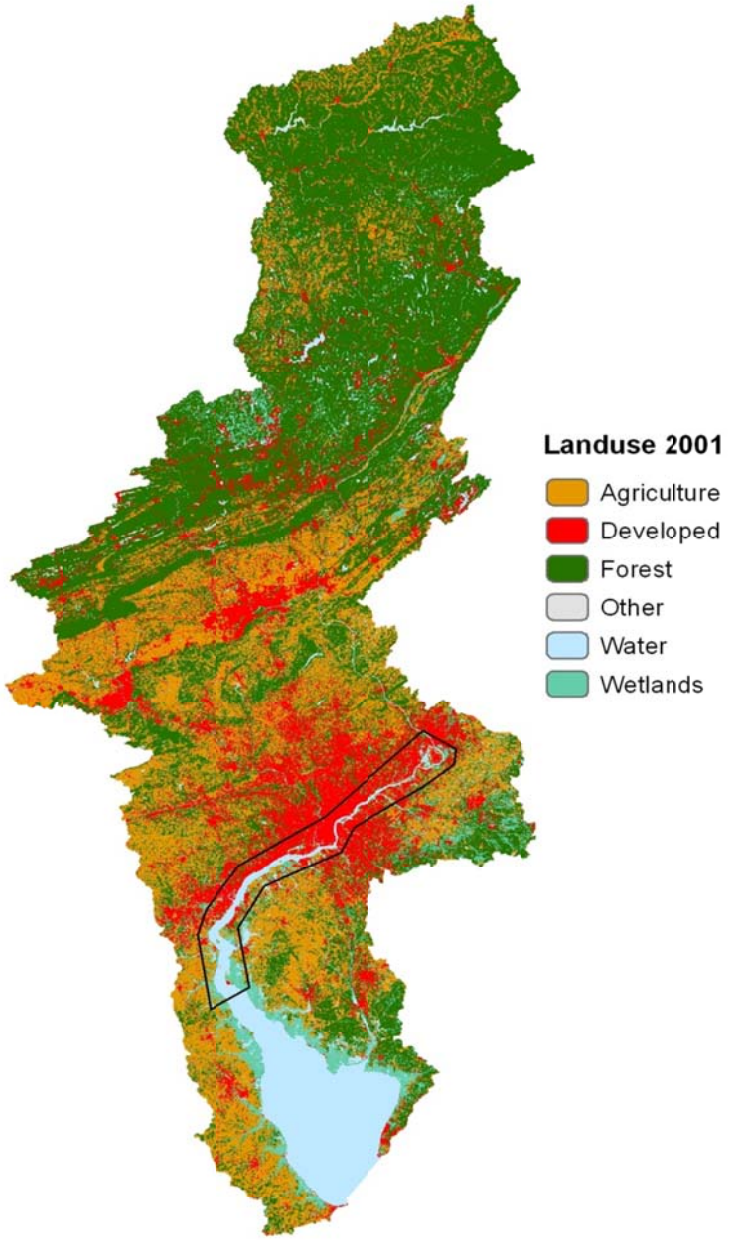

Figure 1. Map of Delaware River Basin Land Use in 2001 from USGS National Land Cover Database

3.0 Pharmaceutical Source Map for the Delaware River Basin

As part of an ongoing effort by the DRBC to assess the vulnerability of the Delaware River Basin to contaminants of emerging concern, a pharmaceutical source map was generated by merging information from a National Pollutant Discharge Elimination System Permit Compliance System (NPDES/PCS) retrieval file and a pharmaceutical manufacturing Standard Industrial Classification (SIC) code file. The merged file includes the facility name, address, NPDES number, SIC, SIC description, and coordinates. The pharmaceutical manufacturing SIC code file for the basin contains twenty-nine entries. Several facilities were deleted from the list based on current information from regulators in basin states. The DRBC pharmaceutical manufacturers list was cross-checked with a similar list obtained from Carey A. Johnston of the U.S. EPA, Office of Water. The EPA list consists of facilities likely to be subject to the Pharmaceutical Manufacturing Effluent Guidelines (Part 439). The EPA list is based on 2004 data from Permit Compliance System (PCS) and Toxics Release Inventory (TRI). Facility identification is done based on the primary SIC code reported to TRI or PCS. Facilities in the following SIC codes are regulated by the Pharmaceuticals Manufacturing Effluent Guidelines source category (Part 439): 2833 Medicinals and botanicals; 2834 Pharmaceutical preparations; 2835 Diagnostic substances; and 2836 Biological products, except diagnostics. In addition, the EPA identified several pharmaceutical manufacturing facilities in SIC Code 2048: Prepared Feed and Feed Ingredients for Animals and Fowls, Except Dogs and Cats. After the DRBC and USEPA lists were found to match, the list was used to generate the pharmaceutical source map. Wastewater treatment plants in the basin were added to the list and map as post-consumer use sources of pharmaceuticals. Figure 2 shows numerous potential sources to the Delaware River, from 584 sewage treatment facilities and 18 manufacturing sites, for only one category of contaminants of emerging concern (pharmaceuticals).

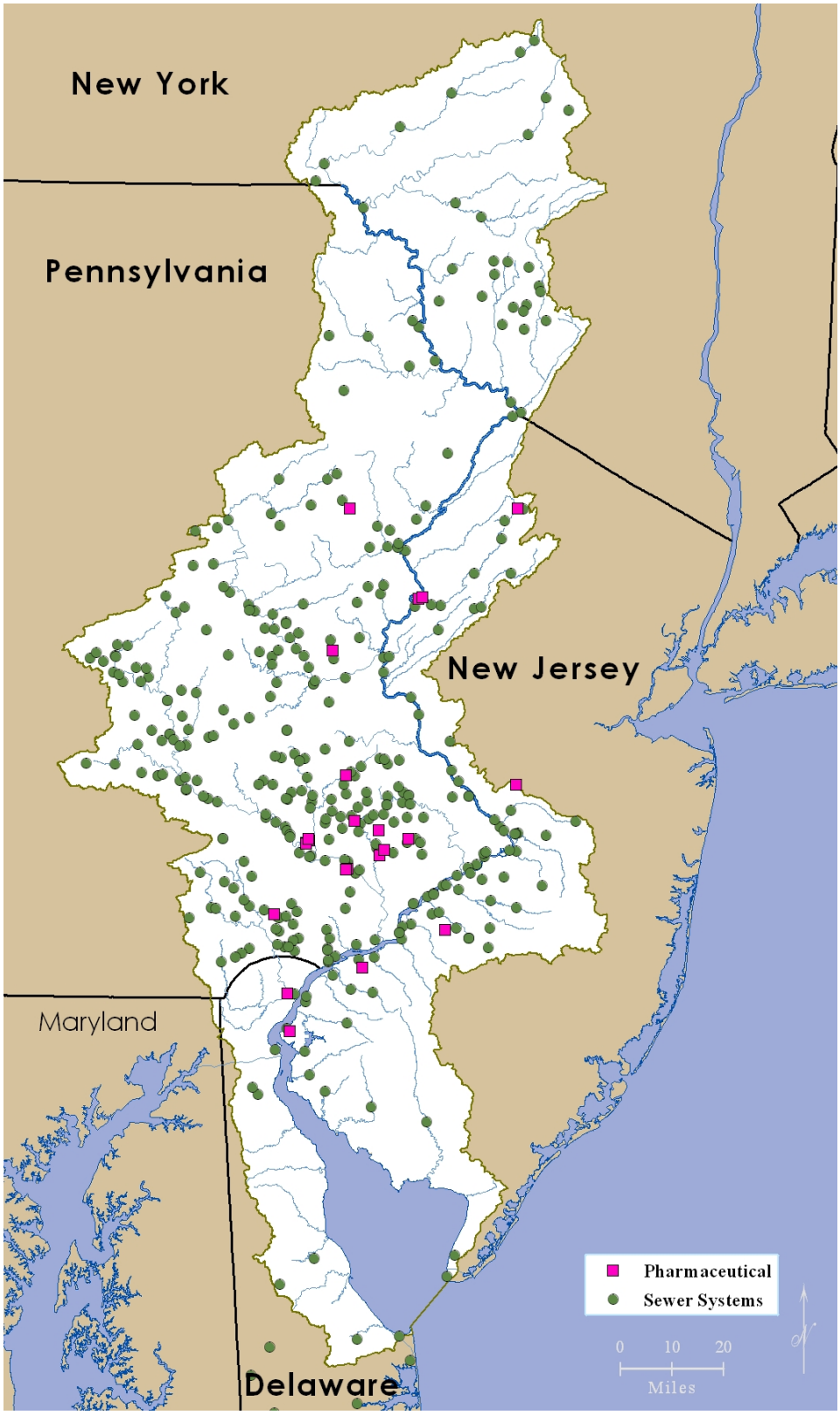

Figure 2. Pharmaceutical Source Map for the Delaware River Basin

4.0 Methods

4.1 Analytical Methods

Pharmaceuticals and personal care products (PPCP) were analyzed using Axys Method MLA-052 Rev 4 in 2007 and 2008 and MLA-075-Rev4 in 2009. Both methods are suitable for determination of pharmaceutical and personal care compounds in aqueous samples. The analysis required extraction at different pH conditions. Prior to extraction and/or clean-up procedures samples were adjusted to the required pH and spiked with surrogates. Aqueous samples were filtered prior to the analysis to remove solid particulate. Aqueous samples were cleaned up by solid phase extraction (SPE) and analyzed by liquid chromatography / electrospray ionization / tandem mass spectrometry (LC/ESI-MS/MS) in positive and negative ionization modes. The method has four modes that target different pharmaceuticals as follows: 1) acid extraction, positive ESI 2) tetracylines, positive ESI; 3) acid extraction, negative ESI; and 4) base extraction, positive ESI. PPCP analytes and estimated detection limits are listed in Appendix A Table A1.

Sterols and hormones were analyzed using Axys Method MLA-055 Rev 2 in 2007 and MLA-068 Rev 1 in 2008. Both methods are suitable for determination of concentration of a suite of steroids in aqueous samples. All samples were spiked with deuterated surrogate standards prior to analysis. Aqueous samples were solvent extracted with extracts cleaned up on a layered alumina Florisil column and derivatized with BSTFA prior to analysis by capillary gas chromatography and low resolution mass spectrometry (LRMS). Hormones were analyzed using Axys MLA-072 Rev 2 in 2009 by LC/MS/MS. The methods used to measure the hormones and sterols in the DRBC study are new analytical methods with few surrogates available and numerous QA/QC qualifiers. Sterol and hormone analytes and estimated detection limits are listed in Tables 6, 7 and 8.

Perfluoroalkyl and polyfluoroalkyl substances (PFASs) were analyzed using the Axys Method MLA-060 Rev 3. After spiking with isotopically-labeled surrogate standards and cleanup on SPE cartridges, samples were analyzed by LC-MS/MS. Final sample concentrations were determined by isotope dilution/internal standard quantification against extracted calibration standards in water. PFASs analytes and limits of quantification are listed in Table 9.

Polybrominated diphenyl ethers (PBDE) were analyzed using Axys Method MLA-033 Rev 3 to determine concentrations of a suite of brominated diphenyl ethers in aqueous samples as described in USEPA Method 1614. The sample is extracted and cleaned up on a series of chromatographic columns. The final extract is spiked with isotopically-labeled recovery (internal) standards prior to instrumental analysis. Analysis of the extract is performed on a high resolution mass spectrometer (HRMS) coupled to a high-resolution gas chromatograph (HRGC) equipped with a DB-5HT chromatography column. PBDE analytes and estimated detection limits are listed in Appendix A, Table A2.

Nonylphenols (NP) and nonylphenol ethoxylates (mono-NPEO1, di-NPEO2) were analyzed using Axys Method MLA-004 Rev 05 for determining the concentration of nonylphenols and nonylphenol ethoxylates in aqueous samples. Samples were spiked with isotopically-labeled surrogate standard prior to extraction. Samples were extracted, underwent acetylation steps, and were cleaned up by column chromatography. The cleaned up extract was analyzed by GC/MS. Nonylphenol and nonylphenol ethoxylates were reported as total concentrations, representing the sum of all the detected isomers in a specific target group. NP and NPEO analytes and estimated detection limits are listed in Table 11.

Analysis of Bisphenol A in aqueous samples containing less than 1% solids was based on Axys method MLA059 for the analysis of urine samples. Aqueous samples were adjusted to a pH of 2 and spiked with labelled ^{13}C quantification standards. Samples were extracted and cleaned up using solid phase extraction (SPE) procedures. The method determined the total of the free bisphenol A (not the glucuronidated metabolites). Analyte concentrations were determined by LC/MS/MS and quantified using the isotope dilution quantification method. The estimated detection limit for Bisphenol A was 0.05 ng/L.

Table 1 presents a summary of all the analytical methods used.

Table 1. Analytical Methods Overview

Parameters	Survey Year		
	2007 # of analytes / method	2008 # of analytes / method	2009 # of analytes / method
PFASs LC/MS/MS	13 MLA060 – Rev03	13 MLA060 – Rev04	13 MLA060 – Rev07
PPCP LC/MS/MS USEPA 1694 plus extended list of analytes	54 MLA052 – Rev04	72 MLA052 – Rev04	119 MLA075 – Rev04
Sterols and Hormones	24 MLA055 – Rev02 GC/LRMS	27 MLA068 – Rev01 GC/LRMS	17 MLA072 – Rev02 Hormones only LC/MS/MS
NP and NPEO GC/MS	3 MLA004 – Rev04	4 MLA004 – Rev05	4 MLA004 – Rev05
bis-phenol-A LC/MS/MS	Not monitored	1 MLA059 – Rev03	1 MLA059 – Rev04
PBDE HRGC/HRMS USEPA 1614	46 MLA033 - Rev03	Not monitored	Not monitored

4.2. Supplemental Water Quality Data Methods

In-field measurements of specific conductivity, salinity, water temperature, dissolved oxygen and pH were performed at all sites on each sample day. Laboratory analysis of transect composite samples from each site was also conducted according to the methods listed in Table 2.

Aquatic toxicity tests for short-term chronic toxicity and *in vitro* tests for estrogenic compounds were also conducted on the ambient water samples. The tests can assess chemical mixtures and possible additive effects as well as assess toxicants with no specific analytical method or chemicals not monitored by the chemical methods utilized (Appendix C).

Table 2. Supplemental Water Quality Methods

PARAMETER	METHOD REFERENCE	MDL[1]	LOQ[2]
CHLORIDE	EPA 300.0	1 mg/L	3 mg/L
CHROMIUM, HEXAVALENT	STD MTH 3500-Cr B	1.4 ug/L	5.0 ug/L
DISSOLVED OXYGEN (field)	STDMTD 18th ed. 4500-O	N/A	0.1 mg/L
ALKALINITY	EPA 2320B	0.9 mg/L	1.0 mg/L
HARDNESS	EPA 2340C	0.3 mg/L	1.0 mg/L
pH (field)	STDMTD 4500 H+	N/A	0.1 unit
SALINITY	STDMTD 2520	N/A	0.1 ppt
SPECIFIC CONDUCTANCE (field)	STDMTD 2510B	N/A	2.0 uS/cm
SUSPENDED SOLIDS, TOTAL (TSS)	EPA 160.2	NA	5.0 mg/L
TEMPERATURE, AIR/WATER (field)	EPA 170.1	N/A	N/A
ORTHOPHOSPHATE, DISSOLVED	EPA 365.1 Rev 2.0	0.002 mg/L	0.010 mg/L
PHOSPHOROUS, TOTAL	EPA 365.4	0.005 mg/L	0.040 mg/L
NH_3 – N	EPA 350.1 Rev 2.0	0.004 mg/L	0.020 mg/L
NO_2 – N	EPA 353.2 Rev 2.0	0.003 mg/L	0.008 mg/L
NO_3 - N	EPA 353.2 Rev 2.0	0.005 mg/L	0.010 mg/L
NO_3 – N & NO_2 - N	EPA 353.2 Rev 2.0	0.005 mg/L	0.010 mg/L
KJELDAHL, TOTAL - N	EPA 351.2 Rev 2.0	0.07 mg/L	0.20 mg/L
CADMIUM, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	0.43 ug/L	5 ug/L

PARAMETER	METHOD REFERENCE	MDL[1]	LOQ[2]
CALCIUM	EPA 200.7 Rev 4.4	NA	1000 µg/L
CHROMIUM, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	3.1 ug/L	10 ug/L
COPPER, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	1.4 ug/L	5 ug/L
LEAD, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	0.9 ug/L	3 ug/L
MAGNESIUM	EPA 200.7 Rev 4.4	NA	50 µg/L
NICKEL, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	1.22 ug/L	20 ug/L
ORGANIC CARBON, DISSOLVED AND TOTAL	5310B	0.3 mg/L	3.0 mg/L
POTASSIUM	EPA 200.7 Rev 4.4	NA	50 µg/L
SILVER, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	1.24 ug/L	10 ug/L
SODIUM	EPA 200.7 Rev 4.4	NA	100 µg/L
SULFATE	EPA 300.0 Rev 2.1	0.04 mg/L	1.5 mg/L
ZINC, TOTAL AND DISSOLVED	EPA 200.7 Rev 4.4	0.4 ug/L	10 ug/L

[1]Method Detection Limit; [2]Limit of Quantitation LOQ represents the lowest standard in the calibration curve or, in instances where a standard curve is not specified by the procedure, LOQ represents the limitations of the method.

[3] EMDL – Estimated Method Detection Limit as per DRBC guidance on PCB sampling at http://www.state.nj.us/drbc/PCB_info.htm; [4] Method Limit

4.3 Sampling Procedures

Samples were collected from six locations in the tidal Delaware River (Table 3; Figure 3). The tidal portion of the Delaware River, from the head of the tide at Trenton, New Jersey, to the Delaware Bay at Liston Point, Delaware, was the study area. The salinity in the tidal Delaware ranges from <1 to 15 ppt. Sites E12 and E16 are in DRBC Water Quality Zones with designated uses that include public water supplies after reasonable treatment. Sites E1, E4, E6 and E9 are not in Zones designated as sources of drinking water.At mid-channel sampling sites, subsurface ambient water was directly sampled into 2L HDPE (high density polyethylene) bottles for analysis of PFASs. A Niskin bottle was used to collect transect composite ambient water at 0.6 of the water column into HDPE pails to be distributed to five 2.5 L amber glass bottles for analysis of other contaminants of emerging concern. A portion of the composite sample was distributed into 2.5 to 5 gal LDPE (low density polyethylene) cubitainers (VWR Int., Brisbane, CA) for chronic toxicity bioassays and into glass bottles for estrogenic assays. Field blanks were collected. Glass bottles and blank water were obtained from the analytical laboratory. The samples were placed on ice in coolers to maintain a temperature of $4^{o}C \pm 2^{o}C$ and transported to the respective laboratories for bioassays and physical-chemical analyses. Temperature inside the cooler for bioassays was tracked during transport with a temperature logger. Temperature of samples for chemical analysis were checked upon arrival at the laboratory.

Table 3. Sampling Sites

Site	RIVER MILE	DRBC ZONE	SITE DESCRIPTION	LATITUDE (dd.ddddd)	LONGITUDE (dd.ddddd)
E1	50	5	Liston Point	39.45500	75.5600
E4	68	5	S. of De. Memorial Bridge	39.65472	75.54667
E7	80	4	Opposite Mouth of Marcus Hook Creek	39.81336	75.39058
E9	90	4	South of Schuylkill River	39.875905	75.195988
E12	105	3	Mouth of Pennsauken Creek	39.99478	75.05978
E16	131	2	Biles Channel	40.181560	74.745050

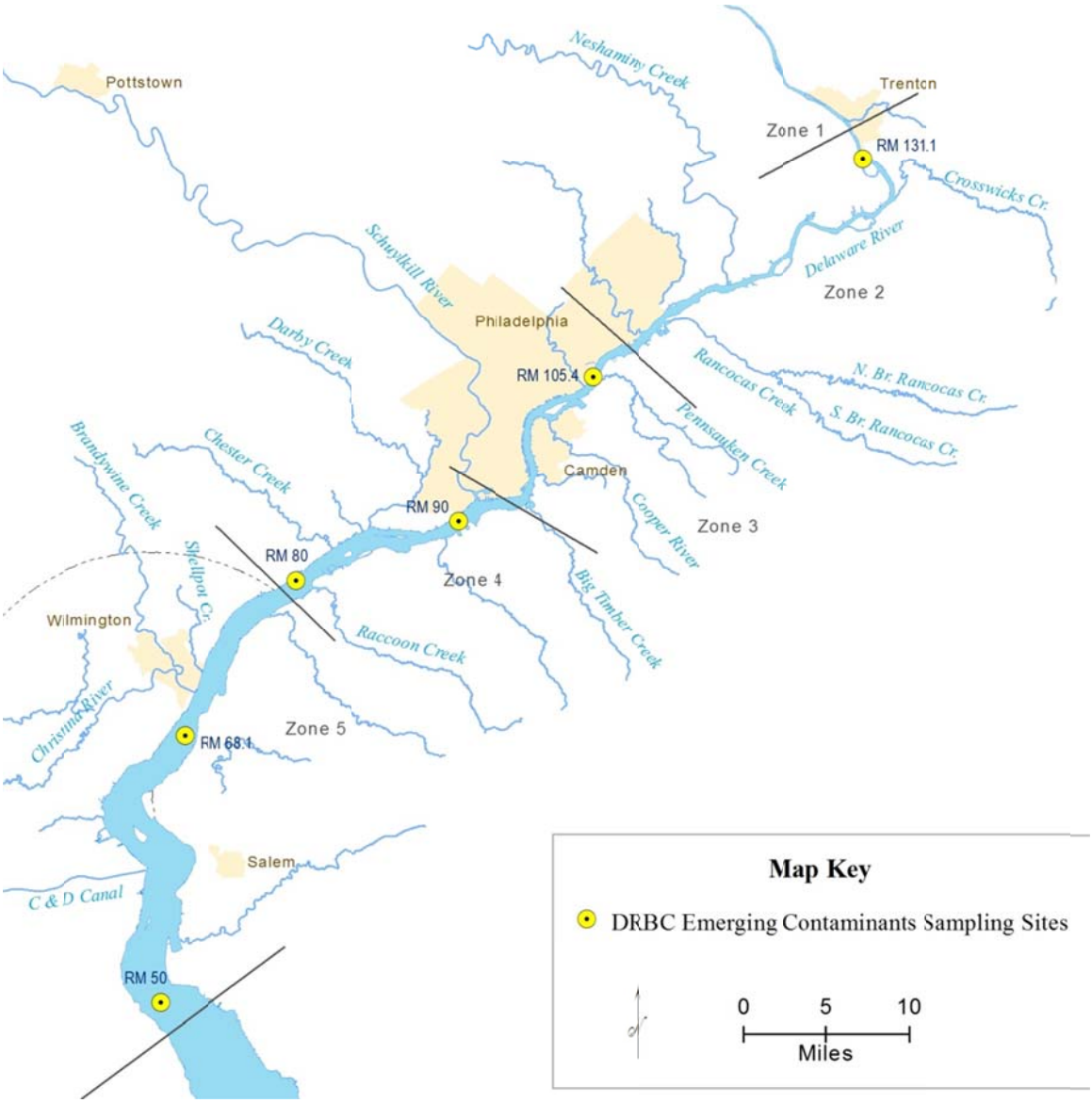

Figure 3. Map of Sampling sites in 2007, 2008 and 2009

4.4 Hydrology

Grab samples of ambient water were collected on October 17, 2007, August 6, 2008 and October 22, 2009 when the mean daily average flows for Delaware River at Trenton, NJ were at 5,390, 4,590 and 5,000 cfs, respectively. The river flows at sampling were below the harmonic mean flow of 6,500 cfs used to calculate protection of human health criteria for carcinogens and above the 30-day flow with a five year recurrence interval (30Q5) of 2,800 cfs used with human health criteria for systemic toxicants. The flows at sampling were also above the minimum flows for aquatic life protection based on a 7Q10 flow of 2,500 cfs. The sampling period is representative of late summer and autumnal river conditions in the tidal Delaware River, but not a worst case scenario.

4.5 Prioritization of PPCP

To evaluate and prioritize individual PPCP on the basis of the risk they pose to the aquatic ecosystem and human health and to add to the knowledge base on the assessment and informed management of the PPCP that pose the greatest risk, a review of the existing knowledge and available data on PPCP exposures and their ecological impacts to the aquatic environment was undertaken using resources listed below.

Databases used in the prioritization included:

US EPA ECOTOX, which currently includes more than 520,000 test results on the effects of more than 8,500 chemicals, including PPCPs, on over 6,400 terrestrial and aquatic species (http://cfpub.epa.gov/ecotox/);

ECOSAR (Ecological Structure Activity Relationship) a computerized predictive system that estimates the aquatic toxicity of chemicals. The program estimates a chemical's acute (short-term) toxicity and chronic (long-term or delayed) toxicity to aquatic organisms such as fish, aquatic invertebrates, and aquatic plants by using Structure Activity Relationships (SARs) (http://www.epa.gov/oppt/newchems/tools/21ecosar.htm);

Center for Coastal Environmental Health and Bimolecular Research (CCEHBR) Pharmaceuticals in the Environment, Information for Assessing Risk (PEIAR) website. A site designed to provide available information for assessing risks to aquatic resources from drugs entering waterways from both point and non-point sources (http://www.chbr.noaa.gov/peiar/default.aspx);

USEPA PPCP Literature Citation Database includes published literature relevant to the issues surrounding PPCPs as environmental contaminants (http://www.epa.gov/ppcp/lit.html).

Additional sources for ecotoxicology data were from the following:

Bergh, K. 2005 unpublished. Ecological Risk Assessment of Pharmaceuticals and Personal Care Products in Surface Water. (http://ir.lib.sfu.ca/retrieve/2491/etd1839.pdf);

Cleuvers, M. 2003. Aquatic ecotoxicity of pharmaceuticals including the assessment of combination effects. Toxicology Letters 142:185-194;

Cunningham, V. et al., 2006. Effects of Human Pharmaceuticals on Aquatic Life: Next Steps. Environment Science & Technology. Vol 40.Issue 11 pp 3456-3462;

Crane, M; C. Watts and T. Boucard. 2006. Chronic aquatic environmental risks from exposure to human pharmaceuticals. Science of the Total Environment 367:23-41;

Fent, K, A Weston and D. Caminanda. 2006. Ecotoxicology of human pharmaceuticals. Aquatic Toxicology 76:122-159;

Kuster, A. et al., 2009. Environmental risk assessment of hman pharmaceuticals in the European union: a case study with the β-blocker atenolol. Integr Environ Assess and Manag 6(1)514-523;

Oakes, K. D.et al., 2010. Environmental Risk assessment for the serotonin re-uptake inhibitor fluoxetine: case study using the European risk assessment framework. Integr Environ Assess and Manag 6(1)524-539;

Schmitt, H et al., 2009. Recommendations on the environmental risk assessment of pharmaceuticals: effect characterization. Integr Environ Assess and Manag 6(1): 588-602;

Winter et al 2008. Defining the chronic impacts of atenolol on embryo-larval development and reproduction in the fathead minnow (Pimephales promelas). Aquat. Toxicol. (3) 361-369;

Material Safety Data Sheets (MSDS); and

TCC Consortium. High Production Volume (HPV) Chemical Challenge Program Data Availablility and Screening Level Assessment for Triclocarban, CAS#:101-20-2. (http://www.epa.gov/hpv/pubs/summaries/tricloca/c14186tc.htm).

Using the available data, a risk based procedure was used to prioritize PPCP based on exposure and effects to aquatic organisms that compared measured environmental concentrations from the DRBC multi-year survey to predicted environmental effects levels from published literature or ECOSAR estimates. Predicted no environmental effects concentrations (PNEC) were estimated from acute toxicity (ECx or LCx) divided by an assessment factor of 1,000 or chronic toxicity (NOEC) divided by an assessment factor of 100. The aquatic toxicity data used were from single species tests. Toxicity

tests with unspecified genus and species were not used. The few available studies on the toxicity of pharmaceutical mixtures also were not included in the assessment (Fent et al., 2006; Kumar et al., 2010).

The list of priority PPCP derived in this way were compared to other prioritization and risk assessment approaches that used multiple prioritization criteria such as estimated environmental concentration, ecological and human toxicity, exposure to stream water and fish consumption, physicochemical properties, analytical feasibility, consumption/sales, prescription numbers, loadings, exposures, degradation, and persistence (Bruce, *et al*., 2010; Collier, 2007; Cooper *et al*., 2008; Cunningham *et al*., 2009; de Voogt *et al*, 2009; Kostich and Lazorchik, 2008; Kumar and Xagoraraki, 2010a, 2010b; Ottmar *et al*., 2010; and Schwab *et al*., 2005; Schmitt *et al*., 2009, Roos *et al*., 2012).

The four key criteria selected for prioritizing PPCP in the tidal Delaware River were the following:

1) environmental occurrence (maximum detected concentration in DRBC surveys),
2) aquatic ecotoxicity (Hazard Quotient of Measured Environmental Concentration to the Predicted No Effect Concentration (MEC/PNEC)),
3) human health effects (reported concerns due to possible carcinogenicity, mutagenicity, reproductive and developmental effects, immunotoxicity, and interactions among drugs when exposed to multiple contaminants especially in sensitive populations),
4) analytical feasibility (USEPA Method 1694 test parameters with validated analytical methods).

Other considerations worth noting in the prioritization include:

1) Occurrence data for some compounds, specifically codeine and metformin, warrant their inclusion for priority study in the Delaware Estuary,
2) A number of compounds such as diclofenac, ethinylestradiol and oxytetracycline that have been identified as priority compounds in other studies were not detected in this study of the tidal Delaware River and were not included in the priority list (Cooper et al., 2008; Collier, 2007),
3) Although aquatic ecotoxicity data were not available for dehydronifedipine, this pharmaceutical has been included in the priority list of pharmaceuticals based on occurrence data and ecological effects (Kumar and Xagoraraki, 2010b).
4) A number of metabolites were detected (benzoylecgonine, desmethyldiltiazem, 1,7-dimethylxanthine, 10-hydroxy amitriptyline and 2-hydroxy-ibuprofen) some at concentrations higher than the parent compound. Although aquatic ecotoxicity data were not available for this compound, 2-hydroxy-ibuprofen was included in the priority list based on occurrence data, toxicity data of the parent compound and the need to further investigate metabolites (Celiz, et al., 2009).
5) The use of physical chemical properties to predict bioaccumulation of PPCP from the water column to fish tissue or aquatic biota is not a key criteria used in this

prioritization because there is limited information on the environmental fate of ionized compounds the chemical form of many PPCP in the environment (Tarazona *et al*, 2009). However, PPCP such as diphenylhydramine, norfluoxetine, sertaline, desmethylsertraline, carbamazepine, diltiazem, fluoxetine, and gemfibrozil that have been detected in fish in EPA studies and by other researchers should be further assessed in the Delaware Estuary (http://water.epa.gov/scitech/swguidance/ppcp/fish-tissue.cfm, Brooks et al., 2005).

5.0 Results and Discussion

5.1 Pharmaceutical and Personal Care Products

A wide range of pharmaceutical and personal care products (PPCP) including prescription medicines, over the counter medicines (OTC), antibiotics, and anti-bacterials used in consumer products were targeted in this study. Until recently, the fate and transport of many common PPCP were not of great concern. However, many of these synthetic compounds may ultimately pose a threat to human health and/or the environment. It has been established that some of these substances, i.e., endocrine disruptors, that affect the function of the endocrine system, have the potential to be detrimental to the development of humans and other organisms by adversely affecting physiology and reproduction (Daughton and Ternes 1999). A number of chemicals have been identified as being of environmental concern including lipid regulators (gemfibrozil), analgesics/anti-inflammatories (codeine, acetaminophen, and ibuprofen), antiepileptics (carbamazapine), antidepressants (fluoxetine), oral contraceptives (ethynylestradiol), and antimicrobial disinfectants (triclosan and triclocarban) (Daughton and Ternes, 1999; Fent et al., 2006). The EPA has listed 2-methoxyethanol, erythromycin, mestranol, and nitroglycerin, as well as, the hormones estrone, estriol, estradiol, equilin, equilenin, 17-α estradiol and 17-α ethynyl estrdiol, mestranol and norethindrone as substances that may require regulation under the Safe Drinking Water Act (SDWA) on the Contaminant Candidate List 3 (CCL3) and/or Unregulated Contaminants Monitoring Rule 3 (UCMR3):
(http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm)
(http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/methods.cfm)

Most PPCP compounds detected and concentrations found in the tidal Delaware River are comparable to those reported in other occurrence studies of ambient waters for pharmaceuticals and other organic wastewater contaminants. Of the PPCP analyzed in water samples from the tidal Delaware River, 57 compounds were detected in 2007, 2008 or 2009 at concentrations in the ng/L range. Ten PPCP were detected in all three years (azithromycin, caffeine, carbamazepine, clarithromycin, codeine, dehydronifedipine, diltiazem, diphenhydramine, erythromycin-hydrate and fluoxetine) (Appendix B, Table B.4). However, the analytical method used was improved each year increasing the number of PPCP analytes from 54 in 2007 to 72 in 2008 and 119 in 2009 (Table 1).

Therefore, most of the chemicals are represented by a single measurement but, chemicals with multiple measurements are represented by mean (standard deviation) in Table 4. With the exception of codeine and metformin, the compounds detected and concentrations found in the tidal Delaware River are comparable to those reported in other studies of ambient waters for the occurrence of pharmaceuticals and other organic wastewater contaminants in Pennsylvania streams (Loper *et al*., 2006); metropolitan Chicago rivers (Mack, unpublished); estuarine environments (Pait *et al*., 2006); and national reconnaissance studies of surface waters by the USGS (Focazio, 2004, Kolpin *et al*., 2002). Most of the PPCP in the DRBC study were below detection limits while those chemicals measured were typically at low concentrations (Table 4 and Appendix B, Tables B1 to B4). It should be noted that the results reported from these studies are based on filtered water samples and are therefore biased toward hydrophilic (soluble) compounds that are less likely to sorb to suspended particles or rapidly transported to river sediment.

The analgesic codeine, one of the top five most highly prescribed prescription pharmaceuticals, had concentrations peak in the Delaware River at the RM 68.1 with a mean of 89.97 ng/L and a range of 38 to 159 ng/L compared to background concentrations in the river of <10 ng/L. Generally, Loper et al., 2006 found non-detects in a Pennsylvania study except in a few creeks with concentrations between 29 to 56 ng/L. Two pharmaceutical facilities that use codeine in their manufacturing processes have been identified near the RM 68.1 site. Both facilities discharge indirectly to the Delaware River through POTWs. Although codeine is reported to have a high removal rate in POTWs, effluent at the two POTWs receiving the indirect discharges had codeine concentrations of 377 and 2,590 ng/L (three day average). This is in comparison to the occurrence of codeine in effluents measured in a national EPA study where six out of nine POTW facilities were found to have concentrations in the range of ND to 25 ng/L and three facilities had concentrations of 628, 642 and 890 ng/L (USEPA, 2009). A recent study of effluent from wastewater treatment facilities with indirect pharmaceutical discharges found 10 to 1000 times higher pharmaceutical concentrations than those typically found in WWTP effluents. The authors concluded that facilities involved in the manufacture of pharmaceutical products are an under-investigated source of pharmaceuticals to the environment (Phillips *et al*., 2010).

Metformin was detected in the DRBC multi-year survey between RM 80 to 90 in the range of 1,000 to 3,500 ng/L compared to no detection in a study of the Chesapeake Bay (Pait et al., 2006) and in the range of several 100 ng/L in most German rivers (Scheurer et al., 2009). This antidiabetic drug has one of the highest pharmaceutical production levels world-wide. A recent publication on pharmaceutical loadings in wastewater treatment plants concluded that metformin and two other PPCP (valacyclovir and gabapentin) warrant study of fate, transport and occurrence due to the fact that these drugs have high effluent concentrations, significant potential for ecotoxicity and have been included in limited prior research (Ottmar et al., 2010).

A Pennsylvania Department of Environmental Protection (PADEP) study in cooperation with United States Geological Survey (USGS) reported analysis of 15 pharmaceuticals

and 31 antibiotics in water samples from streams with inputs from agricultural areas dominated by animal-feeding operations and wells currently used to supply water for livestock on a farm, in south-central Pennsylvania. The study compared the impact of municipal wastewater and agricultural run-off on the occurrence of the target analytes. In streams receiving municipal wastewater effluent (the scenario in the study that is most similar to the urban tidal Delaware River), 13 phamaceuticals and 11 antibiotics were detected. Maximum concentrations observed for caffeine was 4,750 ng/L, para-xanthine (a degradation product of caffeine) was 853 ng/L, carbamazapine was 516 ng/L, ibuprofen was 277 ng/L and individual antibiotic concentration maxima were in the range of 200 to 1,600 ng/L (Loper *et al*., 2006).

In a study of contaminants of emerging concern in metropolitan Chicago rivers, water column samples from three freshwater streams were sampled at low-flow conditions for twenty-five PPCP, hormones and sterols. Land use in the three streams was reported to be 64 to 78% urban. Not surprisingly, in effluent dominated steams, individual fecal sterols were detected at concentrations between 200 to 5,000 ng/L. However, the hormones equilenin, estriol, progesterone, and testosterone were not detected. PPCP were measured at maximum concentrations of 7.8 ng/L for tylosin, 16 ng/L for triclosan, 170 ng/L for carbamazapine, 200 ng/L for trimethoprim, 210 ng/L for cotinine, 310 ng/L for caffeine and 410 ng/L sulfamethoxazole. Concentrations of PPCP in the three Chicago area streams for the most part were higher than those measured in the Delaware River. It should be noted that both the DRBC and Chicago area studies are grab samples representing a single snap shot in time and space of each river under specific flow conditions and season. Nevertheless, it is noteworthy that similar contaminants of emerging concern are present in surface waters of different urban areas within the United States (Mack, 2008).

In national reconnaissance studies of surface waters, the USGS conducted a number of monitoring projects for pharmaceuticals and other organic wastewater contaminants (Focazio, 2004). The USGS criteria for selecting compounds to measure were based on the quantities in use, anticipated environmental behavior, pathways for release, health significance (known and potential), ability to measure the compound, potential as chemical indicators/tracers and stakeholder priorities. The detection of multiple contaminants in surface water was observed in Kolpin et al., 2002. Surface waters were monitored in 1999 through 2000 for 95 organic wastewater contaminants (OWC) in 139 streams including Assunpink Creek in New Jersey. The most commonly detected compounds were coprostranol, cholesterol, N-N-diethyltoluamide (DEET), caffeine, tri(2-chloroethyl)phosphate, triclosan, 4-nonylphenol, nonylphenol monoethoxylate (NPEO1), tris (2-butoxy-ethyl)phosphate, and octylphenol ethoxylate (OPEO1) at low µg/L (ppb) concentrations.

In a NOAA National Status and Trends Program study of three estuarine environments (Chesapeake Bay, Biscayne Bay, and Gulf of Fallones), analytes at most of the sites were below quantifiable concentrations. In the Chesapeake Bay sites, 13 of 24 pharmaceutical and related compounds were detected but fewer were quantified. Erythromycin hydrate was detected at many sites but below quantification levels. Compounds measured at

quantifiable levels were trimethoprim at 1 ng/L, sulfamethoxazole at 11 ng/L, fluoxetine at 3 ng/L, and acetaminophen at 2 μg/L. In Biscayne Bay, two compounds (cotinine and thiabendazole) were above detection limits but below quantification limits. A third compound acetaminophen was quantified at 3 μg/L. In the Gulf of the Farallones, two compounds were above the detection limit but below the quantification limit and no compounds were above the quantification limit. The detection limits and quantification limits (laboratory reporting limits) were not specified in the report (Pait *et al*., 2006). The authors reviewed the ecotoxicology literature on the compounds detected and concluded that the effects of pharmaceuticals in estuarine and coastal waters is not well studied. They recommended future work to document occurrence of pharmaceuticals in both water column and sediments followed by appropriate laboratory and field studies to assess possible impacts.

5.1.1 Aquatic Toxicity

All sites in the survey are within segments of the river with designated uses such as maintenance of resident fish and other aquatic life as well as wildlife protection. A DRBC review of the literature found limited aquatic ecotoxicity data on the detected compounds, primarily on individual compounds using single species tests. Thus, any assessment of risk to aquatic life is preliminary. Nevertheless, a screening level calculation by a risk characterization ratio method which calculates the ratio of the Measured Environmental Concentration to the Predicted No Effect Concentration (MEC/PNEC) indicated a Hazard Quotient of >1.0 for acetaminophen, clarithromycin, fluoxetine, ibuprofen, sulfamethoxazole and triclocarban effects on aquatic organisms. A ratio greater than one estimates that the predicted environmental concentration would be above the no-effects concentration and is generally considered cause for concern (Cunningham et al, 2006). The Hazard Quotient was calculated using maximum concentrations from the DRBC survey and the most sensitive species and endpoints from a limited data set reported in the literature or predicted from an ECOSAR predictive model while using standard adjustment factors for acute and chronic toxicity data as described in the report entitled High Production Volume (HPV) Chemical Challenge Program Data Availability and Screening Level Assessment for Triclocarban CAS #: 101-20-2, Prepared for the HPV Challenge Program by The TCC Consortium on December 27, 2002 (http://www.epa.gov/hpv/pubs/summaries/tricloca/c14186tc.htm).

Using the same approach, Hazard Quotients of 0.1 to 1.0 were calculated for effects on aquatic organisms for carbamazepine, codeine, erythromycin-hydrate, gemfibrozil, lincomycin, and thiabendazole. A ratio greater than one tenth but less than one is considered a low risk but not an insignificant risk by some assessors because of the chemical specificity and potency of many PPCP (Ankley *et al*., 2006, Crane *et al*., 2007 European Medicines Agency. 2006). For a substance with acute ecotoxicity data only, the combined safety factor used in this assessment is 10,000 (1,000 from the calculation of the PNEC and 10 from the use of a 0.1 hazard quotient). This combined safety factor of 10,000 is equivalent to the safety factor recommended as a default aid to prioritization of human pharmaceuticals in the absence of identified specific environmental concerns.

This is a factor of 10 greater than the assessment factor applied to non-biologically active industrial chemicals (Crane *et al*., 2007). A summary of aquatic toxicology data (endpoints, organisms and sources), assessment factors, measured environmental concentrations, estimated PNEC and calculated hazard quotients used in this assessment are tabulated in Appendix C. Aquatic Toxicology Data.

If effects data was not available in the published literature, predictions from ecological structure activity relationship (ECOSAR) were used. Some pharmaceutical compounds detected in the DRBC survey did not have readily available aquatic toxicology data in the literature or ECOSAR predictions of aquatic effects (2-hydroxy-ibuprofen, dehydronifedipine, desmethyldiltiazem, fluticasone propionate and norverapamil). A number of compounds only had acute data readily available. PPCP would be better assessed for ecotoxicity if chronic toxicity, bioavailability, bioaccumulation and duration of exposure data were readily available (Jjemba, 2006).

It should be restated that this is a preliminary screening assessment of the data. Other approaches for assessment might use species sensitivity distribution to estimate PNECs , threshold concentration action levels or integration of river monitoring data in fate and transport models to estimate distribution and effects of contaminants of emerging concern. Assessment of ecotoxicity from contaminants of emerging concern in the tidal Delaware River would be further informed by estrogenicity screening, biomarker measurements and population (sex ratio) surveys.

5.1.2 Human Health Effects

Although the focus of this study was contaminants of emerging concern in surface water and aquatic toxicity, human health effects were considered in the prioritization. Two sites (E12 at RM105.4 and E16 at RM 131.1) in the survey are within segments of the river designated for use as public water supplies after reasonable treatment (Table3 and Figure 3). All sites in the survey are within segments of the river designated for fish ingestion. Numerous studies have concluded that healthy adults are unlikely to be adversely affected at the levels of exposure currently reported for PPCP (Cunningham et al., 2009; Kostich and Lazorchik, 2008; and Schwab et al., 2005). However, potential concerns have been identified for pregnant women, and children from pharmaceutical contaminants in potable water (Collier, 2007). Potential human health effects have also been incorporated in recent risk assessment and prioritization schemes with identified concerns including carcinogenicity, mutagenicity, reproductive and developmental effects, immunotoxicity, allergic reactions, microbial resistance to antibiotics and interactions among drugs when exposured to multiple contaminants (Bruce, *et al*., 2010; Kumar and Xagoraraki, 2010a, 2010b, and Ottmar *et al*., 2010) (Table 5). Human health risk assessment methodologies differ from ecological assessments and may identify other pharmaceuticals from those prioritized in this report that may need consideration (Kumar et al. 2010). It is also important to reiterate that the data presented in this report are from

surface water some of which is source water that will receive additional treatment prior to use as drinking water.

5.1.3 Priority PPCP

Based on the criteria used in this assessment of environmental occurrence, aquatic ecotoxicity, potential human health effects and analytical feasibility, the following PPCP should be considered priority pollutants for future monitoring and assessment in surface waters of the tidal Delaware River: acetaminophen, carbamazepine, clarithromycin, codeine, dehydronifedipine, erythromycin-hydrate, fluoxetine, gemfibrozil, 2-hydroxy-ibuprofen, ibuprofen, lincomycin, metformin, sulfamethoxazole, thiabendazole, and triclocarban (Table 4).

While hormones were detected at low concentrations and a few location in this study and the analytical feasibility of measuring hormones by EPA Method 1698 or other equivalent methods is limited to a few laboratories, hormones should be considered for future study in surface waters of the Delaware River because of their high potential for ecological effects (Kumar and Xagoraraki, 2010b)

In addition, PPCP such as diphenylhydramine, norfluoxetine, sertaline, desmethylsertraline, carbamazepine, diltiazem, fluoxetine, and gemfibrozil that have been detected in fish in EPA studies and by other researchers should be further assessed in the Delaware River (http://water.epa.gov/scitech/swguidance/ppcp/fish-tissue.cfm, Brooks et al., 2005).

Although the value of identifying priority pollutants is well established, because of the limited environmental assessment data available on contaminants of emerging concern, monitoring the environmental occurrence and effects of as many parameters as possible in future studies is recommended. It is also important to note that available information on the environmental effects of PPCP is rapidly increasing and any assessment should be updated periodically using current information.

Table 4. PPCP Detected in 2007, 2008 or 2009 Surveys. Most chemicals represented by a single measurement with multiple measurements represented by the mean (standard deviation).

	Concentration (ng/L) by Site / River Mile					
Compound	**E1 / 50**	**E4 / 68.1**	**E7 / 80**	**E9 / 90**	**E12 / 105.4**	**E16 / 131.1**
Acetaminophen*	ND	ND	ND	105	ND	ND
Albuterol	ND	0.40	0.83	0.84	0.56	0.34
Alprazolam*	0.42	0.46	0.61	0.58	0.38	ND
Amitriptyline*	0.49	1.01	1.17	1.39	0.83	0.76
Amphetamine*	ND	ND	3.83	5.53	4.52	ND
Atenolol*	13.80	20.20	53.80	58.80	28.60	11.60
Azithromycin	ND	ND	ND	ND	ND	9.53(6)
Benzoylecgonine*	6.92	16.20	30.50	39.60	10.60	6.04
Caffeine	18.25(8)	49.53(16)	106.57(15)	158.77(9)	71.50(3)	52.40(8)
Carbadox	ND	ND	7.48	5.89	2.20	ND
Carbamazepine	21.30(8)	47.20(16)	55.63(15)	42.60(9)	23.93(3)	18.50(8)
Clarithromycin	4.78*	ND	2.20(1)	8.38(5)	6.21(1)	4.24(2)
Cocaine*	0.97	0.34	1.21	1.05	2.33	2.58
Codeine	15.52(11)	89.97(62)	11.67(6)	8.96(5)	5.89(2)	3.40#
Cotinine	21.20~	20.00(2)	36.25(3)	36.75(2)	12.60(3)	7.66(2)
DEET*	28.30	32.90	42.80	40.80	17.50	6.34
Dehydronifedipine	0.87(0.3)	1.68(1)	2.19(1)	1.69(1)	0.82(0.2)	ND
Desmethyldiltiazem*	ND	1.31	1.96	3.35	1.38	0.96
Diltiazem	0.47(0.04)	2.57(1)	8.73(3)	10.71(2)	3.67(1)	3.08(1)
Dimethylxanthine	ND	220	193	239	ND	ND
Diphenhydramine	0.85(0.24)	0.89(0.2)	1.13(0.5)	3.38(1)	2.44(2)	4.93(2)
Enalapril*	ND	ND	0.34	ND	ND	ND
Erythromycin-H2O	1.98(1)	5.22(2)	9.15(5)	9.69(5)	4.11(3)	2.87(2)
Fluoxetine	8.02#	4.52(1)	ND	ND	ND	ND
Fluticasone propionate*	ND	ND	2.11	2.06	2.31	2.69
Gemfibrozil	5.35(2)	16.24(8)	30.90(12)	41.03(15)	15.44(10)	9.39(3)
Hydrocodone*	8.11	16.20	5.49	3.21	2.16	ND
10-hydroxy-amitriptyline*	ND	ND	0.25	0.38	0.23	0.21
2-Hydroxy-ibuprofen*	ND	113	346	286	ND	ND
Ibuprofen*	ND	ND	71.20	76.60	30.00	ND
Lincomycin	37.40`	18.40`	ND	ND	ND	ND
Meprobamate*	15.00	32.90	38.20	32.80	17.80	6.23
Metformin	ND	1036.50(1094)	2194.00(1974)	2355.00(1718)	861.00(157)	459.50(296)
Methylprednisolone	ND	0.80	ND	ND	ND	ND
Metoprolol*	11.50	23.70	35.40	42.40	21.20	15.10
Naproxen	ND	7.93(4)	16.34(6)	46.23(21)	31.37(12)	18.70(1)
Norfloxacin	ND	ND	ND	ND	ND	9.70
Norverapamil*	ND	ND	ND	ND	ND	0.17
Ofloxacin	ND	ND	ND	ND	ND	1.60
Oxycodone*	40.70	53.10	20.80	15.30	6.65	1.83
Propoxyphene*	1.02	1.92	2.15	2.96	1.18	1.51
Ranitidine	ND	ND	1.52	2.16	1.23	1.01
Sertraline	ND	ND	ND	1.16	0.64	0.69
Sulfadiazine	ND	2.91	2.36	ND	ND	ND
Sulfadimethoxine	1.64	0.49	0.81	1.42	ND	0.37
Sulfamethoxazole	39.23(18)	107.70(33)	116.93(31)	87.73(11)	37.13(17)	19.80(6)
Sulfanilamide	ND	ND	24.20	ND	ND	ND
Sulfathiazole	2.35	ND	ND	ND	ND	ND
Theophylline*	ND	ND	118.00	145.00	ND	ND

Compound	Concentration (ng/L) by Site / River Mile					
	E1 / 50	**E4 / 68.1**	**E7 / 80**	**E9 / 90**	**E12 / 105.4**	**E16 / 131.1**
Thiabendazole*	2.97	3.45	10.50	73.60	25.20	ND
Triamterene*	3.80	1.06	3.65	4.47	2.92	2.15
Triclocarban	ND	ND	ND	8.52(2)	4.74(1)	7.95(2)
Trimethoprim	4.73#	8.70(2)	16.03(3)	15.13(5)	6.04(2)	5.53(1)
Valsartan*	14.00	38.40	51.10	97.60	91.80	58.50
Verapamil*	ND	ND	ND	0.29	0.24	0.85

detected in 2007 only a mean and standard deviation cannot be calculated
` detected in 2008 only a mean and standard deviation cannot be calculated
~ detected in 2009 only a mean and standard deviation cannot be calculated
* analyzed in 2009 only a mean and standard deviation cannot be calculated
Priority PPCP for the tidal Delaware River are in bold.

Table 5. Prioritization Criteria for PPCP in Surface Waters of The Tidal Delaware River

Compound	Environmental Occurrence	Aquatic Ecotoxicity	Potential Human Health Effects	Analytical Feasibility EPA 1694
Acetaminophen	X	X	X	X
Carbamazepine	X	X	X	X
Clarithromycin	X	X		X
Codeine	X	X	X	X
Dehydronifedipine	X	NA	X	X
Erythromycin-hydrate	X	X	X	X
Fluoxetine	X	X	X	X
Gemfibrozil	X	X	X	X
2-Hydroxy-ibuprofen	X	NA		X
Ibuprofen	X	X	X	X
Lincomycin	X	X	X	X
Metformin	X			X
Sulfamethoxazole	X	X	X	X
Thiabendazole	X	X		X
Triclocarban	X	X		X

X = Data or information was available and used for the prioritization

5.2 Hormones and Sterols

In the 2007 and 2008 surveys, both sterols and hormones were included in the list of analytes. In those surveys, the fecal sterols (coprostanol, epicoprostanol, cholestanol) and a cholesterol precursor (desmosterol) as well as the plant sterols (campesterol, stigmasterol and beta-sitosterol) were detected (Table 6-7). The fecal sterols indicate the presence of human sewage but are not major contributors to ecotoxicity in the river.

In the 2009 survey only hormones were included in the list of analytes. Hormones detected in 2007, 2008 and 2009 at low concentrations and at limited locations include estrone, norethindrone, 17-α-ethynylestradiol, desogestrel and testosterone (Table 8). Concentration for environmental safety such as water quality criteria for aquatic life and human health are not available for hormones however; some information relevant to environmental assessment of these compounds is provided below.

Estrone (a natural hormone used in pharmaceuticals) and norethindrone (a synthetic hormone) were also detected in 2007 only at different sample sites and at concentrations of 1.3 and 4.24 ng/L, respectively (Appendix B, Tables B5 to B7). These values are lower than the median and maximum levels of 27 ng/L and 112 ng/L for estrone and 48 ng/L and 872 ng/L for norethindrone reported in the USGS national reconnaissance survey of streams (Koplin *et al*., 2002). In a study within the Delaware River Basin, Velicu and Suri (2009) report a estrone detection frequency of >90% in 21 surface water locations with concentrations ranging from 0.6 to 2.6 ng/L. Estrone is a steroid estrogen that is generally detected in the greatest quantity in aqueous samples partly because it is a transformation product of 17β-estradiol (Jurgens et al,, 2002). Dilution, sorption and biodegradation in surface waters quickly lowers the concentrations of estrone to the low ng/L levels but higher concentrations of estrone have been reported in sediment (Petrovic *et al*, 2002). Steroid estrogens have been linked to endocrine disruption in fish and their presence in water is attributed to incomplete removal from sewage during treatment (Hurst et al., 2001). Long-term(>60day) and short-term PNEC for use in risk assessment of aquatic organisms have recently been derived for estrone at 6 and 20 ng/L, respectively (Caldwell *et al*, 2012). Using the maximum measured concentration of estrone in the Delaware River, hazard quotients of 0.2 (long-term exposure) and 0.07 (short-term exposure) can be calculated. Since estrone was detected in 2007 only at one site, a short-term exposure to 1.3 ng/L estrone at the site seems to be indicated. Estrone has also been reported to have bioaccumulative properties (Gomes, *et al*, 2004). Neither fish tissue nor other aquatic biota were analyzed for hormones in the DRBC study.

Norethindrone (as reported as norethisterone or 19-nor-17-α-ethynyltestosterone) is a progestogen and a constituent of oral contraceptives that has been measured in streams (Koplin *et al*., 2002) and river sediment (López de Alda *et al*., 2002). Limited information is available on the ecotoxicity of norethindrone. It has been identified as a priority pharmaceutical for further study in Europe (The Environmental Side Effects of Medication. European Molecular Biology Organization (EMBO) Report by Alistair B.A. Boxall (http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1299201).

Norethindrone is also on the California Office of Environmental Health Hazard Assessment list of chemicals known to cause cancer and reproductive toxicity (http://www.oehha.org/prop65/prop65_list/files/P65single091208.pdf). Norethindrone levels reported in drinking water may are a potential concern for pregnant women (Collier, 2007)

The ovulation inhibiting hormone 17-α-ethynylestradiol was detected in 2008 at four sites with concentrations ranging from 1.97 to 4.01 ng/L. The hormone was not detected in 2009 and had interference in analytical detection in 2007 (Appendix B, Table B7). A 7-year, whole-lake experiment showed that chronic exposure of fathead minnow (*Pimephales promelas*) to low concentrations (5 to 6 ng/L) of 17-α-ethynylestradiol led to feminization of males, altered oogenesis in females, and near extirpation of the species from the lake (Kidd et al, 2007). Long-term (>60day) and short-term PNEC for use in risk assessment of aquatic organisms have also recently been derived for 17-α-ethynylestradiol at 0.1 and 0.5 ng/L, respectively (Caldwell *et al*, 2012). Using the maximum measured concentration of estrone in the Delaware River, hazard quotients between 20 to 40 (long-term exposure) and 0.2 to 8 (short-term exposure) can be calculated. In a recent publication, the hormone was also ranked in the top twenty pharmaceuticals, personal care products and endocrine-disrupting chemicals in U.S. surface waters for potential ecological effects (Kumar and Xagoraraki, 2010b). Ethinylestradiol has been identified as a pharmaceutical contaminant in potable water and is of potential concern for pregnant women (Collier, 2007).

In 2009, the ovulation inhibitor hormone desogestrel was measured at 242 and 68 ng/L at two sites and the sex hormone testosterone was measured at 1.37 ng/L at one site (Appendix B, Table B8). The MSDS for desogestrel states that the compound may be very toxic to aquatic life without providing specific ecological toxicity data. Concentrations of desogestrel measured in the Delaware River exceed some predicted acute toxicity values (48 to 336 ng/L LC50) and chronic toxicity values (17 to 237 ng/L ChV) in ECOSAR. Testosterone was ranked in the top ten pharmaceuticals, personal care products and endocrine-disrupting chemicals in U.S. surface waters for potential health effects (Kumar and Xagoraraki, 2010b). However, measured concentrations of testosterone in the Delaware River were orders of magnitude lower than ECOSAR predicted aquatic toxicity values (9 to 87 mg/L LC50) and chronic toxicity values (0.4 to 5 mg/L ChV) as well as observed LC50 at 6.2 mg/L and sublethal chronic toxicity endpoints at 0.31 to 2.48 mg/L in *Daphnia magna* (Barbosa, *et al*., 2008)

The EPA has listed the following hormones which may require regulation under the Safe Drinking Water Act (SDWA) on the Contaminant Candidate List 3 (CCL3) and/or Unregulated Contaminants Monitoring Rule 3 (UCMR3): 17- alpha estradiol, equilenin, equilin, 17-beta estradiol, estriol, estone, 17-alpha ethynylestradiol, mestranol, norethindron, testosterone and 4-androstene-3,17-dione.
(http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm)
(http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/methods.cfm)

Table 6. Sterol and Hormone Analytes in 2007 survey

	Maximum concentration (ng/L)	EDL (ng/l)
androsterone	ND	5.45
androstenedione	ND	6.17
equilenin	ND	0.853
estriol	ND	3.43
17-α-ethynylestradiol	ND	2.74
β-sitosterol	ND	12.8
stigmasterol	ND	9.59
ergosterol	ND	8.12
desmosterol	88.3	5.69
17-α-estradiol	ND	1.44
17-β-estradiol	ND	1.28
estrone	1.3	0.815
norgestrel	ND	13.2
norethindrone	4.24	2.67
equilin	ND	0.986
testosterone	ND	9
estradiol benzoate	ND	0.52
desogestrel	ND	6.08
campesterol	150.86	7.06
mestranol	ND	2.13
17 α-dihydroequlin	ND	2.89
stigmasterol	225	9.59
progesterone	ND	24.2

ND - not detected
EDL – estimated detection limit
17-α-ethinylestradiol was elevated by the presence of interference in 2007 and is not listed.
Hormones and sterols at each sample site are reported in Appendix B.

Table 7. Sterol and Hormone Analytes in 2008 survey

	Maximum concentration (ng/L)	EDL (ng/l)
Androsterone	ND	5.49
Desogestrel	ND	4.56
17-α-Estradiol	ND	1.17
Estrone	ND	1.35
Equilin	ND	1.5
Androstenedione	ND	11.3
17-α-Dihydroequilin	4.01	1.01
17β-Estradiol	ND	1.11
Testosterone	ND	12.4
Equilenin	ND	0.794
Mestranol	ND	1.35
Norethindrone	ND	2.24
17-α-Ethinylestradiol	4.01	2.66
Progesterone	ND	17.4
Norgestrel	ND	7.95
Estriol	ND	
β-Estradiol 3-benzoate	ND	0.384
Coprostanol	267	1.75
Epicoprostanol	18.1	2.29
Cholesterol	2120	6.64
Cholestanol	152	5.57
Desmosterol	1250	9.99
Ergosterol	11.4	5.12
Campesterol	546	6.85
Stigmasterol	643	24.4
β-Sitosterol	856	24.5
β-Stigmastanol	856	22.5

ND - not detected
EDL – estimated detection limit
Hormones and sterols at each sample site are reported in Appendix B.

Table 8. Hormone Analytes in 2009 survey

	Maximum concentration (ng/L)	EDL (ng/l)
17-α-Dihydroequilin	ND	4.01
Equilenin	ND	0.801
Equilin	ND	8.01
17-β-Estradiol	ND	4.01
17-α-Estradiol	ND	4.01
Estrone	ND	4.01
17-α-Ethinylestradiol	ND	5.01
Allyl Trenbolone	ND	0.801
Androstenedione	ND	2
Androsterone	ND	81.5
Desogestrel	242	120
Estriol	ND	16
Mestranol	ND	20
Norethindrone	ND	4.01
Norgestrel	ND	4.01
Progesterone	ND	0.801
Testosterone	1.37	0.801

ND - not detected
EDL – estimated detection limit
Hormones and sterols at each sample site are reported in Appendix B.

5.3 Perfluoroalkyl and polyfluoroalkyl substances

Perfluoroalkyl and polyfluoroalkyl substances (PFASs) are found in a variety of industrial and household products such as stain repellant textiles, fire-fighting foams, and paper coatings. PFASs have unique properties to repel both water and oil. They are a diverse group of compounds that have varying degrees of persistence, toxicity and bioaccumulation in the environment. Overall, PFASs with longer fluorinated carbon chains have greater potential to bioaccumulate especially compounds with greater than seven fluorinated carbons. Furthermore, perfluoroalkyl sulfonic acid and sulfonates (e.g., PFOS) are more bioaccumulative than perfluoroalkyl carboxylic acids (e.g., PFOA) with the same number of carbons. (Conder, *et al*, 2008).

Although national water quality criteria have not been derived for PFASs, benchmarks for PFOA and PFOS have been proposed by state agencies and researchers. Although some of the values discussed below are site-specific and have been developed for waters outside of the Delaware River Basin, they are used as relative benchmarks of environmental health and safety in order to prioritize additional studies. Surface water criteria to protect aquatic life, wildlife, and human health are the most appropriate benchmarks for the waters sampled in this survey. Nevertheless, proposed drinking water criteria are included in this summary, when available, with an acknowledgement that different methodologies are used to derive surface water aquatic life criteria and drinking water criteria. PFOA, PFOS, PFNA, PFHxS, PFHpA, and PFBS have been placed on the EPA contaminant candidate list 3 as contaminants known or anticipated to occur in public water systems and which may require regulation under the Safe Drinking Water Act (SDWA).
(http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm)
(http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/methods.cfm)

PFASs were detected at ng/L levels in the DRBC survey (Table 9 and Appendix B, Tables B8 to B10). All but two PFASs (perfluorododecanoic acid and perfluorooctane sulfonamides) were detected in this survey. PFASs were detected at every site sampled. Although PFASs are increasingly being detected in the environment, little information is available on the ecotoxicology of many PFASs. Additional information is needed especially on longer chain and sulfonated compounds. The following summary includes available information comparing PFASs concentrations in the Delaware River to concentrations found in other locations and to benchmarks developed for environmental health and safety.

Perfluorooctanoate (PFOA) has been described as a ubiquitous contaminant in surface waters and reported as the predominant PFAS in the Hudson River with a median concentration of 35 ng/ L and a range of 22 to 173 ng/L (Sinclair *et al*, 2006). Similar PFOA concentrations were measured in the Delaware River at 3.54 to 75.40 ng/L in the DRBC survey (Table 9 and Figure 4). At the reported concentrations, PFOA in the Delaware River did not exceed the USEPA Provisional Health Advisory short-term value for drinking water short-term exposure at 400 ng/L (USEPA, 2011). Neither did PFOA

concentrations, in areas of the Delaware River used as source waters for drinking water (upstream of RM 95), exceed a NJDEP preliminary health-based guidance value for chronic exposure of 40 ng/L for PFOA in drinking water. (Memorandum to Barker Hamill, Assistant Director for Water Supply Operations, http://www.nj.gov/dep/watersupply/pfoa_dwguidance.pdf)

Perfluorooctanesulfonate (PFOS) was detected in the Delaware River in the range of 2.7 to 8.42 ng/L (Table 9 and Figure 5). Levels described as background (0 to 30 ng/L PFOS) for surface waters of Georgia, Michigan, and New York (Sinclair *et al*, 2006, Konwick *et al*, 2008). The concentrations observed in the Delaware River are also well below PFOS concentration measured in the Conasauga River in Georgia (maximum level at 318.3 ng/L) (Konwick *et al*, 2008) and Lake Onondaga near Syracuse, New York (maximum level at 1,090 ng/L) (Sinclair *et al*, 2006). Nevertheless, the bioaccumulation properties of PFOS indicate the need for low concentrations in water to assure environmental safety. Concentrations of PFOS in the Delaware River did not exceed a USEPA Provisional Health Advisory of 200 ng/L for short-term exposure (USEPA, 2011), an aquatic life chronic benchmark of 5,100 ng/L or an avian wildlife value of 47 ng/L estimated by other authors (Giesy *et al*, 2010; Rostkowski *et al*., 2006) (Figure 5). In the Delaware Estuary, PFOS have been reported as a contaminant in osprey eggs (Toschik *et al*, 2005). Although PFOS concentrations are low in the river water sampled, bioaccumulation of PFOS with potential adverse human health effects from fish consumption and effects on wildlife populations make further studies warranted.

Perfluorononanoate (PFNA) at a maximum of 976 ng/L was the PFAS with the highest concentrations in the DRBC surveys (Table 9). Figure 6 shows the distribution of PFNA in the tidal river. The highest concentrations occur between RM 68.1 and 80. The concentrations found are higher than the 0 to 6 ng/L concentrations of PFNA found in streams of an industrial area in Korea (Rostkowski *et al.*, 2006) and levels measured in the Conasauga River (maximum level at 32.8 to 369 ng/L) near carpet manufacturing facilities in Georgia, USA (Konwick *et al*., 2008). Insufficient information is available to make a preliminary assessment of human health and ecotoxicology for PFNA. However, PFNA (nine fluorinated carbons) has been detected in wildlife indicating the potential for bioaccumulation and biomagnification as reported by Conder *et al*., (2008) substantiating the need for further study of PFN in the Delaware River.

Perfluorohexanesulfonate (PFHxS) was below the detection limit at three sites in this study and detected in the range of 2.97 to 4.48 ng/L at three other sites (Table 9 and Appendix B, Tables B8 to B10). The concentrations measured are similar to concentrations observed in numerous New York state waters (0.7 to 5.6 ng/l) and lower than most observations in Lake Onondaga, New York (4.2 to 8.5 ng/L) (Sinclair *et al*., 2006). Concentration for environmental safety such as water quality criteria for aquatic life and human health are not available for PFHS.

Perfluorohexanoate (PFHxA) was detected at all six sites in each of the three years of this study in the range of 1.4 to 79.80 ng/L (Table 9 and Appendix B, Tables B8 to B10). Higher concentrations were generally observed between RM 50 and 80. In Korean

streams with industrial activity, PFHxA are reported to be in the range of 0.77 to 27 ng/L (Rostkowski *et al.*, 2006). Concentration for environmental safety such as water quality criteria for aquatic life and human health are not available for PFHxA.

Perfluoroundecanoate (PFUnA) was detected at four downriver sites between RM 50 and 90 in concentrations ranging from 1.42 to 26 ng/L (Table 9 and Appendix B, Tables B8 to B10). PFUnA has been reported at concentrations lower than other PFASs such as PFOA in surface water (D'eon *et al*, 2009). However, PFUnA has also been detected in wildlife indicating possible biomagnification and bioaccumulation (Conder *et al*, 2008; Toschik *et al*, 2005). The eleven carbon chain PFUnA was the predominant PFAS observed in recent DRBC fish tissue samples. A comparison of PFUnA concentrations in water and fish tissue from the tidal Delaware River is shown in Figure 7.

Table 9. Perfluoroalkyl and polyfluoroalkyl substances in Ambient Water

	Maximum concentration			
	2007 (ng/L)	2008 (ng/L)	2009 (ng/l)	Limit of Quantification (ng/l)
Perfluorodecanoate (PFDA) C10	10	6	3	1.0
Perfluorododecanoate (PFDoA) C12	NR	NR	NR	1.0
Perfluoroheptanoate (PFHpA) C7	24	16	10	1.0
Perfluorohexanoate (PFHxA) C6	80	80	7	1.0
Perfluorononanoate (PFNA) C9	976	650	546	1.0
Perfluorooctanoate (PFOA) C8	75	48	28	1.0
Perfluoropentanoate (PFPeA) C5	35	32	14	1.0
Perfluoroundecanoate (PFUnA) C11	26	12	4	1.0
Perfluorobutanoate (PFBA) C4	19	26	8	1.0
Perfluorobutanesulfonate (PFBS) C4	3	26	8	2.0
Perfluorohexanesulfonate (PFHxS) C6	4	4	4	2.0
Perfluorooctanesulfonate (PFOS)C8	8	12	8	2.0
Perfluorooctane sulfonamide (PFOSA)	NR	NR	NR	

NR - not reported, below quantification limit
Perfluoroalkyl and polyfluoroalkyl substances at each sample site are reported in Appendix B, Table B.4.

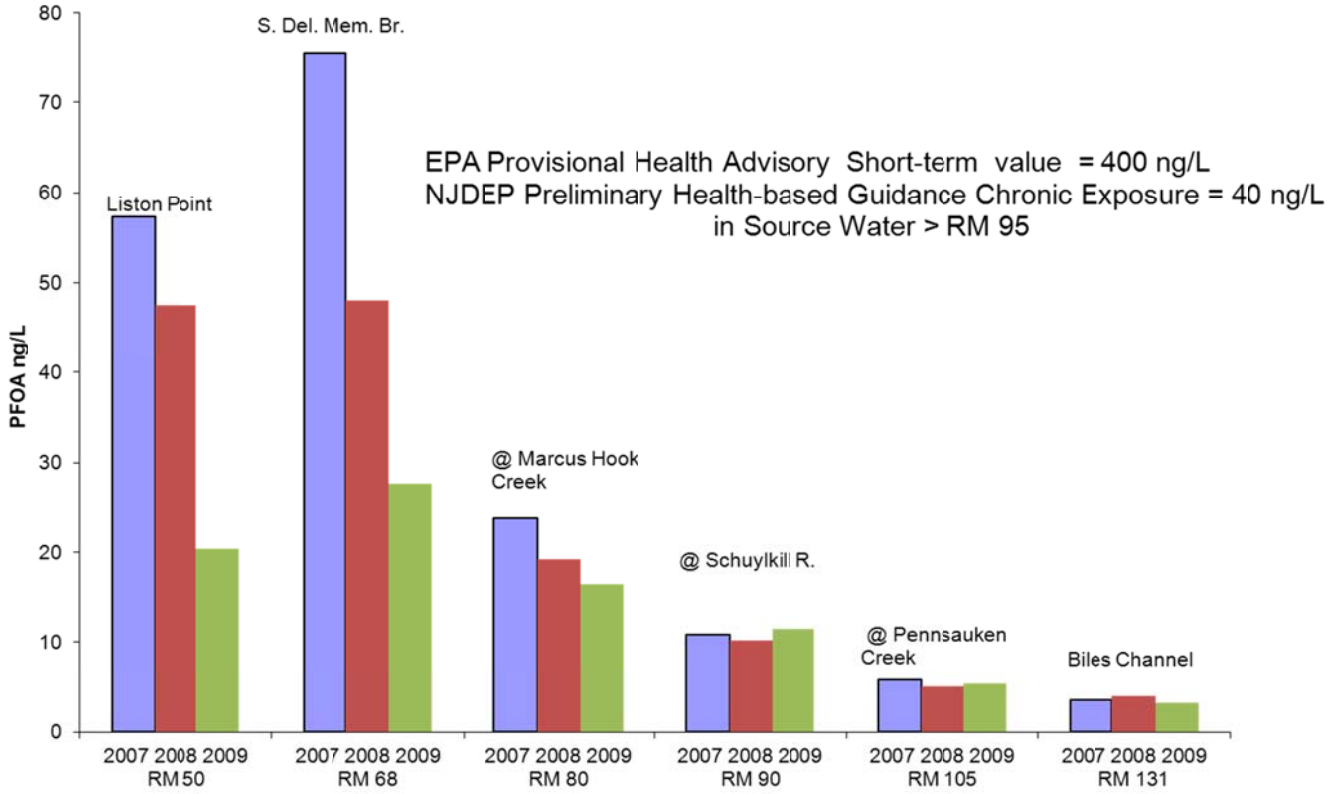

Figure 4. Perfluorooctanoic acid (PFOA) in ambient water

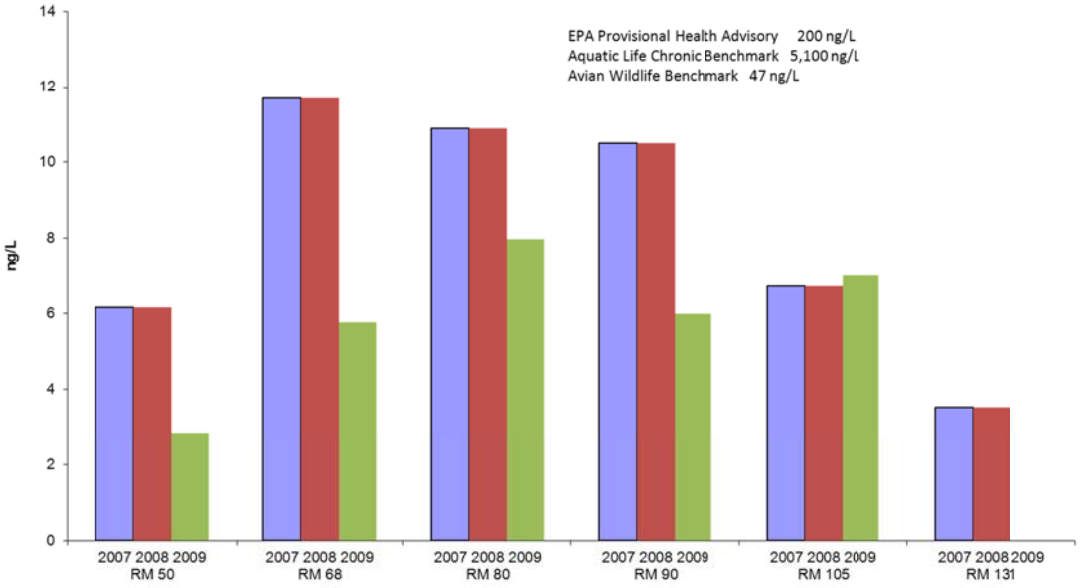

Figure 5. Perfluorooctanesulfonate (PFOS) in ambient water

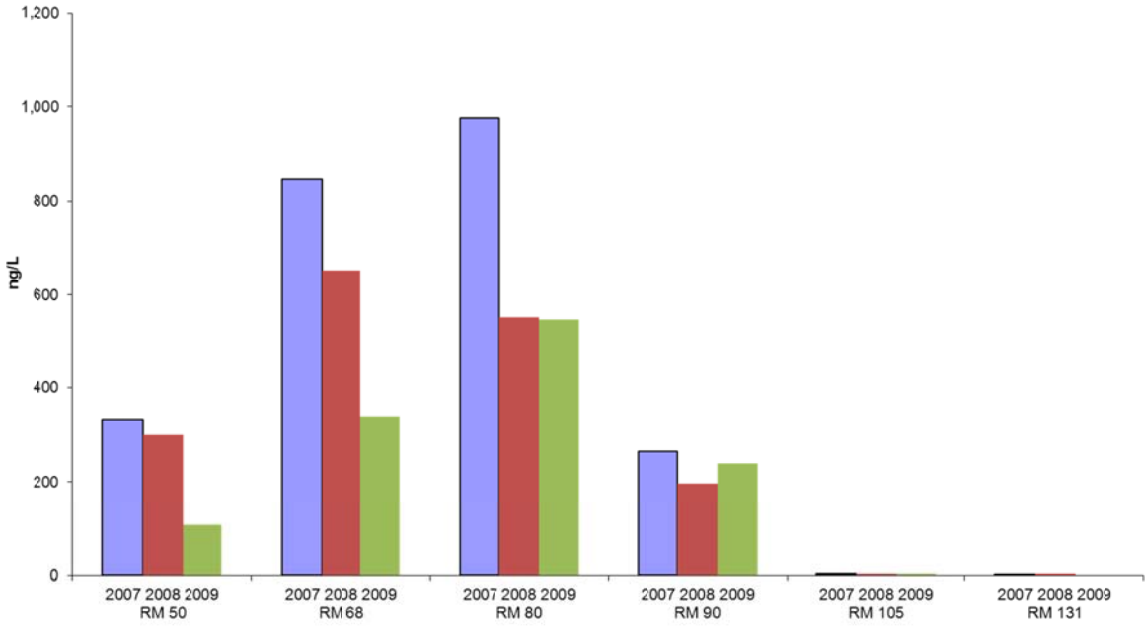

Figure 6. Perfluorononanoic acid (PFNA) in ambient water

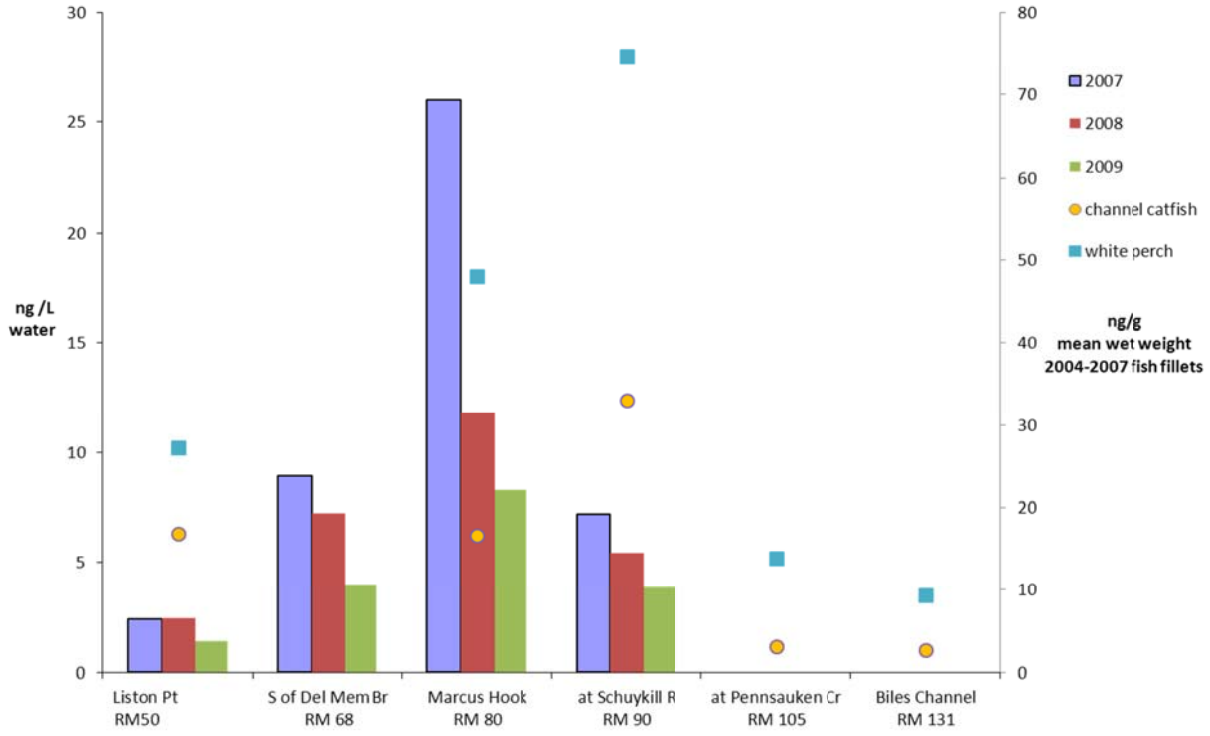

Figure 7. Perfluoroundecanoic acid (PFUdA) in water and fish tissue

5.4 Polybrominated Diphenyl Ethers

5.4.1 Ambient Water

Polybrominated diphenyl ethers (PBDE) are flame retardants found primarily in polymers and plastics. They are widely distributed in the environment and are present at increasing levels in people (U.S. Environmental Protection Agency. Polybrominated Diphenyl Ethers (PBDE) Project Plan, March 2006. http://www.epa.gov/oppt/pbde/pubs/proj-plan32906a.pdf). Flame retardants have been placed on the EPA Unregulated Contaminant Monitoring Rule 2 list to collect data for contaminants suspected to be present in drinking water, but that do not have health-based standards set under the Safe Drinking Water Act (SDWA). The flame retardants are 2,2',4,4'- tetrabromodiphenyl ether (BDE-47), 2,2',4,4',5-pentabromodiphenyl ether (BDE-99), 2,2',4,4',5,5'-hexabromodiphenyl ether (BDE-153) and 2,2',4,4',6-pentabromodiphenyl ether (BDE-100). (http://water.epa.gov/lawsregs/sdwa/ucmr/ucmr2/methods.cfm)

The analytical method used in this study measured forty-six individual PBDE congeners. However, in the interest of simplicity, the congeners are grouped by homologs (compounds with similar structures such as congeners with 5 bromine atoms are grouped together as pentabromodiphenyl ether homologs) (Appendix A, Table A2). In the DRBC ambient water study, the homologs with the maximum concentrations were decabromodiphenyl ethers (DeBDE) and nonabromodiphenyl ethers (NoBDE) (Table 10). The predominant homolog at three river sites was DeBDE detected in the range of 2,090 to 7,630 pg/L (Figure 8). In addition, nonabromodiphenyl ethers (NoBDE), pentabromodiphenyl ethers (PeBDE), and tetrabromodiphenyl ethers (TeBDE) were predominant at different sites detected in the range of 29 to 161 pg/L (Figure 8). Overall, total polybrominated diphenyl ethers (tPBDE) were detected at concentrations between 87 to 9,376 pg/L in ambeint water (Appendix B, Table B11). In unpublished reports, tPBDE have been measured in surface water at levels between 31 to 158 pg/L in Lake Michigan and at 6 pg/L in Lake Ontario (Canadian Environmental Protection Act, 1999 Ecological Screening Assessment Report on Polybrominated Diphenyl Ethers (PBDEs) (http://www.ec.gc.ca/CEPARegistry/documents/subs_list/PBDE_SAR/PBDEs_SAR_EC_June_2006_(en).pdf).

Comparisons among studies are difficult because it is unclear the methods used to measure the PBDE in the other studies and the number of congeners used to calculate the tPBDE. Nevertheless, the homolog distribution observed in the Delaware River is similar to those observed in other North American surface waters. DeBDE is the most prevalent commercial PBDE and is often found in sewage sludge, sediment and water. Any assessment of risk from PBDE should consider the fact that some BDE such as PeBDE and OcBDE have low potential for direct toxicity but can bioaccumulate. The environmental concern from other BDE such as DeBDE is primarily due to persistence and the potential for congeners to transform to bioaccumulative forms.

5.4.2 Fish Tissue

Environmental monitoring programs conducted worldwide during the past decade have shown increasing levels of some BDE congeners in contrast to a general decline in the occurrence of dioxins, PCBs and chlorinated pesticides. PBDEs have been observed in whole or fillet fish tissue at concentrations from non-detect to 1,300 ppb (ng/g) total PBDE wet weight (ww) in U.S. waterways (Wenning et al, 2011). DRBC monitoring in the tidal Delaware River from 2004 to 2007 found ranges for tPBDE of 13 to 168 ng/g ww and 562 to 5,046 ng/g lipid in channel catfish and white perch with BDE 47, 99 and 100 the most abundant congeners. Studies of other biota in the Delaware Estuary found tPBDE at 82 to 572 ng/g ww in osprey eggs and 10 to 5,652 ng/g lipid in American eels with BDE 47 the most abundant congener in both studies (Toschik *et al*., 2005; Ashley *et al*., 2007).

Risk from human consumption based on concentrations observed in fish from the Delaware River was assessed by establishing screening threshold values for four PBDE congeners (BDE-47, BDE-99, BDE-153 and BDE-209) that have oral reference doses listed in EPA-IRIS for non-carcinogenic effects. The fish tissue screening threshold values were established by following USEPA's "Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories – Volume 1, 2 and 3 (http://water.epa.gov/scitech/swguidance/fishshellfish/techguidance/guidance.cfm). The fish tissue screening values (FTSV) are 400 ppb for PBDE-47, PBDE-99, and PBDE-153 and 28,000 ppb for PBDE-209. None of the white perch or channel catfish tissue samples collected in the main stem Delaware River in 2004 to 2007 exceeded these screening values. Maximum concentrations in tidal Delaware River fish tissue were 80 ppb for PBDE-47, 53 ppb for PBDE-99, 8 ppb for PBDE-153 and 1 ppb for PBDE-209.

While total PBDE in Delaware Estuary fish has been reported as greater on average than in fish sampled at other U.S. and international locations, non-cancer risk as a function of fish consumption was reported as relatively low. (Greene, R. 2007. http://www.epa.epa/waterscience/fish/forum/2007/)

FTSVs for carcinogenic effects are not available for PBDE. Although BDE-209 has suggestive evidence of carcinogenic potential, an oral slope factor is not listed in IRS. There are insufficient data currently available to determine if BDE-47, BDE-99, and BDE-153 are potential carcinogens. The DRBC plans to continue to monitor PBDE in fish tissue if adequate funding is available.

Table 10. Polybrominated Diphenyl Ether Homologs in 2007 Ambient Water Survey

	Maximum concentration (pg/L)	Detection limit (ng/l)
Dibromodiphenyl ethers DiBDE	4.06	10
Tribromodiphenyl ethers TriBDE	22.75	10
Tetrabromodiphenyl ethers TeBDE	237.62	10
Pentabromodiphenyl ethers PeBDE	216.02	10
Hexabromodiphenyl ethers HxBDE	50.21	10
Heptabromodiphenyl ethers HpBDE	10.75	20
Octabromodiphenyl ethers OcBDE	44.7	20
Nonabromodiphenyl ethers NoBDE	1,161	100
Decabromodiphenyl ethers DeBDE	7,630	200

PBDE homologs at each sample site are reported in Appendix B.
n=6

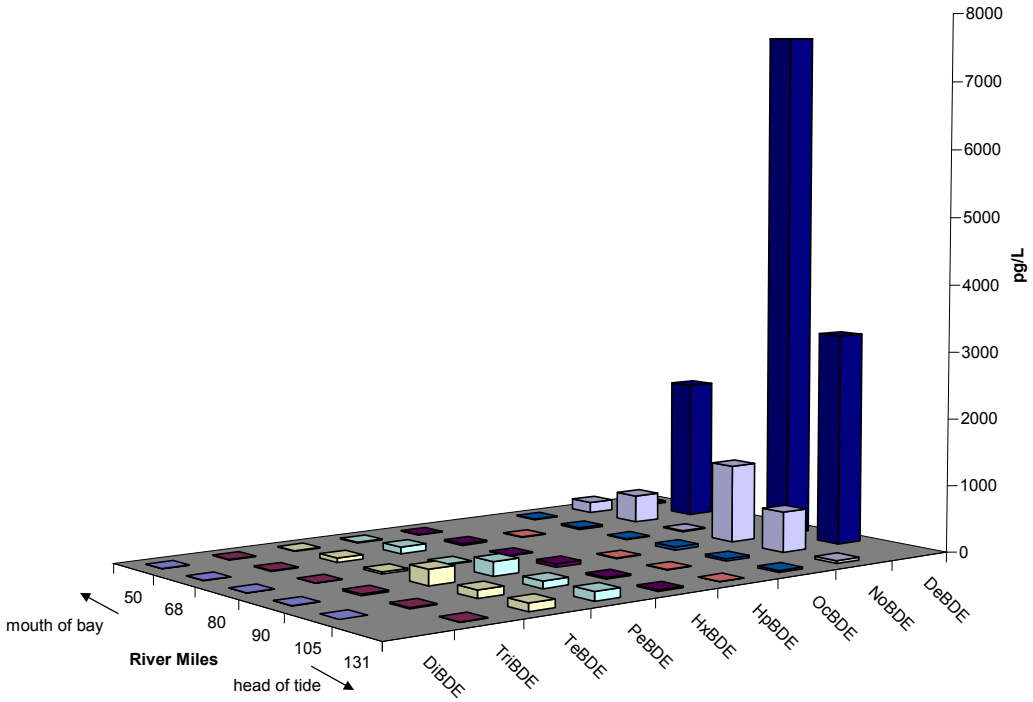

Figure 8. PBDE in ambient waters of the tidal Delaware River

5.5 Nonylphenol and Nonylphenol ethoxylates

Nonylphenol ethoxylates (NPEO) are surfactants used in detergents and other industrial applications. Nonylphenols (NP) are often found in the environment as microbial decay products of NPE. In general, NP are considered more toxic than NPEOs (Vazquez-Duhalt *et al.,* 2005). Although NP are not unregulated, our understanding of their toxicity is still emerging especially in the area of estrogenic effects. In 2006, the USEPA propagated aquatic life criteria for NP (Aquatic Life Ambient Water Quality Criteria Nonylphenol Final, EPA-822-R-05-005, http://www.epa.gov/waterscience/criteria/nonylphenol/final-doc.pdf). NP levels in the tidal Delaware River in the time and space of this limited study did not exceed USEPA criteria of 1.7 µg/L marine Criterion Continuous Concentration (CCC) or 6.6 µg/L freshwater CCC (Table 11 and Figure 9).

In interpreting the concentrations of NP in the environment with regard to the criteria, it should be noted that since studies in the literature that measured estrogenic effects by NP did not meet data quality for deriving criteria, they were not included in the calculation of the USEPA criteria for NP. However, chronic toxicity data used in the derivation of the criteria did include growth and reproduction endpoints. Therefore, to the extent that these chronic toxicity endpoints include the effect of endocrine disruption, the estrogenicity of NP is included in the derivation of the criteria. In short, upon development of standardized tests for estrogenicity, the criteria will certainly be revised.

The concentrations of NP measured in the tidal Delaware River, with a maximum of 0.0876 µg/l, are well below those considered protective (Table 11 and Appendix B, Table B12). Figure 12 shows the distribution of NP in the tidal river. Water concentrations of NP have been reported as high as 644 µg/L in Spanish waters (Sole *et al*., 2000). Maximum concentrations have been reported in the UK at 53 µg/L (Blackburn and Waldock, 1995) and in the Hudson River Estuary at 95 µg/l (Dachs *et a*l., 1999). Published studies including those that measured estrogenicity indicate that surface waters containing < 1 µg/l of NP are at low risk, surface waters containing between 1 to 10 µg/l are at some risk and surface water at >10 µg/l are at a significant risk of environmental harm (Vazquez-Duhalt *et al*., 2005). It should also be noted that the lipohilic NP can bioaccumulate (Vazquez-Duhalt *et al*., 2005). Because of its widespread occurrence in the environment and the evolving knowledge of its ecotoxicity, NP should continue to be characterized as a contaminant of emerging concern in DRBC studies.

Table 11. Nonylphenol and Nonylphenol Ethoxylates in 2007 Survey

	Maximum concentration µg/l / ng/l	Detection limit ng/l
4-nonylphenol	0.0876 / 87.6	10
4-nonylphenol monoethoxylate	0.0398 / 39.8	50
4-nonylphenol diethoxylate	ND	50

ND - not detected

NP and NPEOs at each sample site are reported in Appendix B.

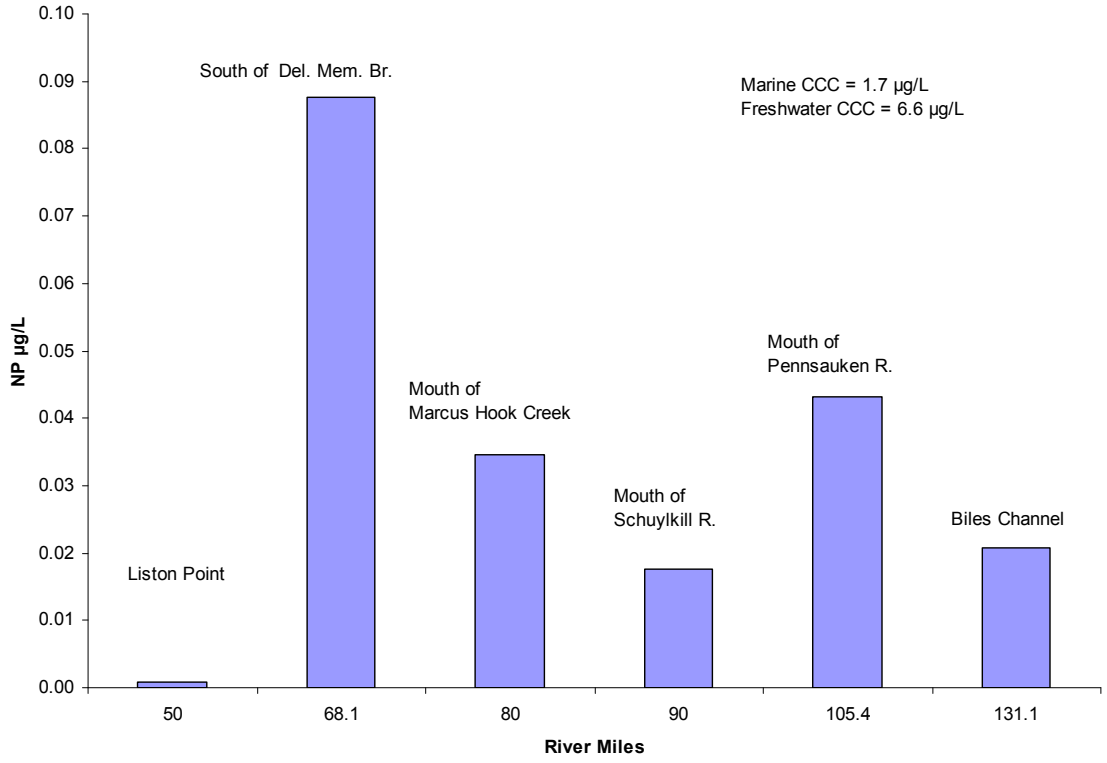

Figure 9. Nonylphenol (NP) in ambient waters of the tidal Delaware River

5.6 Bisphenol A

Bisphenol A was not detected, at a detection limit of 0.05 ng/L, in ambient waters of the tidal Delaware River at any of the six sites sampled in 2008, the only year the parameter was included in the survey.

6.0 Conclusions

Pharmaceuticals and personal care products (PPCP) detected at concentrations of ng/L in the Delaware River were comparable to compounds and concentrations measured in other studies of ambient water in urban areas with the exception of codeine and metformin. Fifteen PPCP were identified for future focused study in surface waters of the tidal Delaware River based on the criteria of environmental occurrence, aquatic ecotoxicity, potential human health effects to sensitive populations, and analytical feasibility. The effects of PPCP in estuarine and coastal waters are not well studied. Future work should evaluate the sources as well as the fate and effects of PPCP in the Delaware River water column, sediments and biota.

Natural and synthetic hormones were detected in ng/L levels in the main stem Delaware River. They have been ranked in the top chemicals in U.S. surface waters for potential ecological effects warranting further study in the Delaware River Basin.

Perfluoroalkyl and polyfluoroalkyl substances (PFASs) were measured in ng/L concentrations in water and fish tissue. The predominant PFAS is perfluorononanoic acid (PFNA) in surface water and perfluoroundecanoic acid (PFUdA) in fish tissue. Although concentrations of PFASs in water appear to be trending downward each year in the Delaware River, additional ecotoxicology and bioaccumulation information is needed for these compounds especially on longer chain and sulfonated PFASs.

Nonylphenol (NP) concentrations in the Delaware River did not exceed current USEPA national water quality criteria. However, because of widespread occurrence in the environment and the evolving knowledge of ecotoxicity, NP and NPEOs should continue to be characterized as a contaminant of emerging concern in Delaware River Basin studies.

Polybrominated diphenyl ethers (PBDE) were measured in pg/L to ng/L concentrations with homolog distributions in the tidal Delaware River similar to those observed in other North American locations. Because of the low levels found in water, future monitoring of PBDE in the Delaware River Basin should focus on bioaccumulation in fish tissue and other biota.

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Appendix A: PPCP and PBDE Analytes

Table A1. PPCP Analytes and Estimated Detection Limits (EDL)

Compound	EDL	UNITS	Compound	EDL	UNITS
Metformin	61.1	NG/L	Albuterol	0.308	NG/L
2-Hydroxy-ibuprofen	90.2	NG/L	Verapamil	0.149	NG/L
Caffeine	14.7	NG/L	Alprazolam	0.298	NG/L
Theophylline	58.7	NG/L	10-hydroxy-amitriptyline	0.147	NG/L
Acetaminophen	58.7	NG/L	Enalapril	0.308	NG/L
Valsartan	3.92	NG/L	Norverapamil	0.149	NG/L
Sulfamethoxazole	0.915	NG/L	Bisphenol A	2460	NG/L
Ibuprofen	14.7	NG/L	Furosemide	49.6	NG/L
Naproxen	2.94	NG/L	Glipizide	5.9	NG/L
Atenolol	1.82	NG/L	Glyburide	2.95	NG/L
Gemfibrozil	1.51	NG/L	Hydrochlorothiazide	19.7	NG/L
Oxycodone	1.08	NG/L	Triclosan	59	NG/L
DEET	0.497	NG/L	Warfarin	1.47	NG/L
Metoprolol	2.09	NG/L	Carbadox	1.47	NG/L
Benzoylecgonine	0.294	NG/L	Cefotaxime	16.6	NG/L
Carbamazepine	1.49	NG/L	Ciprofloxacin	5.9	NG/L
Meprobamate	3.97	NG/L	Clinafloxacin	13.7	NG/L
Codeine	3.71	NG/L	Cloxacillin	1.18	NG/L
Cotinine	1.54	NG/L	Digoxin	14.7	NG/L
Trimethoprim	3	NG/L	Digoxigenin	18.6	NG/L
Hydrocodone	1.85	NG/L	Enrofloxacin	2.95	NG/L
Azithromycin	1.49	NG/L	Flumequine	1.47	NG/L
Methylprednisolone	6.24	NG/L	Fluoxetine	1.47	NG/L
Clarithromycin	1.47	NG/L	Lincomycin	6.88	NG/L
Triclocarban	2.94	NG/L	Lomefloxacin	2.95	NG/L
Diltiazem	0.294	NG/L	Miconazole	1.47	NG/L
Erythromycin-H2O	0.294	NG/L	Norfloxacin	14.7	NG/L
Diphenhydramine	0.597	NG/L	Norgestimate	2.95	NG/L
Amphetamine	1.49	NG/L	Ofloxacin	14.7	NG/L
Triamterene	0.299	NG/L	Ormetoprim	0.59	NG/L
Thiabendazole	1.49	NG/L	Oxacillin	2.95	NG/L
Desmethyldiltiazem	0.147	NG/L	Oxolinic Acid	0.59	NG/L
Ranitidine	0.768	NG/L	Penicillin G	1.18	NG/L
Propoxyphene	0.294	NG/L	Penicillin V	2.95	NG/L
Fluticasone propionate	1.99	NG/L	Roxithromycin	0.295	NG/L
Cocaine	0.147	NG/L	Sarafloxacin	31.3	NG/L
Sulfadimethoxine	0.294	NG/L	Sulfachloropyridazine	1.47	NG/L
Dehydronifedipine	0.596	NG/L	Sulfadiazine	1.47	NG/L
Amitriptyline	0.294	NG/L	Sulfamerazine	0.769	NG/L
Sertraline	0.392	NG/L	Sulfamethazine	0.59	NG/L

Table A1 (cont.). PPCP Analytes and Estimated Detection Limit (EDL).

Compound	EDL	UNITS	Compound	EDL	UNITS
Sulfamethizole	0.693	NG/L	Trenbolone acetate	0.295	NG/L
Sulfanilamide	14.7	NG/L	Anhydrochlortetracycline (ACTC)	60.9	NG/L
Sulfathiazole	1.47	NG/L	Anhydrotetracycline (ATC)	14.7	NG/L
Tylosin	5.9	NG/L	Chlortetracycline (CTC)	5.9	NG/L
Virginiamycin	9.82	NG/L	Demeclocycline	14.7	NG/L
1,7-Dimethylxanthine	147	NG/L	Doxycycline	5.9	NG/L
Amlodipine	1.47	NG/L	4-Epianhydrochlortetracycline (EACTC)	77.6	NG/L
Benztropine	0.295	NG/L	4-Epianhydrotetracycline (EATC)	21.9	NG/L
Betamethasone	1.47	NG/L	4-Epichlortetracycline (ECTC)	14.7	NG/L
Diazepam	0.295	NG/L	4-Epioxytetracycline (EOTC)	5.9	NG/L
Fluocinonide	5.9	NG/L	4-Epitetracycline (ETC)	8.75	NG/L
Hydrocortisone	59	NG/L	Isochlortetracycline (ICTC)	5.9	NG/L
Norfluoxetine	1.47	NG/L	Minocycline	151	NG/L
Paroxetine	3.93	NG/L	Oxytetracyclin (OTC)	5.9	NG/L
Prednisolone	5.9	NG/L	Tetracycline (TC)	5.9	NG/L
Prednisone	19.7	NG/L	Atorvastatin	24.1	NG/L
Promethazine	0.393	NG/L	Cimetidine	2.89	NG/L
Propranolol	1.97	NG/L	Clonidine	24.1	NG/L
Simvastatin	19.7	NG/L			
Trenbolone	3.93	NG/L			

Table A2. PBDE Analytes in 2007 Survey and Their Detection Limits

Polybrominated Diphenyl Ethers EPA Method 1614 (MLA-033)		Detection limit (pg/l)
2,4-DiBDE	BDE - 7	10
2,4'-DiBDE	BDE - 8	10
2,6-DiBDE	BDE - 10	10
3,3'-DiBDE	BDE - 11	10
3,4-DiBDE	BDE - 12	10
3,4'-DiBDE	BDE - 13	10
4,4'-DiBDE	BDE - 15	10
2,2',4-TriBDE	BDE - 17	10
2,3',4-TriBDE	BDE - 25	10
2,4,4'-TriBDE	BDE - 28	10
2,4,6-TriBDE	BDE - 30	10
2,4',6-TriBDE	BDE - 32	10
2',3,4-TriBDE	BDE - 33	10
3,3',4-TriBDE	BDE - 35	10
3,4,4'-TriBDE	BDE - 37	10
2,2',4,4'-TeBDE	BDE - 47	10
2,2',4,5'-TeBDE	BDE - 49	10
2,2',4,6'-TeBDE	BDE - 51	10
2,3',4,4'-TeBDE	BDE - 66	10
2,3',4',6-TeBDE	BDE - 71	10
2,4,4',6-TeBDE	BDE- 75	10
3,3',4,4'-TeBDE	BDE- 77	10
3,3',4,5'-TeBDE	BDE - 79	10
2,2',3,4,4'-PeBDE	BDE - 85	10
2,2',4,4',5-PeBDE	BDE - 99	10
2,2',4,4',6-PeBDE	BDE - 100	10
2,3,3',4,4'-PeBDE	BDE - 105	10
2,3,4,5,6-PeBDE	BDE - 116	10
2,3',4,4',6-PeBDE	BDE - 119	10
2,3',4,5,5'-PeBDE	BDE - 120	10
3,3',4,4',5-PeBDE	BDE - 126	10
2,2',3,3',4,4'-HxBDE	BDE - 128	10
2,2',3,4,4',5'-HxBDE	BDE - 138	10
2,2',3,4,4',6'-HxBDE	BDE - 140	10
2,2',4,4',5,5'-HxBDE	BDE - 153	10
2,2',4,4',5,6'-HxBDE	BDE - 154	10
2,2',4,4',6,6'-HxBDE	BDE - 155	10

Polybrominated Diphenyl Ethers EPA Method 1614 (MLA-033)		Detection limit (pg/l)
2,3,4,4',5,6-HxBDE	BDE - 166	10
2,2',3,4,4',5,6-HpBDE	BDE - 181	20
2,2',3,4,4',5',6-HpBDE	BDE - 183	20
2,3,3',4,4',5,6-HpBDE	BDE - 190	20
2,2',3,4,4',5,5',6-OcBDE	BDE - 203	20
2,2',3,3',4,4',5,5',6-NoBDE	BDE - 206	100
2,2',3,3',4,4',5,6,6'-NoBDE	BDE - 207	100
2,2',3,3',4,5,5',6,6'-NoBDE	BDE - 208	100
2,2',3,3',4,4',5,5',6,6'-DeBDE	BDE - 209	200

Appendix B: Data Tables

Table B1. PPCP in ambient water - 2007 (ng/L)

RM	Azithromycin	Caffeine	Carbamazepine	Clarithromycin	Codeine	Dehydronifedipine	Diltiazem	Diphenhydramine	Erythromycin-H_2O	Fluoxetine
50	ND	12.5	18.8	ND	16	1.1	0.502	1.11	2.68	8.02
68.1	ND	57.2	44.9	ND	159	2.59	3.17	1.02	7.06	3.63
80	ND	24.6	60.4	2.86	14.9	2.97	12.3	1.64	14.4	ND
90	ND	72.2	37.5	10.9	11.4	2.27	12.8	4.56	14.9	ND
105.4	ND	10.4	24.7	5.45	4.52	0.941	4.1	2.21	6.68	ND
131.1	4.96	36.5	11.8	5.18	3.4	ND	2.79	3.47	3.95	ND

RM	Norfloxacin	Ofloxacin	Sulfadiazine	Sulfadimethoxine	Sulfamethoxazole	Sulfanilamide	Trimethoprim	Gemfibrozil	Ibuprofen	Naproxen	Triclocarban
50	ND	ND	ND	1.64	49.8	ND	4.73	6.4	ND	ND	ND
68.1	ND	ND	2.91	ND	124	ND	11.1	22.5	15.9	11.1	ND
80	ND	ND	2.36	ND	150	24.2	16.6	30.5	ND	17.6	ND
90	ND	ND	ND	ND	99.9	ND	11.3	44.3	ND	45	6.34
105.4	ND	ND	ND	ND	37.8	ND	4.36	14.5	ND	27.9	4.36
131.1	9.7	1.6	ND	ND	12.7	ND	ND	6.36	ND	18	6.54

Table B.2. PPCP in ambient water - 2008 (ng/L)

RM	Azithromycin	Caffeine	Carbamazepine	Clarithromycin	Codeine	Dehydronifedipine	Diltiazem	Diphenhydramine	Erythromycin-H_2O	Fluoxetine
50	ND	ND	30.5	ND	4.65	0.637	0.422	0.766	1.19	ND
68.1	ND	44.1	64.5	ND	72.7	1.74	2.26	ND	4.36	5.4
80	ND	117.1	67.3	ND	4.21	2.19	7.01	0.69	6.23	ND
90	ND	166.1	52.9	2.65	3.29	1.61	9.83	1.91	4.94	ND
105.4	ND	86.1	20.2	ND	ND	ND	2.39	1.01	0.61	ND
131.1	ND	74	27.6	1.61	ND	ND	2.68	3.64	0.97	ND

RM	Norfloxacin	Ofloxacin	Sulfadiazine	Sulfadimethoxine	Sulfamethoxazole	Sulfanilamide	Trimethoprim	Gemfibrozil	Ibuprofen	Naproxen	Triclocarban
50	ND	ND	ND	ND	18.8	ND	ND	3.13	ND	ND	ND
68.1	ND	ND	ND	0.485	129	ND	7.29	7.32	ND	ND	ND
80	ND	ND	ND	ND	112	ND	13.1	18.7	ND	9.52	ND
90	ND	ND	ND	ND	80.5	ND	13.8	24.9	ND	26.2	8.53
105.4	ND	ND	ND	ND	20.1	ND	ND	6.31	ND	21.8	3.7
131.1	ND	ND	ND	0.37	24.5	ND	6.27	10.1	ND	18	6.8

RM	Sulfathiazole	Lincomycin	Albuterol	Metformin	Ranitidine	Dimethylxanthine	Cotinine	Carbadox	Thiabendazole
50	2.35	37.4	ND	ND	ND	ND	ND	ND	2.97
68.1	ND	18.4	ND	1810	ND	220	21.2	ND	3.45
80	ND	ND	0.668	3590	0.922	193	34.3	7.48	10.5
90	ND	ND	0.711	3570	1.14	239	35.6	5.89	73.6
105.4	ND	ND	ND	972	ND	ND	10.4	2.2	25.2
131.1	ND	ND	ND	669	0.754	ND	8.99	ND	ND

Table B.3. PPCP in ambient water - 2009 (ng/L)

RM	Azithromycin	Caffeine	Carbamazepine	Clarithromycin	Codeine	Dehydronifedipine	Diltiazem	Diphenhydramine	Erythromycin-H_2O	Fluoxetine
50	ND	24	14.6	4.78	25.9	ND	0.492	0.66	2.07	ND
68.1	ND	47.3	32.2	ND	38.2	0.698	2.28	0.75	4.25	ND
80	ND	178	39.2	1.53	15.9	1.41	6.87	1.07	6.82	ND
90	ND	238	37.4	11.6	12.2	1.19	9.51	3.66	9.24	ND
105.4	ND	118	26.9	6.96	7.25	0.698	4.52	4.11	5.04	ND
131.1	14.1	46.7	16.1	5.93	ND	ND	3.76	7.68	3.7	ND

RM	Norfloxacin	Ofloxacin	Sulfadiazine	Sulfadimethoxine	Sulfamethoxazole	Sulfanilamide	Trimethoprim	Gemfibrozil	Ibuprofen	Naproxen	Triclocarban
50	ND	ND	ND	ND	49.1	ND	ND	6.53	ND	ND	ND
68.1	ND	ND	ND	ND	70.1	ND	7.72	18.9	ND	4.75	ND
80	ND	ND	ND	0.809	88.8	ND	18.4	43.5	71.2	21.9	ND
90	ND	ND	ND	1.42	82.8	ND	20.3	53.9	76.6	67.5	10.7
105.4	ND	ND	ND	ND	53.5	ND	7.71	25.5	30	44.4	6.17
131.1	ND	ND	ND	ND	22.2	ND	4.78	11.7	ND	20.1	10.5

RM	Sulfathiazole	Lincomycin	Albuterol	Metformin	Ranitidine	Dimethylxanthine	Cotinine	Carbadox	Thiabendazole
50	ND	ND	ND	0	ND	ND	21.2	ND	ND
68.1	ND	ND	0.399	263	ND	ND	18.8	ND	ND
80	ND	ND	0.996	798	2.11	ND	38.2	ND	ND
90	ND	ND	0.968	1140	3.18	ND	37.9	ND	ND
105.4	ND	ND	0.555	750	1.23	ND	14.8	ND	ND
131.1	ND	ND	0.344	250	1.27	ND	6.33	ND	ND

RM	Valsartan	Atenolol	Oxycodone	Benzoylecgonine	Meprobamate	Metoprolol	Hydrocodone	Amphetamine	Triamterene	Cocaine	Propoxyphene
50	14	13.8	40.7	6.92	15	11.5	8.11	ND	3.8	0.965	1.02
68.1	38.4	20.2	53.1	16.2	32.9	23.7	16.2	ND	1.06	0.339	1.92
80	51.1	53.8	20.8	30.5	38.2	35.4	5.49	3.83	3.65	1.21	2.15
90	97.6	58.8	15.3	39.6	32.8	42.4	3.21	5.53	4.47	1.05	2.96
105.4	91.8	28.6	6.65	10.6	17.8	21.2	2.16	4.52	2.92	2.33	1.18
131.1	58.5	11.6	1.83	6.04	6.23	15.1	ND	ND	2.15	2.58	1.51

Table B3. continued PPCP in ambient water - 2009 (ng/L)

RM	Amitriptyline	Sertraline	Alprazolam	Enalapril	Methylprednisolone	Norverapamil	Verapamil	Acetaminophen	Fluticasone propionate	Ibuprofen
50	0.491	ND	0.422	ND	ND	ND	ND	ND	ND	ND
68.1	1.01	ND	0.456	ND	0.8	ND	ND	ND	ND	ND
80	1.17	ND	0.612	0.343	ND	ND	ND	ND	2.11	71.2
90	1.39	1.16	0.583	ND	ND	ND	0.287	105	2.06	76.6
105.4	0.834	0.636	0.375	ND	ND	ND	0.238	ND	2.31	30
131.1	0.759	0.692	ND	ND	ND	0.172	0.852	ND	2.69	ND

RM	Theophylline	2-Hydroxy-ibuprofen	Desmethyldiltiazem	10-hydroxy-amitriptyline	DEET	Thiabendazole
50	ND	ND	ND	ND	28.3	2.47
68.1	ND	113	1.31	ND	32.9	3.66
80	118	346	1.96	0.245	42.8	3.27
90	145	286	3.35	0.382	40.8	2.63
105.4	ND	ND	1.38	0.227	17.5	1.5
131.1	ND	ND	0.961	0.214	6.34	1.78

Table B.4. PPCP 2007 to 2009 mean ng/L

RM	Azithromycin	Caffeine	Carbamazepine	Clarithromycin	Codeine	Dehydronifedipine	Diltiazem	Diphenhydramine	Erythromycin-H_2O	Fluoxetine
50	ND	18	21	5	16	1	0	1	2	8
68.1	ND	50	47	ND	90	2	3	1	5	5
80	ND	107	56	2	12	2	9	1	9	ND
90	ND	159	43	8	9	2	11	3	10	ND
105.4	ND	72	24	6	6	1	4	2	4	ND
131.1	10	52	19	4	3	ND	3	5	3	ND

Table B5. Sterols and Hormones in ambient water - 2007 (ng/L)

RM	Estrone	Norethindrone	Coprostanol	Epicoprostanol	Cholesterol	Cholestanol	Desmosterol	Campesterol	Stigmasterol	beta-Sitosterol
50	ND	4.24	6.84	2.47	390	47.46	52.5	100.86	87	312
68.1	ND	ND	57.9	15	595	137.16	88.3	150.86	139	286
80	ND	ND	90	16.7	761	143.16	60.1	145.86	225	294
90	ND	ND	82.8	10.9	309	64.66	27.7	53.56	160	293
105.4	ND	ND	87.6	11.4	256	50.96	34.3	66.36	195	415
131.1	1.3	ND	198	15.3	643	67.16	49.6	73.16	198	534

ND= not detected

17α-ethinyl estradiol concentrations in 2007 were elevated by the presence of interference and are not reported.

Table B6. Sterols and Hormones in ambient water - 2008 (ng/L)

RM	Estrone	Norethindrone	Coprostanol	Epicoprostanol	Cholesterol	Cholestanol	Desmosterol	Campesterol	Stigmasterol
50	ND	ND	10.1	ND	766	55.5	94.7	226	86.4
68.1	ND	ND	22.5	6.35	904	110	105	350	175
80	ND	ND	50.9	12.3	1130	152	239	308	341
90	ND	ND	70.2	14.4	1220	115	343	281	322
105.4	ND	ND	67.4	10.8	2120	115	1250	546	396
131.1	ND	ND	267	18.1	1790	104	83	324	643

RM	17 alpha-Ethinyl-Estradiol	beta Stigmastanol	beta-Sitosterol	Ergosterol
50	ND	31.1	335	6.22
68.1	1.97	68	306	11.4
80	2.3	60	476	9.65
90	ND	43.4	403	ND
105.4	4.01	46.2	546	ND
131.1	2.4	39.3	856	6.44

ND= not detected

Table B7. Hormones in ambient water - 2009 (ng/L)

RM	Estrone	Norethindrone	17 alpha-Ethinyl-Estradiol	Desogestrel	17 alpha-Dihydroequilin	Equilenin	Equilin	17 beta-Estradiol
50	ND	ND	ND	242	ND	ND	ND	ND
68.1	ND	ND	ND	239	ND	ND	ND	ND
80	ND	ND	ND	ND	ND	ND	ND	ND
90	ND	ND	ND	ND	ND	ND	ND	ND
105.4	ND	ND	ND	ND	ND	ND	ND	ND
131.1	ND	ND	ND	ND	ND	ND	ND	ND

RM	17 alpha-Estradiol	Allyl Trenbolone	Androstenedione	Androsterone	Estriol	Mestranol	Norgestrel	Testosterone	Progesterone
50	ND	ND	ND	ND	ND	ND	ND	ND	ND
68.1	ND	ND	ND	ND	ND	ND	ND	ND	ND
80	ND	ND	ND	ND	ND	ND	ND	ND	ND
90	ND	ND	ND	ND	ND	ND	ND	ND	ND
105.4	ND	ND	ND	ND	ND	ND	ND	ND	ND
131.1	ND	ND	ND	ND	ND	ND	ND	1.37	ND

ND= not detected

Table B8. PFASs (ng/L) in ambient water - 2007

	Liston Point RM 50	De. Mem Bridge RM 68.1	Marcus Hook Creek RM 80	Schuylkill R. RM 90	Pennsauken Creek RM 105.4	Biles Channel RM 131.1
Perfluorobutanoate (PFBA) C4	17.60	19.00	5.89	2.92	1.43	1.71
Perfluoropentanoate (PFPeA) C5	35.40	30.50	10.40	5.09	4.56	ND
Perfluorohexanoate (PFHxA) C6	57.90	79.80	16.20	6.00	3.59	1.40
Perfluoroheptanoate (PFHpA) C7	19.80	24.30	12.30	4.55	2.85	ND
Perfluorooctanoate (PFOA) C8	57.30	75.40	23.80	10.80	5.80	3.54
Perfluorononanoate (PFNA) C9	331.00	847.00	976.00	265.00	3.32	1.71
Perfluorodecanoate (PFDA) C10	6.97	9.97	4.62	2.07	1.11	ND
Perfluoroundecanoate (PFUnA) C11	2.42	8.92	26.00	7.22	ND	ND
Perfluorododecanoate (PFDoA) C12	ND	ND	ND	ND	ND	ND
Perfluorobutanesulfonate (PFBS) C4	2.79	2.34	ND	ND	ND	ND
Perfluorohexanesulfonate (PFHxS) C6	2.97	ND	4.48	4.12	ND	ND
Perfluorooctanesulfonate (PFOS) C8	5.96	7.27	7.05	8.42	7.49	2.70
Perfluorooctane sulfonamide (PFOSA) C8	ND	ND	ND	ND	ND	ND

ND= not detected

Table B9. PFASs (ng/L) in ambient water - 2008

	Liston Point RM 50	De. Mem Bridge RM 68.1	Marcus Hook Creek RM 80	Schuylkill R. RM 90	Pennsauken Creek RM 105.4	Biles Channel RM 131.1
Perfluorobutanoate (PFBA) C4	25.9	20.6	4.51	4.87	2.6	1.63
Perfluoropentanoate (PFPeA) C5	31.6	28.2	8.9	5.88	ND	ND
Perfluorohexanoate (PFHxA) C6	80.1	53.8	10.5	6.26	5.02	2.92
Perfluoroheptanoate (PFHpA) C7	16.3	15.4	7.23	4.51	2.72	2.03
Perfluorooctanoate (PFOA) C8	47.5	48	19.2	10.2	5.1	3.99
Perfluorononanoate (PFNA) C9	**301**	650	552	196	4.25	3.24
Perfluorodecanoate (PFDA) C10	5.25	6.21	2.33	1.69	ND	ND
Perfluoroundecanoate (PFUnA) C11	2.47	7.23	11.8	5.44	ND	ND
Perfluorododecanoate (PFDoA) C12	ND	ND	ND	ND	ND	ND
Perfluorobutanesulfonate (PFBS) C4	24.4	15.4	3.73	3.24	ND	ND
Perfluorohexanesulfonate (PFHxS) C6	2.12	3.55	2.1	3.17	ND	ND
Perfluorooctanesulfonate (PFOS) C8	6.16	11.7	10.9	10.5	6.71	3.53
Perfluorooctane sulfonamide (PFOSA) C8	ND	ND	ND	ND	ND	ND

ND= not detected

Table B10. PFASs (ng/L) in ambient water - 2009

	Liston Point RM 50	De. Mem Bridge RM 68.1	Marcus Hook Creek RM 80	Schuylkill R. RM 90	Pennsauken Creek RM 105.4	Biles Channel RM 131.1
Perfluorobutanoate (PFBA) C4	7.46	8.15	3.37	2.84	1.87	ND
Perfluoropentanoate (PFPeA) C5	13.8	11.7	3.98	3.17	2.23	1.34
Perfluorohexanoate (PFHxA) C6	40	47.4	6.54	6.07	5.45	3.27
Perfluoroheptanoate (PFHpA) C7	7.78	9.81	5.02	4.01	1.95	1.14
Perfluorooctanoate (PFOA) C8	20.4	27.7	16.4	11.5	5.37	3.29
Perfluorononanoate (PFNA) C9	108	338	546	240	3.68	1.65
Perfluorodecanoate (PFDA) C10	2.08	2.62	1.75	ND	ND	ND
Perfluoroundecanoate (PFUnA) C11	1.42	3.97	8.3	3.86	ND	ND
Perfluorododecanoate (PFDoA) C12	ND	ND	ND	ND	ND	ND
Perfluorobutanesulfonate (PFBS) C4	9.88	8.57	2.62	ND	ND	ND
Perfluorohexanesulfonate (PFHxS) C6	ND	2.74	3.62	3.05	2.89	ND
Perfluorooctanesulfonate (PFOS) C8	2.86	5.75	7.97	5.99	7	ND
Perfluorooctane sulfonamide (PFOSA) C8	ND	ND	ND	ND	ND	ND

ND= not detected

Table B11. PBDE in ambient water – 2007 (pg/L)

RM	DiBDE	TriBDE	TeBDE	PeBDE	HxBDE	HpBDE	OcBDE	NoBDE	DeBDE	tPBDE
50	4.06	8.34	10.88	12.1	7.56	ND	12.2	161	30	246
68.1	4.3	11.97	65.16	101.187	24.97	7.78	25.6	413	2,090	2,744
80	3.08	6.85	28.62	12.07	12.02	ND	11.4	13.3	ND	87
90	3.38	22.75	237.62	216.02	50.21	10.75	44.7	1161	7,630	9,376
105.4	1.95	16.22	119.59	108.65	21.8	6.45	32.1	608	3,170	4,085
131.1	ND	7.71	118.81	136.57	20.28	3.13	27.6	40.5	ND	355

ND= not detected

Table B12.Nonylphenols and Nonylphenol Ethoxylates in Ambient Water – 2007 (ng/L)

RM	NP	NP1EO
50	0.8	ND
68.1	87.6	18.4
80	34.6	17.6
90	17.7	24.6
105.4	43.1	39.8
131.1	20.7	7.97

NP = nonylphenol
NPEO1 = nonylphenol monoethoxylate

Appendix C: Aquatic Toxicity Data

Table C1: Aquatic Toxicology Data for Pharmaceuticals and Personal Care Products						
Parameter	acute EC_x or LC_x mg/L	chronic NOEC or ChV mg/L	Assessment Factor	PNEC ng/L	Organism	Source
1,7-dimethylxanthine	100		1000	100,000	Scenedesmus subspicatus	MSDS
1,7-dimethylxanthine	178		1000	178,000	Daphnia magna	MSDS
1,7-dimethylxanthine	100		1000	100,000	Leucisus idus	MSDS
2-hydroxy-ibuprofen	NA	NA				
10-hydroxy-amitriptyline	NA	NA				
acetaminophen	378		1000	378,000	Brachydanio rerio	Cunningham et al., 2006
Albuterol		1.3	100	13,000	Daphnid	ECOSAR (phenol amine)
Albuterol		2.591	100	25,910	Daphnid	ECOSAR (aliphatic amine)
alprazolam		0.018	100	180	Daphnid	ECOSAR (triazoles)
amitriptyline		0.017	100	170	Daphnid	ECOSAR
amoxicillin	0.0037		1000	4	Microcystis aeruginosa	Schmitt et al 2009
amphetamine		11.871	100	118,710	Daphnid	ECOSAR
Atenolol		3.2	100	32,000	Pimephales promelas	Winter et al, 2008
Atenolol		1.8	100	18,000	Daphnia magna	Kuster et al, 2010
azithromycin	>120		1000	120,000	Daphnid	Cunningham et al., 2006
azithromycin		1.023	100	10,230	fish	ECOSAR
benzoylecgonine		1.531	100	15,310	fish	ECOSAR (esters)
benzoylecgonine		24.708	100	247,080	Daphnid	ECOSAR (esters)
benzoylecgonine		5.81	100	58,100	green algae	ECOSAR (esters)
caffeine	151		1000	151,000	Pimephales	Cunningham et al., 2006
carbadox		38.699	100	386,990	fish	ECOSAR
carbamazepine		0.377	100	3,770	Brachionus calyciflorus	Ferrari et al., 2003
carbamazepine		0.025	100	250	Ceriodaphnia	Ferrari et al., 2003

Parameter	acute EC_x or LC_x mg/L	chronic NOEC or ChV mg/L	Assessment Factor	PNEC ng/L	Organism	Source
carbamazepine	25.5		1000	25,500	Lemna minor	Cleuvers, 2003
ciprofloxacin		0.106	100	1,060	Lemna gibba	Crane et al., 2006
ciprofloxacin	0.005		1000	5	Microcystis aeruginosa	Schmitt et al 2009
clarithromycin	0.002		1000	2	algae	Cunningham et al., 2006
clarithromycin	18.66		1000	18,660	Ceriodaphnia	Cunningham et al., 2006
clarithromycin	0.002		1000	2	Pseudokirchneriella subcapitata	Schmitt et al. 2009
cocaine	5.482		1000	5,482	Daphnid	ECOSAR
codeine	7.438		1000	7,438	Fish	ECOSAR (vinyl/allyl alcohol)
codeine	0.976		1000	976	Daphnid	ECOSAR (vinyl/allyl alcohol)
codeine		18.345	100	183,450	green algae	ECOSAR (aliphatic amines)
cotinine		12.95	100	129,500	fish	ECOSAR (aliphatic amines)
cotinine		1.425	100	14,250	Daphnid	ECOSAR (aliphatic amines)
cotinine		5.732	100	57,320	green algae	ECOSAR (aliphatic amines)
cotinine		1000	100	10,000,000	Lemna gibba	Brain et al, 2004 (max test conc)
DEET		0.091	100	910	fish	ECOSAR
dehydronifedipine	NA	NA				
Desmethyl-diltiazem	NA	NA				
digoxigenin		70.837	100	708,370	Daphnid	ECOSAR
digoxin		<0.01	100	100	Hydra vulgaris	Crane et al., 2006
diltiazem		0.092	100	920	fish	ECOSAR (amines)
diphenhydramine		1.289	100	12,890	Daphnid	ECOSAR
enalapril		220	100	2,200,000	green algae	ECOSAR
enrofloxacine		123	100	1,230,000	green algae	ECOSAR
erythromycin hydrate	0.94		1000	940	Brachionus calcyciflous	Cunningham et al., 2006

Parameter	acute EC_x or LC_x mg/L	chronic NOEC or ChV mg/L	Assessment Factor	PNEC ng/L	Organism	Source
erythromycin hydrate	0.02		1000	20	Pseudokirchneriella subcapitata	Cunningham et al., 2006
fluoxetine	0.0001		1000	0.10	Gammurus pulex	Schmitt et al, 2009
fluoxetine		0.00064	100	6.4	Potamopyrgus antipodarum	Schmitt et al 2009
fluoxetine		0.0006	100	6	Desmodesmus subspicatus	Oakes et al., 2010
fluoxetine		0.089	100	890	Daphnid	Oakes et al., 2010
fluticasone propionate	NA	NA				
furosemide		1.216	100	12,160	fish	ECOSAR (amides - acids)
furosemide		38.677	100	386,770	Daphnid	ECOSAR (amides - acids)
furosemide		21.238	100	212,380	green algae	ECOSAR (amides - acids)
gemfibrozil	74.3		1000	74,300	Daphnia magna	CCEHBR
gemfibrozil	0.53		1000	530	Ceriodaphnia	CCEHBR
gemfibrozil	0.44		1000	440	Brachionus calyciflorus	CCEHBR
Hydrocodone		9.37	100	93,700	Daphnid	ECOSAR
ibuprofen	0.00001		1000	0.01	Gammarus pulex	Schmitt et al, 2009
ibuprofen	0.001		1000	1.00	Lemna minor	Schmitt et al 2009
ibuprofen				1,000.00		pers.com. Dan Caldwell
lincomycin	0.07		1000	70	Pseudokirchneriella subcapitata	Cunningham et al., 2006
lincomycin	0.68		1000	680	Brachionus calyciflorus	Cunningham et al., 2006
Meprobamate		10.674	100	106,740	fish	ECOSAR (esters)
Meprobamate		243.393	100	2,433,930	Daphnid	ECOSAR (esters)
Meprobamate		24.473	100	244,730	green algae	ECOSAR (esters)
Metformin	110		1000	110,000	Lemna	Cleuvers 2003
Metformin	64		1000	64,000	Daphnia magna	Cleuvers 2003
Metformin	130		1000	130,000	Daphnid	CCEHBR

Parameter	acute EC_x or LC_x mg/L	chronic NOEC or ChV mg/L	Assessment Factor	PNEC ng/L	Organism	Source
Metformin	110		1000	110,000	Daphnia magna	pers.com. Dan Caldwell
Metformin	110		1000	110,000	Pseudokirchneriella	pers.com. Dan Caldwell
Metformin		100	100	1,000,000	Daphnia magna	pers.com. Dan Caldwell recommended
Metformin		10.3	100	103,000	Brachydanio rerio	pers.com. Dan Caldwell
Methylprednisolone		39.231	100	392,310	fish	ECOSAR
Metoprolol	7.3		1000	7,300	Desmodesmus subspicatus	Cleuvers, 2003
naproxen	24.2		1000	24,200	Lemna minor	Cleuvers, 2003
naproxen	174		1000	174,000	Daphnia	Cleuvers, 2003
norfloxacin		0.206	100	2,060	Lemna gibba	Crane et al., 2006
norverapamil	NA	NA				
ofloxacin		0.005	100	50	Synechococcus (cyanobacteria)	Ferrari et al., 2004
oxycodone		42.895	100	428,950	fish	ECOSAR (aliphatic amines)
oxycodone		3.304	1000	3,304	Daphnid	ECOSAR (aliphatic amines)
oxycodone		15.584	1000	15,584	green algae	ECOSAR (aliphatic amines)
Propoxyphene		0.338	100	3,380	fish	ECOSAR
Ranitidine	150		1000	150,000	algae	MSDS
sertraline		0.034	100	340	fish	ECOSAR
sulfachloropyridazine	2.3		1000	2,300	Lemna minor	Schmitt et al 2009
sulfadiazine	221		1000	221,000	Lemna minor	Crane et al., 2006
sulfadimethoxine		0.1	100	1,000	Lemna gibba	ECOTOX
sulfadimethoxine	19.49		1000	19,490	Artemia	ECOTOX
sulfamethazine	>1.0		1000	1,000	Lemna gibba	Crane et al., 2006
sulfamethoxazole		0.25	100	2,500	Ceriodaphnia	Ferrari et al., 2004
sulfamethoxazole		0.0059	100	59	Synechococcus (cyanobacteria)	Ferrari et al., 2004

Parameter	acute EC_x or LC_x mg/L	chronic NOEC or ChV mg/L	Assessment Factor	PNEC ng/L	Organism	Source
sulfanilamide	14		1000	13,700	Daphnia magna	ECOTOX
sulfanilamide		1	100	10,000	Oryzia latipes	ECOTOX
sulfathiazole		0.923	100	9,230	fish	ECOSAR
theophylline	100		1000	100,000	Leuciscus idus	MSDS
theophylline	178		1000	178,000	Daphnia magna	MSDS
thiabendazole		0.012	100	120	rainbow trout	MSDS
thiabendazole		0.11	100	1,100	fathead minnow	MSDS
thiabendazole	0.55		1000	550	rainbow trout	MSDS
Triamterene		56	100	560,000	Daphnid	ECOSAR
triclocarban		0.000056	100	0.56	Americamysis bahia	ECOTOX
triclocarban		0.00025	100	2.50	Daphnid	ECOTOX
trimethoprim		1	100	10,000	Lemna gibba	ECOTOX
trimethoprim		6	100	60,000	Daphnia magna	ECOTOX
tylosin		22.4	100	224,000	Brachionus plicatilus	ECOTOX
tylosin		22.4	100	224,000	Brachionus calyciflorus	ECOTOX
tylosin		45	100	450,000	Daphnia magna	ECOTOX
tylosin		1	100	10,000	Lemna gibba	ECOTOX
valsartan		58	100	580,000	green algae	FDA Novartis EA 2009
valsartan		280	100	2,800,000	Daphnia magna	FDA Novartis EA 2009
valsartan		100	100	1,000,000	Salmo gairdneri	FDA Novartis EA 2009
verapamil		0.029	100	290	Daphnid	ECOSAR

EC_{50} – effective concentration of the tested chemical at which mortality or immobility occurs at 50 % of organisms

LC_{50} -lethal concentration of the tested chemical at which mortality occurs at 50 % of organisms

NOEC – no observed effect concentration

ChV – chronic value is the geometric mean of the no observed effect concentration (NOEC) and the lowest observed effect concentration (LOEC) as reported in ECOSAR

Aquatic Toxicity Source References

Databases used:

US EPA ECOTOX, which currently includes more than 520,000 test results on the effects of more than 8,500 chemicals, including PPCPs, on over 6,400 terrestrial and aquatic species (http://cfpub.epa.gov/ecotox/);

Center for Coastal Environmental Health and Bimolecular Research (CCEHBR) Pharmaceuticals in the Environment, Information for Assessing Risk (PEIAR) website. A site designed to provide available information for assessing risks to aquatic resources from drugs entering waterways from both point and non-point sources (http://www.chbr.noaa.gov/peiar/default.aspx);

ECOSAR (Ecological Structure Activity Relationship) a computerized predictive system that estimates the aquatic toxicity of chemicals. The program estimates a chemical's acute (short-term) toxicity and chronic (long-term or delayed) toxicity to aquatic organisms such as fish, aquatic invertebrates, and aquatic plants by using Structure Activity Relationships (SARs) (http://www.epa.gov/oppt/newchems/tools/21ecosar.htm);

FDA Novartis EA 2009 Environmental Assessment Report, NDA 22-217, Aliskiren/Valsartan Film-Coated Tablets. http://www.accessdata.fda.gov/drugsatfda_docs/nda/2009/022217s000ea.pdf

Material Safety Data Sheets (MSDS) prepared by the manufacturer of a product for the purpose of providing information on the safe use, handling and potential hazards of a product;

TCC Consortium. High Production Volume (HPV) Chemical Challenge Program Data Availablility and Screening Level Assessment for Triclocarban, CAS#:101-20-2. (http://www.epa.gov/hpv/pubs/summaries/tricloca/c14186tc.htm); and

USEPA PPCP Literature Citation Database includes published literature relevant to the issues surrounding PPCPs as environmental contaminants (http://www.epa.gov/ppcp/lit.html).

Additional sources for ecotoxicology data were from the following publication:

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Crane, M; C. Watts and T. Boucard. 2006. Chronic aquatic environmental risks from exposure to human pharmaceuticals. Science of the Total Environment 367:23-41.

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Fent, K, A Weston and D. Caminanda. 2006. Ecotoxicology of human pharmaceuticals. Aquatic Toxicology 76:122-159

Ferrari B. et al. 2004. Environmental risk assessment of six human pharmaceuticals: are the current environmental risk assessment procedures sufficient for the protection of the aquatic environment. Environ Toxicol and Chem 23(5)1344-1354.

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Oakes, K. D.et al., 2010. Environmental Risk assessment for the serotonin re-uptake inhibitor fluoxetine: case study using the European risk assessment framework. Integr Environ Assess and Manag 6(1)524-539

Schmitt, H et al., 2009. Recommendations on the environmental risk assessment of pharmaceuticals: effect characterization. Integr Environ Assess and Manag 6(1): 588-602.

Winter et al 2008. Defining the chronic impacts of atenolol on embryo-larval development and reproduction in the fathead minnow (Pimephales promelas). Aquat. Toxicol. (3) 361-369.

Table C2. Risk Characterization of DRBC Contaminants of Emerging Concern 2007 Survey					
Parameter	MEC ng/L	PNEC ng/L	Hazard Quotient	Quotient > 0.1 Priority	Quotient >1.0 Higher Priority
azithromycin	4.96	120,000	0.00	FALSE	FALSE
azithromycin	4.96	10,230	0.00	FALSE	FALSE
caffeine	72.2	151,000	0.00	FALSE	FALSE
carbamazepine	60.4	3,770	0.02	FALSE	FALSE
carbamazepine	60.4	250	0.24	TRUE	FALSE
carbamazepine	60.4	25,500	0.00	FALSE	FALSE
clarithromycin	10.9	2	5.4500	TRUE	TRUE
clarithromycin	10.9	18,660	0.00	FALSE	FALSE
clarithromycin	10.9	2	5.45	TRUE	TRUE
codeine	159	7,438	0.02	FALSE	FALSE
codeine	159	976	0.16	TRUE	FALSE
codeine	159	183,450	0.00	FALSE	FALSE
dehydronifedipine	2.97	NA			
diltiazem	12.8	920	0.01	FALSE	FALSE
diphenhydramine	4.56	12,890	0.00	FALSE	FALSE
erythromycin hydrate	14.9	940	0.02	FALSE	FALSE
erythromycin hydrate	14.9	20	0.75	TRUE	FALSE
fluoxetine	8.02	0.1	80.20	TRUE	TRUE
fluoxetine	8.02	6	1.25	TRUE	TRUE
fluoxetine	8.02	6	1.34	TRUE	TRUE
fluoxetine	8.02	890	0.01	FALSE	FALSE
gemfibrozil	44.3	74,300	0.00	FALSE	FALSE
gemfibrozil	44.3	530	0.08	FALSE	FALSE
gemfibrozil	44.3	440	0.10	TRUE	FALSE
ibuprofen	15.9	0.01	1590.00	TRUE	TRUE
ibuprofen	15.9	1	15.90	TRUE	TRUE
ibuprofen	15.9	1,000	0.02	FALSE	FALSE
naproxen	27.9	24,200	0.001153	FALSE	FALSE
naproxen	27.9	174,000	0.00016	FALSE	FALSE
norfloxacin	9.7	2,060	0.00	FALSE	FALSE
ofloxacin	1.6	50	0.03	FALSE	FALSE
Parameter	MEC	PNEC	Hazard	Quotient	

	ng/L	ng/L	Quotient	> 0.1 Priority	Quotient >1.0 Higher Priority
sulfadiazine	2.91	221,000	0.00	FALSE	FALSE
sulfadimethoxine	1.64	1,000	0.00	FALSE	FALSE
sulfadimethoxine	1.64	19,490	0.00	FALSE	FALSE
sulfamethoxazole	150	2,500	0.06	FALSE	FALSE
sulfamethoxazole	150	59	2.54	TRUE	TRUE
sulfanilamide	16.6	13,700	0.001212	FALSE	FALSE
sulfanilamide	16.6	10,000	0.00166	FALSE	FALSE
triclocarban	6.54	0.56	11.67857	TRUE	TRUE
triclocarban	6.54	2.5	2.616	TRUE	TRUE
trimethoprim	16.6	10,000	0.00	FALSE	FALSE
trimethoprim	16.6	60,000	0.00	FALSE	FALSE

MEC = measured environmental concentration
PNEC = predicted no effect concentration most sensitive species
Hazard Quotient = MEC/PNEC
NA = not available

Table C3. Risk Characterization of DRBC Contaminants of Emerging Concern 2008 Survey

Parameter	MEC ng/L	PNEC ng/L	Hazard Quotient	Quotient > 0.1 Priority	Quotient >1.0 Higher Priority
1,7-dimethylxanthine	239	100,000	0.00239	FALSE	FALSE
1,7-dimethylxanthine	239	100,000	0.00239	FALSE	FALSE
1,7-dimethylxanthine	239	178,000	0.0013427	FALSE	FALSE
Albuterol	0.711	13,000	7.9888E-07	FALSE	FALSE
caffeine	166.1	151,000	0.0011	FALSE	FALSE
carbadox	7.48	386,990	1.9329E-05	FALSE	FALSE
carbamazepine	67.3	3,770	0.01785146	FALSE	FALSE
carbamazepine	67.3	250	0.2692	TRUE	FALSE
carbamazepine	67.3	25,500	0.00263922	FALSE	FALSE
clarithromycin	2.65	2	1.325	TRUE	TRUE
clarithromycin	2.65	18,660	0.00014202	FALSE	FALSE
codeine	72.7	7,438	0.01628584	FALSE	FALSE
codeine	72.7	976	0.0744877	FALSE	FALSE
codeine	72.7	183,450	0.00039629	FALSE	FALSE
codeine	72.7	129,500	0.00056139	FALSE	FALSE
cotinine	35.6	129,500	0.0002749	FALSE	FALSE
cotinine	35.6	14,250	0.00249825	FALSE	FALSE
cotinine	35.6	57,320	0.00062107	FALSE	FALSE
cotinine	35.6	10,000,000	0.00000356	FALSE	FALSE
dehydronifedipine	2.19	708,370	3.0916E-06	FALSE	FALSE
diltiazem	9.83	920	0.00076261	FALSE	FALSE
diphenhydramine	3.64	12,890	0.00028239	FALSE	FALSE
erythromycin hydrate	6.23	940	0.00662766	FALSE	FALSE
erythromycin hydrate	6.23	20	0.3115	TRUE	FALSE
fluoxetine	5.4	0	54	TRUE	TRUE
fluoxetine	5.4	6	0.84375	TRUE	FALSE
fluoxetine	5.4	6	0.9	TRUE	FALSE
fluoxetine	5.4	890	0.00606742	FALSE	FALSE
gemfibrozil	24.9	100,000	0.000249	FALSE	FALSE
lincomycin	37.4	70	0.53428571	TRUE	FALSE
lincomycin	37.4	680	0.055	FALSE	FALSE
Metformin	3590	110,000	0.03263636	FALSE	FALSE
Metformin	3590	64,000	0.05609375	FALSE	FALSE
Metformin	3590	130,000	0.02761538	FALSE	FALSE
Metformin	3590	110,000	0.03263636	FALSE	FALSE
Metformin	3590	110,000	0.03263636	FALSE	FALSE
Metformin	3590	1,000,000	0.00359	FALSE	FALSE
Metformin	3590	103,000	0.03485437	FALSE	FALSE

Parameter	MEC ng/L	PNEC ng/L	Hazard Quotient	Quotient > 0.1 Priority	Quotient >1.0 Higher Priority
naproxen	26.2	24,200	0.00108264	FALSE	FALSE
naproxen	26.2	174,000	0.00015057	FALSE	FALSE
Ranitidine	0.922	1,000	0.000922	FALSE	FALSE
sulfadimethoxine	0.485	1,000	0.000485	FALSE	FALSE
sulfadimethoxine	0.485	19,490	2.4885E-05	FALSE	FALSE
sulfamethoxazole	2.35	2,500	0.00094	FALSE	FALSE
sulfamethoxazole	2.35	59	0.03983051	FALSE	FALSE
thiabendazole	73.6	120	0.61333333	TRUE	FALSE
thiabendazole	73.6	1,100	0.06690909	FALSE	FALSE
thiabendazole	73.6	550	0.13381818	TRUE	FALSE
triclocarban	8.53	1	15.2321429	TRUE	TRUE
triclocarban	8.53	3	3.412	TRUE	TRUE
trimethoprim	13.1	10,000	0.03983051	FALSE	FALSE
trimethoprim	13.1	60,000	0.03983051	FALSE	FALSE

MEC = highest measured environmental concentration
PNEC = predicted no effect concentration most sensitive species
Hazard Quotient = MEC/PNEC
NA = not available

Table C4. Risk Characterization of DRBC Contaminants of Emerging Concern 2009 Survey

Parameter	MEC ng/L	PNEC ng/L	Hazard Quotient	Quotient > 0.1 Priority	Quotient >1.0 Higher Priority
2-hydroxy-ibuprofen	346	NA			
10-hydroxy-amitriptyline	0.382	NA			
acetaminophen	105	100	1.05	TRUE	TRUE
Albuterol	0.996	13,000	0.00	FALSE	FALSE
Alprazolam	0.612	180	0.00	FALSE	FALSE
Amitriptyline	1.39	170	0.01	FALSE	FALSE
Amphetamine	5.53	118,710	0.00	FALSE	FALSE
Atenolol	58.8	32,000	0.00	FALSE	FALSE
Atenolol	58.8	18,000	0.00	FALSE	FALSE
azithromycin	14.1	120,000	0.0001175	FALSE	FALSE
azithromycin	14.1	10,230	0.0013783	FALSE	FALSE
Benzoylecgonine	39.6	15,310	0.00258654	FALSE	FALSE
caffeine	238	151,000	0.00	FALSE	FALSE
carbadox	7.48	386,990	1.9329E-05	FALSE	FALSE
carbamazepine	39.2	3,770	0.01	FALSE	FALSE
carbamazepine	39.2	250	0.16	TRUE	FALSE
carbamazepine	39.2	25,500	0.00	FALSE	FALSE
clarithromycin	11.6	2	5.80	TRUE	TRUE
clarithromycin	11.6	18,660	0.00	FALSE	FALSE
clarithromycin	11.6	2	5.80	TRUE	TRUE
cocaine	2.58	5,482	0.00	FALSE	FALSE
codeine	38.2	7,438	0.01	FALSE	FALSE
codeine	38.2	976	0.04	FALSE	FALSE
codeine	38.2	18,345	0.00	FALSE	FALSE
cotinine	38.2	1,000	0.04	FALSE	FALSE
DEET	42.8	900	0.05	FALSE	FALSE
dehydronifedipine	1.41	NA			
Desmethyldiltiazem	3.35	NA			
diltiazem	9.51	920	0.01	FALSE	FALSE
diphenhydramine	7.68	12,890	0.00	FALSE	FALSE
Enalapril	0.343	2,200,000	0.00	FALSE	FALSE
erythromycin hydrate	9.24	940	0.01	FALSE	FALSE
erythromycin hydrate	9.24	20	0.46	TRUE	FALSE
Fluticasone propionate	2.69	NA			
gemfibrozil	53.9	440	0.12	TRUE	FALSE

Parameter	MEC ng/L	PNEC ng/L	Hazard Quotient	Quotient > 0.1 Priority	Quotient >1.0 Higher Priority
Hydrocodone	16.2	93,700	0.00	FALSE	FALSE
ibuprofen	76.6	0.01	7660.00	TRUE	TRUE
ibuprofen	76.6	1	76.60	TRUE	TRUE
ibuprofen	76.6	1,000	0.08	FALSE	FALSE
Meprobamate	38.2	106,740	0.00	FALSE	FALSE
Meprobamate	38.2	244,730	0.00	FALSE	FALSE
Metformin	1140	110,000	0.01	FALSE	FALSE
Metformin	1140	64,000	0.02	FALSE	FALSE
Metformin	1140	130,000	0.01	FALSE	FALSE
Metformin	1140	110,000	0.01	FALSE	FALSE
Metformin	1140	110,000	0.01	FALSE	FALSE
Metformin	1140	1,000,000	0.00	FALSE	FALSE
Metformin	1140	103,000	0.01	FALSE	FALSE
Methylprednisolone	0.8	392,310	0.00	FALSE	FALSE
Metoprolol	42.4	7,300	0.01	FALSE	FALSE
naproxen	67.5	24,200	0.00	FALSE	FALSE
Norverapamil	0.172	NA			
oxycodone	53.1	3,304	0.02	FALSE	FALSE
Propoxyphene	2.96	3,380	0.00	FALSE	FALSE
Ranitidine	3.18	150,000	0.00	FALSE	FALSE
Sertraline	1.16	340	0.00	FALSE	FALSE
sulfadimethoxine	1.42	1,000	0.00	FALSE	FALSE
sulfamethoxazole	88.8	2,500	0.04	FALSE	FALSE
sulfamethoxazole	88.8	59	1.51	TRUE	TRUE
sulfathiazole	2.35	9,230	0.00	FALSE	FALSE
theophylline	145	116,750	0.00	FALSE	FALSE
thiabendazole	3.66	10,000	0.00	FALSE	FALSE
thiabendazole	3.66	1,000	0.00	FALSE	FALSE
Triamterene	4.47	560,000	0.00	FALSE	FALSE
triclocarban	10.7	0.56	19.11	TRUE	TRUE
triclocarban	10.7	2.50	4.28	TRUE	TRUE
trimethoprim	20.3	10,000	0.00	FALSE	FALSE
trimethoprim	20.3	60,000	0.00	FALSE	FALSE
valsartan	97.6	90,000		FALSE	FALSE
Verapamil	0.852	290	0.00	FALSE	FALSE

MEC = measured environmental concentration
PNEC = predicted no effect concentration most sensitive species
Hazard Quotient = MEC/PNEC
NA = not available

Appendix D: Bioassays

Short-term Chronic Toxicity Tests

Bioassays were included in the study because they assess chemical mixtures and possible additive effects as well as assess toxicants with no specific analytical detection method or chemicals that are not being monitored by chemical methods used in the study. Concurrent with sampling for contaminants of emerging concern in years 2007 and 2008, short-term chronic toxicity tests were conducted with ambient water from the six sampling stations in the main-stem river as described in the contaminants of emerging concern sampling . Samples were split and transported on the day collected to the respective laboratories for toxicity testing and physical-chemical analysis. Samples for toxicity testing were transported to American Aquatic Testing Laboratory Inc., Allentown, PA. Following USEPA Methods, short-term chronic toxicity tests were performed using *Pimephales promelas*, *Americamysis bahia*, *Menidia beryllina*, and *Ceriodaphnia dubia* in 7-day tests; *Pseudokirchneriella subcapitata* in a 96-hour test; and Hyalella azteca in a 10-day water-only test. Test organism survival, growth, and when possible, reproduction were measured (USEPA 2000, 2002a and 2002b) (Table C1). Results from this study indicate that water collected from six main-stem sites in the tidal Delaware River Basin caused little or no significant adverse effects for the endpoints measured in short-term chronic toxicity tests for the species tested. Survival, growth and reproduction results indicated the lack of effects (MacGillivray et al., 2011). Bioassays can provide a cost effective approach for prioritization of monitoring surface water quality in a large watershed providing information for environmental management over the largest time scale and spatial coverage with limited resources. Using bioassays, sites that exhibit harmful effects can be targeted for additional evaluation using more expensive chemical analysis, toxicity identification evaluation and toxicity reduction evaluation.

Table D1. Mainstem toxicity bioassays[a]

Site	River Mile	Latitude Longitude	Salinity ppt	2007	2008
E1	50	39.455 -75.56	11.4 – 15.5	Ab, Mb, Ha	Ab, Mb, Ha
E4	68.1	39.65472 -75.54667	4.3 – 4.9	Ab, Mb, Ha	Pp, Cd, Ps
E7	80	39.81336 -75.39058	0.9 – 1.6	Pp, Cd, Ps	Pp, Cd, Ps
E9	90	39.8835 -75.18616	<1	Pp, Cd, Ps	Pp, Cd, Ps
E12	105.4	39.99478 -75.05978	<1	Pp, Cd, Ps	Pp, Cd, Ps
E16	131.1	40.18156 -74.74505	<1	Pp, Cd, Ps	Pp, Cd, Ps

[a]Abbreviations for bioassays are Ab (*A. bahia),* Mb (*M. beryllina), Ha (H. azteca), Pp (P. promelas), Cd (C. dubia), and Ps (P. subcapitata)* Sampling occurred on October 15 and 17, 2007 and August 4, 6, 8 and 11, 2008.

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Assays for Estrogenic Compounds

A number of the compounds detected in the 2007 pilot study have been proven or suspected to be estrogenic including hormones and nonylphenols. Therefore, as part of a weight of evidence approach to environmental assessment, the 2008 study included both chemical analysis for contaminants of emerging concern and bioassays for estrogenicity. The bioassays were included because they assess chemical mixtures and possible additive effects as well as assess toxicants with no specific analytical detection method or chemicals that are not being monitored by chemical methods used in the study. Affordable bioassays integrated with more costly analytical testing can provide a cost effective approach for monitoring surface water quality for endocrine disrupting activity (Quiros. *et al*., 2005). Samples were collected for estrogenicity bioassays at six sites. Ambient water samples were split and transported on the day collected to the respective laboratories for bioassays and physical-chemical analysis. Samples for estrogenic compounds assays were shipped to the University of Pittsburgh. The extraction process described in Soto et al. (2004) was followed. Each water sample was processed in 250 mL fractions. The fraction was poured onto a 1 liter separatory funnel. Fifteen mLs of dichloromethane (DCM) was added to each fraction. The separatory funnel was vigorously shaken for 2 minutes and allowed to settle for 15 minutes. The DCM is heavier than water and sinks to the bottom of the funnel where it is removed. This process was repeated two more times for a total of three extractions for each fraction. The DCM from each water sample was combined and concentrated using nitrogen and an NEVAP evaporator (Organomation Associates, Berlin, MA, USA) maintained at 40°C. The residue was suspended in 1 mL ethanol: glycerol (70:30) and stored at -20C, under nitrogen, until tested in the both assays. The efficiency of this extraction procedure was tested by adding a known quantity of tritiated estradiol to distilled water. Following the above procedure, recoveries of 88 to 92% of the tritiated estradiol was obtained. The ambient water was tested in two types of bioassaysE-screen assay and estrogen receptor binding assay (Soto *et al*, 2004; Eagon *et al*. 1980; Porter *et al*., 1983; Rogerson and Eagon, 1986).

E-Screen Assay

The E-Screen assay utilized three human breast cancer cell lines, MCF-7 which is mostly estrogen receptor (ER)-alpha positive, T47D which is mostly ER-beta positive and BT-20 which is ER-negative as described in Soto et al, (2004). This assay was performed in phenol-red free RPMI supplemented with charcoal-dextran stripped fetal bovine serum. After 72 hours of exposure to various concentrations (1/4000, 1/3000, 1/1500, 1/1000, 1/500, 1/200, and 1/100) of ambient river water water in steroid-free medium, the 96-well plates were stained using sulforhodamine B and absorbance at 564 nm was measured

using a plate reader. In the E-screen assay, none of the cell lines exhibited a proliferative response to extracts derived from water samples RM 50, RM 68.1, RM 80, RM90, and Blank. MCF-7 did exhibit a weak response, at higher concentrations (1/200 and 1/100) of extract derived from water samples RM 105.4 and RM 131.1. This response was muted with 1x10-6M 4-hydroxytamoxifen, an ER blocker, suggesting this is an estrogenic response. MCF-7 exhibited an antagonistic response, again at higher concentrations, to sample RM 131.1 in the presence of 1x10-9M E2. T47D exhibited an antagonistic response at higher concentrations (1/200, and 1/100) to samples RM 105.4 and RM 131.1 in the presence of 1x10-9M E2. BT-20, the negative control cell line, did not respond to any of the test conditions. This indicates the absence of non-specific growth factors and cytotoxic compounds (Table C2)

Table D2. Summary of E-Screen Assay

	RM 50	RM 68.1	RM 80	RM 90	RM 105.4	RM 131.1	Blank
MCF-7	-	-	-	-	-/+	-/+	-
MCF-7+E2	-	-	-	-	-	*	-
T47D	-	-	-	-	-	-	-
T47D+E2	-	-	-	-	*	*	-
BT-20	-	-	-	-	-	-	-
BT-20+E2	-	-	-	-	-	-	-

Key:
-: No Response
-/+: Weak Response
+: Moderate Response
++: Strong Response
*: Antagonistic Response

Competitive ER-Binding Assay

The competitive in vitro estrogen receptor (ER) binding assay, previously described by Porter et al. (1983), Rogerson et al. (1986), and Eagon et al. (1980), is used to identify the presence of estrogenic compounds that can bind to the ER. Aliquots of cytosol were prepared using either mature rabbit uteri as a source of ER-alpha or male rat prostate as a source of ER-beta. The cytosols were incubated with 5nM [3H]-E2 in the absence (control) and presence of ambient river water extracts along with standards of known estrogenic potency such as estradiol (E2) and diethylstilbestrol (DES), a potent estrogen. The cytosol, [3H]-E2, and various concentrations (1/20, 1/5, 1/1) of sample were combined and allowed to incubate over night at 4^0C. The next morning, the bound ligand was separated from the free ligand using P6 resin spin columns. The effluent passing through the spin column was added to scintillation vials containing 8 mLs of Biosafe II counting cocktail. The disintegrations per minute (DPMs) was measured using a beta counter. The mean DPMs for each test condition were calculated. The lower the DPMs the more displacement that has taken place suggesting that the test agent is estrogenic. Generally, the greater the competition of the water, as expressed relative to the results of control binding in the absence of river water, the greater the likelihood the sample contains an estrogenic compound. The Competitive ER-alpha binding assay measures the displacement of tritiated estradiol ([3H]-E2) from estrogen receptor. The cell proliferation assay quantitating growth of hormone-sensitive cells based on three human breast cancer cell lines BT-20, MCF-7 and T47D in the presence and absence of estradiol as described in Soto et al., (2004). The screening level assays for estrogenic compounds

indicated either low concentrations or weak effects for the ambient water sampled from the tidal Delaware River. All of the river water samples tested were at least weak estrogen competitors and that some of the samples were strong competitors (Table C3). In addition, the samples are arranged based on the strength of competition in Table C4. At the 1/1 dilution of the water sample, the samples fall into three groups. Samples from RM 68.1 and the blank were weak estrogen competitors, while samples from RM 131.1, RM 105.4 and RM 50 were moderate estrogen competitors and finally, E7 was a strong competitor. Future monitoring should include assays for estrogenic compounds in fish tissue where bioaccumulation of estrogenic compounds may occur causing a stronger response to be observed.

Table D3. Summary of Competitive ER-alpha Binding Assay (DPM)

River Mile	1/20 Dilution	1/5 Dilution	1/1 Dilution
50	0.80 (-/+)	0.76 (+)	0.58 (+)
68.1	0.80 (-/+)	0.86 (-/+)	0.84 (-/+)
80	0.81 (-/+)	0.68 (+)	0.54 (+/++)
90	0.72 (+)	NR	NR
105.4	0.86 (-/+)	0.76 (+)	0.68 (+)
131.1	0.79 (+)	0.82 (-/+)	0.74 (+)
Blank	0.86 (-/+)	0.83 (-/+)	0.80 (-/+)

- : No Competition (100% - 90% of Control)
-/+: Weak Competition (89% - 80% of Control)
+: Moderate Competition (79% - 55% of Control)
++: Strong Competition (<=55% of Control)
NR – not reported
DPM – disintegrations per minute

Table D4. Samples Arranged by Concentration and Increasing ER-alpha Competition (DPM)

1/20 Dilution	1/5 Dilution	1/1 Dilution
105.4 0.86 (-/+)	RM 68.1 0.86 (-/+)	RM 68.1 0.84 (-/+)
Blank 0.86 (-/+)	Blank 0.83 (-/+)	Blank 0.80 (-/+)
RM 80 0.86 (-/+)	RM 131.1 0.82 (-/+)	RM 131.1 0.74 (+)
RM 50 0.80 (-/+)	RM 50 0.76 (+)	RM 105.4 0.68 (+)
RM 68.1 0.80 (-/+)	RM 105.4 0.76 (+)	RM 50 0.58 (+)
RM 131.1 0.79 (+)	RM 80 0.68 (+)	RM 80 0.54 (++)

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