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The Occurrence, Fate, Environmental Impact, and Management Implications of Pharmaceutical and Personal Care Products in Wastewater and the Environment

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THE OCCURRENCE, FATE, ENVIRONMENTAL IMPACT, AND MANAGEMENT IMPLICATIONS OF PHARMACEUTICAL AND PERSONAL CARE PRODUCTS IN WASTEWATER AND THE ENVIRONMENT

by

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ABSTRACT
THE OCCURRENCE, FATE, ENVIRONMENTAL IMPACT, AND MANAGEMENT IMPLICATIONS OF PHARMACEUTICAL AND PERSONAL CARE PRODUCTS IN WASTEWATER AND THE ENVIRONMENT

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Pharmaceuticals and personal care products (PPCPs) are a critical part of modern life. However, there is growing evidence that the levels of PPCPs detected in wastewater effluent and in the environment have the potential to cause damage to aquatic organisms. These pollutants enter the aquatic environment primarily through human use, disposal in the drain or toilet, land application of biosolids, and veterinary sources. Numerous questions remain regarding the occurrence, fate, and impacts of PPCPs in wastewater, along with limited feasible management recommendations that would adequately mitigate the risk from these pollutants. This dissertation will present three advances in the field of PPCPs in the aquatic environment: 1) An experiment that describes the occurrence of PPCPs in a large scale urban wastewater treatment plant and the assessment of a model that predicts the removal of these PPCPs across the different wastewater treatment processes; 2) A study that monitors the occurrence of PPCPs in Lake Michigan and an assessment of the ecological risks at the detected levels; and 3) A case study that provides an analysis of current wastewater treatment regulations (e.g. requiring phosphorus removal from

wastewater) and whether the regulations can be modified to remove unregulated PPCPs. Overall, this research provides an assessment of the fate, occurrence, and corresponding ecological damage from PPCPs in wastewater and the environment, along with an evaluation of a potential management technique. The major contribution of this work is to further the understanding of the distribution and fate of PPCPs in the aquatic environment, which can ultimately be used to assist in constructing relevant policy and management recommendations.

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LIST OF ABBREVIATIONS

Abbreviation	Extended Nomenclature
CAS	conventional activated sludge
BDL	below detection limit
HQ	hazard quotient
JIWRF	Jones Island Water Reclamation Facility
MDL	minimum detection limit
MBR	membrane bioreactor
MEC	maximum environmental concentration
MF	microfiltration
MGD	million gallons per day
MQL	minimum quantification limit
NF	nanofiltration
PNEC	predicted no-effect concentration
PPCPs	pharmaceutical and personal care products
RQ	risk quotient
SSWRF	South Shore Water Reclamation Facility
UF	ultrafiltration
WWTP	wastewater treatment plant

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Chapter 1: INTRODUCTION

Since the mid-19th century, pharmaceutical use has become a mainstay of the health care system. To illustrate this trend, over the last 10 years, the percentage of Americans who took at least one prescription drug in the past month increased from 44% to 48%, the percentage who use two or more drugs increased from 25% to 31%, and the percentage who use five or more drugs increased from 6% to 11% (Gu et al., 2010). More recent studies have found 68% of the population used at least one prescription drug per year (Zhong et al., 2013). The advances in the development and widespread use of pharmaceutical medicines has played a partial role in the American life span increasing from an average of 47 years in 1850 to 79 years in 2010 (Daimmrich and Bowden 2005; Murphy et al., 2013). Overall, it is reasonable to expect that the worldwide usage of pharmaceuticals and personal care products (PPCPs) will continue to increase as populations and affluence rises.

Thirty years ago, concerns were raised regarding the probable presence of pharmaceuticals and personal care products (PPCPs) in the aquatic environment (e.g., Aherne et al., 1985). In spite of these concerns, pharmaceuticals in the aquatic environment received relatively little attention until Ternes et al. (1998) detected pharmaceuticals in wastewater effluent and Jobling et al. (1998) and Desbrow et al. (1998) found that pharmaceuticals were impacting aquatic organisms (Sumpter 2010). Together, these findings led the way for a surge in research and thousands of papers have since been published regarding PPCPs in, and the potential impact on, the aquatic

environment (Daughton and Scuderi, 2013). Despite the significant research efforts regarding PPCPs in the aquatic environment, numerous questions remain unanswered.

Background and Significance

The term PPCPs is used to describe a set of compounds consisting primarily of human and veterinary pharmaceutical medicines, over-the-counter drugs, stimulants, hormones, cosmetics, soaps, and fragrances. These pollutants enter the aquatic environment through human use, disposal in the drain or toilet, land application of biosolids, and veterinary sources such as waste overflow and land application (Kolpin et al., 2002; Lapen et al., 2008). Many PPCPs enter the waste stream through the recommended use: orally consumed PPCPs are excreted unchanged or partly metabolized through urine or feces while topically applied PPCPs are washed off (Kasprzyk-Hordern et al., 2009).

When PPCPs enter the waste stream through excretion or disposal, these complex pollutants are partially removed from wastewater and a fraction of the influent concentration is discharged into the environment. Municipal wastewater treatment plants (WWTPs) were not designed to remove PCPPs, as they have been implemented with the aim of removing easily or moderately biodegradable carbon, nitrogen and phosphorus compounds and microbiological organisms (Verlicchi et al., 2012). The removal of PPCPs through the most common WWTP configuration, primary treatment followed by a conventional active sludge process and disinfection, can be explained through three primary mechanisms: sorption to sludge, biological degradation, and volatilization, but

other additional factors can impact the removal (Khan and Ongerth, 2004; Verlicchi et al. 2012).

Antibiotics, pharmaceuticals, stimulants, recreational drugs, hormones, non-prescription drugs, and personal care products have all been found in surface and ground waters across the world (Cahill et al., 2004; Fatta-Kassinos et al., 2011; Fent et al., 2006; Focazio et al., 2008; Halling-Sorensen et al., 1998; Kolpin et al., 2002; Ternes, 1998). The concentration of PPCPs detected in the environment can depend on the wastewater treatment processes used, the PPCPs' chemical characteristics, the flow of the waste stream, and the different PPCPs usage patterns that vary by region and over time (Dickenson et al., 2011; Le-Minh et al., 2010; Oulton et al., 2010; USEPA, 2010; Verlicchi et al., 2012). Large water bodies have been generally disregarded due to the expected low levels of PPCPs from dilution and the complex hydrodynamics in a lake as large as one of the Great Lakes; however, insignificant levels of PPCPs cannot be assumed. Therefore, few studies have been conducted on PPCPs in the Great Lakes and they have focused on locations close to shore, in harbors, or where rivers enter the lakes (Csiszar et al., 2010; Ferguson et al., 2013; Li et al., 2010; Metcalf et al., 2003).

PPCPs have been shown to cause a wide range of impacts on aquatic organisms. One of the most well-known studies assessing the environmental harm of PPCPs was an experiment conducted in a formerly pristine lake at the Experimental Lakes Area in Ontario, Canada where it was found that the addition of synthetic estrogen (17α-ethynylestradiol [EE2]) to the lake led to feminization of males and caused the collapse of a fish population (Kidd et al., 2007). As another example of the environmental impacts

of PPCPs, the non-steroidal anti-inflammatory drug (NSAID) diclofenac used by veterinarians for the treatment of inflammation and fever in domestic livestock, caused the near extinction of three types of Old World vultures in southeast Asia because the vultures fed on the livestock carcasses (Oaks et al., 2004).

Overall, a complete understanding of the impacts of PPCPs on aquatic organisms and the ecosystem is lacking. Studies have assessed the acute toxicity of PPCPs on aquatic organisms with a limited number of endpoints; however, more research is needed on the potential effects and risks of these pollutants, in particular at the concentrations PPCPs have been detected in the environment (Boxall et al., 2011). In the absence of a complete understanding of the toxicity of PPCPs, risk assessment studies have evaluated the potential impacts that PPCPs may have on ecosystem health using existing data (e.g. Verlicchi et al., 2012). For example, the risk quotient (RQ, also known as the hazard quotient or risk index) is defined as the ratio of the maximum observed concentration to the predicted no-effect concentration and has been used to determine the potential ecological impact of PPCPs (Al Aukidy et al., 2012; Gros et al., 2010; Hernando et al., 2006; Tewari et al., 2013; Verlicchi et al., 2012). To determine the predicted no-effect concentration, a wide range of toxicity endpoints have been used and these include endocrine disruption, changes in growth, and changes in behaviors important to feeding, reproduction, and predator avoidance, along with modeled toxicity endpoints (Brausch and Rand, 2011; Brodin et al., 2013; Kidd et al., 2007; Nassef et al., 2010; USEPA 2012; Weinberger and Klaper 2013). In addition, research suggests that the effects of a mixture of PPCPs can cause a greater impact than the individual effect from a specific PPCP,

particularly if they share a similar mechanism of action (Crago and Klaper, 2012; DeLorenzo and Flemming, 2008).

Although research has been completed on the potential impacts and biological degradation of PPCPs in the aquatic environment, experimental data that assesses these characteristics are sparse (Dickenson et al., 2010). The paucity of these data causes great difficulty in determining the degradation of PPCPs in wastewater and the environment, along with an inability to determine the predicted no-effect concentration. A common thread throughout this work will be the use of quantitative structure–activity relationship (QSAR) models, which have been used to fill in many of the gaps in assessing the degradation and ecological impacts of PPCPs. For example, BIOWIN from the EPA's EPI Suite is a model used to predict the biological degradation of pollutants in the environment, and it has been proposed for use in WWTP aerobic basins (Khan and Ongerth, 2004). Also, the model ECOSAR from the EPA is used to predict the ecosystem toxicity of pollutants in response to Pre-Manufacture Notices mandated under the Toxic Substances Control Act (TSCA). In addition, ECOSAR has been used in assessing the risk quotient for PPCPs (USEPA 2012; Verlicchi et al., 2012). Using a combination of experimental data and modeled characteristics allows PPCPs to be assessed using the best data available.

With over 5,000 different pharmaceuticals and thousands more personal care products currently available for use (Williams and Brooks, 2012), significant complications surround the management of these pollutants. At this time, no regulations exist for PPCPs in drinking or natural waters in the United States (Ryu et al., 2014). The

efforts to reduce PPCPs in the aquatic environment often lack the support of critical stakeholders in the regulatory process. For example, previous research has found that investment in advanced waste or drinking water treatment to reduce pharmaceuticals is opposed by both wastewater treatment companies and drinking water suppliers (Titz and Döll 2008). The management and policy solutions currently proposed in the academic literature consist of the following strategies: take-back and landfill disposal of unused PPCPs, tertiary wastewater treatment, urine separation, dilution through watershed management, producing pharmaceuticals that would cause less harm to the environment, selecting PPCPs possessing environment-friendly excretion profiles, improving drug delivery, and prescribing patients the minimum therapeutic dosage (Borsuk et al. 2008; Cook et al., 2012; Daughton and Ruhoy 2013; Eckstein and Sherk 2012; Glassmeyer et al., 2009; Khetan and Collins, 2007; Schimmelpfennig et al., 2012). Overall, given the increasing concerns over PPCPs in wastewater and the environment, it has been speculated that PPCPs will be eventually regulated in the United States (Eckstein, 2012; Eckstein and Sherk, 2011). Therefore, research is needed at this time that addresses the fate, occurrence, and environmental damage of PPCPs, along with assessing the regulatory framework and management implications to prevent the release of PPCPs into the aquatic environment.

Objectives

This research seeks to provide data that can help to fill the gaps in the current literature surrounding PPCPs in wastewater and the environment. Therefore, the goal of this work is to advance the understanding of PPCPs in the aquatic environment and to

then use this knowledge to engage in the management and policy discussions regarding these complex pollutants. In doing so, this research aims to answer three primary questions:

1) Is it possible to predict the removal of PPCPs from a WWTP that utilizes a conventional active sludge (CAS) system?
2) What is the fate, occurrence, and corresponding environmental risk quotient for PPCPs in Lake Michigan and the Milwaukee Harbor?
3) Is it feasible to modify wastewater treatment regulations to remove unregulated PPCPs to levels that will minimize environmental harm?

To answer these questions, two experiments and a case study are presented. The objective of **Chapter 2**, published in *Science of the Total Environment*, was to assess the fate and occurrence of PPCPs across the different stages of a wastewater treatment plant. This chapter presented two advances in the field of PPCPs in wastewater: a dataset that assessed 54 PPCPs on six dates for a conventional actived sludge WWTP and the assessment of a model that determined the fate across an activated sludge treatment process. The results showed that of the 54 PPCPs assessed, 48 were detected above the level of detection at some time in wastewater. The *in situ* data were then used to show that PPCPs with a log octanol-water partitioning coefficient (K_{ow}) that is greater than 4.5 are removed through a combination of sorption to solids and biodegradation. This study demonstrated that BIOWIN4 from the USEPA's EPI Suite can be used to establish a basic understanding of the biological degradation of PPCPs across an aerobic biological treatment process. Overall, this chapter advanced the understanding of PPCPs in

wastewater through the use of the log K_{ow} along with BIOWIN4 integrated into pseudo-first order kinetics to predict the removal of easily degradable and recalcitrant PPCPs from a WWTP.

The objective of **Chapter 3**, published in *Chemosphere*, was to assess the occurrence of PPCPs in the water and sediments of Lake Michigan, including sites 3.2 km (2 miles) from shore. The sampling sites included the area surrounding the South Shore Water Reclamation Facility outfall and within the Milwaukee Harbor. Using the risk quotient, this chapter presented that PPCPs were detected at concentrations that are estimated to cause environmental concern and these results are of great importance because these area are near locations for fish spawning and aquatic organisms. Of the 54 PPCPs assessed, 32 PPCPs were detected in Lake Michigan water and 30 PPCPs were detected in the sediment above the level of detection. Using the risk quotient, it was found that medium or high risk was associated with twenty-four compounds in the final effluent, and fourteen were found to be of medium or high risk in Lake Michigan.

Further research is needed to understand how emerging wastewater treatment processes impact the fate of PPCPs so that we can minimize the discharge and associated risks of PPCPs in the environment. The objective of **Chapter 4** was to assess whether current wastewater regulations could be modified to significantly reduce the emission of PPCPs. More specifically, this chapter will assess whether the regulations and technologies to address phosphorus emissions from WWTPs could also remove PPCPs. To accomplish this objective, this chapter applied a meta-analysis of PPCPs removal from tertiary treatment technologies to a case study using the potential WWTPs upgrades

under a statewide phosphorus reduction policy. Two of the technologies proposed to meet the improved phosphorus effluent regulations also significantly reduced the risk quotient from PPCPs, the tertiary membrane bioreactor and nanofiltration, and the median risk quotient from PPCPs was estimated to be reduced by 71 and 81 percent, respectively. In addition, ultrafiltration was estimated to reduce the median risk quotient by 28 percent, with no additional cost under the current phosphorus regulations. Therefore, the conclusion was reached that evaluating nutrient reduction policies to minimize emissions of PPCPs should be considered by managers and policy makers.

Implications of the Present Studies

Collectively, the results of these projects will help us further understand the fate, occurrence, and ecological damage from PPCPs in wastewater and the aquatic environment, along with advancing the debate on how to accomplish meaningful reductions in these pollutants. The overarching goal of this work is to contribute to the scientific and political debate on how to achieve a significant reduction in the amount of PPCPs being emitting into the aquatic environment.

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Chapter 2: EVALUATION OF A MODEL FOR THE REMOVAL OF PHARMACEUTICALS, PERSONAL CARE PRODUCTS, AND HORMONES FROM WASTEWATER

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Abstract

Current wastewater treatment processes are insufficient at removing many pharmaceutical and personal care products (PPCPs) from wastewater and it is necessary to identify the chemical characteristics that determine their fate. Models that predict the fate of various chemicals lack verification using in situ data, particularly for PPCPs. BIOWIN4 is a quantitative structure–activity relationship (QSAR) model that has been proposed to estimate the removal of PPCPs from wastewater, but data verifying the accuracy of its predictions is limited. In this study, the in situ soluble and suspended solid concentrations were assessed from raw influent, primary effluent, secondary effluent, and final effluent for 54 PPCPs and hormones over six dates. When assessing the removal efficiency across the different stages of the WWTP, the majority of the removal occurred across the secondary treatment process for the majority of the compounds. The primary treatment and disinfection process had limited impacts on the removal of most PPCPs. Sorption to solids was found to influence the removal for compounds with a log octanol–water partitioning coefficient greater than 4.5 across the secondary treatment process. For other compounds, the removal of PPCPs across the secondary treatment process was significantly correlated with the biodegradation predicted by BIOWIN4. Removal efficiencies across the aerobic secondary treatment process were predicted by integrating BIOWIN4 into pseudo-first order kinetics of PPCPs and these predicted values were compared to the in situ data. This study determines that under a certain set of operating

conditions, two chemical characteristics — the expected hydrophobic interaction and the modeled biological degradation from BIOWIN4 — were found to predict the removal of highly degradable and recalcitrant PPCPs from a wastewater secondary treatment process.

Introduction

Pharmaceuticals and personal care products (PPCPs) have been detected in surface waters worldwide and risk analysis studies have led to the concern that these PPCPs may have a negative impact on ecosystem and human health (Al Aukidy et al., 2012; Cahill et al., 2004; Fatta-Kassinos et al., 2011; Fent et al., 2006; Focazio et al., 2008; Gros et al., 2010; Halling-Sorensen et al., 1998; Kolpin et al., 2002; Ternes, 1998). In general, the removal of PPCPs through wastewater treatment plants (WWTPs) has been shown to depend on the PPCPs' chemical characteristics, the treatment processes used, and the concentration found in the influent, but other variables can influence the removal efficiencies (Le-Minh et al., 2010; Oulton et al., 2010; USEPA, 2010; Verlicchi et al., 2012).

The understanding of the fate of PPCPs in wastewater is limited, in particular, the removal efficiency across the varying configurations of WWTPs. WWTPs that utilize conventional activated sludge (CAS) systems have been found to present a wide range of removal efficiencies for different PPCPs (Miege et al., 2009; Oulton et al., 2010; Verlicchi et al., 2012). The removal of PPCPs through primary and secondary treatment can be explained through three mechanisms: sorption to sludge, biological degradation,

and volatilization (Khan and Ongerth, 2002). The incomplete removal of many PPCPs is primarily due to the resistance of these compounds to biological degradation (Joss et al., 2006). The hydrophobic interaction, which explains sorption to solids, is expected to be significant for PPCPs with a log octanol– water partitioning coefficient (K_{ow}) greater than 4.0 (Thompson et al., 2011) and removal by sorption was concluded to be a minor pathway for most PPCPs (Radjenovic et al., 2009). Additionally, Khan and Ongerth (2004) estimated that removal by sorption accounted for 10% or less of the overall removal of 47 of the top 50 PPCPs in use in Australia. Volatilization can be considered negligible for the majority of PPCPs (Joss et al., 2006).

The biological degradation rate constant (K_{biol}) has been suggested to be a strong indicator of the removal efficiency of PPCPs due to biological transformation being the major elimination mechanism (Abegglen et al., 2009; Salgado et al., 2012; Thompson et al., 2011). However, the K_{biol} values for many PPCPs are not available (Dickenson et al., 2010). Since the biological degradation is the key mechanism for the removal of many PPCPs, finding these values is critical to understanding the removal efficiency. The K_{biol} values have been found for a limited number of compounds using pseudo-first-order kinetics (Joss et al., 2006; Schwarzenbach et al., 2003). At this time, the majority of K_{biol} values used in modeling the removal of PPCPs are estimated using their chemical composition and characteristics (Dickenson et al., 2010). However, with over 3000 pharmaceuticals currently in use (Monteiro and Boxall, 2010), being able to predict the removal of these PPCPs without in situ testing is necessary.

BIOWIN, a quantitative structure–activity relationship (QSAR) based model that is included in the Environmental Protection Agency's EPI Suite, estimates the probability of biodegradation based on mathematical models for predicting aerobic biodegradability from chemical structure (Boethling et al., 1994). BIOWIN models have been used to predict the biological half-life of pollutants with varying success (Aronson et al., 2006). One of the BIOWIN models, BIOWIN4, was designed as an expert survey model for primary biodegradation estimation and it calculates the time required to achieve primary biodegradation in a typical aquatic environment (Boethling et al., 1994). It has been predicted that BIOWIN4 can be used to determine the aerobic biodegradation in WWTP systems (Clark et al., 1995; Khan and Ongerth, 2002).

While many studies have investigated the fate of PPCPs during secondary treatment, these studies are seldom combined with an in situ evaluation of a model. This is often due to the difficulties in predicting the fate of PPCPs in WWTPs and the number of variables that influence the removal efficiency. The purpose of this study was to detect the concentration of 54 PPCPs with varying chemical characteristics across the stages of a WWTP and to use these data to verify a model assessing the fate of PPCPs in wastewater. This paper presents a substantial PPCP monitoring data set by collecting these PPCPs at the raw influent, primary effluent, secondary effluent, and final effluent stages. With the use of these data, the observed PPCPs removal efficiencies are compared to the predicted removal efficiencies from a simple model that integrates BIOWIN4 into pseudo-first order kinetics.

Materials and methods

PPCPs were measured at South Shore Water Reclamation Facility (SSWRF) in Oak Creek, WI, which is a facility that services the greater Milwaukee, WI area. Samples of raw influent, primary effluent, secondary effluent and final effluent were collected on six dates over a two-year period (Spring 2009–Fall 2010). SSWRF uses preliminary treatment (7 bar screens/grit channels), primary treatment (16 primary clarifiers), activated sludge treatment (28 aeration basins and 24 secondary clarifiers) and chlorine disinfection (2 5-pass contact channels). SSWRF has a treatment capacity of 1,135,000 m^3 day^{-1} (300 MGD (million gallons day^{-1})) with an average flow of approximately 379,000 m^3 day^{-1} (100 MGD). The base flow is 246,000 to 284,000 m^3 day^{-1} (65 to 75 MGD) in dry conditions. The disinfection process used variable levels of sodium hypochlorite and sodium bisulfite and the dosage was adjusted to obtain the required disinfecting performance and to obtain zero residual chlorine. The pH on the sampling dates ranged from 6.73 to 7.06, with the average being 6.91. All samples were taken over a 24-hour period. The sampling for the raw influent and final effluent were flow proportional and the primary effluent and secondary effluent were time proportional. The influent was sampled using a Hach/Sigma SD 900 instrument that was set to collect a composite sample in flow pace with a target of 6–8 L. The system would cycle every 2000 to 5000 gal depending on influent flow to the plant and collected 50 mL of sample each cycle. The primary effluent was sampled using a Sanford instrument where 20 mL

was collected every 4 min for a final volume of 6–8 L. The secondary effluent was sampled using a grab method of 250 mL every 3 h. The final effluent was sampled using an ISCO 6712FR instrument with the same flow dependent setup as the influent sample. All samples were then well mixed and 1 L was sampled and extracted.

PPCP analysis

Methods are based upon US EPA Method 1694 (USEPA, 2007a), which determines PPCPs in environmental samples and US EPA Method 1698 (USEPA, 2007b), which determines steroids and hormones in environmental samples by high performance liquid chromatography combined with tandem mass spectrometry (HPLC/MS/MS). One liter liquid samples were filtered through Whatman GF-A glass fiber filter media to retain particulate material. The filtrate pH was adjusted to 2 with concentrated sulfuric acid and 0.5 g of EDTA was added to chelate minerals for acid analytes (or pH adjusted to 10 with concentrated ammonium hydroxide for basic extraction analytes).

Liquid samples were then spiked with a suite of mass labeled internal standard compounds and extracted with 20 mL, 1 g Waters Oasis HLB cartridges and eluted sequentially with 12 mL methanol, 6 mL methanol:acetone (50:50), and 6 mL MTBE:methanol (90:10) for acid analytes or 6 mL of methanol followed by 9 mL methanol with 2% formic acid for basic analytes. The elution solvents were concentrated under nitrogen to approximately 0.2 mL and quantitatively transferred to 1.0 mL final volume with methanol pending analysis.

Liquid samples for hormones were spiked with a suite of mass labeled internal standard compounds and extracted with 6 mL, 200 mg Biotage Isolute ENV+ cartridges and eluted sequentially with 6 mL methanol and 6 mL methanol:ethyl acetate (50:50). The elution solvents were concentrated under nitrogen to approximately 0.2 mL and quantitatively transferred to 0.5 mL final volume with methanol pending analysis.

Particulates were captured on glass fiber filters from the April 9, 2010 sampling date and these data are presented separate from the liquid concentration in the results. This study did not assess the concentration of PPCPs in the waste activated sludge. An extraction of the particulates that were filtered was conducted and then an additional solids extraction was completed for those particulates. Aliquots (1 g) of filtered solids were placed in 50 mL polypropylene centrifuge tubes with pH 2 phosphate buffer: acetonitrile, spiked with a suite of mass labeled internal standard compounds, and extracted three times by sonication. The pooled acetonitrile was removed from the extract using a rotary evaporator and the aqueous extract was brought to 200 mL volume with 18 MΩ/cm water before further processing by the liquids method described above. From the extract, 15 µL was injected onto a Phenomenex Synergi MAX-RP 250×4.6 mm, 4 µm column and separated by a binary gradient employing an Agilent 1100 HPLC system. Detection was achieved with an Applied Biosystems/ MDS SCIEX API 4000 MS/MS system operating with Turbo Ion Spray ionization and multiple reaction monitoring (MRM) detection. The data are presented in Appendix A.

BIOWIN assessment

The USEPA's Estimation Program Interface (EPI) Suite v4.10 was used to find the BIOWIN4 values and log K_{ow} (USEPA, 2011). The QSAR model used was BIOWIN4 and it was designed as an expert survey model to determine the time needed for primary biodegradation (Boethling et al., 1994). BIOWIN4 was used in this paper due to it providing the best estimate of biodegradation when compared with measured first-order rate constants (Dickenson et al., 2010). The BIOWIN4 values are interpreted as the environmental biological degradation timeframe, where 2=months, 3=weeks, and 4=days. Along with predicting the environmental biological degradation timeframe, BIOWIN4 has been predicted to determine the aerobic biodegradation timeframe in WWTP systems using the relationship developed by Khan and Ongerth (2002). Also, ultimate degradation was not studied in the present paper, which reinforced the decision to use the primary biodegradation estimation model, BIOWIN4.

The median suspended solid concentration (X_{ss}) on the five sampling dates was used for the pseudo first-order calculation. On the sampling dates, the median hydraulic retention time in the aeration basin was 9.8 h and the median hydraulic retention time in the secondary clarifier was 7.3 h. Using the settling velocity and activated sludge blanket depth, it was estimated that the soluble compounds were in contact with the activated sludge for approximately 30% of hydraulic retention time in the secondary clarifier. When the compounds were not in contact with the activated sludge, it was assumed that the biodegradation was negligible. When optimizing the biological degradation equation to correspond with the experimental data only the compounds with a K_{ow} of less than 4.5

were used. For this assessment, the compounds with negative removal efficiency were considered to have no removal. If the secondary effluent concentration was below the minimum detection limit, the compound was considered to be 100% removed. The removal efficiencies were found for each date and the median of these values was used for this analysis. Calculations are shown in Appendix B.

Detection limits and statistics

The minimum detection limit (MDL) and minimum quantification limit (MQL) were found using the USEPA CFR 40, part 136 (USEPA, 2003). PASW Statistics v18 was used to find the statistical correlation and significance when finding the relationships between BIOWIN4 and removal efficiency and between the modeled and observed removal efficiency.

Results and discussion

SSWRF PPCP Levels

The concentrations of 54 PPCPs, at the raw influent, primary effluent, secondary effluent, and final effluent sites, are shown in Table 1. The minimum detection limit and minimum quantification limit are also shown. The suspended solids were filtered from the April 9, 2010 samples and these were assessed for sorption of PPCPs to the solids and the results are shown in Table 2. All compounds not shown in Table 2 had levels below MDL for all 4 stages across the WWTP. The compounds with log K_{ow} values greater than 4.5, triclosan and triclocarban, had the highest concentrations found in the suspended

solids. Of the 54 compounds listed in Table 1, 48 of them were detected at a level above the MDL at some time in SSWRF. However, substantial variations were seen for the majority of the compounds across dates and the stages of SSWRF.

When assessing the removal efficiency across the different stages of the WWTP, the majority of the removal occurred across the secondary treatment process for the majority of the compounds. For example, caffeine which is shown in Fig. 1, had a high removal efficiency, but caffeine also had notable values in the final effluent. In contrast to caffeine, many compounds, such as codeine, had poor removal efficiencies as shown in Fig. 2.

The removal efficiency was low for the majority of PPCPs across the primary clarifier, but some PPCPs had notable removal efficiencies. One compound, ibuprofen, had a high removal efficiency (88% median removal efficiency) in the primary treatment process. Some PPCPs that had moderate removal across the primary treatment process were acetaminophen (22% median removal efficiency), estrone (59% median removal efficiency), fluoxetine (45% median removal efficiency), metformin (24% median removal efficiency), paraxanthine (22% median removal efficiency), and triclosan (32% median removal efficiency).

Minimal PPCPs were removed across the disinfection process, which used sodium hypochlorite for disinfection and sodium bisulfite for de-chlorination. The compounds that had notable removal across the disinfection stage were caffeine (69% median removal efficiency), gemfibrizol (60% median removal efficiency), naproxen (73% median removal efficiency), paraxanthine (78% median removal efficiency), and

sulfanilamide (41% median removal efficiency). Caffeine has been shown to have no interaction with chlorine and gemfibrizol was implied to be chlorinated (Glassmeyer and Shoemaker, 2005). The removal of caffeine across the disinfection process could be due to the biological degradation prior to complete disinfection. Some compounds, such as naproxen, have been shown to have a high removal efficiency (>80%) from chlorine disinfection (Benotti et al., 2009).

A few compounds had negative removal efficiencies across the entire treatment plant where lower concentrations were seen in the raw influent than the final effluent. Also, many compounds had a negative removal efficiency across one of the stages assessed at SSWRF. Numerous explanations are available for the observed negative removal efficiency and five possible explanations apply specifically to this study. First, shifting the primary effluent, secondary effluent, and final effluent sampling periods by the hydraulic retention time or sampling using the residence time distribution may lower the potential for errors (Majewsky et al., 2011a). Second, 24-hour composite samples may be insufficient to determine PPCP removal in WWTPs (Ort et al., 2010). Third, some compounds have also been proposed to have conjugate compounds that are not detected at the influent but retransformed into the original compound due to biological processes (Monteiro and Boxall, 2010; Salgado et al., 2012). Fourth, desorption from the return activated sludge may occur during the secondary treatment process (Salgado et al., 2012). Finally, PPCPs may be released from fecal particles as the feces are being broken down by microbes (Göbel et al., 2007).

BIOWIN4 results

The median removal efficiency from each date was used when assessing BIOWIN4. When using very low concentrations to establish a relationship with removal efficiencies, the low concentrations will cause unavoidable instrumental errors that may affect their observed removal values (Verlicchi et al., 2012). To minimize this error in the removal efficiency calculations, the compounds selected for assessment using BIOWIN had at least three of the five samples with a primary effluent concentration greater than the MQL. Also, since using 24-hour composite samples may not adequately determine the removal of PPCPs (Ort et al., 2010), using the median value from the three to five sampling dates will create a stronger representative removal efficiency value. Only the data from the dates where the detection was greater than the MQL at the primary effluent site were used. If the secondary effluent concentration was less than the MQL, the removal efficiency assigned to this compound was 100%. The following eighteen met these criteria and were further analyzed: acetaminophen, caffeine, carbamazepine, codeine, cotinine, diltiazem, diphenhydramine, fluoxetine, ibuprofen, metformin, naproxen, paraxanthine, ofloxacin, ranitidine, sulfamethoxazole, triclocarban, triclosan, and trimethoprim.

As shown in Fig. 3, a statistically significant correlation (Pearson correlation=0.773, $p<0.001$) between the observed removal efficiency across the secondary treatment process and the BIOWIN4 estimated biological degradation in the environment was found. This relationship does not include the compounds with a log K_{ow}

greater than 4.5, therefore, the relationship is based on the remaining 16 PPCPs with primary effluent concentrations consistently above their respective MDLs. Triclosan and triclocarban were two compounds assessed that had a log K_{ow} greater than 4.5. These two compounds' high removal efficiency and lowBIOWIN4 value, implies removal by sorption to solids as well as limited biological degradation.

BIOWIN and half-life

It has been approximated that the BIOWIN4 values can be converted to biological degradation half-lives (t½) in an aeration tank with using the following relationship (Khan and Ongerth, 2004):

$$t_{½} = 10^{(5-\text{BIOWIN4})} \quad (1)$$

Using the expected biological degradation half-life from Eq. (1), the intrinsic biological rate constant (K_{biol}) can be found using Eq. (2):

$$K_{biol} = \ln 2 / t_{½} \quad (2)$$

The pseudo first-order kinetic equation (Joss et al., 2006) was used to convert the biological half-life to removal efficiency across the secondary treatment process:

$$\frac{dC_t}{dt} = -K_{biol} X_{ss} C_0 \quad (3)$$

where, C_t is the soluble compound concentration at time t (ng L^{-1}), t is hydraulic retention time (day), K_{biol} is the biological rate constant (L g_{ss}^{-1} day^{-1}), X_{ss} is the concentration of suspended solids (g_{ss} L^{-1}), and C_0 is the initial soluble compound

concentration (ng L^{-1}). This median concentration of mixed liquor suspended solids at SSWRF was 1.74 g L^{-1}. By integrating Eq. (3) and combining it with Eq. (2), the results are shown in Eq. (4):

$$\ln(C_t/C_0) = -\left[\frac{\ln 2}{t_{1/2}}\right] X_{ss} t \tag{4}$$

Using Eq. (4), the predicted results relate significantly to the actual removal observed at SSWRF (Pearson correlation=0.792, p<.001). The PPCPs with a log K_{ow} greater than 4.5 were omitted from this analysis. These results are shown in Fig. 4. This relationship demonstrates that BIOWIN4 paired with pseudo-first order kinetics may be used to predict the removal of PPCPs across a secondary treatment process for compounds that are expected to biological degrade quickly (i.e. acetaminophen, naproxen) and the recalcitrant compounds (i.e. codeine, ofloxacin).

The compounds with a BIOWIN4 value between 3.3 and 3.7 are more difficult to predict. For example, caffeine and carbamazepine have similar BIOWIN4 values, 3.57 and 3.51 respectively, but caffeine had a high removal efficiency and carbamazepine was not removed. Other studies have reported incomplete removal efficiencies for carbamazepine while caffeine has been shown to be degraded in aWWTP (>50% removal efficiency) (Santos et al., 2007; Radjenovic et al., 2009; Rosal et al., 2010; Vieno et al., 2007). This demonstrates the difficulty in assessing the removal of PPCPs from wastewater and the variability seen in the removal efficiencies for many compounds.

The results from the use of BIOWIN4 with the removal of PPCPs across the aeration basin were more accurate than reported by Dickenson et al. (2010). These results

suggest that using a combination of BIOWIN models may not be necessary to accurately predict removal of PPCPs across an aeration basin as proposed by Posthumus et al. (2005); BIOWIN4 was found to have a statistically significant relationship with the removal of PPCPs across the aeration basin.

Other variables have been shown to impact removal, such as the fraction of active biomass, solids retention time (SRT), recirculation rate, water pH, temperature, reactor configuration, molecular charge, and hydraulic retention time (Abegglen et al., 2009; Fatta-Kassinos et al., 2011; Majewsky et al., 2011b; Suarez et al., 2012; Tadkaew et al., 2010; Verlicchi et al., 2012). For example, the SRT at SSWRF was between 8.0 and 15.5 days. WWTPs with a SRT of 8 days or higher used for nitrification will have higher removal efficiencies for PPCPs in comparison with WWTPs without nitrification (Abegglen et al., 2009). Furthermore, the solubility should also be considered in relation to the pH of the matrix in which it is present (Fatta-Kassinos et al., 2011); however, the average pH at SSWRF on the sampling dates was 6.9. Therefore, these variables must all be considered when assessing the removal of PPCPs from a WWTP.

Since this analysis focuses on primary biological degradation and sorption to solids, it may be neglecting other degradation or removal processes. For example, triclosan has been shown to create polychlorodibenzo-p-dioxin (PCDD) photoproducts when exposed to UV light combined with the chlorination process that is commonly used in the wastewater processes and this is important because these PCDD photoproducts have been found in the environment (Buth et al., 2009). An additional limitation of this study is that BIOWIN4 assesses primary degradation and future research should also

place an emphasis on metabolites and transformation products since it is necessary to evaluate the fate of these transformation products (Farre et al., 2012).

Conclusion

This paper presented two advances in the field of PPCPs in wastewater: a dataset that assesses the occurrence and fate of 54 PPCPs on six dates for a CAS WWTP and the assessment of a model that determines the fate across an activated sludge treatment process. Of the 54 PPCPs assessed, 48 were detected in SSWRF and the concentrations at each treatment stage were determined. The large number of variables that could influence the removal efficiency causes great difficulty in determining an intrinsic biological degradation rate constant. These in situ data showed that PPCPs with a log K_{ow} that is greater than 4.5 are removed through a combination of sorption to solids and biodegradation. This study also demonstrates that BIOWIN4 can be used to establish a basic understanding of the removal of PPCPs across an aerobic biological treatment process. Overall, using log K_{ow} along with BIOWIN4 integrated into pseudo-first order kinetics, it was possible to predict the removal of easily degradable and recalcitrant PPCPs from the aerobic treatment process at SSWRF.

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Table 1: Classification, Minimum Detection Limit (MDL), Minimum Quantification Limit (MQL), range and median values for compounds assessed at SSWRF. Below Detection Limit (BDL) values are below the MDL.

	Classification	MDL	MQL	Raw Influent	Primary Effluent	Secondary Effluent	Final Effluent
				Min - Max, Median	Min - Max, Median	Min - Max, Median	Min - Max, Median
		ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}
17,20-dihydroxyprogesterone	Sex Hormone	1.4	4.2	BDL - 3.8*, BDL	BDL - 2.9*, BDL	BDL - BDL, BDL	BDL - BDL, BDL
17-alpha-estradiol	Sex Hormone	1.2	3.5	BDL - 10000, BDL	BDL - 760000, BDL	BDL - 2900, BDL	BDL - 4700, BDL
17-beta-estradiol	Sex Hormone	1.3	3.8	BDL - 9.4, BDL	BDL - 11, BDL	BDL - BDL, BDL	BDL - 2.8*, BDL
4-androstene-3,17-dione†	Sex Hormone	0.5	1.4	BDL - 150, 12	BDL - 73, 0.8*	BDL - 1.9, BDL	BDL - 2.3, BDL
5-alpha-androstane-3,17-dione	Anabolic Agent	2.3	6.9	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - 24, BDL
Acetaminophen	Antipyretic, Analgesic	2.5	7.5	5900 - 150000, 18000	8000 - 150000, 14000	BDL - 22000, 29	BDL - 650, 39
Albuterol	Antiasthmatic	1.4	4.2	BDL - 23, 6.3	BDL - 69, 7.2	BDL - 12, BDL	BDL - 2.6*, BDL
Azithromycin†	Macrolide Antibiotic	3.7	11.0	BDL - 280, BDL	BDL - 340, 6.9*	BDL - 47, 6.5*	BDL - 350, 110
Boldenone	Anabolic Steroid	1.3	4.0	BDL - 170, 13	BDL - 16, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Caffeine†	Stimulant	3.1	9.3	3300 - 130000, 9200	4200 - 110000, 9400	34 - 7800, 1000	BDL - 1400, 310
Carbadox†	Quinoxaline Antibiotic	3.4	10.1	BDL - 44, BDL	BDL - 68, BDL	BDL - BDL, BDL	BDL - 15, BDL
Carbamazepine	Anticonvulsant	2.7	8.2	21 - 310, 72	24 - 310, 73	33 - 170, 88	27 - 340, 180
Cimetidine	Anti-acid reflux	1.3	3.8	BDL - 39, BDL	BDL - 120, BDL	BDL - 18, BDL	BDL - BDL, BDL
Ciprofloxacin	Quinoline antibiotic	3.3	9.9	BDL - 87, BDL	BDL - 19, BDL	BDL - 16, BDL	BDL - BDL, BDL
Clarithromycin	Macrolide antibiotic	3.2	9.6	BDL - 5.6*, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - 19, BDL
Codeine	Opiate	3.6	10.7	15 - 540, 45	15 - 460, 43	9.6* - 170, 62	BDL - 230, 100
Cotinine	Nicotine metabolite	3.5	10.6	BDL - 130, 18	BDL - 810, 28	BDL - 810, BDL	BDL - BDL, BDL
Digoxigenin	Cardanolide Steroid	4.4	13.2	BDL - 850, 26	BDL - 710, 34	BDL - 68, BDL	BDL - BDL, BDL

Diltiazem†	Antihypertensive	3.5	10.4	20 - 640, 52	17 - 720, 41	BDL - 160, 38	BDL - 510, 45
Diphenhydramine†	Antihistamine	3.6	10.9	11 - 420, 35	7* - 420, 24	5.8* - 140, 22	BDL - 360, 54
Estriol	Sex Hormone	2.0	6.1	BDL - 22, BDL	BDL - 44, 3*	BDL - 6.1, BDL	BDL - BDL, BDL
Estrone	Sex Hormone	2.2	6.7	BDL - 350, 64	BDL - 290, 26	BDL - BDL, BDL	BDL - BDL, BDL
Fluoxetine	SSRI Antidepressant	3.5	10.5	6.1* - 95, 20	4* - 120, 11	5* - 25, 8.3*	BDL - 96, 28
Gemfibrozil	Antilipemic	1.6	4.8	29 - 1200, 180	62 - 1100, 500	85 - 1100, 420	30 - 1100, 170
Ibuprofen	Analgesic	4.7	14.0	670 - 11000, 2100	BDL - 14000, 260	BDL - 4000, BDL	BDL - BDL, BDL
Lincomycin	Lincosamide antibiotic	3.1	9.3	BDL - 25, BDL	BDL - 29, BDL	BDL - BDL, BDL	BDL - 15, BDL
Lomefloxacin	Quinoline antibiotic	4.7	14.2	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Melengestrol	Steroid Hormone	1.3	4.0	BDL - 43, BDL	BDL - 49, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Melengestrol Acetate	Steroid Hormone	0.6	1.7	BDL - 1300, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Metformin†	Anti-diabetic drug	0.5	1.5	3200 - 100000, 55000	9800 - 92000, 42000	800 - 33000, 27000	640 - 47000, 26000
Miconazole	Tetracycline antibiotic	2.7	8.1	BDL - 81, BDL	BDL - 69, BDL	BDL - 6.4*, BDL	BDL - 25, 3*
Naproxen	NSAIDs	1.0	2.9	780 - 9400, 3000	260 - 11000, 3000	19 - 4000, 520	8.3 - 580, 140
Norfloxacin	Quinoline antibiotic	5.1	15.3	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Ofloxacin	Quinoline antibiotic	3.9	11.7	BDL - 980, 16	BDL - 530, BDL	BDL - 220, 11*	BDL - 670, 44
Oxacillin	β-lactam antibiotics	2.5	7.4	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Paraxanthine	Caffeine Metabolite	6.1	18.2	740 - 15000, 3800	1500 - 13000, 3000	25 - 2700, 540	BDL - 770, 120
Progesterone†	Sex Hormone	0.7	2.0	BDL - 23, 3.7	BDL - 8.7, BDL	BDL - 0.8*, BDL	BDL - 2.9, BDL
Ranitidine	Anti-acid reflux	0.9	2.6	BDL - 130, 16	BDL - 330, 53	BDL - 29, BDL	BDL - 13, BDL
Roxithromycin†	Macrolide antibiotic	4.3	13.0	BDL - 1500, BDL	BDL - 88, BDL	BDL - BDL, BDL	BDL - 110, 9.2*
Sarafloxacin	Fluoroquinolone antibiotic	5.4	16.3	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Sulfachloropyridazine	Sulfonamide antibiotic	4.1	12.3	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Sulfadiazine	Sulfonamide antibiotic	2.8	8.5	BDL - 2.8*, BDL	BDL - BDL, BDL	BDL – 3*, BDL	BDL - 5.7*, BDL
Sulfadimethoxine	Sulfonamide antibiotic	2.4	7.1	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - 13, BDL
Sulfamerazine	Sulfonamide antibiotic	2.1	6.2	BDL - BDL, BDL	BDL - BDL, BDL	BDL - 3.2*, BDL	BDL - BDL, BDL
Sulfamethazine	Sulfonamide antibiotic	4.0	12.1	BDL - BDL, BDL	BDL - 48, BDL	BDL - BDL, BDL	BDL - BDL, BDL

Sulfamethizole	Sulfonamide antibiotic	4.2	12.7	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Sulfamethoxazole	Sulfonamide antibiotic	4.1	12.4	54 - 1200, 140	21 - 1300, 93	34 - 300, 67	17 - 810, 180
Sulfanilamide	Sulfonamide antibiotic	2.9	8.6	BDL - 900, 57	BDL - 2500, 21	BDL - 68, 42	BDL - 900, 25
Sulfathiazole	Sulfonamide antibiotic	2.6	7.8	BDL - 3.8*, BDL	BDL – 5.0*, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Testosterone†	Sex Hormone	1.1	3.2	BDL - 25, 1.7*	BDL - 13, BDL	BDL - BDL, BDL	BDL - BDL, BDL
Thiabendazole	Fungicide	1.8	5.3	BDL - 26, BDL	BDL - 19, BDL	BDL - 9.5, BDL	BDL - 16, 6.8
Triclocarban	Antimicrobial	0.5	1.4	3.3 - 5900, 120	17 - 2800, 260	61 - 120, 74	27 - 980, 120
Triclosan†	Antimicrobial	0.5	1.6	89 - 9100, 650	250 - 5700, 440	24 - 350, 120	BDL - 850, 97
Trimethoprim	Pyrimidine antibiotic	3.4	10.1	18 - 590, 49	19 - 510, 44	21 - 260, 41	BDL - 660, 120

* Value above MDL, but below MQL

† Compound where values above the MDL were found in the method blanks, see Appendix A for values

Table 2: PPCPs found in suspended solids on April 9th, 2010

Compound	Raw Influent (ng g^{-1})	Primary Effluent (ng g^{-1})	Secondary Effluent (ng g^{-1})	Final Effluent (ng g^{-1})
Acetaminophen	12	BDL	BDL	BDL
Caffeine	142	33	BDL	BDL
Diltiazem	7*	BDL	BDL	BDL
Diphenhydramine	23	BDL	BDL	BDL
Fluoxetine	18	BDL	BDL	BDL
Naproxen	BDL	8	BDL	BDL
Ofloxacin	45	BDL	8*	BDL
Triclocarban	3280	108	BDL	155
Triclosan	5601	182	BDL	31

* Value above MDL, but below MQL.

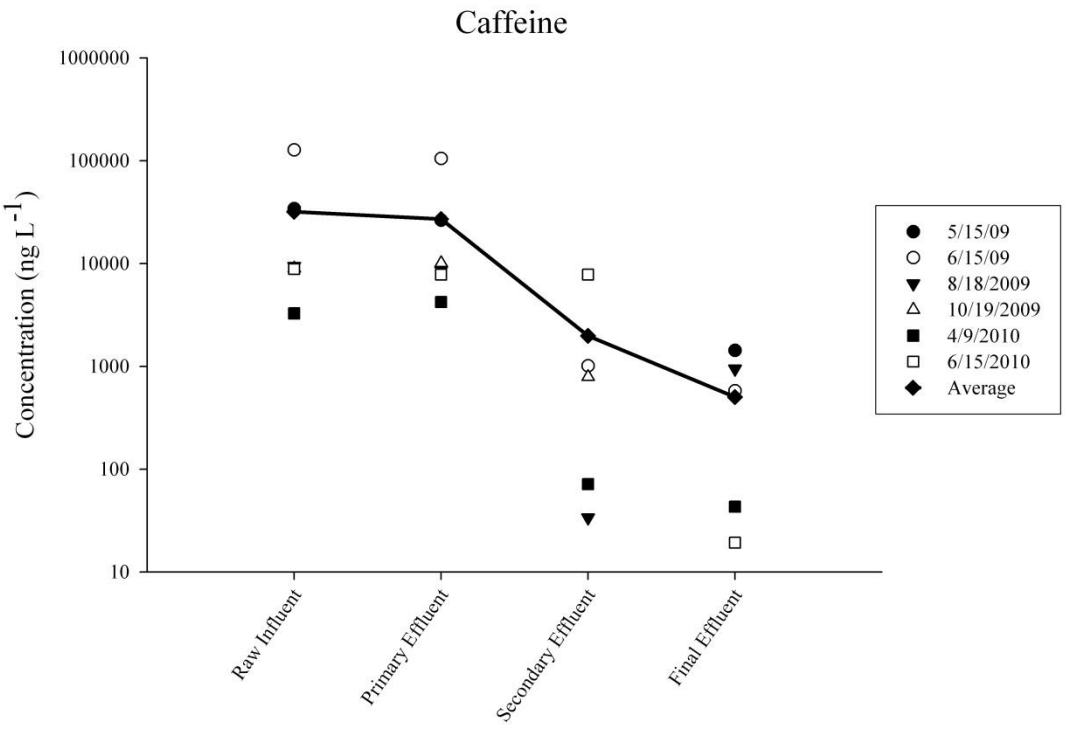

Figure 1. Concentration of Caffeine across the Stages of SSWRF on Six Dates

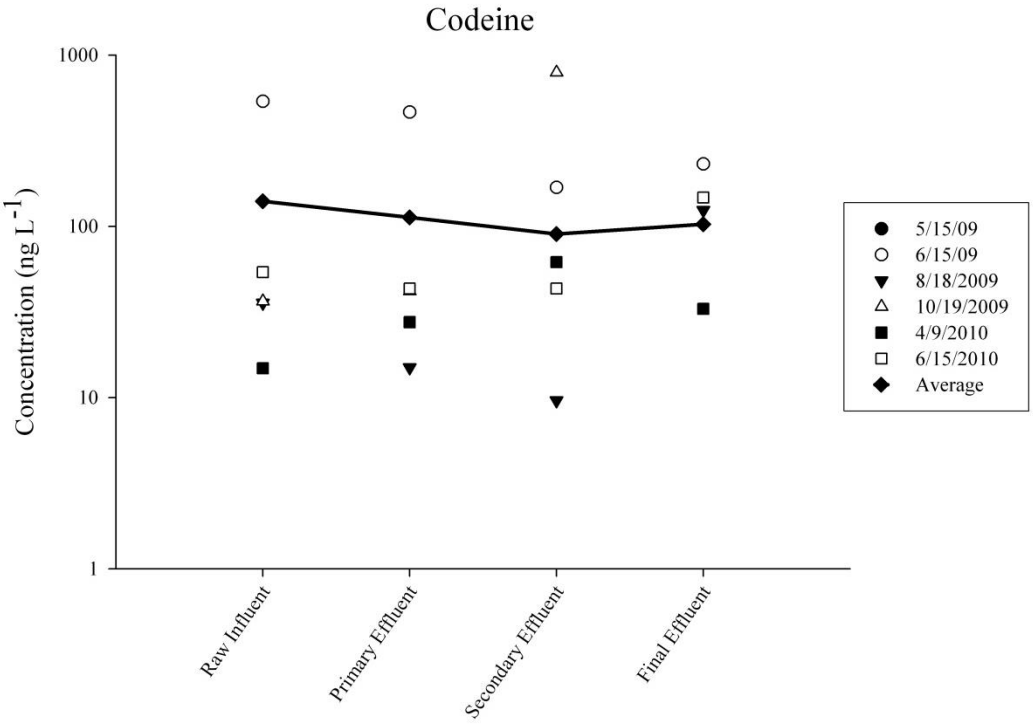

Figure 2. Concentration of Codeine across the Stages of SSWRF on Six Dates

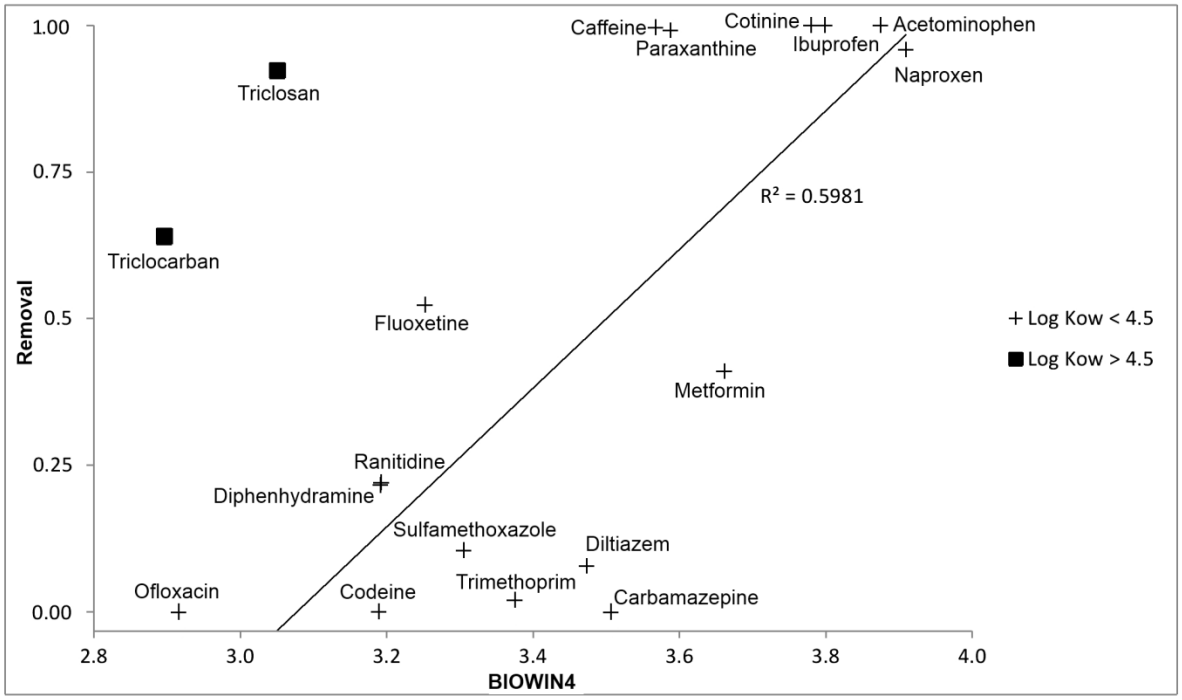

Figure 3. Median Removal Efficiency across the Secondary Treatment Process at SSWRF Compared to BIOWIN4 (Pearson Correlation = 0.773, $p < 0.001$, coefficient of determination and correlation includes only log K_{ow} values < 4.5)

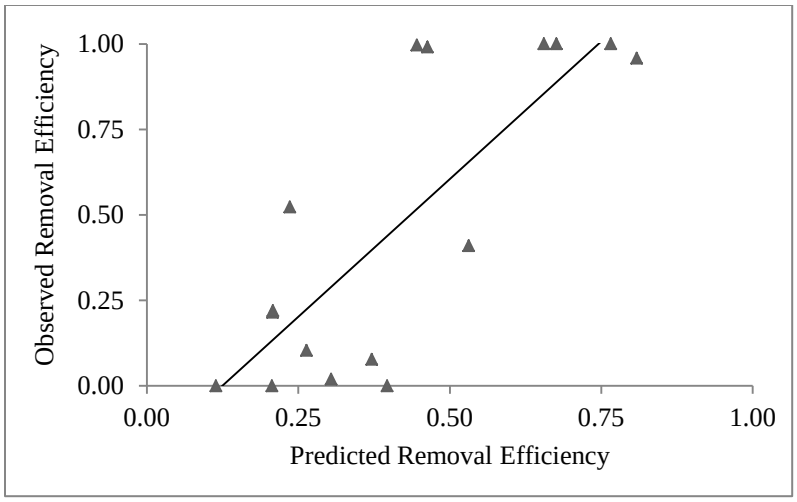

Figure 4. Predicted Removal Efficiency using Pseudo First-order Removal with the BIOWIN4 Converted Biodegradation Rate Constant Compared to Observed Data from South Shore (Pearson Correlation = 0.792, $p < .001$)

Chapter 3: PHARMACEUTICALS AND PERSONAL CARE PRODUCTS FOUND IN THE GREAT LAKES ABOVE CONCENTRATIONS OF ENVIRONMENTAL CONCERN

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Abstract

The monitoring of pharmaceuticals and personal care products (PPCPs) has focused on the distribution in rivers and small lakes, but data regarding their occurrence and effects in large lake systems, such as the Great Lakes, are sparse. Wastewater treatment processes have not been optimized to remove influent PPCPs and are a major source of PPCPs in the environment. Furthermore, PPCPs are not currently regulated in wastewater effluent. In this experiment we evaluated the concentration, and corresponding risk, of PPCPs from a wastewater effluent source at varying distances in Lake Michigan. Fifty-four PPCPs and hormones were assessed on six different dates over a two-year period from surface water and sediment samples up to 3.2 km from a wastewater treatment plant and at two sites within a harbor. Thirty-two PPCPs were detected in Lake Michigan and 30 were detected in the sediment, with numerous PPCPs being detected up to 3.2 km away from the shoreline. The most frequently detected PPCPs in Lake Michigan were metformin, caffeine, sulfamethoxazole, and triclosan. To determine the ecological risk, the maximum measured environmental concentrations were compared to the predicted no-effect concentration and 14 PPCPs were found to be of medium or high ecological risk. The environmental risk of PPCPs in large lake systems, such as the Great Lakes, has been questioned due to high dilution; however, the concentrations found in this study, and their corresponding risk quotient, indicate a significant threat by PPCPs to the health of the Great Lakes, particularly the near shore organisms.

Introduction

Pharmaceutical and personal care products (PPCPs) have been found in wastewater worldwide (Aydin and Talinli, 2013; Gomez et al., 2007; Miege et al., 2009; Suarez et al., 2012; Ternes, 1998; Tewari et al., 2013; Vieno et al., 2007). The level of removal has been found to vary widely depending on the chemical, the operating conditions, and the treatment technologies (Blair et al., 2013; Miege et al., 2009; Oulton et al., 2010; Verlicchi et al., 2012). Variable removal of PPCPs through WWTPs has led to detection of these compounds in the aquatic environment, albeit mostly in microgram to nanogram per liter concentrations (Cahill et al., 2004; Focazio et al., 2008; Glassmeyer et al., 2005; Halling-Sorensen et al., 1998; Kolpin et al., 2002; Kümmerer, 2009; Li et al., 2010; Scheurer et al., 2009; Snyder 2008; Yu and Chu 2009). Higher pharmaceutical concentrations in WWTP effluent have been measured under certain circumstances, such as WWTPs that receive a substantial amount of their flow rate from pharmaceutical manufacturing (Phillips et al., 2010, Larsson et al., 2007). Research has shown that certain PPCPs may have an impact on the environment at the microgram to nanogram per liter concentrations with a range of potential impacts (Al Aukidy et al., 2012; Brodin et al., 2013; Brooks et al., 2002; Christensen et al., 2009; Fent et al., 2006; Gros et al., 2010; Han et al., 2006; Hernando et al., 2006; Tewari et al., 2013).

The emission of PPCPs into the environment from wastewater can depend on the wastewater treatment processes, the flow of the waste stream, and different PPCPs usage patterns that vary by region and season (Dickenson et al., 2011, Yu et al., 2013). In an aquatic environment the fate and concentration of PPCPs be can reliant on the receiving

water body flow rate, partitioning to sediments or biological entities, uptake up by biota, volatilization, biological degradation, photodegradation, or transformed through other abiotic transformations such as hydrolysis (Yamamoto et al., 2009). In the Great Lakes, which contains 84% of North America's freshwater (USEPA 2012a), dilution from the source may also be a major factor in the occurrence and detection of PPCPs in surface water and sediments.

Limited studies are available that assess PPCPs offshore in large water bodies due to the expected low levels of PPCPs from dilution and the complex hydrodynamics in a lake as large as one of the Great Lakes. Site selection for PPCPs research has focused on bodies of water that are potentially contaminated from human, industrial, and agricultural wastewater (Kolpin et al., 2002). Four previous studies have looked at PPCPs levels in the Great Lakes (Csiszar et al. 2011; Li et al., 2010; Metcalf et al., 2003; Wu et al., 2009) with a wide range of results and they have focused near shore, in harbors, and in rivers that are tributaries to the Great Lakes. No previous studies have assessed PPCPs offshore in Lake Michigan. Lake Michigan is the sixth largest lake in the world by volume and fifth by area (Beeton, 2002) and understanding the concentration of these pollutants in Lake Michigan is critical. Additionally, no previous studies have assessed the extent of the temporal and spatial distribution of PPCPs from a large, urban WWTP into the Great Lakes.

Using a risk quotient (RQ), which is defined as the ratio of the maximum measured environmental concentration (MEC) to the predicted no-effect concentration (PNEC), the ecosystem risk from pollutants can be gauged (Hernando, 2006). However,

calculating this ratio can be challenging due to a lack of information regarding the effects of PPCPs in the environment and difficulty in establishing the PNEC. Researchers have used the RQ to assess the low levels of PPCPs on the ecosystem health with varying results. Recent studies have found limited ecological risk is expected for many PPCPs, which may be due to the risk being partially mitigated by high dilution (Al Aukidy et al., 2012; Gros et al., 2010; Yu et al., 2013). Conversely, other studies have found PPCPs of high or medium risk in secondary effluent, rivers, and small lakes (Christensen et al., 2009; Tewari et al., 2013; Valcárcel et al., 2011; Verlicchi et al. 2012). Additionally, levels of concern have been found in sewage sludge (Yu et al., 2013).

Studies have not been conducted evaluating the occurrence and risk of PPCPs in Lake Michigan and other studies on the Great Lakes have assessed a small number of PPCPs. A better understanding of the occurrence of PPCPs in large water systems, particularly in areas with substantial urban development, needs further investigation. The purpose of our study was to assess the risk of 54 PPCPs in Lake Michigan from varying proximities to a major effluent discharge site and to assess the risk potential to the environment. PPCPs were measured in both surface water and sediment samples over six dates. The sampling pattern was selected due to the prevailing southern current in this portion of the Lake Michigan basin (Rao and Schwab, 2007). When possible, a RQ was estimated to determine which compounds are at a level of concern based on existing effects data or models.

Materials and Methods

South Shore Water Reclamation Facility **(**SSWRF) and Jones Island Water

Reclamation Facility (JIWRF) services the greater Milwaukee, Wisconsin area. Fifty-four PPCPs were measured in Lake Michigan and compared to the related data on wastewater effluent from Blair et al. (2013). Both SSWRF and JIWRF uses preliminary treatment (bar screens/grit channels), primary clarifiers, activated sludge treatment and chlorine disinfection. SSWRF has a treatment capacity of 1,135,000 m^3 day^{-1} (300 MGD (million gallons per day)) with an average flow of approximately 379,000 m^3 day^{-1} (100 MGD). JIWRF has a treatment capacity of 1,457,000 m^3 day^{-1} (385 MGD) with an average flow of approximately 473,000 m^3 day^{-1} (125 MGD).

Surface water and sediment samples were collected in Lake Michigan the day following the sampling at SSWRF. Sampling was conducted using a Niskin bottle at a depth of 5 m over sites up to 3.6 km away from the effluent discharge site (Figure 5). SSWRF discharges directly into Lake Michigan whereas JIWRF discharges into the Milwaukee Harbor. Field blanks were collected on each date using distilled water. Grab sediment samples were collected on 5/15/2009 and 4/9/2010. Water and sediment samples were also collected in the Milwaukee Harbor near JIWRF as a comparison site that has lower dilution and potentially higher PPCPs concentration than the open lake. The final effluent was sampled using a 24-hour composite sample as described by Blair et al. (2013).

PPCPs Analysis

PPCPs were extracted and analyzed based upon US EPA Method 1694 (USEPA, 2007a) for pharmaceuticals and US EPA Method 1698 (USEPA, 2007b) for steroids and hormones by using high performance liquid chromatography combined with tandem mass

spectrometry (HPLC/MS/MS) with modifications as published by Blair et al. (2013). The PPCPs were selected for this study based on the EPA methods. Forty-one PPCPs were assessed under EPA 1694 and thirteen hormones were assessed under EPA 1698. Sediment samples were collected for a subset of the sampling dates and these data are presented separate from the liquid concentration. The same 54 PPCPs were assessed in both the water and sediment samples.

Risk Quotient

To determine the risk quotient (RQ) for each compound, the PNECs were found using the review paper from Verlicchi et al. (2012) and ECOSAR v1.11 from the US EPA (USEPA 2012b). When the values found by Verlicchi et al. (2012) were from an older version of ECOSAR, or if the data were not available, the lowest freshwater toxicity value from ECOSAR v1.11 was used. The PNEC selected from these values also included the chronic values from ECOSAR. An assessment factor (1000) was introduced to take into account the effect on other, potentially more sensitive, aquatic species (Hernando et al., 2006; Al Aukidy et al., 2012). An accepted definition was used for the RQ, where low risk is below 0.1, medium risk is from 0.1 to 1, and high risk is greater than 1 (Hernando et al., 2006; Verlicchi et al., 2011). When a PPCP had a concentration in the blank above the MQL, this value was subtracted from the maximum concentration before the RQ was calculated.

Results and Discussion

Surface Water Concentration

Over six sampling dates, 38 of the 54 compounds were detected from effluent or

Lake Michigan samples. Four compounds were detected with greater than 50% frequency at all of the sampling sites in Lake Michigan and the Milwaukee Harbor: metformin (100%), caffeine (97.6%), sulfamethoxazole (83.3%), and triclosan (71.4%). Table 3 has the mean and maximum levels from the six samples dates along with the MDL, MQL, and maximum value found in the method blanks. The frequency of detection and general classifications for the compounds assessed are available in Appendix C. The complete data are available in Appendix C.

The most widely detected pharmaceutical in our study was the antidiabetic metformin, which was detected above the minimum detection limit with 100% frequency in Lake Michigan (Figure 6a). Metformin was detected at sites up to 3.2 km away from the shore, which was unanticipated given the volume of such a large lake system and the predominant southern current in this portion of Lake Michigan. Although metformin is less frequently measured than other compounds in PPCP studies, we have found, along with others, that metformin is prevalent in WWTP influent at concentrations as high as 129,000 ng L^{-1} but the removal efficiency ranges from 41% to over 98% (Blair et al., 2013; Oosterhuis et al., 2011; Scheurer et al., 2009; Scheurer et al., 2012; Trautwein and Kümmerer, 2011). The median value for metformin in Lake Michigan was greater than 100 ng L^{-1}, comparable to stream and small lake studies where metformin has been observed in 4.8% of samples with estimated levels of 110 ng L^{-1} in the U.S. (Kolpin et al., 2002) and was detected at all of the sites assessed at concentration up to 2,000 ng L^{-1} in German rivers (Scheurer et al., 2009; Scheurer et al., 2012). Given the prevailing southern water current, the concentration of metformin was expected to vary at the

different sampling sites depending on the direction from source. Yet average metformin concentrations were similar to levels found in smaller water bodies and the prevailing currents did not seem to lead to differences in concentration with location. Other compounds that followed the same general trend as metformin were caffeine, paraxanthine, sulfamethoxazole, and triclosan.

As a contrast to metformin, the anticonvulsant compound carbamazepine, shown in Figure 6b, was detected on all of the sampling dates in the final effluent at SSWRF but rarely in Lake Michigan water or sediment. Carbamazepine has been found to be highly persistent in wastewater since it is expected to resist biological degradation (Blair et al., 2013; Gomez et al., 2007; Radjenovic et al., 2009; Rosal et al., 2010; Santos et al., 2007). However, carbamazepine was not detected in the water or sediment samples surrounding SSWRF. Dilution of the wastewater effluent may have been adequate to reduce the concentration to below the MDL. However, carbamazepine was detected with 66.7% frequency at both locations in the Milwaukee harbor at levels above the MDL. Given the lack of detection of carbamazepine around SSWRF, the fate of carbamazepine in Lake Michigan is unknown.

Twenty-seven PPCPs were detected at notable levels at the JI outfall and South gap in the Milwaukee harbor. JI Water Reclamation Facility discharges into the Milwaukee harbor and this is a potential source of these PPCPs, although effluent levels were not assessed at this WWTP. Additionally, the Milwaukee River also flows into the harbor and is an additional potential source of the PPCPs that were detected. As shown in Table 3, the PPCPs concentrations in the Milwaukee Harbor were overall higher than the

area surrounding SSWRF. This was used as a reference site as previous research has shown chronic fecal pollution in the harbor (Newton et al., 2011). These results agree with other studies assessing harbors on the Great Lakes (Csiszar et al., 2011; Metcalf et al., 2003).

Hormones were not consistently detected above the minimum detection limit in Lake Michigan. The concentrations of hormones were low and inconsistent in the final effluent at SWWRF which may be due to the high expected removal from a WWTP through adsorption, biodegradation, and exposure to chlorine (Benotti et al., 2009; Esperanza et al., 2007; Huerta-Fontela et al., 2011; Joss et al., 2006). Given the inconsistent and low levels detected in the effluent and the high dilution from entering Lake Michigan, the levels of hormones in the lake from the WWTP would be expected to be below the detection limit.

Sediment Levels

Thirty compounds were detected in the sediment at levels above the MDL in Lake Michigan and these compounds are listed in Table 4. The most commonly detected compounds were: azithromycin, clarithromycin, diphenhydramine, metformin, triclosan and triclocarban. Of these compounds, all of them were regularly detected in the final effluent, with the exception of the macrolide antibiotic clarithromycin, which was detected only once. Given the low occurrence in the final effluent and across the stages of SSWRF (Blair et al, 2013), the regular and widespread occurrence of clarithromycin in sediment needs further investigation. Azithromycin and clarithromycin were found to have limited sorption to sludge in WWTPs (Verlicchi et al., 2012) therefore their

detection in sediment needs further investigation. Triclosan and triclocarban were detected in Lake Michigan sediment due to their regular occurrence in effluent and their known hydrophobic characteristics (Loranzo et al., 2013). Additionally, the detection of metformin and diphenhydramine in Lake Michigan sediment needs further research.

Other PPCPs detected in the sediment cannot be clearly contributed to the effluent from SSWRF. For example, thiabendazole, a fungicide, was detected at low levels in the final effluent, but was only located in the sediment at the 1.6 km east and 3.2 km east sampling locations and was not detected in the surface water. The detection at these locations implies the potential source is from land runoff, not discharged from SSWRF. With detection only at the eastern locations, not the southern locations, the source may be from area north of the WWTP. Significant agricultural developments are not present in the area north of SSWRF, but a residential area that includes many parks and golf courses are a possible source of this fungicide.

Ecological Risk Quotient

Overall, a total of twenty-four compounds were detected in the final effluent or Lake Michigan at a level of medium or high risk. As shown in Figure 7, fourteen compounds were detected in Lake Michigan itself with high or medium risk. Metformin, the most widespread compound, did not correspond with high or medium risk at the concentrations detected; however, this may be due to the lack of predictive toxicity data on the chronic effects of this compound. When comparing the final effluent RQs to the values in Lake Michigan, many compounds drop below the threshold to medium or low risk after the compound is discharged from the WWTP, such as gemfibrozil, diltiazem.

However, dilution is not sufficient to reduce the risk of all compounds to below the high and medium threshold, even at a distance of 3.2 km from shore, such as for sulfamethoxazole and codeine.

Conclusion

The detection of such a large number of PPCPs with high or medium risk in the Great Lakes is novel and of concern. The area surrounding the SS outfall and the sites within the Milwaukee Harbor are important as they are near locations for fish spawning and aquatic organisms, such as perch, can be found congregating around the effluent pipes of SSWRF and are exposed to effluent concentration with little dilution. Knowing that PPCPs can impact the behavior of aquatic organisms (Brodin et al.,2013, Brooks et al., 2003) leads to the conclusion that the endpoints used to assess the PNEC values for PPCPs may not properly address the ecological impacts and further testing is needed to identify the PPCPs of greatest concern. Additionally, the RQ may also underestimate risk due to potential mixture effects of PPCPs with similar mechanisms of action that may be additive in their impact. Reliance on a model such as ECOSAR is useful for identification of PPCPs that warrant further research, but these models are not a replacement for experimental tests to determine the full ecological impacts from PPCPs.

PPCPs were frequently detected in the water and sediments at the ng L^{-1} level, including sites 3.2 km from shore in Lake Michigan at concentrations that are estimated to cause environmental concern. At the concentrations detected, medium or high risk was associated with twenty four compounds in the final effluent, and fourteen were found to be of medium or high risk in Lake Michigan. The most frequently detected PPCPs were

metformin, caffeine, sulfamethoxazole, and triclosan. Given the widespread detection of PPCPs, these pollutants are not ephemeral and pose an environmental risk to the sixth largest lake in the world. Therefore, high dilution is not adequate to mitigate the risk from this cocktail of PPCPs and the potential ecological risk for large lake systems is much higher than previously understood.

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Table 3: Concentration of PPCPs at the final effluent and at seven locations in Lake Michigan (Below Detection Limit (BDL)

	Field Blank			Outfall	1.6 km East (1 mi. East)	1.6 km South (1 mi. South)	3.2 km East (2 mi. East)	3.2 km South (2 mi. South)	JI Outfall	South Gap
	Max	MDL	MQL	Mean, Max	Mean, Max	Mean, Max	Mean, Max	Mean, Max	Mean, Max	Mean, Max
	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}	ng L^{-1}
17,20-dihydroxyprogesterone	BDL	1.4	4.2	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
17-alpha-estradiol	BDL	1.2	3.5	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
17-beta-estradiol	BDL	1.3	3.8	BDL, 1.7*	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, 1.3*
4-androstene-3,17-dione	4.5	0.5	1.4	97, 580	BDL, 2.0	0.9*, 5.3	3.1, 17	0.8*, 3.5	0.3*, 1.4	BDL, 0.9*
5-alpha-androstane-3,17-dione	BDL	2.3	6.9	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Acetaminophen	BDL	2.5	7.5	4.2*, 21	BDL, BDL	BDL, BDL	BDL, BDL	BDL, 2.5*	17, 73	13, 45
Albuterol	BDL	1.4	4.2	BDL, BDL	BDL, BDL	BDL, 5.9	BDL, BDL	BDL, BDL	BDL 4.3	BDL, BDL
Azithromycin	15.9	3.7	11	BDL, BDL	BDL, 12	BDL, 12	BDL, 7.5*	BDL, 11	BDL, 22	BDL, 12
Boldenone	BDL	1.3	4.0	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Caffeine	62.1	3.1	9.3	44, 110	18, 42	37, 86	21, 35	24, 39	67, 230	71, 190
Carbadox	44.9	3.4	10	7.2*, 20	BDL, 17	12, 49	BDL, 6	6.7*, 33	6.1*, 22	4.2*, 19
Carbamazepine	BDL	2.7	8.2	BDL, 6.2*	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	15, 38	6.4*, 17
Cimetidine	BDL	1.3	3.8	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Ciprofloxacin	BDL	3.3	9.9	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Clarithromycin	BDL	3.2	9.6	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Codeine	BDL	3.6	11	BDL, 11	BDL, 8.7*	BDL, 9.2*	BDL, 5.4*	BDL, 7.2*	4.4*, 11	5.3*, 15
Cotinine	BDL	3.5	11	BDL, 7.4*	BDL, 6.5*	BDL, 5*	BDL, 6.1*	BDL, 11	BDL, 20	3.5*, 21
Digoxigenin	BDL	4.4	13.2	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Diltiazem	5.4	3.5	10	BDL, 7.9*	BDL, 5.5*	BDL, 7.8*	BDL, BDL	BDL, BDL	6.3, 21	BDL, 10
Diphenhydramine	4.2	3.6	11	4.1*, 14	BDL, 6.6*	BDL, 9.2*	BDL, 4.9*	BDL, 6.7*	10*, 43	3.6*, 12
Estriol	BDL	2	6.1	BDL, BDL	BDL, 3.9*	BDL, BDL	BDL, 5.0*	BDL, BDL	BDL, BDL	BDL, 4.9*
Estrone	BDL	2.2	6.7	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, 3.4*	BDL, BDL	BDL, BDL

Fluoxetine	BDL	3.5	11	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	8.2*, 49	10*, 62
Gemfibrozil	BDL	1.6	4.8	9.1, 42	BDL, BDL	1.6*, 4.5*	BDL, BDL	3.1*, 19	14, 36	13, 43
Ibuprofen	BDL	4.7	14	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Lincomycin	BDL	3.1	9.3	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Lomefloxacin	BDL	4.7	14.2	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Melengestrol	BDL	1.3	4	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Melengestrol Acetate	BDL	0.6	1.7	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Metformin	35.5	0.5	1.5	1200, 3800	240, 820	270, 840	120, 160	110, 160	4100, 9200	1200, 2400
Miconazole	BDL	2.7	8.1	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Naproxen	BDL	1	2.9	4.9, 19	BDL, BDL	2.5*, 15	BDL, BDL	BDL, BDL	8.4, 31	4.8, 18
Norfloxacin	BDL	5.1	15.3	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Ofloxacin	BDL	3.9	12	10*, 61	BDL, 21	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Oxacillin	BDL	2.5	7.4	BDL, BDL	BDL, BDL	BDL, BDL	2.9*, 17	BDL, BDL	BDL, BDL	BDL, BDL
Paraxanthine	11.6	6.1	18	6.3*, 23	BDL, 9.7*	11*, 39	BDL, BDL	BDL, 8.7*	15*, 57	15*, 45
Progesterone	17.8	0.7	2	2.0, 11	1.0*, 4.9	1.5*, 8.7	15, 88	2.6, 13	BDL, BDL	BDL, BDL
Ranitidine	BDL	0.9	2.6	BDL, BDL	BDL, 3.7	BDL, BDL	BDL, BDL	BDL, BDL	5.4, 27	BDL, BDL
Roxithromycin	5.5	4.3	13	BDL, 8.7*	BDL, BDL	BDL, 7.5*	4.5*, 15	6.5*, 39	BDL, 9.2*	BDL, BDL
Sarafloxacin	BDL	5.4	16	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Sulfachloropyridazine	BDL	4.1	12	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Sulfadiazine	BDL	2.8	8.5	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, 3.8*	BDL, BDL
Sulfadimethoxine	BDL	2.4	7.1	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Sulfamerazine	BDL	2.1	6.2	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, 3.6*
Sulfamethazine	BDL	4.0	12	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Sulfamethizole	BDL	4.2	13	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Sulfamethoxazole	BDL	4.1	12	6.9*, 14	BDL, 6.2*	5.1,* 7.0*	BDL, 7.3*	4.5*, 10*	29, 77	16, 30
Sulfanilamide	BDL	2.9	8.6	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	6.3*, 20	BDL, BDL
Sulfathiazole	BDL	2.6	8	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL
Testosterone	12.4	1.1	3.2	2.2*, 13	BDL, 4.5	1.5*, 9.1	6.4, 38	1.4*, 7.0	BDL, BDL	BDL, BDL
Thiabendazole	BDL	1.8	5.3	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL

Triclocarban	BDL	0.5	1.4	2.6, 16	BDL, BDL	BDL, BDL	BDL, BDL	BDL, BDL	3.9, 9.9	BDL, BDL
Triclosan	5.4	0.5	1.6	9.9, 41	0.8*, 2.1	3.0, 16	2.7, 7.4	1.4*, 6.5	7.7, 24	5.0, 11
Trimethoprim	BDL	3.4	10	BDL, 3.4*	BDL, BDL	BDL, 6.0*	BDL, BDL	BDL, BDL	17, 52	6.9*, 13

*Value above MDL, but below MQL.

Table 4: PPCPs Levels in Sediment from Lake Michigan (Compounds listed in Table 3 that were not detected above the MDL in the sediment samples are omitted from this table)

			SS Outfall	1.6 km East (1 mi. East)	1.6 km South (1 mi. South)	3.2 km East (2 mi. East)	3.2 km South (2 mi. South)	JI Outfall	South Gap
	MDL	MQL	Mean	Mean	Mean	Mean	Mean	Mean	Mean
	ng g^{-1}	ng g^{-1}	ng g^{-1}	ng g^{-1}	ng g^{-1}	ng g^{-1}	ng g^{-1}	ng g^{-1}	ng g^{-1}
Acetaminophen	2.5	7.5	BDL	18	29	BDL	BDL	BDL	29
Azithromycin	3.7	11	490	16	72	19	25	59	350
Caffeine	3.1	9.3	25	BDL	24	30	14	4.2*	BDL
Carbadox	3.4	10	BDL	BDL	BDL	BDL	BDL	BDL	14
Ciprofloxacin	3.3	9.9	42	7.7*	9.0*	46	52	43	BDL
Clarithromycin	3.2	9.6	33	28	130	5*	120	90	BDL
Codeine	3.6	11	BDL	BDL	BDL	BDL	BDL	4*	BDL
Cotinine	3.5	11	8.0*	BDL	BDL	BDL	BDL	39	BDL
Digoxigenin	4.4	13	BDL	BDL	BDL	BDL	BDL	4.9*	9.2*
Diltiazem	3.5	10	4.0*	BDL	BDL	BDL	BDL	5.2*	3.9*
Diphenhydramine	3.6	11	81	13	43	7.3*	82	150	160
Enrofloxacin	1.4	4.1	BDL	BDL	BDL	6.6	BDL	BDL	BDL
Erythromycin	9.9	30	BDL	BDL	BDL	BDL	BDL	25*	BDL
Flumequine	5.2	16	BDL	BDL	BDL	6.9*	BDL	BDL	6.0*
Fluoxetine	3.5	11	7.6*	BDL	BDL	BDL	BDL	20	12
Ibrupoven	4.7	14	BDL	BDL	8.8*	BDL	BDL	BDL	No Data
Lincomycin	3.1	9.3	BDL	BDL	BDL	BDL	BDL	BDL	5*
Metformin	0.51	1.5	50	43	3.8	16	2.3	59	140
Miconazole	2.7	8.1	7.6	BDL	BDL	BDL	BDL	3.7*	8.4
Naproxen	0.97	2.9	4.8	1.0*	BDL	BDL	BDL	2.6*	No Data
Norfloxacin	5.1	15	BDL	BDL	BDL	12*	36	BDL	BDL
Ofloxacin	3.9	12	4.3*	BDL	BDL	7.7*	BDL	BDL	7.3*
Oxacillin	2.5	7.4	BDL	BDL	BDL	BDL	2.8*	BDL	9.1

Paraxanthine	6.1	18	BDL	BDL	BDL	BDL	BDL	BDL	15*
Roxithromycin	4.3	13	28	BDL	31	BDL	44	71	BDL
Sarafloxacin	5.4	16	BDL	BDL	BDL	9.9*	BDL	BDL	BDL
Thiabendazole	1.8	5.3	BDL	230	BDL	68	BDL	BDL	BDL
Triclocarban	0.48	1.4	170	4.5	33	BDL	11	510	No Data
Triclosan	0.53	1.6	37	18	22	26	12	150	No Data
Tylosin	3.5	11	9.4*	12	3.9*	BDL	14	20	BDL

*Value above MDL, but below MQL.

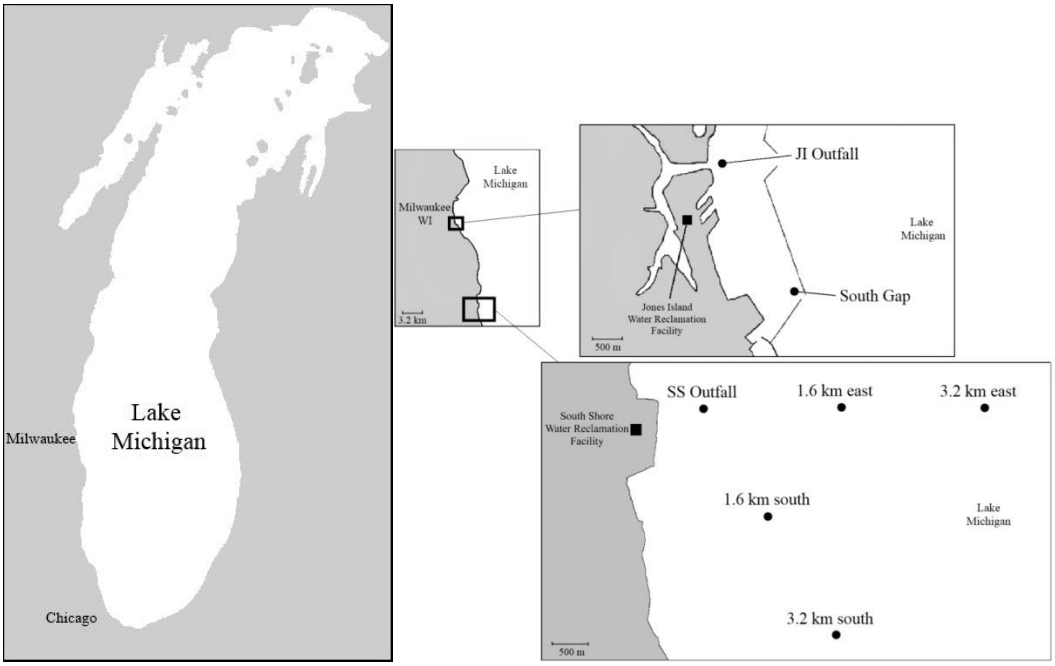
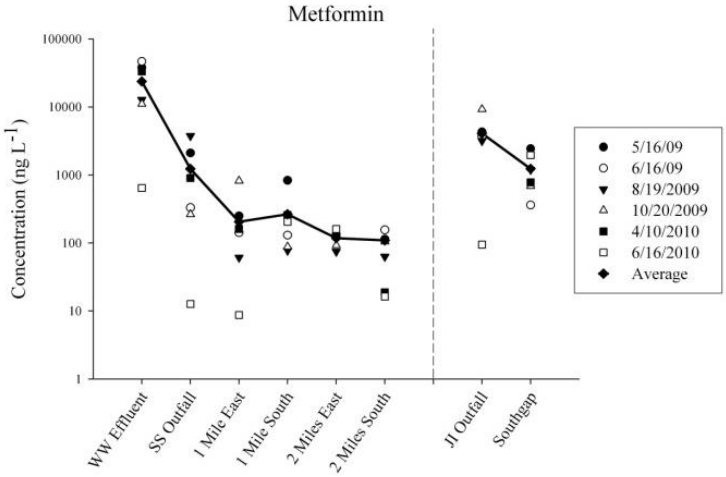
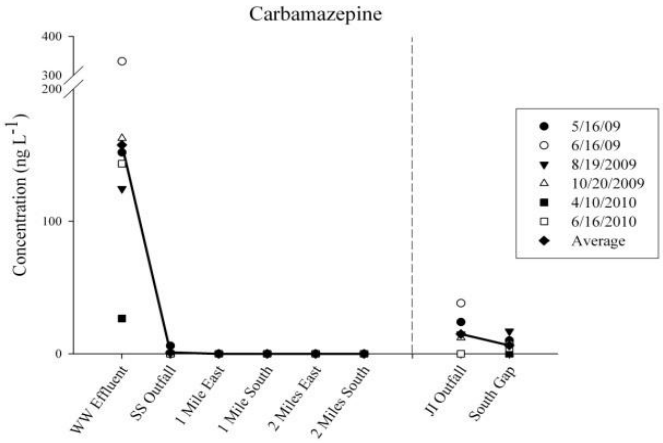


Figure 5. Lake Michigan and the Sampling locations in Lake Michigan near Milwaukee, Wisconsin, USA. Boxes represent the two WWTPs discussed: JIWRF and SSWRF.



a



b

Figure 6. Concentration of metformin (a) and carbamazepine (b) in wastewater effluent and in Lake Michigan on six dates

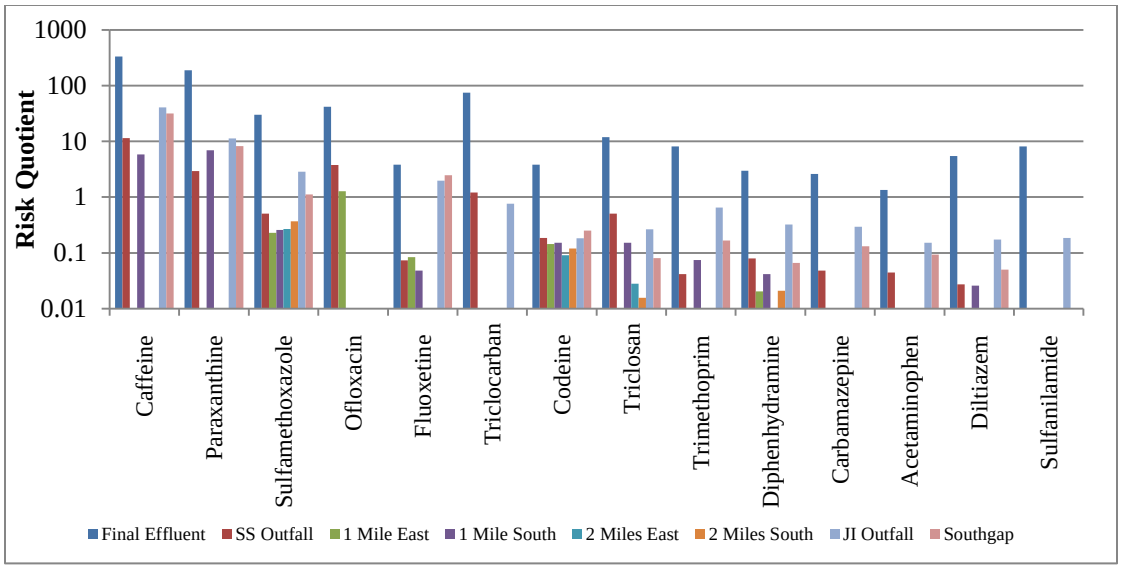


Figure 7. Risk Quotient for 14 PPCPs in Wastewater Effluent and in Lake Michigan (RQ > 1 is high risk, RQ from 0.1 to 1 is medium risk, and RQ < 0.1 is low risk

Chapter 4: ASSESSING CURRENT WASTEWATER REGULATIONS TO MINIMIZE THE RISK QUOTIENT OF PHARMACEUTICALS AND PERSONAL CARE PRODUCTS (PPCPS): A CASE STUDY IN WISCONSIN, USA

Abstract

Phosphorus and pharmaceutical and personal care products (PPCPs) are pollutants that can cause a wide array of negative environmental impacts. Phosphorus is a regulated pollutant in many industrial countries, while PPCPs are widely unregulated. Since many technologies designed to remove phosphorus from wastewater also have the potential to remove PPCPs, the purpose of this work is to explore the ability of these technologies to also reduce the emission of unregulated PPCPs. We examine this potential ancillary benefit by presenting a novel method using the PPCPs' risk quotient (RQ) to measure the effectiveness of different wastewater treatment technologies. The RQ is then applied via a case study that uses the phosphorus effluent regulations in Wisconsin, USA to determine the ability of the recommended technologies to also mitigate PPCPs. The result show that the tertiary membrane bioreactor and nanofiltration processes recommended to remove phosphorus can reduce the median risk quotient from PPCPs by 71% and 81%, respectively. The ultrafiltration technology was estimated to reduce the median risk quotient from PPCPs by 28% with no cost in addition to the costs expected under the current phosphorus effluent regulations. Additionally, higher quality effluent is expected with a membrane bioreactor and the cost of upgrading to this technology from a low-pressure membrane system was found to be $11.76 per capita per year. Using these results, we discuss the management implications, including watershed management, alternative PPCPs reduction strategies, adaptive management, and water quality trading. In conclusion, this study suggests the management of practices to remove regulated pollutants could significantly reduce the emission of PPCPs from wastewater.

1. Introduction.

When establishing a regulatory framework to reduce the emission of a specific pollutant (e.g. phosphorus), the ancillary benefit of removing unregulated pollutants is often ignored. In this work, we focus on the regulations to remove phosphorus from wastewater and the potential concurrent removal of unregulated pharmaceuticals and personal care products (PPCPs). An emphasis is placed on phosphorus and PPCPs because the levels of these pollutants in the environmental may be damaging to aquatic organisms and the ecosystem (Anderson et al., 2002; Blair et al., 2013a; Verlicchi et al., 2011). For example, PPCPs have the potential to cause endocrine disruption, changes in growth, and changes in behaviors important to feeding, reproduction, and predator avoidance in aquatic organisms while excess phosphorus has the potential to cause eutrophication, deoxygenation of the water, and harmful algal blooms (Anderson et al., 2002; Brausch and Rand, 2011; Brodin et al., 2013; Kidd et al., 2007; Nassef et al., 2010; Weinberger and Klaper 2013).

Policies have been implemented to reduce phosphorus emissions into the environment (e.g. US Federal Water Pollution Control Act 2002); however, no regulations exist for PPCPs in drinking or natural waters in the United States (Ryu et al., 2014). Recently, calls have been made for the initiation of programs that would remove PPCPs from wastewater in the United States and the European Union (EC 2013; Molinos-Senante et al., 2013; USEPA 2013a). The European Commission has proposed adding three PPCPs (17 alpha-ethinylestradiol, 17 beta-estradiol, Diclofenac) to the list of pollutants that are monitored and controlled in European Union waters (EC 2013).

Likewise, a number of state and local governments across the United States have started to develop programs to reduce the amount of PPCPs entering the aquatic environment (Kotchen et al., 2009).

Current municipal wastewater treatment plant (WWTP) configurations are ineffective at removing many PPCPs and have difficulty meeting ultra-low phosphorus (<0.1 mg P /L) effluent concentrations (Michael et al., 2013; Outlon et al., 2010; Verlicchi et al., 2011). To meet the ultra-low phosphorus effluent regulations, tertiary technologies such as membrane bioreactors and membrane filtration are being considered for municipal WWTPs and these technologies also have the potential to remove PPCPs from wastewater (Strand Associates, 2008; Snyder et al., 2007). However, many of the tertiary wastewater treatment processes that are being implemented to meet phosphorus effluent regulations also have the potential to remove PPCPs (Oulton et al., 2010; Wenzel et al., 2008).

In general, there is no consensus regarding the best method to manage PPCPs in the aquatic environment. The most commonly implemented programs focus on the disposal of unused PPCPs through take-back programs or education regarding trash disposal (Daughton, 2010; Kotchen et al., 2009). However, many PPCPs enter the waste stream through normal use: orally consumed PPCPs are excreted through urine or feces unchanged or partly metabolized (Kasprzyk-Hordern et al., 2009). In addition, people can use PPCPs topically and wash them off, for example, triclosan, a widely used antibacterial agent used in soaps. Therefore, programs focusing on the disposal of unused PPCPs are only partially effective at mitigating the risk from these pollutants. Along with

the efforts advocating trash disposal or take-back programs of unused PPCPs, the currently proposed solutions consist of tertiary wastewater treatment, urine separation, dilution through watershed management, producing pharmaceuticals that would cause less harm to the environment, selecting PPCPs possessing environment-friendly excretion profiles, improving drug delivery, and prescribing patients the minimum therapeutic dosage (Borsuk et al., 2008; Cook et al., 2012; Daughton and Ruhoy, 2013; Eckstein and Sherk, 2012; Khetan and Collins, 2007; Glassmeyer et al., 2009; Schimmelpfennig et al., 2012). Nevertheless, establishing a regulatory framework to reduce the damage from PPCPs is challenging. This is because we have an incomplete understanding of the ecological and human health impacts, a limited capability to effectively remove PPCPs from wastewater using conventional technologies, and face high costs associated with systems shown to minimize PPCPs emissions (Blair et al., 2013b; Brodin et al., 2013; EC 2013; Molinos-Senante et al., 2013; Verlicchi et al., 2012).

The goal of this work is to assess via case study the feasibility of utilizing a regulation requiring ultra-low phosphorus effluent levels in wastewater to also achieve a meaningful reduction in PPCPs. It is critical to assess the removal of unregulated pollutants when evaluating processes to meet updated regulatory standards, particularly when it involves significant capital upgrades. To demonstrate the potential ancillary benefits of a phosphorus reduction policy, this study will assess the change in the RQ for 11 PPCPs across four different phosphorus reduction technologies. At this time, no study has quantitatively assessed the change in the PPCPs risk quotient (RQ) from the implementation of a policy to remove phosphorus from wastewater. We then apply the

RQ to a case study that uses the recently modified phosphorus effluent regulations in Wisconsin, USA to explore whether a phosphorus reduction policy could minimize the release of unregulated PPCPs into the environment. The purpose is not to provide an exhaustive review of PPCPs removal using different WWTP configurations. Rather, this study will provide an illustration of the potential ancillary benefits from reducing regulated pollutants from wastewater, along with a recommendation to prioritize research on the potential impacts of PPCPs on environmental health. We will also address management implications including the use of alternative PPCPs strategies, adaptive management including water quality trading, and site-specific concerns. An emphasis will also be placed on the estimated cost of these technologies.

This chapter proceeds as follows: section 1.1 of this article provides a review of the policies related to phosphorus reduction in the State of Wisconsin; section 1.2 presents the RQ and the application in understanding environmental damage from PPCPs. Section 2 provides the data analytic methods and the development of the RQ. Section 3.1 provides the results from the meta-analysis; section 3.2 provides the limitations of the meta-analysis and data collection; section 4.0 discusses the management implications; and section 4.1 presents the cost of the technologies. Section 5.0 offers an overall conclusion.

1.1. Summary of Phosphorus Reduction Regulations in Wisconsin.

In the United States, the adoption of nutrient reduction policies will require the significant advancement of statewide nutrient reduction efforts. Many of these efforts will focus on agricultural practices and enhancing wastewater treatment. States within the

United States are encouraged to establish numeric criteria for nutrients (phosphorus and nitrogen) in all bodies of water in compliance with the EPA's National Strategy for Development of Regional Nutrient Criteria under the Clean Water Act (USEPA 1998). However, only 10% of states in the United States currently have statewide numeric criteria for phosphorus in streams and rivers and only 12% of states have these criteria for lakes and reservoirs (USEPA 2012a). Wisconsin and New Jersey are the only states currently with statewide phosphorus criteria for lakes/reservoirs and rivers/streams (USEPA 2012a).

We use the State of Wisconsin for a case study because they recently set some of the lowest effluent standards for municipal wastewater treatment facilities in the United States and the wastewater phosphorus effluent limits will be as low as 50 µg/L (WI Code NR102 and WI Code NR217). Based on the EPA nutrient criteria guidelines, the State of Wisconsin implemented changes to the Natural Resources (NR) Codes 102 (2010) and 217 (2010) to address phosphorus loading in surface waters. At this time, there are no water quality standards criteria for total nitrogen in Wisconsin (WIDNR 2013a). The Wisconsin Legislature and Wisconsin Department of Natural Resources (DNR) have enacted surface water phosphorus limits and modified the point and nonpoint regulations to meet the desired surface water phosphorus concentration.

Wisconsin NR Code 102 outlines the phosphorus limits to be achieved in surface waters. The surface water levels range from 5-7 µg/L for Lake Superior and Lake Michigan to 100 µg/L for specifically identified rivers. The overarching foundation

behind the changes to Wisconsin NR Code 102 sets the limits at levels that are near naturally occurring levels for a given body of water.

To meet these surface water levels, Wisconsin NR Code 217 set the effluent standards for municipal wastewater treatment facilities. Phosphorus emission limits are variable for each WWTP and the limits are a function of the water quality criterion from NR102, receiving water flow, effluent flow, fraction of the effluent flow withdrawn from the receiving water, and the upstream concentration. The phosphorus wastewater effluent limit for reservoirs and lakes is equal to the level in the receiving water or downstream water. The wastewater phosphorus effluent limits may be as low as 50 µg/L. However, for the majority of the wastewater treatment plants, the effluent limit will be in the range of 100 µg/L. The previous effluent standard for phosphorus in Wisconsin was 1.0 mg/L. The Wisconsin DNR estimated that the changes to these policies will require additional WWTP capital and operating/maintenance costs between $1.1 and $2.1 billion (WIDNR 2012).

1.2 Phosphorus Removal from Wastewater.

The engineering challenges are significant to meet ultra-low phosphorus effluent limits. Once the total phosphorus limit drops below 0.1 mg/L, separate stages of chemical precipitation/solids separation and filtration tend to be required (Strand Associates, 2008; Metcalf Eddy, 2013). The average phosphorus effluent concentration in Wisconsin was found to 0.59 mg/L and wastewater treatment plants are expected to use several technological upgrades to meet these required phosphorus levels (Strand Associates, 2008). This article will evaluate four tertiary technologies that reduce phosphorus that

also have the potential to remove PPCPs from wastewater: 1) ultrafiltration (UF) which uses a pressure-driven membrane filtration process that typically employs hollow-fiber membranes with a pore size range of approximately 0.01 – 0.05 µm; 2) microfiltration (MF) a pressure-driven membrane filtration process that typically employs hollow-fiber membranes with a pore size range of approximately 0.1 – 0.2 µm; 3) tertiary membrane bioreactor (MBR) which uses a suspended growth bioreactor paired with MF or UF; and 4) nanofiltration (NF) a pressure-driven membrane separation process with a pore size of 0.001–0.01 µm that employs the principles of reverse osmosis to remove dissolved contaminants from water, but at a lower operating pressure than reverse osmosis (USEPA 2005, USEPA 2013b). A rapid mix and flocculation step will likely precede these technologies (Strand Associates 2008). MF and UF are the technologies expected to be implemented to meet the phosphorus regulations, however, the MBR and NF technologies will also be assessed due to their ability to remove phosphorus and potentially mitigate PPCPs pollution.

Based on pore size, MF, UF, and NF are expected to remove the particulate phosphorus as an operational definition of particulate phosphorus is that it is retained by a 0.45-micron membrane filter paper, however, in practice, defects in the membrane can cause colloidal particles to escape to the product water (WERF, 2008). Under similar operating and influent conditions, the removal of total phosphorus is the greatest for NF, followed by MBR, UF, and MF. It is important to note that the observed removal efficiency of total phosphorus may not be the best benchmark for comparing these technologies, since the makeup of total phosphorus (soluble and particulate) can vary

significantly. To illustrate this relationship, MF and UF have 0 to 2% rejection of phosphate ions while NF has the ability to reject these ions (Metcalf Eddy, 2013; Visvanathan and Roy, 1997). In addition, the type and amount of coagulant used will significantly alter the removal efficiency (Metcalf Eddy, 2013). In general, with adequate coagulation and flocculation, these technologies have the potential to meet the lower required Wisconsin effluent regulations for phosphorus (50 µg/L) under normal operating and influent conditions (Chon et al., 2012; Gnirss and Dittrich, 2000; Strand Associates 2008).

1.3 Assessing Environmental Damage from PPCPs using the Risk Quotient.

Many of the wastewater treatment processes that are being implemented to meet phosphorus effluent regulations also have the potential to remove PPCPs. However, we must evaluate the potential damage associated with PPCPs concentrations in comparison to a toxicity endpoint. The RQ (also referred to as the hazard quotient or risk index) is often used to estimate the risks associated with low levels of PPCPs on environmental health and is defined as the ratio between the measured PPCPs concentration and its corresponding predicted no-effect concentration (PNEC) (Al Aukidy et al., 2012; Blair et al., 2013a; Christensen et al., 2009; Deblonde et al., 2011; Valcárcel et al., 2011). However, at this time, no study has quantitatively assessed the change in the PPCPs risk quotient (RQ) from the implementation of a policy to remove regulated pollutants from wastewater. In addition, calculating the RQ can be challenging due to a lack of information regarding the effects of PPCPs in the environment and difficulty in establishing the PNEC. Recent studies have found PPCPs of high or medium RQs in

wastewater effluent, waste sludge, and the aquatic environment (Tewari et al., 2013; Valcárcel et al., 2011; Verlicchi et al., 2012; Yu et al., 2013). By compiling the data related to PPCPs in wastewater, we can begin to assess the total PPCPs and RQ reduction from the changes to the Wisconsin NR codes.

2. Methods.

2.1. Data Analytic Plan.

This assessment focused on four different tertiary technologies for phosphorus removal as a possible addition to a conventional activated sludge (CAS) WWTP: UF, MF, MBR, and NF. These technologies were selected due to their ability to meet the phosphorus effluent regulations and the potential to remove PPCPs. These technologies were then assessed for their ability to remove PPCPs and a meta-analysis was completed to evaluate the removal efficiencies of pollutants based on different wastewater treatment facility configurations and technologies. For the meta-analysis, 1,085 influent concentration and removal efficiency data points were compiled from 57 peer-reviewed articles for 11 PPCPs. The data were compiled from recent papers along with guidance from the recent reviews by Verlicchi et al. (2012) and Oulton et al. (2010). We used observed concentrations from the literature review for the influent concentration. Bench scale or experiments with spiked PPCPs concentration, along with observed concentrations, were used for the removal efficiencies. Appendix E presents the concentrations and removal efficiencies, along with the corresponding citations.

From the meta-analysis, we collected data for the influent concentration and the removal efficiencies for CAS, MBR, MF, UF, and NF treatment processes. To determine

the range of PPCPs concentrations and removal efficiencies, resampling was completed using 10,000 iterations of a Monte Carlo Simulation using bootstrapping (Efron, 1987) for each variable using Matlab R2011a. Negative removal rates, where the effluent concentration was greater than the influent concentration, were changed to zero removal efficiency in the simulation, although many scenarios can explain the negative removal efficiencies (Blair et al., 2013b). Appendix E includes an example of the code used for ibuprofen. To determine the effluent concentration for each technology for each PPCP, we applied the relationship shown in equation 1 to the resampled distributions:

Effluent Concentration = Influent Concentration * (1 - Removal Efficiency) (1)

In addition, we determined the worst-case scenario, defined as the maximum observed concentration and minimum observed removal efficiency, for each PPCP with each technology.

2.2 Risk Quotient.

The RQ was calculated using the aquatic ecological PNEC with a safety factor (SF) of 1000 (Hernando et al., 2006). Equation 2 was used to calculate the RQ:

RQ = Observed Concentration * SF / PNEC (2)

An RQ greater than 1 is defined as high risk, an RQ from 0.1 to 1 is medium risk, and an RQ less than 0.1 is low risk. We used PNEC values from Verlicchi et al., (2012), ECOSAR v1.11 (USEPA 2012b), along with other published values. Appendix E shows these values, and the corresponding citations. When the values found in the literature were from an older version of ECOSAR, or if the data were not available, we used the

lowest freshwater toxicity value from ECOSAR v1.11. The PNEC selected from these values included the acute and chronic values from ECOSAR.

2.3. Present Value Calculations

Present value costs were calculated for the tertiary technologies. Cost information was obtained for UF and MF (Simultaneous Compliance Tool), MBR (AMTA, 2007), and NF (Costa and Pinho, 2006). For the comparison, capital, O&M, and annual costs were converted to a 20-year present value. A discount rate of 4.375 percent was used, as recommended by the WI DNR NR 110.09(1)(a), Wisconsin Administrative Code.

3. Results and Discussion.

3.1. Tertiary Wastewater Treatment to Remove PPCPs and Phosphorus.

The influent concentration range for the 11 PPCPs selected for this study was found to cover less than approximately one order of magnitude for the 25^{th} to 75^{th} percentiles, as shown in Figure 8. In addition, a summary from the meta-analysis displaying the mean influent concentration and removal efficiency is shown in Table 5.

Many PPCPs detected in wastewater effluent at high or medium RQ levels have the ability to be removed to lower RQ levels. We use the RQ to compare the effectiveness of technologies to remove PPCPs: this allows the use of a metric to determine what levels of the most harmful PPCPs are removed from wastewater, rather than just assessing the removal efficiency. For example, Figure 9a shows a simulation conducted for the concentration of the nonsteroidal anti-inflammatory drug ibuprofen after treatment by a conventional activated sludge system (labeled WWTP) and then with the addition of the four different tertiary treatment technologies selected to remove

phosphorus from wastewater. Figure 9b presents the RQ for ibuprofen, revealing that the majority of the effluent concentrations from a WWTP without tertiary treatment were high risk. However, the technologies used to remove phosphorus can potentially reduce the RQ. The findings reveal that MF and UF have limited potential to remove ibuprofen. A MBR is expected to reduce the median value from high to medium risk, but the removal efficiencies have a wide range, so the 25^{th} percentile is considered low risk and the maximum is considered high risk. With NF, the 25^{th} to 75^{th} percentile range decrease from high to low risk.

In contrast to ibuprofen, the RQ for some PPCPs can only be partially reduced with the use of the tertiary treatment processes used to remove phosphorus. Figure 10a and 10b show the concentration and RQ respectively for the anti-epileptic and mood stabilizing drug carbamazepine. Similar to ibuprofen, the carbamazepine effluent values from a WWTP without tertiary treatment were found to be high risk. However, with NF, the the 25^{th} to 75^{th} percentile range decreases from high to medium risk. The findings show that MBR, MF and UF have a limited potential to remove carbamazepine.

After treatment by a CAS WWTP, 100% of the maximum concentrations and 64% of the median concentrations were found to have a high RQ. Figure 11 reveals the maximum and median RQs across the different treatment technologies for 11 PPCPs. MF was found to have a minimal impact on the RQ. The MF technology may be able to remove PPCPs via adsorption on to membrane polymers or interaction with natural organic matter in wastewater (Luo et al., 2014), however, this low-pressure membrane has pore sizes that are insufficient to retain PPCPs (Oulton et al., 2010). The results show

that the median RQ decreased by 7% with the MF the technology. This also confirms that the MF used in a tertiary MBR process will provide limited removal of pharmaceuticals and the removal of PPCPs through a MBR using MF is completed primarily by biological degradation and sorption to solids (Snyder et al., 2007).

The results indicate that using NF, MBR, or UF technologies have the ability to reduce the RQ. The UF was found to reduce the worst-case scenario RQ by 21% and the median RQ by 28%. Similar to MF, UF has pore sizes insufficient to remove PPCPs, however, potential electrostatic repulsion or adsorption may explain the increased observed removal (Oulton et al., 2010). NF lowered the total PPCPs concentration and RQ more than the other treatment processes. For the worst-case scenario, using NF reduced the aggregate RQ by 72%. For median concentration values, the aggregate RQ decreased by 81% with NF. The MBR had removal rates approaching the removal seen by NF, lowering the maximum RQ by 69% and the median RQ by 71%.

3.2. Limitations.

Pharmaceuticals consumption patterns vary by region and the occurrence pattern of PPCPs is not static (Dickenson et al., 2011). Therefore, using observed influent data from one region may not represent the influent characteristics for other regions or WWTPs. In addition, different treatment processes within the same category have a wide range of removal efficiencies, even with the same PPCPs being assessed. For example, Chon et al. (2012) assessed three different nanofiltration membranes and the removal efficiencies had a range of 50% or more for the same pharmaceutical with different nanofiltration membranes. Further research must examine removal efficiencies for full-

scale WWTPs, especially membrane filters and their change in removal efficiencies under different conditions and with fouling (Bellona et al., 2004; Xu et al., 2006; Botton et al., 2012). Also, the chemical coagulation used for phosphorus or suspended solids removal is inadequate for PPCPs removal and a further understanding of the relationship between hydrophobicity and the removal of PPCPs is required (Alexander et al., 2012).

Other variables have the potential to impact removal of PPCPs from wastewater, such as the solids retention time, hydraulic retention time, concentration of suspended solids, fraction of autotrophic biomass, pH, and other operating and influent conditions (Constantine et al., 2006; Majewsky et al., 2011a; Majewsky et al., 2011b; Morton et al., 2013; Oulton et al., 2010; Verlicchi et al., 2012). An additional understanding of these variables is necessary to predict the removal of PPCPs from wastewater. Future models have the potential to include a mechanistic approach to predicting the removal efficiencies.

The PNEC values for PPCPs must constantly evolve as a better understanding of the environmental impacts of these pollutants and their metabolites becomes available. Using a model such as ECOSAR will allow an initial investigation to determine PPCPs of concern, but these models are not a replacement for complete toxicological testing of individual PPCPs and their mixtures on aquatic organisms. Additionally, we need an understanding of the impacts that PPCPs at low levels have on mammals, birds, and amphibians (Boxall et al., 2012). The incomplete aquatic toxicity data echoes the call for mandated toxicity testing of chemicals and epidemiologic monitoring of exposed human populations, particularly children (Landrigan and Goldman, 2011). Also, the PNEC may

not be adequate and a more thorough ranking may be needed, such as that reflected in the process developed by Kumar et al. (2010) or Ortiz de García et al. (2013).

The treatment options presented in this analysis do not represent all of the technologies available for the removal of phosphorus, and other systems have been shown to have high removal of phosphorus and PPCPs. For example, a system that pairs a 20 mg/L dosage of powdered activated charcoal paired with a ferric chloride coagulant demonstrated PPCP removal rates ranging between 67% and 97% for ten different PPCPs, and the phosphorus effluent levels were at concentrations between 20 and 50 µg/L (Treguer et al., 2012). Other systems that are potentially effective at removing phosphorus and PPCPs are reverse osmosis and advanced oxidation processes (Kim et al., 2007; Larsen et al., 2004; Luo et al., 2014; Oulton et al., 2010; Snyder et al., 2007; Urtiaga et al., 2013).

Developing a model to predict the removal of PPCPs from all configurations of WWTPs is not feasible at this time and it is critical to develop a better understanding of the mechanisms that determine the removal of PPCPs. Furthermore, the number of studies assessing the removal of PPCPs through tertiary MBR and membrane filtration is low and more studies are needed. Currently, over 3,000 PPCPs are in use and further monitoring of PPCPs is necessary since many of the widely prescribed pharmaceuticals lack environmental monitoring (Daughton, 2014; Monteiro and Boxall, 2010).

4.0. Management Implications.

The potential abatement of PPCPs from wastewater is a particularly timely issue due to the expected increase in worldwide PPCP usage and the potential for regulation of

PPCPs in the environment (Boxall et al., 2012; EC 2013; Eckstein and Sherk, 2011; Eckstein, 2012; USEPA 2013a). Numerous management implications emerge from this work. First, federal, state, and municipal policy makers and wastewater managers should consider the ability to meet phosphorus effluent regulations while minimizing the environmental damage from PPCPs and other pollutants. Due to the substantial costs affiliated with phosphorus reduction (USEPA, 2008), we recommend a broad monitoring study prior to implementing a phosphorus reduction technology to assess the level of PPCPs in the effluent. If these pollutants are found at concentrations that imply medium or high risk, a tertiary process should be considered that could meet both the phosphorus effluent regulations and reduce the risk from the contaminants of emerging concern.

Wastewater treatment plant managers and policy makers must consider whether the fate and transport of PPCPs along with dilution will mitigate the risk from PPCPs, as discussed by Al Aukidy et al., (2012) and Gros et al. (2010). Therefore, the ratio of the flow rate of a WWTP to the flow rate or volume of receiving water must be evaluated. Dilution may not be adequate to minimize the risk from PPCPs from large, urban WWTPs, even in bodies of water as large as the Great Lakes (Blair et al., 2013a). Additionally, we must consider the number of WWTPs in a watershed before we can model the total loading of PPCPs and the fate and transport of PPCPs and nutrients for a watershed (Schimmelpfennig et al., 2012). We should also identify other sources of PPCPs for a watershed, such as agricultural sources or from land application of biosolids (Lapen et al., 2008; Xia et al., 2005). The fate and transport of PPCPs in varying watersheds must be further addressed. As PPCPs effluent discharge increases with

expanding populations, the dilution afforded by receiving waters is expected to diminish (Daughton, 2003).

The technologies used for tertiary treatment could also eliminate many other pollutants such as metals, total suspended solids, and viruses, thereby further improving the quality of the wastewater effluent (USEPA 2013b). For example, the technology with the smallest pore size assessed in this research, NF, was found to reject between 58% and >99% of copper ions, but influent concentration and operating conditions such as pressure, pH, and biofouling were found to impact overall removal efficiency (Al-Rashdi et al., 2013). Likewise, NF and UF are both expected to remove viruses (USEPA 2013b), whereas MF will have a limited impact on viruses (Metcalf Eddy, 2013). In contrast, recent research has determined that high-pressure membrane filtration does not consistently achieve total nitrogen levels less than 1.0 mg/L and further treatment would be needed to reduce this concentration (Merlo et al., 2012). Overall, the removal of PPCPs and additional pollutants would potentially make it easier to treat water downstream for the production of drinking water. Policy makers should consider these benefits, particularly due to the unknown human health impacts from long-term exposures of PPCPs in drinking water (Bruce et al., 2010; Kumar et al., 2010; Jelic et al., 2012).

Tertiary treatment may not adequately remove some PPCPs such as carbamazepine and further actions may be necessary to achieve the desired level of environmental protection. If monitoring has indicated a specific pollutant has reliable occurrence above the RQ, the tertiary technology is not expected to adequately reduce the

RQ, and dilution is not sufficient to reduce the RQ, the next objective would be to address other alternative strategies. Potential short-term management options include discouraging disposal down the toilet/sink and encouraging medical providers to prescribe the minimum therapeutic dosage or drug alternatives (Cook et al., 2012; Daughton and Ruhoy, 2013). Potential long-term solutions include encouraging the development of green pharmaceuticals, using innovative toilet technologies, watershed management, and additional tertiary treatment for WWTPs such as NF, reverse osmosis, or advanced oxidation processes (Borsuk et al., 2008; Daughton, 2003; Eckstein and Sherk, 2012; Khetan and Collins, 2007; Luo et al., 2014; Schimmelpfennig et al., 2012; Snyder et al., 2007).

As an additional long-term solution, the ancillary benefits of phosphorus reduction technologies could be integrated into water quality trading (WQT). Wisconsin is implementing an adaptive management policy that allows phosphorus trading within a watershed between point and nonpoint sources (WIDNR 2013b). Water quality trading would allow WWTPs to purchase offsets from nonpoint sources, primarily the agricultural sector, rather than adding a tertiary treatment technology or upgrading current technologies to meet the desired phosphorus discharge levels. Although this strategy has the potential to meet the desired water quality standards for phosphorus, it overlooks the additional benefits of PPCPs risk mitigation from wastewater treatment for phosphorus. This information should be incorporated in the WQT assessment. For example, the removal of PPCPs with tertiary wastewater treatment using ozonation may have a positive economic environmental benefit of avoiding the discharge of

contaminants into water bodies (Molinos-Senante et al., 2013) and these values should be integrated in the WQT framework.

4.1. Cost of Technologies.

NF is expected to be the most costly process proposed in this article, followed by the tertiary MBR, UF, and MF, respectively, as shown in Table 6. Determining the costs of a tertiary treatment process depends on the selected technology, the quality and characteristics of the secondary effluent, the flow rate, the size/footprint constraints, along with other site specific demands and the values in Table 6 can vary based on these variables.

UF was found to reduce the RQ from PPCPs with no costs in addition to the expected cost to meet the phosphorus effluent limits. In Wisconsin, policy makers and WWTP managers will likely prefer the MF or UF technologies to meet these effluent regulations due to the relative lower costs. UF was estimated to reduce the median RQ by 28% and MF was found to reduce the median RQ by 7%. Therefore, the UF technology could be recommended over the MF technology to meet the phosphorus effluent limits due to the negligible different in cost and the ancillary benefit of increased PPCPs removal.

As an illustration of the cost of phosphorus reduction in Wisconsin, the Milwaukee Metropolitan Sewerage District (MMSD) in Wisconsin USA estimated it would cost $600 million (20 year present value) to meet the effluent regulations using a UF or MF range filtration process (Strand Associates, 2008). Using the same relationships shown in Table 6, the estimated costs for MMSD (two plants, design flow

of 140 MGD and 120 MGD and assuming these systems are bypassed during rain events) were estimated. The UF and MF technologies were found to have a 20 year present value of $533.0 million, the MBR was $791.7 million, and NF was $1,373 million.

For this municipality, upgrading from a UF or MF to a tertiary MBR would cost an estimated $259 million over 20 years. Knowing that MMSD serves 1.1 million individuals (NRDC, 2011), the estimated cost to upgrade from UF or MF to MBR would be $11.76 per capita per year. To put this into context, the willingness to pay to support pharmaceuticals take-back programs was estimated to be $14 per year per capita (Kotchen et al., 2009). It is not possible to assume that individuals are willing to pay similar amounts for infrastructure upgrades to remove PPCPs from wastewater as they would for take-back programs, however, this displays that upgrading from UF or MF to an MBR to increase PPCPs removal is a potentially feasible recommendation. Additional research is currently being conducted to assess the relationship between willingness to pay and tertiary wastewater treatment to reduce PPCPs.

The Wisconsin DNR estimated the net benefit of the phosphorus reduction rules to be $18.8 million, with a standard deviation of $97.1 million (WIDNR 2012). However, this analysis did not include the ancillary benefit of PPCPs removal. Under these rules, many WWTPs will upgrade their current configuration to include UF technologies which was estimated to reduce the RQ from PPCPs by 28%. Including the ancillary benefit of this reduction in the RQ is critical to assessing the net benefit of a policy and further research is needed on the valuation of PPCPs reduction.

Previous research has found that investment in advanced waste or drinking water

treatment to remove PPCPs is opposed by both wastewater treatment companies and drinking water suppliers (Titz and Döll, 2008). Therefore, utilizing existing regulations, such as those to remove phosphorus, may overcome the resistance to implementing technologies for PPCPs removal, particularly when significant capital infrastructure costs are involved.

5.0. Conclusion.

The presence of excess phosphorus and PPCPs in treated wastewater and surface waters are a significant environmental concern. This study assessed the change in PPCPs emissions, and the corresponding change in RQs, from WWTPs under a statewide phosphorus reduction policy that is similar to policies implemented across the world. This study found that it is possible to implement a wastewater treatment infrastructure that can mitigate the environmental damage from PPCPs while meeting strict phosphorus regulations. UF was estimated to reduce the median RQ from PPCPs by 28% while meeting the phosphorus effluent limits. In addition, the MBR and NF reduced the median RQ from PPCPs by an estimated 71% and 81%, respectively. However, the MBR and NF technologies come at a greater cost than the MF or UF technologies.

Using the results from our meta-analysis, this study presented numerous management options regarding the removal of PPCPs from wastewater. Primarily, it recommended the use of a meta-analysis, along with the utilization of the RQ, to determine the potential reduction in environment harm from PPCPs. From these results, managers and policy makers can then prioritize the specific needs of the WWTP and the watershed. If the findings revealed a specific PPCP had poor removal with tertiary

treatment technologies and insufficient dilution to mitigate the RQ, the study presented mechanisms that have the potential to mitigate PPCPs pollution. These options include short-term and long-term alternative mitigation strategies and adaptive management including water quality trading.

With the expansion of nutrient criteria for watersheds, nutrient abatement efforts, and the expected increase in PPCPs usage, this is an ideal time to address both nutrient and PPCPs reduction. At an estimated cost of $11.76 per capita per year to use a tertiary MBR rather than UF or MF, this upgrade should be considered by WWTP managers and policy makers. With the opposition by both wastewater treatment companies and drinking water suppliers to expand infrastructure to reduce PPCPs (Titz and Döll, 2008), utilizing existing regulations may overcome the resistance to implementing technologies for PPCPs removal.

Enhancing the wastewater treatment infrastructure to remove phosphorus and PPCPs is an essential long-term goal to minimize the environmental and human health impacts from wastewater. Overall, the novel approach developed in this research validated that phosphorus reduction policies can be utilized to reduce the environmental damage caused by PPCPs and we recommend this approach be integrated into future nutrient abatement policies and regulations.

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Table 5: Summary from meta-analysis of influent concentration and fraction removed for 11 PPCPs and 4 tertiary technologies.

		Mean	Mean Fraction Removed				
Pollutant	Classification	Influent (ng/L)	CAS	MBR	UF	NF	MF
Acetaminophen	Antipyretic, analgesic	50,000	0.95	1.00	0.06	0.40	0.04
Caffeine	Stimulant	22,000	0.85	0.99	0.07	1.00	0.00
Carbamazepine	Anticonvulsant	420	0.17	0.15	0.14	0.73	0.11
Estradiol (E2)	Sex Hormone	33	0.85	0.96	0.97	0.52	0.00
Estriol (E3)	Sex Hormone	160	0.84	0.99	0.41	0.98	0.00
Gemfibrozil	Antilipemic	1,100	0.42	0.63	0.07	0.50	0.00
Ibuprofen	Analgesic	39,000	0.87	0.91	0.08	1.00	0.55
Naproxen	NSAID	3,800	0.61	0.80	0.18	0.86	0.00
Sulfamethoxazole	Sulfonamide antibiotic	280	0.45	0.58	0.09	0.64	0.00
Triclosan	Antimicrobial	2,500	0.73	0.78	0.44	0.99	0.00
Trimethoprim	Pyrimidine antibiotic	370	0.51	0.47	0.20	0.95	0.00

Table 6: Estimated 20 year present value (PV, in millions of US dollars, using i=4.875%) for selected technologies with a design flow of 100 MGD.

	Estimated 20 Year PV (in millions)	Source
UF	$205.0	Simultaneous Compliance Tool
MF	$205.0	Simultaneous Compliance Tool
MBR	$304.5	AMTA 2007
NF	$528.0	Costa and Pinho, 2006

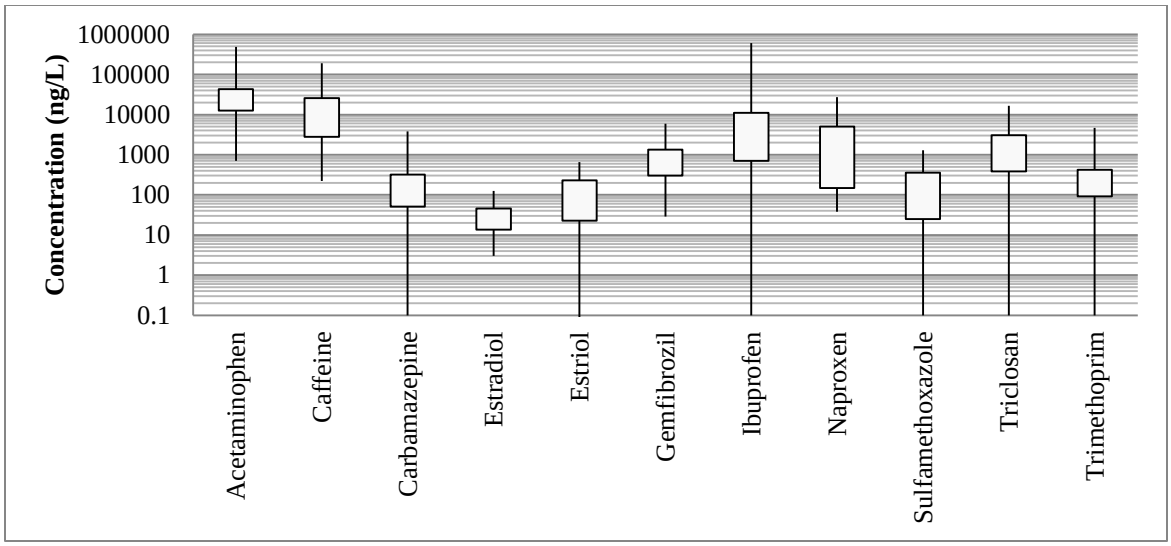


Figure 8: Influent concentration results of 11 PPCPs from meta-analysis. Box plot represents minimum, 25th percentile, 75th percentile, and maximum.

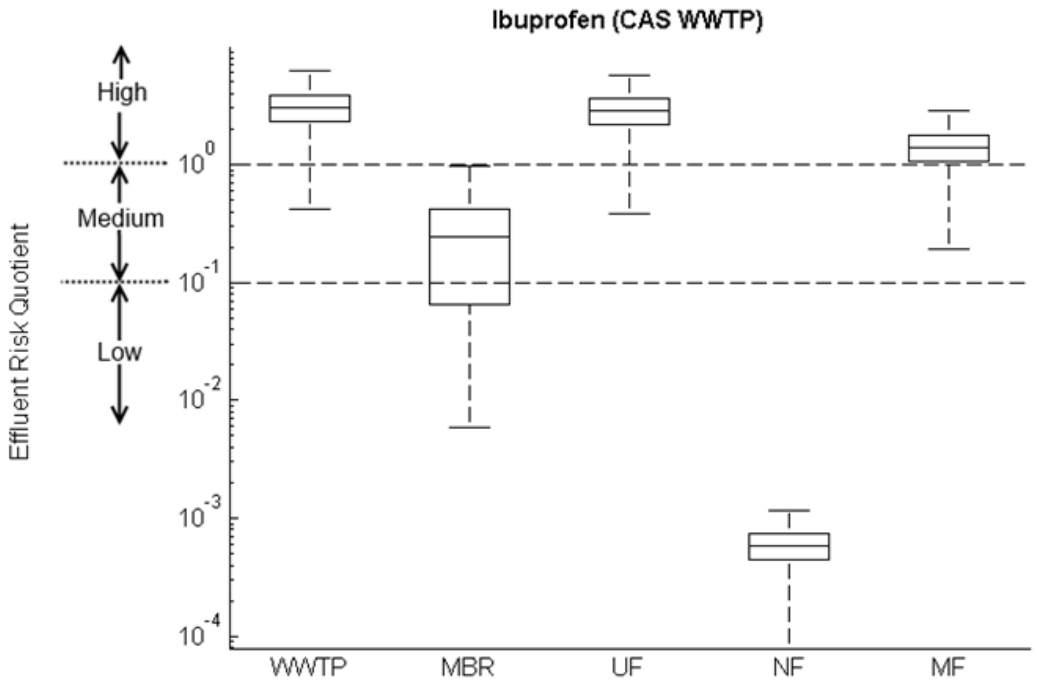


Figure 9: Effluent risk quotient for ibuprofen after treatment from a CAS then tertiary treatment with MBR, UF, NF, and MF systems. Central mark is the median, the edges of the box are the 25th and 75th percentiles, and the whiskers extend to the most extreme data points not considered outliers.

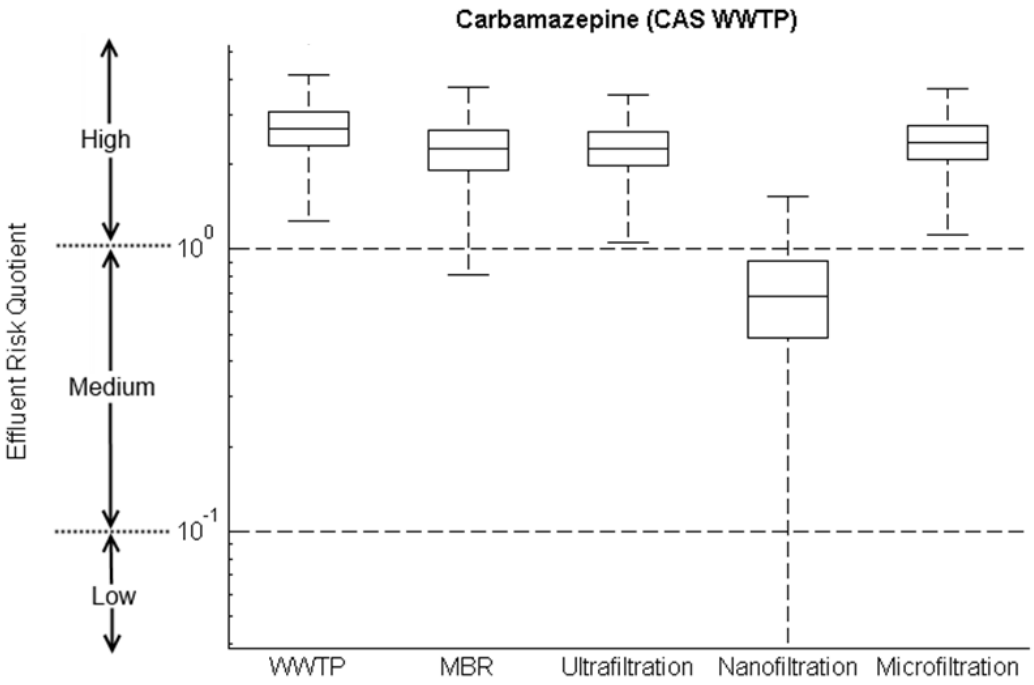
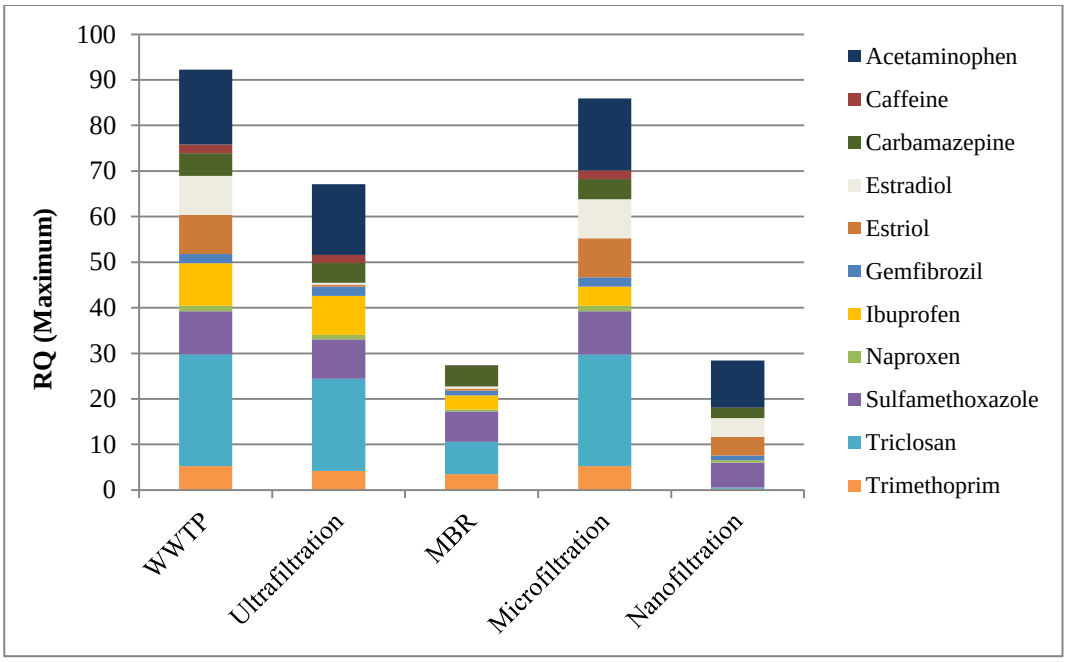
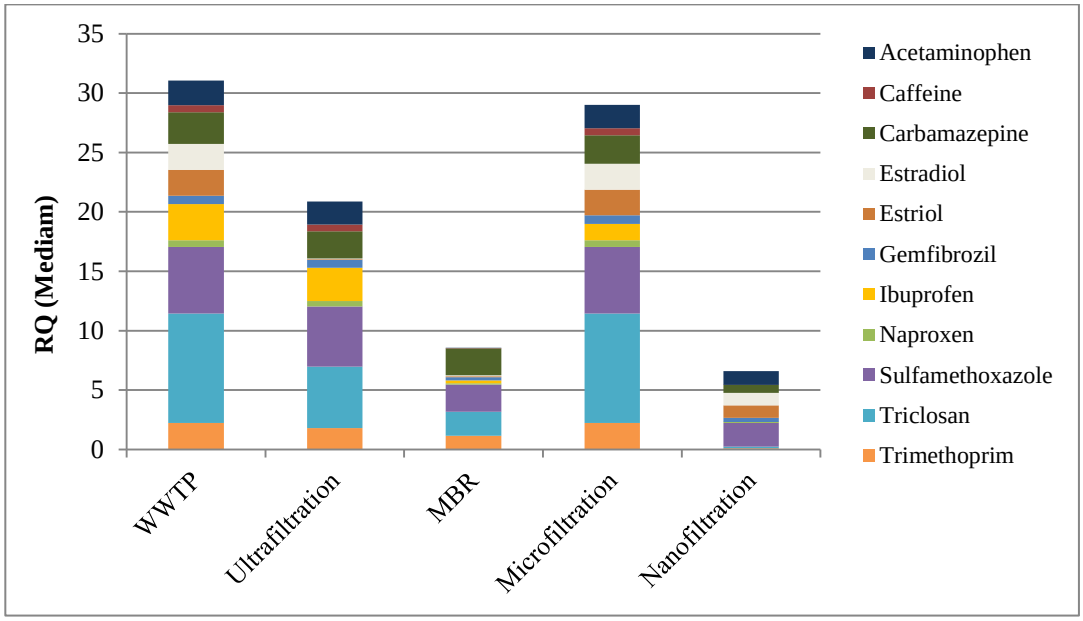


Figure 10: Effluent risk quotient for carbamazepine after treatment from a CAS then tertiary treatment with MBR, UF, NF, and MF systems. Central mark is the median, the edges of the box are the 25th and 75th percentiles, and the whiskers extend to the most extreme data points not considered outliers.



(a)



(b)

Figure 11: (a) Maximum RQ and (b) Median RQ for 11 PPCPs

Chapter 5: CONCLUSION AND DISCUSSION

The omnipresent usage of pharmaceuticals and personal care products (PPCPs) has led to detection in the aquatic environmental at levels that are potentially damaging to aquatic organisms (Blair et al., 2013a; Brodin et al. 2013; Christensen et al., 2009; Valcárcel et al., 2011; Verlicchi et al., 2011). The chapters of this dissertation presented noteworthy advances in the understanding of PPCPs in wastewater and the environment, with an emphasis on the potential environmental harm from these pollutants. The recommendations for future research based on each data chapter are presented below. In addition, debating and evaluating the feasibility of the various solutions recommended to reduce PPCPs emissions is needed. Therefore, a section comparing the current recommended solutions to mitigate PPCPs pollution is included.

Future Research

The Fate of PPCPs in Wastewater

Predicting the removal of PPCPs from wastewater is challenging. However, modeling efforts based on the known chemical characteristics has been found to advance the prediction of the fate of these complex pollutants. Overall, a further understanding of the variables that influence fate of PPCPs in a wastewater treatment plant is required to predict the removal of PPCPs from aerobic treatment processes. A critical characteristic necessary to determine the removal efficiency of PPCPs from a conventional activated sludge wastewater treatment plant is the biological degradation rate constant (Kb),

however, the Kb values have not been developed for many of the commonly used PPCPs (Dickenson et al., 2010). The predominant method to assess biological degradation of PPCPs is pseudo-first-order kinetics (Joss et al., 2006; Schwarzenbach et al., 2003). Recent research has raised concerns regarding the inability to assess the activity of the total suspended solids using pseudo-first-order kinetics and methods have been developed to assess the activity of the biomass (Majewsky et al., 2011a). Therefore, future research will examine the relationship between the activity of the biomass in activated sludge and the biological degradation rate of PPCPs.

Many studies assessing the fate of PPCPs in wastewater have encountered issues with negative removal efficiencies, where lower concentrations were seen in the influent than the effluent and this requires further research. As summarized by Blair et al. (2013b), reasons have been proposed to explain the observed negative removal efficiency. These include improperly addressing the fluid dynamics of a WWTP (Majewsky et al., 2011b; Ort et al., 2010), the conjugate compounds that are not detected at the influent could be retransformed into the original compound due to biological processes (Monteiro and Boxall, 2010; Salgado et al., 2012), desorption from the return activated sludge may occur during the secondary treatment process (Salgado et al., 2012), and PPCPs may be released from fecal particles as the feces are being broken down by microbes (Göbel et al., 2007). Understanding the cause of negative removal efficiencies is a future research goal.

Ecological Risk and Occurrence of PPCPs in Lake Michigan:

It was found that the distribution of PPCPs in a large lake system was more

widespread than previously thought and the levels detected are of concern. Future research on PPCPs in wastewater and the environment will require an interdisciplinary approach combining engineering, environmental science, economics, policy, and management. It is critical to point out that further research is needed on the fate and corresponding ecological and human health impacts surrounding PPCPs in the aquatic environment. In particular, experimental data assessing the potential impacts and biological degradation of PPCPs in the aquatic environment are needed since the QSAR models used in this dissertation have significant limitations. Future research on large lake systems should also more closely assess the hydrodynamic characteristics of these systems to further evaluate the distribution in the aquatic environment.

A better understanding the various point and nonpoint sources of PPCPs is needed. An unexplored potential source of PPCPs is from meat processing plants. In Wisconsin, many slaughterhouses and meat packing facilities discharge into the waste stream without pretreatment (UW Extension, 2012). As an example, a widely used veterinary pharmaceutical in the treatment of bacterial infection in swine is carbadox. This pharmaceutical is not to be used 42 days prior to slaughter in the United States and has been banned for use in Canada, the European Union, and Australia due to the potential carcinogenic properties and the potential to cause birth defects (Human Metabolome Database, 2013). Carbadox was detected at SSWRF and Lake Michigan in the 2009-2010 study and consistently during the more recent 2013 study. It is unknown whether the source was from agricultural sources, which are sparse in the Milwaukee area, or one of the swine processing plants, which are numerous in Milwaukee, and more

research is needed on this potential, unexplored, source of pharmaceuticals entering the waste stream.

Using Phosphorus Regulations to Mitigate the Risk from PPCPs:

When assessing the recommended technologies to meet wastewater effluent standards, it is recommended to consider the net environmental benefit of the technologies rather than focusing on a single pollutant. Therefore, completing a Life Cycle Assessment (LCA) on the technologies to remove PPCPs from wastewater would be a valuable assessment to determine the overall change in emissions from the use of advanced tertiary technologies. The objective of wastewater treatment is to increase the effluent quality through the reduction of endpoints such as environmental toxicity, hormone effects, and pathogenic effects of the effluent. Using a LCA, it would be possible to assess the environmental trade-off since the increased quality of the effluent will happen at the expense of increased resource and energy consumption (Wenzel et al., 2008). Future research would assess the technologies implemented to remove regulated pollutants and determine their abilities to remove PPCPs and other unregulated pollutants.

Overall, the need for economic, management, and policy research on PPCPs in the aquatic environment is significant. Future research should focus on comparing the marginal benefits of removal with the marginal abatement costs. It is possible to evaluate the public's perception of the environmental impacts PPCPs and to estimate the marginal abatement costs. In addition, expert stakeholders' views on the management of pharmaceuticals in the environment has been assessed (Doerr-MacEwen and Haight,

2006), however, applying other policy research tools to these issues is an additional future research goal.

Recommendations for PPCPs Mitigation.

Debating and evaluating the feasibility of the various solutions recommended to reduce PPCPs emissions is needed. Short-term and long-term alternative PPCPs mitigation strategies were presented in Chapter 4 and some of these concepts are more plausible than others. The advantages and limitations of the current efforts to reduce PPCPs pollution will now be presented where the proposed solutions will consist of: disposal of unused PPCPs through trash disposal or take-back programs, green pharmaceuticals, minimum therapeutic doses, utilizing toilets with waste separation, and advanced wastewater treatment technologies.

The majority of the current efforts to reduce PPCPs from entering the aquatic environment focus on disposal of unused PPCPs (Daughton, 2010; Kotchen et al., 2009). The major goals of disposing unused PPCPs are to prevent accident exposure or abuse along with protecting the environment. For some PPCPs, such as narcotics and fentanyl transdermal patches, it has been determined that the potential for accidental exposure or abuse outweighs the environmental protection concerns at this time, and these pharmaceuticals are recommended for toilet disposal (USFDA, 2012).

PPCPs mitigation through the efforts advocating non-toilet disposal options may face diminishing returns. Along with some PPCPs being recommended for toilet disposal, it is also important to note that disposal of unused pharmaceuticals through take-back programs or trash disposal does not address an overarching problem with PPCPs: many

PPCPs are excreted from the body remaining partially unchanged when excreted (Kasprzyk-Hordern et al., 2009). It was found that 0% – 17% of respondents in the UK and 28% of respondents in the US disposed of unused pharmaceuticals in the toilet/drain and the majority of respondents disposed of their pharmaceuticals in the trash bin (Bound et al., 2005; Kotchen et al., 2009). Disposal to trash also increases the possibility of human or pet exposure to unused PPCPs and could also allow environmental contamination (Glassmeyer et al., 2009). Overall, the efforts to encourage trash disposal may have a limited overall impact on the amount of PPCPs entering the environment.

Pharmaceutical take-back programs have shown limited success, such as in Wisconsin, where an estimated 2% of unused pharmaceuticals were recovered (WIDNR 2012). In addition, no studies have succeeded in linking drug collection programs to either reductions in environmental levels or to reductions in human poisonings (Daughton, 2010). Due to the limited number of take-back options, take-back programs could increase the risk of accidental poisonings, diversion, or abuse due to stockpiling of unused medications awaiting take-back (Cook et al., 2012a). Concerns have also been raised regarding the net environmental benefit of incineration and pollution from transportation of PPCPs returned through take-back programs (Cook et al., 2012b).

Pharmaceutical take-back programs currently have significant regulatory issues in the United States. Under the Controlled Substances Act, the U.S. Drug Enforcement Administration requires controlled substances to be surrendered to the proper law enforcement officials (Lubick 2010). Therefore, take-back programs for medical employees (e.g. nursing home employees) are subject to the same rules that are meant to

keep controlled substances from reentering the supply chain either legally or illegally which has caused stockpiling of unused pharmaceuticals in medical settings. In addition, most pharmacies across the United States cannot accept unused pharmaceuticals due to regulations under the Controlled Substance Act (Barlas, 2009; Lubick, 2010).

The green pharmaceutical movement calls for pharmaceutical developers and manufacturers to increase uptake of the compound by the body (thereby requiring lower doses), creating compounds that maximize their susceptibility to environmental biodegradation or photolysis, or creating compounds that are less dangerous to the environment (Daughton 2003). Meeting these requirements is difficult at this time due to the cost and issues associated with the development of pharmaceuticals (Adams and Brantner, 2006; Sumpter, 2010) and the environmental release of pharmaceuticals from wastewater is largely unregulated across the world at this time (Ryu et al., 2014). Overall, the incentives to develop new compounds that have an increased uptake or lower environmental toxicity do not currently exist.

Using the minimum therapeutic dosage or drug alternatives has been proposed to lower the concentration of PPCPs in wastewater (Daughton and Ruhoy 2013). However, over the last 10 years, the percentage of Americans who took at least one prescription drug in the past month increased from 44% to 48%, the percentage who use two or more drugs increased from 25% to 31%, and the percentage who use five or more drugs increased from 6% to 11% (CDC 2010). In addition, resistance may be met by critical stakeholders (e.g. medical doctors) due to a primary goal of pharmaceutical interventions is to get patients to the therapeutic range as fast as possible and starting at the minimum

therapeutic dosage could cause additional hospital visits and testing. Therefore, the possibility of reversing this trend or requesting patients to start their dosage at the minimum dosage rather than the mean dosage is challenging at this time.

Human waste separation combined with onsite treatment to remove PPCPs has shown to be a promising concept (Borsuk et al., 2008; Lamichhane, 2012). However, the high cost associated with upgrading all toilets (public and private), along with the regulatory framework needed, causes this concept to be difficult to implement in the short-term. In addition, many PPCPs are rinsed off and this concept would not address these pollutants.

As discussed in Chapter 4, advanced wastewater treatment technologies have the potential to remove PPCPs, but also carry significant capital, operating, and maintenance costs. In addition, further expanding a WWTPs capability to remove PPCPs may also be beneficial due to some pharmaceuticals being recommended for disposal in toilets to lower accidental contact risks, such as fentanyl transdermal patches and narcotics (USFDA 2012). In addition, advanced wastewater treatment processes can remove other contaminants of concerns, such as heavy metals, viruses, industrial chemicals, and household chemicals. Overall, additional wastewater treatment would have the potential to remove PPCPs that are disposed of through the toilet/drain, PPCPs that are excreted in feces or urine, and PPCPs that are rinsed or washed off, thereby removing a majority of the PPCPs in the waste stream.

Overall, a multifaceted approach is the best long-term solution to prevent PPCPs emissions into the environment. While reducing the disposal of PPCPs through the

toilet/drain is a great first step, exploring other safe disposal methods is needed. In addition, green pharmaceuticals, waste separating toilets, and recommending the minimum therapeutic dosage are useful concepts that warrant further research, but have significant obstacles to their implementation. Updating wastewater treatment plants is costly, but infrastructure upgrades have the potential to offer significant environmental benefits and research should continue to evaluate the feasibility of advanced wastewater treatment technologies.

Conclusion.

The chapters of this dissertation presented advances in the understanding of PPCPs in wastewater and the environment. The experiments carried out in this investigation provided data that are essential for obtaining a comprehensive understanding of the occurrence, fate, and transport of PPCPs in a conventional active sludge wastewater treatment plant and Lake Michigan. This work also furthered the debate on how to achieve meaningful reduction in PPCPs from wastewater through the optimization of current regulations that utilize tertiary treatment technologies.

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Appendix A: Supporting Data for Chapter 2. All units are ng/L.

		Metformin	Albuterol	Cimetidine	Ranitidine	Acetaminophen
	MDL	0.51	1.4	1.3	0.87	2.5
	MQL	1.5	4.2	3.8	2.6	7.5
Raw Influent	5/15/2009	34000	No Detection	No Detection	10	26000
Primary Effluent	5/15/2009	38000	No Detection	No Detection	No Detection	8000
Secondary Effluent	5/15/2009	33000	No Detection	No Detection	No Detection	29
Final Effluent	5/15/2009	38000	No Detection	No Detection	No Detection	0
Raw Influent	6/15/2009	75000	No Detection	No Detection	130	150000
Primary Effluent	6/15/2009	34000	No Detection	No Detection	91	150000
Secondary Effluent	6/15/2009	33000	No Detection	No Detection	No Detection	29
Final Effluent	6/15/2009	47000	No Detection	No Detection	13	650
Raw Influent	8/18/2009	100000	23	No Detection	No Detection	12000
Primary Effluent	8/18/2009	78000	14	No Detection	No Detection	12000
Secondary Effluent	8/18/2009	15000	12	No Detection	No Detection	54
Final Effluent	8/18/2009	19000	No Detection	No Detection	No Detection	85
Raw Influent	10/19/2009	100000	19	No Detection	1.3	14000
Primary Effluent	10/19/2009	92000	19	No Detection	330	16000
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	11000	No Detection	No Detection	4.3	52
Raw Influent	4/9/2010	22000	No Detection	14	21	5900
Primary Effluent	4/9/2010	46000	No Detection	9.6	16	9700
Secondary Effluent	4/9/2010	27000	No Detection	7.2	18	0
Final Effluent	4/9/2010	33000	No Detection	No Detection	No Detection	14
Raw Influent	6/15/2010	3200	13	39	28	22000
Primary Effluent	6/15/2010	9800	69	120	89	22000
Secondary Effluent	6/15/2010	800	9	18	29	22000
Final Effluent	6/15/2010	640	2.6	No Detection	No Detection	27

		Azithromycin	Caffeine	Carbadox	Carbamazepine	Ciprofloxacin
	MDL	3.7	3.1	3.4	2.7	3.3
	MQL	11	9.3	10	8.2	9.9
Raw Influent	5/15/2009	5.9	34000	44	230	No Detection
Primary Effluent	5/15/2009	14	26000	68	96	No Detection
Secondary Effluent	5/15/2009	6.5	1000	No Detection	170	No Detection
Final Effluent	5/15/2009	16	1400	6.9	150	No Detection
Raw Influent	6/15/2009	280	130000	No Detection	310	No Detection
Primary Effluent	6/15/2009	340	110000	No Detection	310	No Detection
Secondary Effluent	6/15/2009	6.5	1000	No Detection	170	No Detection
Final Effluent	6/15/2009	350	580	15	340	No Detection
Raw Influent	8/18/2009	No Detection	9400	No Detection	73	No Detection
Primary Effluent	8/18/2009	No Detection	8600	No Detection	70	No Detection
Secondary Effluent	8/18/2009	No Detection	34	No Detection	88	No Detection
Final Effluent	8/18/2009	230	940	No Detection	230	No Detection
Raw Influent	10/19/2009	No Detection	9000	No Detection	51	87
Primary Effluent	10/19/2009	No Detection	10000	No Detection	76	19
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	160	No Detection
Raw Influent	4/9/2010	No Detection	3300	11	21	No Detection
Primary Effluent	4/9/2010	No Detection	4200	21	24	No Detection
Secondary Effluent	4/9/2010	No Detection	71	No Detection	33	No Detection
Final Effluent	4/9/2010	No Detection	43	No Detection	27	No Detection
Raw Influent	6/15/2010	32	8800	No Detection	72	13
Primary Effluent	6/15/2010	47	7800	No Detection	59	16
Secondary Effluent	6/15/2010	47	7800	No Detection	59	16
Final Effluent	6/15/2010	210	19	No Detection	200	No Detection

		Clarithromycin	Codeine	Cotinine	Digoxigenin	Diltiazem
	MDL	3.2	3.6	3.5	4.4	3.5
	MQL	9.6	11	11	13	10
Raw Influent	5/15/2009	No Detection	160	No Detection	No Detection	170
Primary Effluent	5/15/2009	No Detection	84	No Detection	No Detection	150
Secondary Effluent	5/15/2009	No Detection	170	No Detection	No Detection	160
Final Effluent	5/15/2009	No Detection	82	No Detection	No Detection	57
Raw Influent	6/15/2009	No Detection	540	No Detection	850	640
Primary Effluent	6/15/2009	No Detection	460	No Detection	710	720
Secondary Effluent	6/15/2009	No Detection	170	No Detection	No Detection	160
Final Effluent	6/15/2009	No Detection	230	No Detection	No Detection	510
Raw Influent	8/18/2009	No Detection	36	37	No Detection	46
Primary Effluent	8/18/2009	No Detection	15	65	No Detection	34
Secondary Effluent	8/18/2009	No Detection	9.6	No Detection	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	120	No Detection	No Detection	220
Raw Influent	10/19/2009	No Detection	37	36	61	57
Primary Effluent	10/19/2009	No Detection	42	56	76	44
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	No Detection	15	No Detection	No Detection	20
Primary Effluent	4/9/2010	No Detection	28	No Detection	No Detection	17
Secondary Effluent	4/9/2010	No Detection	62	No Detection	No Detection	22
Final Effluent	4/9/2010	No Detection	33	No Detection	No Detection	No Detection
Raw Influent	6/15/2010	5.6	54	130	52	41
Primary Effluent	6/15/2010	No Detection	43	810	68	38
Secondary Effluent	6/15/2010	No Detection	43	810	68	38
Final Effluent	6/15/2010	19	150	No Detection	No Detection	34

		Paraxanthine	Diphenhydramine	Fluoxetine	Lincomycin
	MDL	6.1	3.6	3.5	3.1
	MQL	18	11	11	9.3
Raw Influent	5/15/2009	5700	75	61	No Detection
Primary Effluent	5/15/2009	5000	78	100	No Detection
Secondary Effluent	5/15/2009	540	140	25	No Detection
Final Effluent	5/15/2009	770	87	41	No Detection
Raw Influent	6/15/2009	15000	420	95	25
Primary Effluent	6/15/2009	13000	420	120	29
Secondary Effluent	6/15/2009	540	140	25	No Detection
Final Effluent	6/15/2009	210	360	96	15
Raw Influent	8/18/2009	3000	33	18	3.2
Primary Effluent	8/18/2009	2800	21	9.6	No Detection
Secondary Effluent	8/18/2009	25	5.8	5	No Detection
Final Effluent	8/18/2009	370	98	78	No Detection
Raw Influent	10/19/2009	2400	37	23	3.7
Primary Effluent	10/19/2009	3100	26	12	4
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	740	11	6.1	No Detection
Primary Effluent	4/9/2010	1500	7	4	No Detection
Secondary Effluent	4/9/2010	31	10	7	No Detection
Final Effluent	4/9/2010	14	5.6	5.1	No Detection
Raw Influent	6/15/2010	4500	34	12	No Detection
Primary Effluent	6/15/2010	2700	22	8.3	No Detection
Secondary Effluent	6/15/2010	2700	22	8.3	No Detection
Final Effluent	6/15/2010	30	22	14	No Detection

		Lomefloxacin	Miconazole	Norfloxacin	Ofloxacin
	MDL	4.7	2.7	5.1	3.9
	MQL	14	8.1	15	12
Raw Influent	5/15/2009	No Detection	36	No Detection	200
Primary Effluent	5/15/2009	No Detection	15	No Detection	150
Secondary Effluent	5/15/2009	No Detection	6.4	No Detection	220
Final Effluent	5/15/2009	No Detection	10	No Detection	88
Raw Influent	6/15/2009	No Detection	81	No Detection	980
Primary Effluent	6/15/2009	No Detection	69	No Detection	530
Secondary Effluent	6/15/2009	No Detection	6.4	No Detection	220
Final Effluent	6/15/2009	No Detection	6	No Detection	670
Raw Influent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	25	No Detection	200
Raw Influent	10/19/2009	No Detection	No Detection	No Detection	32
Primary Effluent	10/19/2009	No Detection	No Detection	No Detection	6.1
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Primary Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	4/9/2010	No Detection	No Detection	No Detection	11
Final Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Raw Influent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Primary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Final Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection

		Oxacillin	Roxithromycin	Sarafloxacin	Sulfachloropyridazine
	MDL	2.5	4.3	5.4	4.1
	MQL	7.4	13	16	12
Raw Influent	5/15/2009	No Detection	420	No Detection	No Detection
Primary Effluent	5/15/2009	No Detection	64	No Detection	No Detection
Secondary Effluent	5/15/2009	No Detection	No Detection	No Detection	No Detection
Final Effluent	5/15/2009	No Detection	28	No Detection	No Detection
Raw Influent	6/15/2009	No Detection	1500	No Detection	No Detection
Primary Effluent	6/15/2009	No Detection	88	No Detection	No Detection
Secondary Effluent	6/15/2009	No Detection	No Detection	No Detection	No Detection
Final Effluent	6/15/2009	No Detection	110	No Detection	No Detection
Raw Influent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	18	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Primary Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Final Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Raw Influent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Primary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Final Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection

		Sulfadiazine	Sulfadimethoxine	Sulfamerazine	Sulfamethazine
	MDL	2.8	2.4	2.1	4
	MQL	8.5	7.1	6.2	12
Raw Influent	5/15/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	5/15/2009	No Detection	No Detection	1.3	No Detection
Secondary Effluent	5/15/2009	No Detection	No Detection	3.2	No Detection
Final Effluent	5/15/2009	No Detection	No Detection	1.6	No Detection
Raw Influent	6/15/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	6/15/2009	No Detection	No Detection	No Detection	48
Secondary Effluent	6/15/2009	No Detection	No Detection	3.2	No Detection
Final Effluent	6/15/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	13	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Primary Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	4/9/2010	3	No Detection	No Detection	No Detection
Final Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Raw Influent	6/15/2010	2.8	No Detection	No Detection	No Detection
Primary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Final Effluent	6/15/2010	5.7	No Detection	No Detection	No Detection

		Sulfamethizole	Sulfamethoxazole	Sulfanilamide	Sulfathiazole
	MDL	4.2	4.1	2.9	2.6
	MQL	13	12	8.6	7.8
Raw Influent	5/15/2009	No Detection	210	500	No Detection
Primary Effluent	5/15/2009	No Detection	110	No Detection	No Detection
Secondary Effluent	5/15/2009	No Detection	300	68	No Detection
Final Effluent	5/15/2009	No Detection	150	69	No Detection
Raw Influent	6/15/2009	No Detection	1200	900	2.5
Primary Effluent	6/15/2009	No Detection	1300	2500	5
Secondary Effluent	6/15/2009	No Detection	300	68	No Detection
Final Effluent	6/15/2009	No Detection	810	900	No Detection
Raw Influent	8/18/2009	No Detection	110	59	3.8
Primary Effluent	8/18/2009	No Detection	75	42	4.2
Secondary Effluent	8/18/2009	No Detection	67	42	No Detection
Final Effluent	8/18/2009	No Detection	360	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	120	55	No Detection
Primary Effluent	10/19/2009	No Detection	160	93	4.2
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	31	49	No Detection
Raw Influent	4/9/2010	No Detection	54	No Detection	No Detection
Primary Effluent	4/9/2010	No Detection	21	No Detection	No Detection
Secondary Effluent	4/9/2010	No Detection	34	No Detection	No Detection
Final Effluent	4/9/2010	No Detection	17	No Detection	No Detection
Raw Influent	6/15/2010	No Detection	170	No Detection	No Detection
Primary Effluent	6/15/2010	No Detection	57	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	57	No Detection	No Detection
Final Effluent	6/15/2010	No Detection	210	No Detection	No Detection

		Thiabendazole	Trimethoprim	Triclosan	Triclocarban	Naproxen
	MDL	1.8	3.4	0.53	0.48	0.97
	MQL	5.3	10	1.6	1.4	2.9
Raw Influent	5/15/2009	5.4	220	4300	5200	4200
Primary Effluent	5/15/2009	7.2	120	1600	1200	2800
Secondary Effluent	5/15/2009	9.5	260	120	120	520
Final Effluent	5/15/2009	11	62	74	150	250
Raw Influent	6/15/2009	26	590	9100	5900	9400
Primary Effluent	6/15/2009	19	510	5700	2800	11000
Secondary Effluent	6/15/2009	9.5	260	120	120	520
Final Effluent	6/15/2009	16	660	270	250	320
Raw Influent	8/18/2009	No Detection	36	89	3.3	2900
Primary Effluent	8/18/2009	No Detection	31	330	120	260
Secondary Effluent	8/18/2009	No Detection	21	24	74	110
Final Effluent	8/18/2009	14	190	850	980	580
Raw Influent	10/19/2009	No Detection	52	130	35	1800
Primary Effluent	10/19/2009	No Detection	54	530	400	3100
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	27	8.3
Raw Influent	4/9/2010	No Detection	18	610	110	780
Primary Effluent	4/9/2010	No Detection	19	250	17	460
Secondary Effluent	4/9/2010	No Detection	41	150	61	19
Final Effluent	4/9/2010	No Detection	13	46	40	26
Raw Influent	6/15/2010	No Detection	45	680	140	3100
Primary Effluent	6/15/2010	No Detection	34	350	67	4000
Secondary Effluent	6/15/2010	No Detection	34	350	67	4000
Final Effluent	6/15/2010	2.8	170	120	99	19

		Gemfibrozil	Ibuprofen	Estriol	17-alpha-estradiol
	MDL	1.6	4.7	2	1.2
	MQL	4.8	14	6.1	3.5
Raw Influent	5/15/2009	29	3000	No Detection	No Detection
Primary Effluent	5/15/2009	62	No Detection	No Detection	No Detection
Secondary Effluent	5/15/2009	85	No Detection	No Detection	No Detection
Final Effluent	5/15/2009	53	No Detection	No Detection	No Detection
Raw Influent	6/15/2009	150	11000	10	No Detection
Primary Effluent	6/15/2009	220	14000	44	No Detection
Secondary Effluent	6/15/2009	85	No Detection	No Detection	No Detection
Final Effluent	6/15/2009	270	No Detection	No Detection	No Detection
Raw Influent	8/18/2009	200	1300	No Detection	No Detection
Primary Effluent	8/18/2009	460	No Detection	No Detection	No Detection
Secondary Effluent	8/18/2009	420	210	No Detection	No Detection
Final Effluent	8/18/2009	66	No Detection	No Detection	No Detection
Raw Influent	10/19/2009	97	840	No Detection	No Detection
Primary Effluent	10/19/2009	630	No Detection	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	30	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	510	670	22	10000
Primary Effluent	4/9/2010	550	520	6	7500
Secondary Effluent	4/9/2010	1100	No Detection	No Detection	2900
Final Effluent	4/9/2010	750	No Detection	No Detection	4700
Raw Influent	6/15/2010	1200	4800	No Data	No Data
Primary Effluent	6/15/2010	1100	4000	41	760000
Secondary Effluent	6/15/2010	1100	4000	6.1	No Detection
Final Effluent	6/15/2010	1100	No Detection	No Data	No Data

		Estrone	17-Beta-estradiol	Testosterone	Androsterone
	MDL	2.2	1.3	1.1	0.55
	MQL	6.7	3.8	3.2	1.6
Raw Influent	5/15/2009	64	No Detection	1.7	48
Primary Effluent	5/15/2009	52	No Detection	No Detection	No Detection
Secondary Effluent	5/15/2009	No Detection	0.91	No Detection	No Detection
Final Effluent	5/15/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	6/15/2009	350	9.4	19	470
Primary Effluent	6/15/2009	290	11	13	520
Secondary Effluent	6/15/2009	No Detection	0.91	No Detection	No Detection
Final Effluent	6/15/2009	No Detection	No Detection	No Detection	14
Raw Influent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	2.8	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Primary Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	200	No Detection	25	380
Primary Effluent	4/9/2010	120	No Detection	2.7	57
Secondary Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Final Effluent	4/9/2010	No Detection	No Detection	No Detection	No Detection
Raw Influent	6/15/2010	No Data	No Data	No Data	No Data
Primary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection	No Detection	No Detection
Final Effluent	6/15/2010	No Data	No Data	No Data	No Data

		5-alpha-androstane-3,17-dione	4-androstene-3,17-dione	Progesterone
	MDL	2.3	0.48	0.66
	MQL	6.9	1.4	2
Raw Influent	5/15/2009	No Detection	12	3.7
Primary Effluent	5/15/2009	No Detection	1.7	5.7
Secondary Effluent	5/15/2009	No Detection	No Detection	0.82
Final Effluent	5/15/2009	24	0.35	No Detection
Raw Influent	6/15/2009	No Detection	88	6.9
Primary Effluent	6/15/2009	No Detection	73	8.7
Secondary Effluent	6/15/2009	No Detection	No Detection	0.82
Final Effluent	6/15/2009	No Detection	2.3	2.9
Raw Influent	8/18/2009	No Detection	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	No Detection	No Detection
Primary Effluent	10/19/2009	No Detection	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection	No Detection
Raw Influent	4/9/2010	No Detection	150	23
Primary Effluent	4/9/2010	No Detection	13	No Detection
Secondary Effluent	4/9/2010	No Detection	1.9	No Detection
Final Effluent	4/9/2010	No Detection	1.1	No Detection
Raw Influent	6/15/2010	No Data	No Data	No Data
Primary Effluent	6/15/2010	No Detection	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection	No Detection
Final Effluent	6/15/2010	No Data	No Data	No Data

		17,20-dihydroxyprogesterone	Boldenone
	MDL	1.4	1.3
	MQL	4.2	4
Raw Influent	5/15/2009	No Detection	13
Primary Effluent	5/15/2009	No Detection	No Detection
Secondary Effluent	5/15/2009	0.82	No Detection
Final Effluent	5/15/2009	No Detection	No Detection
Raw Influent	6/15/2009	3.8	56
Primary Effluent	6/15/2009	2.9	15
Secondary Effluent	6/15/2009	0.82	No Detection
Final Effluent	6/15/2009	No Detection	No Detection
Raw Influent	8/18/2009	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	No Detection
Primary Effluent	10/19/2009	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection
Raw Influent	4/9/2010	No Detection	170
Primary Effluent	4/9/2010	No Detection	16
Secondary Effluent	4/9/2010	No Detection	No Detection
Final Effluent	4/9/2010	No Detection	No Detection
Raw Influent	6/15/2010	No Data	No Data
Primary Effluent	6/15/2010	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection
Final Effluent	6/15/2010	No Data	No Data

		Melengestrol	Melengestrol acetate
	MDL	1.3	0.58
	MQL	4	1.7
Raw Influent	5/15/2009	No Detection	No Detection
Primary Effluent	5/15/2009	No Detection	No Detection
Secondary Effluent	5/15/2009	No Detection	No Detection
Final Effluent	5/15/2009	No Detection	No Detection
Raw Influent	6/15/2009	43	No Detection
Primary Effluent	6/15/2009	49	No Detection
Secondary Effluent	6/15/2009	No Detection	No Detection
Final Effluent	6/15/2009	0.86	0.51
Raw Influent	8/18/2009	No Detection	No Detection
Primary Effluent	8/18/2009	No Detection	No Detection
Secondary Effluent	8/18/2009	No Detection	No Detection
Final Effluent	8/18/2009	No Detection	No Detection
Raw Influent	10/19/2009	No Detection	No Detection
Primary Effluent	10/19/2009	No Detection	No Detection
Secondary Effluent	10/19/2009	No Data	No Data
Final Effluent	10/19/2009	No Detection	No Detection
Raw Influent	4/9/2010	19	1300
Primary Effluent	4/9/2010	No Detection	No Detection
Secondary Effluent	4/9/2010	No Detection	No Detection
Final Effluent	4/9/2010	No Detection	No Detection
Raw Influent	6/15/2010	No Data	No Data
Primary Effluent	6/15/2010	No Detection	No Detection
Secondary Effluent	6/15/2010	No Detection	No Detection
Final Effluent	6/15/2010	No Data	No Data

Appendix B: BIOWIN4 Calculations

Compound	Acetaminophen	Caffeine	Carbamazepine	Codeine	Cotinine	Diltiazem
Number of Applicable Samples	5	5	5	5	4	5
Biowin 4 Value*	3.8748	3.5676	3.5068	3.1895	3.7802	3.4732
Intrinsic Half Life (hr)	13.3	27.1	31.1	64.6	16.6	33.6
Predicted Kbiol ($L\ g_{ss}^{-1}\ hr^{-1}$)	0.05	0.02	0.02	0.01	0.04	0.02
Predicted Removal Efficiency In Aeration Basin (%)	0.59	0.35	0.32	0.17	0.51	0.30
Predicted Removal Efficiency in Secondary Clarifier (%)	0.18	0.09	0.08	0.04	0.15	0.08
Sum Aeration Basin and Secondary Clarifier Removal Efficiency (%)	0.77	0.45	0.40	0.21	0.66	0.37
Observed Median Removal Efficiency (%)	1.00	1.00	0.00	0.00	1.00	0.08

Compound	Diphenhydramine	Fluoxetine	Ibuprofen	Metformin	Naproxen	Ofloxacin
Number of Applicable Samples	4	3	3	5	5	3
Biowin 4 Value*	3.1914	3.2523	3.7986	3.6614	3.9097	2.9163
Intrinsic Half Life (hr)	64.4	55.9	15.9	21.8	12.3	121.3
Predicted Kbiol ($L\ gss^{-1}\ hr^{-1}$)	0.011	0.012	0.044	0.032	0.056	0.006
Predicted Removal Efficiency In Aeration Basin (%)	0.17	0.19	0.52	0.42	0.62	0.09
Predicted Removal Efficiency in Secondary Clarifier (%)	0.04	0.05	0.15	0.11	0.19	0.02
Sum Aeration Basin and Secondary Clarifier Removal Efficiency (%)	0.21	0.24	0.68	0.53	0.81	0.11
Observed Median Removal Efficiency (%)	0.22	0.52	1.00	0.41	0.96	0.00

Compound	Paraxanthine	Ranitidine	Sulfamethoxazole	Triclocarban	Triclosan	Trimethoprim
Number of Applicable Samples	5	3	5	5	5	5
Biowin 4 Value*	3.5878	3.1927	3.3054	2.8964	3.0508	3.3749
Half Life (hr)	25.8	64.2	49.5	126.9	89.0	42.2
Predicted Kbiol ($L\ g_{ss}^{-1}\ hr^{-1}$)	0.027	0.011	0.014	0.005	0.008	0.016
Predicted Removal Efficiency In Aeration Basin (%)	0.37	0.17	0.21	0.09	0.12	0.24
Predicted Removal Efficiency in Secondary Clarifier (%)	0.10	0.04	0.05	0.02	0.03	0.06
Sum Aeration Basin and Secondary Clarifier Removal Efficiency (%)	0.46	0.21	0.26	0.11	0.15	0.30
Observed Median Removal Efficiency (%)	0.99	0.22	0.10	0.64	0.92	0.02

Appendix C: Detection Frequency and Classification of Compounds

Table C1: Detection frequency at five locations with varying proximities to SSWRF, two locations in the Milwaukee Harbor, and the average across all seven sampling sites.

	SS Outfall	1 Mile East	1 Mile South	2 Miles East	2 Miles South	JI Outfall	South Gap	Average
	Detection Frequency	Detection Frequency	Detection Frequency	Detection Frequency	Detection Frequency	Detection Frequency	Detection Frequency	Detection Frequency
Metformin	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Caffeine	83.3%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	97.6%
Sulfamethoxazole	83.3%	66.7%	100.0%	66.7%	66.7%	100.0%	100.0%	83.3%
Triclosan	50.0%	66.7%	50.0%	83.3%	50.0%	100.0%	100.0%	71.4%
Paraxanthine	50.0%	16.7%	50.0%	16.7%	33.3%	83.3%	66.7%	45.2%
Carbadox	66.7%	50.0%	50.0%	16.7%	33.3%	33.3%	33.3%	40.5%
Diphenhydramine	33.3%	33.3%	33.3%	33.3%	33.3%	33.3%	33.3%	33.3%
4-androstene-3,17-dione	33.3%	50.0%	16.7%	50.0%	20.0%	16.7%	33.3%	31.4%
Diltiazem	33.3%	16.7%	33.3%	33.3%	16.7%	33.3%	33.3%	28.6%
Codeine	16.7%	0.0%	33.3%	16.7%	16.7%	50.0%	66.7%	28.6%
Trimethoprim	16.7%	0.0%	33.3%	0.0%	0.0%	66.7%	66.7%	26.2%
Acetaminophen	33.3%	16.7%	0.0%	0.0%	16.7%	50.0%	50.0%	23.8%
Naproxen	33.3%	0.0%	16.7%	0.0%	0.0%	50.0%	66.7%	23.8%
Azithromycin	0.0%	33.3%	16.7%	33.3%	33.3%	16.7%	33.3%	23.8%
Roxithromycin	33.3%	16.7%	16.7%	33.3%	16.7%	16.7%	16.7%	21.4%
Carbamazepine	16.7%	0.0%	0.0%	0.0%	0.0%	66.7%	66.7%	21.4%
Cotinine	16.7%	16.7%	16.7%	33.3%	16.7%	16.7%	16.7%	19.0%
Progesterone	33.3%	16.7%	16.7%	16.7%	20.0%	0.0%	0.0%	14.8%
Testosterone	16.7%	16.7%	16.7%	33.3%	20.0%	0.0%	0.0%	14.8%
Estriol	0.0%	33.3%	0.0%	33.3%	0.0%	0.0%	33.3%	14.3%
Triclocarban	16.7%	0.0%	0.0%	0.0%	0.0%	66.7%	0.0%	11.9%
Fluoxetine	16.7%	0.0%	16.7%	0.0%	0.0%	16.7%	16.7%	9.5%
17-Beta-estradiol	16.7%	0.0%	0.0%	0.0%	0.0%	0.0%	33.3%	7.1%
Sulfadiazine	0.0%	0.0%	16.7%	0.0%	0.0%	33.3%	0.0%	7.1%
Sulfamerazine	0.0%	0.0%	0.0%	0.0%	16.7%	16.7%	16.7%	7.1%
Albuterol	0.0%	0.0%	16.7%	0.0%	0.0%	20.0%	0.0%	5.2%
Sulfanilamide	0.0%	0.0%	0.0%	0.0%	0.0%	33.3%	0.0%	4.8%
Thiabendazole	0.0%	0.0%	0.0%	0.0%	0.0%	33.3%	0.0%	4.8%
Ranitidine	0.0%	0.0%	0.0%	0.0%	0.0%	20.0%	0.0%	2.9%
Estrone	0.0%	0.0%	0.0%	0.0%	20.0%	0.0%	0.0%	2.9%
Ofloxacin	16.7%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	2.4%
Oxacillin	0.0%	0.0%	0.0%	16.7%	0.0%	0.0%	0.0%	2.4%

Table C2: List of compounds assessed and their general classification

	Classification
17,20-dihydroxyprogesterone	Sex Hormone
17-alpha-estradiol	Sex Hormone
17-beta-estradiol	Sex Hormone
4-androstene-3,17-dione	Sex Hormone
5-alpha-androstane-3,17-dione	Anabolic Agent
Acetaminophen	Antipyretic, Analgesic
Albuterol	Antiasthmatic
Azithromycin	Macrolide Antibiotic
Boldenone	Anabolic Steroid
Caffeine	Stimulant
Carbadox	Quinoxaline Antibiotic
Carbamazepine	Anticonvulsant
Cimetidine	Anti-acid reflux
Ciprofloxacin	Quinoline antibiotic
Clarithromycin	Macrolide antibiotic
Codeine	Opiate
Cotinine	Nicotine metabolite
Digoxigenin	Cardanolide Steroid
Diltiazem	Antihypertensive
Diphenhydramine	Antihistamine
Estriol	Sex Hormone
Estrone	Sex Hormone
Fluoxetine	SSRI Antidepressant
Gemfibrozil	Antilipemic
Ibuprofen	Analgesic
Lincomycin	Lincosamide antibiotic
Lomefloxacin	Quinoline antibiotic
Melengestrol	Steroid Hormone
Melengestrol Acetate	Steroid Hormone
Metformin	Anti-diabetic drug
Miconazole	Tetracycline antibiotic
Naproxen	NSAIDs
Norfloxacin	Quinoline antibiotic
Ofloxacin	Quinoline antibiotic

Oxacillin	β-lactam antibiotics
Paraxanthine	Caffeine Metabolite
Progesterone	Sex Hormone
Ranitidine	Anti-acid reflux
Roxithromycin	Macrolide antibiotic
Sarafloxacin	Fluoroquinolone antibiotic
Sulfachloropyridazine	Sulfonamide antibiotic
Sulfadiazine	Sulfonamide antibiotic
Sulfadimethoxine	Sulfonamide antibiotic
Sulfamerazine	Sulfonamide antibiotic
Sulfamethazine	Sulfonamide antibiotic
Sulfamethizole	Sulfonamide antibiotic
Sulfamethoxazole	Sulfonamide antibiotic
Sulfanilamide	Sulfonamide antibiotic
Sulfathiazole	Sulfonamide antibiotic
Testosterone	Sex Hormone
Thiabendazole	Fungicide
Triclocarban	Antimicrobial
Triclosan	Antimicrobial
Trimethoprim	Pyrimidine antibiotic

Appendix D: Supporting information for Chapter 3. All units ng/L.

Note: Concentration of "0" is equal to below the BDL, it does not mean a concentration of zero.

		Metformin	Albuterol	Cimetidine	Ranitidine	Acetaminophen	Azithromycin	Caffeine	Carbadox
		ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L
	MDL	0.5	1.4	1.3	0.9	2.5	3.7	3.1	3.4
	MQL	1.5	4.2	3.8	2.6	7.5	11.0	9.3	10.1
1 Mile East	5/15/2009	249.8	0.0	0.0	0.0	0.0	12.0	5.0	1.9
1 Mile East	6/15/2009	142.2	0.0	0.0	0.0	0.0	11.1	29.8	16.7
1 Mile East	8/18/2009	60.8	0.0	0.0	0.0	0.0	0.0	41.7	0.0
1 Mile East	10/19/2009	818.2	0.0	0.0	3.7	0.0	0.0	10.0	0.0
1 Mile East	4/9/2010	160.9	0.0	0.0	0.0	0.0	0.0	23.8	0.0
1 Mile East	6/15/2010	8.7	0.0	0.0	0.0	0.0	0.0	19.0	0.0
1 Mile South	5/15/2009	836.0	0.0	0.0	0.0	0.0	12.3	85.5	13.4
1 Mile South	6/15/2009	130.4	0.0	0.0	0.0	0.0	0.0	42.5	49.0
1 Mile South	8/18/2009	77.4	0.0	0.0	0.0	0.0	0.0	42.5	0.0
1 Mile South	10/19/2009	87.8	5.9	0.0	0.0	0.0	0.0	10.8	0.0
1 Mile South	4/9/2010	258.5	0.0	0.0	0.0	0.0	0.0	17.8	0.0
1 Mile South	6/15/2010	205.0	0.0	0.0	0.0	0.0	0.0	25.0	6.6
2 Miles East	5/15/2009	133.6	0.0	0.0	0.0	0.0	6.8	18.6	0.0
2 Miles East	6/15/2009	122.8	0.0	0.0	0.0	0.0	7.5	34.9	0.0
2 Miles East	8/18/2009	74.6	0.0	0.0	0.0	0.0	0.0	16.6	0.0
2 Miles East	10/19/2009	92.6	0.0	0.0	0.0	0.0	0.0	12.0	0.0
2 Miles East	4/9/2010	126.0	0.0	0.0	0.0	0.0	0.0	18.9	6.0
2 Miles East	6/15/2010	160.0	0.0	0.0	0.0	0.0	0.0	23.0	0.0
2 Miles South	5/15/2009	114.0	0.0	0.0	0.0	0.0	11.4	39.3	6.8
2 Miles South	6/15/2009	155.4	0.0	0.0	0.0	2.5	5.7	22.7	33.5
2 Miles South	8/18/2009	63.2	0.0	0.0	0.0	0.0	0.0	24.0	0.0
2 Miles South	10/19/2009	105.2	0.0	0.0	0.0	0.0	0.0	9.2	0.0
2 Miles South	4/9/2010	No Data	No Data	No Data	No Data	0.0	0.0	16.7	0.0

		Metformin	Albuterol	Cimetidine	Ranitidine	Acetaminophen	Azithromycin	Caffeine	Carbadox
2 Miles South	6/15/2010	No Data	No Data	No Data	No Data	0.0	0.0	29.1	0.0
JI Outfall	5/15/2009	4285.3	0.0	0.0	0.0	72.8	0.0	226.6	0.0
JI Outfall	6/15/2009	3709.3	0.0	0.0	0.0	15.8	21.7	80.2	22.4
JI Outfall	8/18/2009	3168.0	0.0	0.0	0.0	0.0	0.0	10.6	0.0
JI Outfall	10/19/2009	9240.0	4.3	0.0	26.9	0.0	0.0	13.5	0.0
JI Outfall	4/9/2010	No Data	No Data	No Data	No Data	13.0	0.0	32.1	0.0
JI Outfall	6/15/2010	94.3	0.0	0.0	0.0	0.0	0.0	41.7	14.0
Southgap	5/15/2009	2434.0	0.0	0.0	0.0	44.8	5.7	189.4	5.7
Southgap	6/15/2009	362.0	0.0	0.0	0.0	19.1	12.3	118.1	0.0
Southgap	8/18/2009	1192.0	0.0	0.0	0.0	0.0	0.0	15.5	0.0
Southgap	10/19/2009	688.0	0.0	0.0	0.0	0.0	0.0	19.2	0.0
Southgap	4/9/2010	776.8	0.0	0.0	0.0	14.1	0.0	46.5	0.0
Southgap	6/15/2010	1954.0	0.0	0.0	0.0	0.0	0.0	40.0	19.4
SS Outfall	5/15/2009	2108.0	0.0	0.0	0.0	3.6	0.0	108.3	7.0
SS Outfall	6/15/2009	331.6	0.0	0.0	0.0	0.0	0.0	54.2	20.1
SS Outfall	8/18/2009	3752.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	264.0	0.0	0.0	0.0	0.0	0.0	32.0	0.0
SS Outfall	4/9/2010	901.8	0.0	0.0	0.0	21.3	0.0	40.2	5.5
SS Outfall	6/15/2010	12.6	0.0	0.0	0.0	0.0	0.0	26.8	10.6

		Carbamazepine	Ciprofloxacin	Clarithromycin	Codeine	Cotinine	Digoxigenin	Diltiazem	Paraxanthine	Diphenhydramine
		ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L
	MDL	2.7	3.3	3.2	3.6	3.5	4.4	3.5	6.1	3.6
	MQL	8.2	9.9	9.6	10.7	10.6	13.2	10.4	18.2	10.9
1 Mile East	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	4.4	0.0	4.7
1 Mile East	6/15/2009	0.0	0.0	0.0	8.7	6.5	0.0	5.5	0.0	6.6
1 Mile East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	9.7	0.0
1 Mile East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.4	0.0
1 Mile South	5/15/2009	0.0	0.0	0.0	5.5	5.0	0.0	5.9	39.4	6.6
1 Mile South	6/15/2009	0.0	0.0	0.0	9.2	0.0	0.0	7.8	0.0	9.2
1 Mile South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	22.8	0.0
1 Mile South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	3.8	0.0
2 Miles East	5/15/2009	0.0	0.0	0.0	5.4	6.1	0.0	2.0	0.0	4.4
2 Miles East	6/15/2009	0.0	0.0	0.0	0.0	6.1	0.0	2.3	0.0	4.9
2 Miles East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	3.9	0.0
2 Miles East	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	5/15/2009	0.0	0.0	0.0	0.0	11.3	0.0	1.8	0.0	4.1
2 Miles South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	6.7
2 Miles South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0	0.0
2 Miles South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		Carbamazepine	Ciprofloxacin	Clarithromycin	Codeine	Cotinine	Digoxigenin	Diltiazem	Paraxanthine	Diphenhydramine
2 Miles South	6/15/2010	0.0	0.0	0.0	7.2	0.0	0.0	0.0	8.7	0.0
JI Outfall	5/15/2009	24.2	0.0	0.0	10.9	0.0	0.0	16.3	57.0	19.6
JI Outfall	6/15/2009	38.2	0.0	0.0	0.0	20.4	0.0	21.3	14.6	43.2
JI Outfall	8/18/2009	14.8	0.0	0.0	0.0	0.0	0.0	0.0	5.5	0.0
JI Outfall	10/19/2009	12.2	0.0	0.0	0.0	0.0	0.0	0.0	7.4	0.0
JI Outfall	4/9/2010	0.0	0.0	0.0	9.0	0.0	0.0	0.0	8.2	0.0
JI Outfall	6/15/2010	0.0	0.0	0.0	6.2	0.0	0.0	0.0	0.0	0.0
Southgap	5/15/2009	10.2	0.0	0.0	15.0	0.0	0.0	10.0	26.4	9.6
Southgap	6/15/2009	7.2	0.0	0.0	4.2	20.9	0.0	8.1	44.5	12.1
Southgap	8/18/2009	17.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	5.4	0.0
Southgap	4/9/2010	0.0	0.0	0.0	7.5	0.0	0.0	0.0	11.9	0.0
Southgap	6/15/2010	4.2	0.0	0.0	5.4	0.0	0.0	0.0	0.0	0.0
SS Outfall	5/15/2009	6.2	0.0	0.0	11.2	0.0	0.0	7.2	23.3	10.8
SS Outfall	6/15/2009	0.0	0.0	0.0	0.0	7.4	0.0	7.9	0.0	13.7
SS Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	6.6	0.0
SS Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.5	0.0
SS Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		Fluoxetine	Lincomycin	Lomefloxacin	Miconazole	Norfloxacin	Ofloxacin	Oxacillin	Roxithromycin	Sarafloxacin	Sulfachloropyridazine	Sulfadiazine
		ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L
	MDL	3.5	3.1	4.7	2.7	5.1	3.9	2.5	4.3	5.4	4.1	2.8
	MQL	10.5	9.3	14.2	8.1	15.3	11.7	7.4	13.0	16.3	12.3	8.5
1 Mile East	5/15/2009	2.1	0.0	0.0	0.0	0.0	0.0	0.0	4.1	0.0	0.0	0.0
1 Mile East	6/15/2009	0.0	0.0	0.0	0.0	0.0	20.5	0.0	0.0	0.0	0.0	0.0
1 Mile East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.5	0.0	0.0	0.0
1 Mile South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	6/15/2010	1.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.6
2 Miles East	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	17.2	15.1	0.0	0.0	0.0
2 Miles East	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	11.7	0.0	0.0	0.0
2 Miles East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	38.9	0.0	0.0	0.0
2 Miles South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		Fluoxetine	Lincomycin	Lomefloxacin	Miconazole	Norfloxacin	Ofloxacin	Oxacillin	Roxithromycin	Sarafloxacin	Sulfachloropyridazine	Sulfadiazine
2 Miles South	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2009	49.3	0.0	0.0	0.0	0.0	0.0	0.0	9.2	0.0	0.0	0.0
JI Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.5
JI Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	3.8
Southgap	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	6/15/2009	61.6	0.0	0.0	0.0	0.0	0.0	0.0	2.3	0.0	0.0	0.0
Southgap	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	5/15/2009	1.8	0.0	0.0	0.0	0.0	0.0	0.0	8.7	0.0	0.0	0.0
SS Outfall	6/15/2009	0.0	0.0	0.0	0.0	0.0	60.7	0.0	3.1	0.0	0.0	0.0
SS Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		Fluoxetine	Lincomycin	Lomefloxacin	Miconazole	Norfloxacin	Ofloxacin	Oxacillin	Roxithromycin	Sarafloxacin	Sulfachloropyridazine	Sulfadiazine
2 Miles South	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2009	49.3	0.0	0.0	0.0	0.0	0.0	0.0	9.2	0.0	0.0	0.0
JI Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.5
JI Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	3.8
Southgap	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	6/15/2009	61.6	0.0	0.0	0.0	0.0	0.0	0.0	2.3	0.0	0.0	0.0
Southgap	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	5/15/2009	1.8	0.0	0.0	0.0	0.0	0.0	0.0	8.7	0.0	0.0	0.0
SS Outfall	6/15/2009	0.0	0.0	0.0	0.0	0.0	60.7	0.0	3.1	0.0	0.0	0.0
SS Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		Sulfadimethoxine	Sulfamerazine	Sulfamethazine	Sulfamethizole	Sulfamethoxazole	Sulfanilamide	Sulfathiazole	Thiabendazole	Trimethoprim	Triclosan
		ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L
	MDL	2.4	2.1	4.0	4.2	4.1	2.9	2.6	1.8	3.4	0.5
	MQL	7.1	6.2	12.1	12.7	12.4	8.6	7.8	5.3	0.0	1.6
1 Mile East	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.4
1 Mile East	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	8/18/2009	0.0	1.5	0.0	0.0	4.8	0.0	0.0	0.0	0.0	0.0
1 Mile East	10/19/2009	0.0	0.0	0.0	0.0	6.2	0.0	0.0	0.0	0.0	0.5
1 Mile East	4/9/2010	0.0	0.0	0.0	0.0	5.3	0.0	0.0	0.0	0.0	2.1
1 Mile East	6/15/2010	0.0	0.0	0.0	0.0	6.2	0.0	0.0	0.0	0.0	1.9
1 Mile South	5/15/2009	0.0	0.0	0.0	0.0	2.3	0.0	0.0	0.0	0.0	16.1
1 Mile South	6/15/2009	0.0	0.0	0.0	0.0	3.6	0.0	0.0	0.0	6.0	0.0
1 Mile South	8/18/2009	0.0	0.0	0.0	0.0	4.5	0.0	0.0	0.0	0.0	0.0
1 Mile South	10/19/2009	0.0	0.0	0.0	0.0	6.3	0.0	0.0	0.0	0.0	0.0
1 Mile South	4/9/2010	0.0	0.0	0.0	0.0	6.6	0.0	0.0	0.0	0.0	1.3
1 Mile South	6/15/2010	0.0	0.0	0.0	0.0	7.0	0.0	0.0	0.0	2.8	0.6
2 Miles East	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.4
2 Miles East	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.6
2 Miles East	8/18/2009	0.0	0.0	0.0	0.0	5.2	0.0	0.0	0.0	0.0	4.3
2 Miles East	10/19/2009	0.0	0.0	0.0	0.0	6.4	0.0	0.0	0.0	0.0	0.0
2 Miles East	4/9/2010	0.0	0.0	0.0	0.0	5.3	0.0	0.0	0.0	0.0	1.2
2 Miles East	6/15/2010	0.0	0.0	0.0	0.0	7.3	0.0	0.0	0.0	0.0	1.7
2 Miles South	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	6.5
2 Miles South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	8/18/2009	0.0	1.8	0.0	0.0	2.5	0.0	0.0	0.0	0.0	0.0
2 Miles South	10/19/2009	0.0	0.0	0.0	0.0	6.7	0.0	0.0	0.0	0.0	0.0
2 Miles South	4/9/2010	0.0	0.0	0.0	0.0	7.7	0.0	0.0	0.0	0.0	1.2

		Sulfadimethoxine	Sulfamerazine	Sulfamethazine	Sulfamethizole	Sulfamethoxazole	Sulfanilamide	Sulfathiazole	Thiabendazole	Trimethoprim	Triclosan
2 Miles South	6/15/2010	0.0	0.0	0.0	0.0	10.0	0.0	0.0	0.0	0.0	0.9
JI Outfall	5/15/2009	0.0	1.3	0.0	0.0	21.5	0.0	0.0	1.3	34.8	24.1
JI Outfall	6/15/2009	0.0	0.0	0.0	0.0	77.1	0.0	0.0	0.9	52.4	11.6
JI Outfall	8/18/2009	0.0	0.0	0.0	0.0	33.6	20.3	0.0	0.0	0.0	5.5
JI Outfall	10/19/2009	0.0	0.0	0.0	0.0	21.5	17.7	0.0	0.0	0.0	0.7
JI Outfall	4/9/2010	0.0	0.0	0.0	0.0	7.7	0.0	0.0	0.0	5.4	2.2
JI Outfall	6/15/2010	0.0	0.0	0.0	0.0	14.5	0.0	0.0	0.0	7.3	2.3
Southgap	5/15/2009	0.0	0.0	0.0	0.0	15.8	0.0	0.0	0.0	12.5	5.0
Southgap	6/15/2009	0.0	0.0	0.0	0.0	30.3	0.0	0.0	0.0	13.4	11.1
Southgap	8/18/2009	0.0	3.6	0.0	0.0	11.9	0.0	0.0	0.0	0.0	5.8
Southgap	10/19/2009	0.0	0.0	0.0	0.0	13.2	0.0	0.0	0.0	0.0	1.4
Southgap	4/9/2010	0.0	0.0	0.0	0.0	8.0	0.0	0.0	0.0	6.5	3.3
Southgap	6/15/2010	0.0	0.0	0.0	0.0	13.8	0.0	0.0	0.0	9.1	3.3
SS Outfall	5/15/2009	0.0	0.0	0.0	0.0	5.5	0.0	0.0	0.0	3.4	41.5
SS Outfall	6/15/2009	0.0	0.0	0.0	0.0	13.6	0.0	0.0	0.0	0.0	13.8
SS Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	0.0	0.0	0.0	0.0	9.9	0.0	0.0	0.0	0.0	0.0
SS Outfall	4/9/2010	0.0	0.0	0.0	0.0	7.0	0.0	0.0	0.0	0.0	4.2
SS Outfall	6/15/2010	0.0	0.0	0.0	0.0	5.4	0.0	0.0	0.0	0.0	0.0

		Triclocarban	Naproxen	Gemfibrozil	Ibuprofen	Estriol	Estrone	17-Beta-estradiol	Testosterone
		ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L
	MDL	0.5	1.0	1.6	4.7	2.0	2.2	1.3	1.1
	MQL	1.4	2.9	4.8	14.0	6.1	6.7	3.8	3.2
1 Mile East	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.5
1 Mile East	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	8/18/2009	0.0	0.9	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	6/15/2010	0.0	0.0	0.0	0.0	3.9	0.0	0.0	0.0
1 Mile South	5/15/2009	0.0	15.3	0.0	0.0	0.0	0.0	0.0	9.1
1 Mile South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	38.0
2 Miles East	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6
2 Miles East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	4/9/2010	0.0	0.0	0.0	0.0	3.3	0.0	0.0	0.0
2 Miles East	6/15/2010	0.0	0.0	0.0	0.0	5.0	0.0	0.0	0.0
2 Miles South	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.0
2 Miles South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	4/9/2010	0.0	0.0	0.0	0.0	0.0	3.4	0.0	0.0

		Triclocarban	Naproxen	Gemfibrozil	Ibuprofen	Estriol	Estrone	17-Beta-estradiol	Testosterone
2 Miles South	6/15/2010	0.0	0.0	No Data	No Data	No Data	No Data	No Data	No Data
JI Outfall	5/15/2009	6.3	30.8	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2009	9.9	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	8/18/2009	4.3	13.8	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	10/19/2009	2.8	5.9	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	5/15/2009	0.0	18.1	0.0	0.0	0.0	0.0	1.3	0.0
Southgap	6/15/2009	0.0	5.0	0.0	0.0	0.0	0.0	0.8	0.0
Southgap	8/18/2009	0.0	3.6	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	10/19/2009	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	4/9/2010	0.0	0.0	0.0	0.0	4.9	0.0	0.0	0.0
Southgap	6/15/2010	0.0	0.0	0.0	0.0	3.6	0.0	0.0	0.0
SS Outfall	5/15/2009	15.7	18.8	0.0	0.0	0.0	0.0	1.7	13.2
SS Outfall	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	4/9/2010	0.0	10.6	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		5-alpha-androstane-3,17-dione	4-androstene-3,17-dione	Progesterone	17,20-dihydroxyprogesterone	Boldenone	Melengestrol	Melengestrol acetate	17-alpha-estradiol
		ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L
	MDL	2.3	0.5	0.7	1.4	1.3	1.3	0.6	1.2
	MQL	6.9	1.4	2.0	4.2	4.0	4.0	1.7	3.5
1 Mile East	5/15/2009	0.0	2.0	4.9	0.0	0.0	0.0	0.0	0.0
1 Mile East	6/15/2009	0.0	0.0	2.1	0.0	0.0	0.0	0.0	0.0
1 Mile East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile East	6/15/2010	0.0	0.8	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	5/15/2009	0.0	5.3	8.7	0.0	0.0	0.0	0.0	0.0
1 Mile South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1 Mile South	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	5/15/2009	0.0	16.6	88.2	0.0	0.0	0.0	0.0	0.0
2 Miles East	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	4/9/2010	0.0	1.2	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles East	6/15/2010	0.0	0.8	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	5/15/2009	0.0	3.5	13.1	0.0	0.0	0.0	0.0	0.0
2 Miles South	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2 Miles South	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

		5-alpha-androstane-3,17-dione	4-androstene-3,17-dione	Progesterone	17,20-dihydroxyprogesterone	Boldenone	Melengestrol	Melengestrol acetate	17-alpha-estradiol
2 Miles South	6/15/2010	No Data	No Data	No Data	No Data	No Data	No Data	No Data	No Data
JI Outfall	5/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	4/9/2010	0.0	1.4	0.0	0.0	0.0	0.0	0.0	0.0
JI Outfall	6/15/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	5/15/2009	0.0	0.9	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	6/15/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Southgap	6/15/2010	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	5/15/2009	0.0	7.3	11.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	6/15/2009	0.0	0.0	1.3	0.0	0.0	0.0	0.0	0.0
SS Outfall	8/18/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	10/19/2009	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	4/9/2010	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SS Outfall	6/15/2010	0.0	577.6	0.0	0.0	0.0	0.0	0.0	0.0

Appendix E: Supporting Data for Chapter 4

Type	Compound	Concentration or Removal	Cite
Influent Concentration (ng/L)	Acetaminophen	61000.00	Benotti et al., 2007
Influent Concentration (ng/L)	Acetaminophen	26000.00	Blair et al., 2013
Influent Concentration (ng/L)	Acetaminophen	150000.00	Blair et al., 2013
Influent Concentration (ng/L)	Acetaminophen	12000.00	Blair et al., 2013
Influent Concentration (ng/L)	Acetaminophen	14000.00	Blair et al., 2013
Influent Concentration (ng/L)	Acetaminophen	5900.00	Blair et al., 2013
Influent Concentration (ng/L)	Acetaminophen	22000.00	Blair et al., 2013
Influent Concentration (ng/L)	Acetaminophen	56944.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	34021.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	18729.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	22325.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	48097.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	25461.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	22706.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	13046.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	18286.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	28756.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	23407.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	13284.00	Choi et al., 2008
Influent Concentration (ng/L)	Acetaminophen	29000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Acetaminophen	246000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Acetaminophen	134000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Acetaminophen	68107.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Acetaminophen	482687.00	Kasprzyk-Hordern et al., 2009

Influent Concentration (ng/L)	Acetaminophen	10400.00	Khan and Ongerth, 2005
Influent Concentration (ng/L)	Acetaminophen	710.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Acetaminophen	1140.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Acetaminophen	5529.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Acetaminophen	6924.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Acetaminophen	69570.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Acetaminophen	23202.00	Rosal et al., 2010
Influent Concentration (ng/L)	Acetaminophen	37458.00	Rosal et al., 2010
Influent Concentration (ng/L)	Acetaminophen	1571.00	Rosal et al., 2010
Influent Concentration (ng/L)	Acetaminophen	17200.00	Snyder et al., 2006
Influent Concentration (ng/L)	Acetaminophen	960.00	Yu et al., 2006
MBR Removal	Acetaminophen	1.00	Kim et al., 2007
MBR Removal	Acetaminophen	1.00	Kim et al., 2007
MBR Removal	Acetaminophen	1.00	Radjenovic et al., 2007
MBR Removal	Acetaminophen	1.00	Radjenovic et al., 2009
MBR Removal	Acetaminophen	0.99	Radjenovic et al., 2009
MBR Removal	Acetaminophen	1.00	Snyder et al., 2007
Microfiltration Removal	Acetaminophen	0.04	Snyder et al., 2007
Nanofiltration Removal	Acetaminophen	0.13	Chon et al., 2012
Nanofiltration Removal	Acetaminophen	0.16	Chon et al., 2012
Nanofiltration Removal	Acetaminophen	0.31	Chon et al., 2012
Nanofiltration Removal	Acetaminophen	1.00	Kim et al., 2007
Primary + CAS Removal	Acetaminophen	1.00	Blair et al., 2013
Primary + CAS Removal	Acetaminophen	1.00	Blair et al., 2013
Primary + CAS Removal	Acetaminophen	1.00	Blair et al., 2013
Primary + CAS Removal	Acetaminophen	1.00	Blair et al., 2013
Primary + CAS Removal	Acetaminophen	0.00	Blair et al., 2013
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008

Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Choi et al., 2008
Primary + CAS Removal	Acetaminophen	1.00	Gómez et al., 2007
Primary + CAS Removal	Acetaminophen	0.87	Jones et al., 2007
Primary + CAS Removal	Acetaminophen	0.94	Jones et al., 2007
Primary + CAS Removal	Acetaminophen	0.95	Jones et al., 2007
Primary + CAS Removal	Acetaminophen	0.94	Jones et al., 2007
Primary + CAS Removal	Acetaminophen	0.94	Kasprzyk-Hordern et al., 2009
Primary + CAS Removal	Acetaminophen	1.00	Khan and Ongerth, 2005
Primary + CAS Removal	Acetaminophen	0.98	Radjenovic et al., 2007
Primary + CAS Removal	Acetaminophen	1.00	Radjenovic et al., 2009
Primary + CAS Removal	Acetaminophen	1.00	Roberts and Thomas, 2006
Primary + CAS Removal	Acetaminophen	1.00	Rosal et al., 2010
Primary + CAS Removal	Acetaminophen	1.00	Yu et al., 2006
Ultrafiltration Removal	Acetaminophen	0.06	Snyder et al., 2007
Influent Concentration (ng/L)	Caffeine	42000.00	Benotti et al., 2007
Influent Concentration (ng/L)	Caffeine	34000.00	Blair et al., 2013
Influent Concentration (ng/L)	Caffeine	130000.00	Blair et al., 2013
Influent Concentration (ng/L)	Caffeine	9400.00	Blair et al., 2013
Influent Concentration (ng/L)	Caffeine	9000.00	Blair et al., 2013
Influent Concentration (ng/L)	Caffeine	3300.00	Blair et al., 2013
Influent Concentration (ng/L)	Caffeine	8800.00	Blair et al., 2013
Influent Concentration (ng/L)	Caffeine	36856.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	30615.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	18405.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	9750.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	33821.00	Choi et al., 2008

Influent Concentration (ng/L)	Caffeine	21070.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	18706.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	14313.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	24236.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	20750.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	29491.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	25758.00	Choi et al., 2008
Influent Concentration (ng/L)	Caffeine	118000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Caffeine	52000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Caffeine	192000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Caffeine	65625.00	Rosal et al., 2010
Influent Concentration (ng/L)	Caffeine	5010.00	Rosal et al., 2010
Influent Concentration (ng/L)	Caffeine	22849.00	Rosal et al., 2010
Influent Concentration (ng/L)	Caffeine	2170.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	2510.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	680.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	750.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	220.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	6100.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	6000.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	11440.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	9260.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	3840.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	2870.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	2340.00	Santos et al., 2007
Influent Concentration (ng/L)	Caffeine	7370.00	Santos et al., 2009

Influent Concentration (ng/L)	Caffeine	2330.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	27900.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	4870.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	540.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	2610.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	7090.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	750.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	43900.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	5340.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	220.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	22000.00	Santos et al., 2009
Influent Concentration (ng/L)	Caffeine	2448.00	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Caffeine	4865.00	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Caffeine	2769.00	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Caffeine	628.00	Yu and Chu, 2009
MBR Removal	Caffeine	0.99	Kim et al., 2007
MBR Removal	Caffeine	0.99	Kim et al., 2007
MBR Removal	Caffeine	1.00	Snyder et al., 2007
MBR Removal	Caffeine	1.00	Snyder et al., 2007
Microfiltration Removal	Caffeine	0.00	Snyder et al., 2007
Nanofiltration Removal	Caffeine	1.00	Kim et al., 2007
Primary + CAS Removal	Caffeine	0.64	Benotti et al., 2007
Primary + CAS Removal	Caffeine	0.97	Blair et al., 2013
Primary + CAS Removal	Caffeine	0.99	Blair et al., 2013
Primary + CAS Removal	Caffeine	1.00	Blair et al., 2013
Primary + CAS Removal	Caffeine	0.98	Blair et al., 2013
Primary + CAS Removal	Caffeine	0.11	Blair et al., 2013
Primary + CAS Removal	Caffeine	0.98	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.97	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.99	Choi et al., 2008
Primary + CAS Removal	Caffeine	1.00	Choi et al., 2008

Primary + CAS Removal	Caffeine	0.98	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.99	Choi et al., 2008
Primary + CAS Removal	Caffeine	1.00	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.97	Choi et al., 2008
Primary + CAS Removal	Caffeine	1.00	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.99	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.99	Choi et al., 2008
Primary + CAS Removal	Caffeine	1.00	Choi et al., 2008
Primary + CAS Removal	Caffeine	0.90	Gómez et al., 2007
Primary + CAS Removal	Caffeine	0.43	Santos et al., 2007
Primary + CAS Removal	Caffeine	0.85	Santos et al., 2007
Primary + CAS Removal	Caffeine	0.73	Santos et al., 2007
Primary + CAS Removal	Caffeine	0.50	Santos et al., 2007
Primary + CAS Removal	Caffeine	0.78	Santos et al., 2009
Primary + CAS Removal	Caffeine	0.50	Santos et al., 2009
Primary + CAS Removal	Caffeine	0.76	Santos et al., 2009
Primary + CAS Removal	Caffeine	0.57	Santos et al., 2009
Primary + CAS Removal	Caffeine	1.00	Spongberg and Witter, 2008
Primary + CAS Removal	Caffeine	1.00	Spongberg and Witter, 2008
Primary + CAS Removal	Caffeine	0.99	Spongberg and Witter, 2008
Primary + CAS Removal	Caffeine	0.86	Yu and Chu, 2009
Ultrafiltration Removal	Caffeine	0.07	Snyder et al., 2007
Influent Concentration (ng/L)	Carbamazepine	1680.00	Bendz et al., 2005
Influent Concentration (ng/L)	Carbamazepine	100.00	Benotti et al., 2007
Influent Concentration (ng/L)	Carbamazepine	230.00	Blair et al., 2013
Influent Concentration (ng/L)	Carbamazepine	310.00	Blair et al., 2013
Influent Concentration (ng/L)	Carbamazepine	73.00	Blair et al., 2013
Influent Concentration (ng/L)	Carbamazepine	51.00	Blair et al., 2013
Influent Concentration (ng/L)	Carbamazepine	21.00	Blair et al., 2013
Influent Concentration (ng/L)	Carbamazepine	72.00	Blair et al., 2013
Influent Concentration (ng/L)	Carbamazepine	13.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	201.00	Choi et al., 2008

Influent Concentration (ng/L)	Carbamazepine	203.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	0.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	451.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	242.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	0.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	156.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	223.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	6.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	283.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	29.00	Choi et al., 2008
Influent Concentration (ng/L)	Carbamazepine	325.00	Clara et al., 2005a
Influent Concentration (ng/L)	Carbamazepine	670.00	Clara et al., 2005a
Influent Concentration (ng/L)	Carbamazepine	704.00	Clara et al., 2005a
Influent Concentration (ng/L)	Carbamazepine	1200.00	Clara et al., 2005a
Influent Concentration (ng/L)	Carbamazepine	1850.00	Clara et al., 2005a
Influent Concentration (ng/L)	Carbamazepine	120.00	Gómez et al., 2007
Influent Concentration (ng/L)	Carbamazepine	150.00	Gómez et al., 2007
Influent Concentration (ng/L)	Carbamazepine	310.00	Gómez et al., 2007
Influent Concentration (ng/L)	Carbamazepine	104.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Carbamazepine	3110.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Carbamazepine	500.00	Khan and Ongerth, 2005
Influent Concentration (ng/L)	Carbamazepine	32.00	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	24.00	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	119.00	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	16.50	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	270.00	Nakada et al., 2006

Influent Concentration (ng/L)	Carbamazepine	116.00	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	51.40	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	52.10	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	248.00	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	60.50	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	80.40	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	100.00	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	56.20	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	14.90	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	16.70	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	48.50	Nakada et al., 2006
Influent Concentration (ng/L)	Carbamazepine	54.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Carbamazepine	220.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Carbamazepine	156.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Carbamazepine	106.00	Rosal et al., 2010
Influent Concentration (ng/L)	Carbamazepine	173.00	Rosal et al., 2010
Influent Concentration (ng/L)	Carbamazepine	129.00	Rosal et al., 2010
Influent Concentration (ng/L)	Carbamazepine	120.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	0.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	0.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	0.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	940.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	1380.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	2150.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	280.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	300.00	Santos et al., 2007

Influent Concentration (ng/L)	Carbamazepine	290.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	360.00	Santos et al., 2007
Influent Concentration (ng/L)	Carbamazepine	0.00	Santos et al., 2009
Influent Concentration (ng/L)	Carbamazepine	3780.00	Santos et al., 2009
Influent Concentration (ng/L)	Carbamazepine	530.00	Santos et al., 2009
Influent Concentration (ng/L)	Carbamazepine	39.30	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Carbamazepine	50.90	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Carbamazepine	24.80	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Carbamazepine	660.00	Wick et al, 2009
Influent Concentration (ng/L)	Carbamazepine	1662.00	Zhou et al., 2009
Influent Concentration (ng/L)	Carbamazepine	1237.00	Zhou et al., 2009
Influent Concentration (ng/L)	Carbamazepine	1833.00	Zhou et al., 2009
MBR Removal	Carbamazepine	0.12	Clara et al, 2005b
MBR Removal	Carbamazepine	0.04	Clara et al, 2005b
MBR Removal	Carbamazepine	0.00	Clara et al, 2005b
MBR Removal	Carbamazepine	0.05	Clara et al., 2005a
MBR Removal	Carbamazepine	0.00	Kim et al., 2007
MBR Removal	Carbamazepine	0.00	Kim et al., 2007
MBR Removal	Carbamazepine	0.00	Snyder et al., 2007
MBR Removal	Carbamazepine	0.95	Snyder et al., 2007
Microfiltration Removal	Carbamazepine	0.11	Snyder et al., 2007
Nanofiltration Removal	Carbamazepine	0.41	Chon et al., 2012
Nanofiltration Removal	Carbamazepine	0.72	Chon et al., 2012
Nanofiltration Removal	Carbamazepine	0.82	Chon et al., 2012
Nanofiltration Removal	Carbamazepine	0.98	Kim et al., 2007
Primary + CAS Removal	Carbamazepine	0.30	Bendz et al., 2005
Primary + CAS Removal	Carbamazepine	0.37	Benotti et al., 2007
Primary + CAS Removal	Carbamazepine	0.26	Blair et al., 2013
Primary + CAS Removal	Carbamazepine	0.45	Blair et al., 2013
Primary + CAS Removal	Carbamazepine	0.00	Blair et al., 2013
Primary + CAS Removal	Carbamazepine	0.00	Blair et al., 2013
Primary + CAS Removal	Carbamazepine	0.18	Blair et al., 2013

Primary + CAS Removal	Carbamazepine	0.54	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.43	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.47	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.65	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.57	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.01	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.46	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.00	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.50	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.00	Choi et al., 2008
Primary + CAS Removal	Carbamazepine	0.00	Clara et al, 2005b
Primary + CAS Removal	Carbamazepine	0.00	Clara et al, 2005b
Primary + CAS Removal	Carbamazepine	0.00	Clara et al, 2005b
Primary + CAS Removal	Carbamazepine	0.14	Clara et al, 2005b
Primary + CAS Removal	Carbamazepine	0.00	Clara et al, 2005b
Primary + CAS Removal	Carbamazepine	0.00	Clara et al, 2005b
Primary + CAS Removal	Carbamazepine	0.00	Clara et al., 2005a
Primary + CAS Removal	Carbamazepine	0.00	Clara et al., 2005a
Primary + CAS Removal	Carbamazepine	0.00	Clara et al., 2005a
Primary + CAS Removal	Carbamazepine	0.13	Gómez et al., 2007
Primary + CAS Removal	Carbamazepine	0.13	Kasprzyk-Hordern et al., 2009
Primary + CAS Removal	Carbamazepine	0.57	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.03	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.78	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.47	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.40	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.00	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.24	Nakada et al., 2006
Primary + CAS Removal	Carbamazepine	0.40	Nakada et al., 2006

Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2007
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2007
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2007
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2007
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2009
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2009
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2009
Primary + CAS Removal	Carbamazepine	0.00	Santos et al., 2009
Primary + CAS Removal	Carbamazepine	0.00	Spongberg and Witter, 2008
Primary + CAS Removal	Carbamazepine	0.00	Spongberg and Witter, 2008
Primary + CAS Removal	Carbamazepine	0.00	Spongberg and Witter, 2008
Primary + CAS Removal	Carbamazepine	0.07	Suarez et al., 2005
Primary + CAS Removal	Carbamazepine	0.00	Vieno et al., 2007
Primary + CAS Removal	Carbamazepine	0.43	Zhou et al., 2009
Primary + CAS Removal	Carbamazepine	0.49	Zhou et al., 2009
Primary + CAS Removal	Carbamazepine	0.54	Zhou et al., 2009
Ultrafiltration Removal	Carbamazepine	0.16	Snyder et al., 2007
Ultrafiltration Removal	Carbamazepine	0.13	Snyder et al., 2006
Influent Concentration (ng/L)	Estradiol (17B - E2)	15.80	Anderson et al., 2003
Influent Concentration (ng/L)	Estradiol (17B - E2)	54.00	Clara et al, 2005b
Influent Concentration (ng/L)	Estradiol (17B - E2)	82.00	Clara et al, 2005b
Influent Concentration (ng/L)	Estradiol (17B - E2)	125.00	Clara et al, 2005b
Influent Concentration (ng/L)	Estradiol (17B - E2)	24.50	Clara et al., 2005b
Influent Concentration (ng/L)	Estradiol (17B - E2)	67.00	Clara et al., 2005b
Influent Concentration (ng/L)	Estradiol (17B - E2)	46.00	Clara et al., 2005b
Influent Concentration (ng/L)	Estradiol (17B - E2)	4.90	Joss et al., 2004
Influent Concentration (ng/L)	Estradiol (17B - E2)	7.60	Joss et al., 2004
Influent Concentration (ng/L)	Estradiol (17B - E2)	11.00	Joss et al., 2004
Influent Concentration (ng/L)	Estradiol (17B - E2)	14.10	Nakada et al., 2006
Influent Concentration (ng/L)	Estradiol (17B - E2)	13.30	Nakada et al., 2006
Influent Concentration	Estradiol (17B -	20.60	Nakada et al., 2006

(ng/L)	E2)		
Influent Concentration (ng/L)	Estradiol (17B - E2)	25.80	Nakada et al., 2006
Influent Concentration (ng/L)	Estradiol (17B - E2)	22.90	Nakada et al., 2006
Influent Concentration (ng/L)	Estradiol (17B - E2)	20.00	Thomas et al., 2007
Influent Concentration (ng/L)	Estradiol (17B - E2)	44.00	Thomas et al., 2007
Influent Concentration (ng/L)	Estradiol (17B - E2)	3.00	Thomas et al., 2007
MBR Removal	Estradiol (17B - E2)	1.00	Clara et al, 2005b
MBR Removal	Estradiol (17B - E2)	0.96	Clara et al, 2005b
MBR Removal	Estradiol (17B - E2)	0.95	Clara et al, 2005b
MBR Removal	Estradiol (17B - E2)	0.92	Joss et al., 2004
Microfiltration Removal	Estradiol (17B - E2)	0.00	Snyder et al., 2007
Nanofiltration Removal	Estradiol (17B - E2)	0.52	Yoon et al., 2006
Primary + CAS Removal	Estradiol (17B - E2)	0.94	Anderson et al., 2003
Primary + CAS Removal	Estradiol (17B - E2)	0.96	Clara et al, 2005b
Primary + CAS Removal	Estradiol (17B - E2)	0.95	Clara et al, 2005b
Primary + CAS Removal	Estradiol (17B - E2)	1.00	Clara et al, 2005b
Primary + CAS Removal	Estradiol (17B - E2)	0.80	Clara et al., 2005b
Primary + CAS Removal	Estradiol (17B - E2)	0.93	Clara et al., 2005b
Primary + CAS Removal	Estradiol (17B - E2)	0.89	Clara et al., 2005b
Primary + CAS Removal	Estradiol (17B - E2)	0.80	Joss et al., 2004
Primary + CAS Removal	Estradiol (17B - E2)	0.93	Joss et al., 2004
Primary + CAS Removal	Estradiol (17B - E2)	0.95	Joss et al., 2004
Primary + CAS Removal	Estradiol (17B - E2)	0.84	Nakada et al., 2006
Primary + CAS Removal	Estradiol (17B - E2)	0.84	Nakada et al., 2006
Primary + CAS Removal	Estradiol (17B - E2)	0.90	Nakada et al., 2006
Primary + CAS Removal	Estradiol (17B - E2)	0.85	Thomas et al., 2007

Primary + CAS Removal	Estradiol (17B - E2)	0.22	Zorita et al., 2009
Ultrafiltration Removal	Estradiol (17B - E2)	0.99	Snyder et al., 2007
Ultrafiltration Removal	Estradiol (17B - E2)	0.95	Yoon et al., 2006
Influent Concentration (ng/L)	Estriol (E3)	0.00	Blair et al., 2013
Influent Concentration (ng/L)	Estriol (E3)	10.00	Blair et al., 2013
Influent Concentration (ng/L)	Estriol (E3)	0.00	Blair et al., 2013
Influent Concentration (ng/L)	Estriol (E3)	0.00	Blair et al., 2013
Influent Concentration (ng/L)	Estriol (E3)	22.00	Blair et al., 2013
Influent Concentration (ng/L)	Estriol (E3)	336.00	Clara et al, 2005b
Influent Concentration (ng/L)	Estriol (E3)	23.50	Clara et al, 2005b
Influent Concentration (ng/L)	Estriol (E3)	143.00	Clara et al, 2005b
Influent Concentration (ng/L)	Estriol (E3)	372.00	Clara et al, 2005b
Influent Concentration (ng/L)	Estriol (E3)	660.00	Clara et al., 2005b
Influent Concentration (ng/L)	Estriol (E3)	326.00	Clara et al., 2005b
Influent Concentration (ng/L)	Estriol (E3)	129.00	Nakada et al., 2006
Influent Concentration (ng/L)	Estriol (E3)	225.00	Nakada et al., 2006
Influent Concentration (ng/L)	Estriol (E3)	137.00	Nakada et al., 2006
Influent Concentration (ng/L)	Estriol (E3)	92.60	Nakada et al., 2006
Influent Concentration (ng/L)	Estriol (E3)	83.00	Nakada et al., 2006
Influent Concentration (ng/L)	Estriol (E3)	128.00	Thomas et al., 2007
Influent Concentration (ng/L)	Estriol (E3)	54.00	Thomas et al., 2007
Influent Concentration (ng/L)	Estriol (E3)	237.00	Thomas et al., 2007
MBR Removal	Estriol (E3)	1.00	Clara et al, 2005b
MBR Removal	Estriol (E3)	1.00	Clara et al, 2005b
MBR Removal	Estriol (E3)	1.00	Clara et al, 2005b
MBR Removal	Estriol (E3)	0.97	Kim et al., 2007
MBR Removal	Estriol (E3)	0.97	Kim et al., 2007
MBR Removal	Estriol (E3)	0.99	Snyder et al., 2007

Microfiltration Removal	Estriol (E3)	0.00	Snyder et al., 2007
Nanofiltration Removal	Estriol (E3)	0.98	Kim et al., 2007
Primary + CAS Removal	Estriol (E3)	0.18	Clara et al, 2005b
Primary + CAS Removal	Estriol (E3)	0.28	Clara et al, 2005b
Primary + CAS Removal	Estriol (E3)	0.99	Clara et al, 2005b
Primary + CAS Removal	Estriol (E3)	1.00	Clara et al, 2005b
Primary + CAS Removal	Estriol (E3)	1.00	Clara et al., 2005b
Primary + CAS Removal	Estriol (E3)	1.00	Clara et al., 2005b
Primary + CAS Removal	Estriol (E3)	1.00	Nakada et al., 2006
Primary + CAS Removal	Estriol (E3)	1.00	Nakada et al., 2006
Primary + CAS Removal	Estriol (E3)	1.00	Nakada et al., 2006
Primary + CAS Removal	Estriol (E3)	0.98	Thomas et al., 2007
Ultrafiltration Removal	Estriol (E3)	0.41	Snyder et al., 2007
Influent Concentration (ng/L)	Gemfibrozil	710.00	Bendz et al., 2005
Influent Concentration (ng/L)	Gemfibrozil	29.00	Blair et al., 2013
Influent Concentration (ng/L)	Gemfibrozil	150.00	Blair et al., 2013
Influent Concentration (ng/L)	Gemfibrozil	200.00	Blair et al., 2013
Influent Concentration (ng/L)	Gemfibrozil	97.00	Blair et al., 2013
Influent Concentration (ng/L)	Gemfibrozil	510.00	Blair et al., 2013
Influent Concentration (ng/L)	Gemfibrozil	1200.00	Blair et al., 2013
Influent Concentration (ng/L)	Gemfibrozil	1500.00	Khan and Ongerth, 2005
Influent Concentration (ng/L)	Gemfibrozil	453.00	Lishman et al., 2006
Influent Concentration (ng/L)	Gemfibrozil	2000.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Gemfibrozil	5900.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Gemfibrozil	3080.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Gemfibrozil	415.00	Rosal et al., 2010
Influent Concentration (ng/L)	Gemfibrozil	3525.00	Rosal et al., 2010
Influent Concentration (ng/L)	Gemfibrozil	415.00	Rosal et al., 2010
Influent Concentration (ng/L)	Gemfibrozil	181.80	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Gemfibrozil	450.20	Spongberg and Witter, 2008

Influent Concentration (ng/L)	Gemfibrozil	451.30	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Gemfibrozil	410.00	Yu et al., 2006
MBR Removal	Gemfibrozil	0.42	Radjenovic et al., 2009
MBR Removal	Gemfibrozil	0.33	Radjenovic et al., 2009
MBR Removal	Gemfibrozil	0.86	Snyder et al., 2007
MBR Removal	Gemfibrozil	0.89	Snyder et al., 2007
Microfiltration Removal	Gemfibrozil	0.00	Snyder et al., 2007
Nanofiltration Removal	Gemfibrozil	0.50	Yoon et al., 2006
Primary + CAS Removal	Gemfibrozil	0.75	Bendz et al., 2005
Primary + CAS Removal	Gemfibrozil	0.00	Blair et al., 2013
Primary + CAS Removal	Gemfibrozil	0.43	Blair et al., 2013
Primary + CAS Removal	Gemfibrozil	0.00	Blair et al., 2013
Primary + CAS Removal	Gemfibrozil	0.00	Blair et al., 2013
Primary + CAS Removal	Gemfibrozil	0.08	Blair et al., 2013
Primary + CAS Removal	Gemfibrozil	0.85	Khan and Ongerth, 2005
Primary + CAS Removal	Gemfibrozil	0.96	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.27	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.28	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.25	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.24	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.00	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.41	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.00	Lee et al., 2003
Primary + CAS Removal	Gemfibrozil	0.46	Lishman et al., 2006
Primary + CAS Removal	Gemfibrozil	1.00	Spongberg and Witter, 2008
Primary + CAS Removal	Gemfibrozil	0.91	Spongberg and Witter, 2008
Primary + CAS Removal	Gemfibrozil	0.81	Spongberg and Witter, 2008
Primary + CAS Removal	Gemfibrozil	0.68	Yu et al., 2006
Ultrafiltration Removal	Gemfibrozil	0.00	Snyder et al., 2007
Ultrafiltration Removal	Gemfibrozil	0.21	Snyder et al., 2006
Ultrafiltration Removal	Gemfibrozil	0.00	Yoon et al., 2006
Influent Concentration (ng/L)	Ibuprofen	3590.00	Bendz et al., 2005
Influent Concentration (ng/L)	Ibuprofen	3590.00	Bendz et al., 2005
Influent Concentration (ng/L)	Ibuprofen	3000.00	Blair et al., 2013

Influent Concentration (ng/L)	Ibuprofen	11000.00	Blair et al., 2013
Influent Concentration (ng/L)	Ibuprofen	1300.00	Blair et al., 2013
Influent Concentration (ng/L)	Ibuprofen	840.00	Blair et al., 2013
Influent Concentration (ng/L)	Ibuprofen	670.00	Blair et al., 2013
Influent Concentration (ng/L)	Ibuprofen	4800.00	Blair et al., 2013
Influent Concentration (ng/L)	Ibuprofen	2750.00	Carballa et al., 2004
Influent Concentration (ng/L)	Ibuprofen	5700.00	Carballa et al., 2004
Influent Concentration (ng/L)	Ibuprofen	2640.00	Carballa et al., 2004
Influent Concentration (ng/L)	Ibuprofen	1480.00	Clara et al., 2005a
Influent Concentration (ng/L)	Ibuprofen	2679.00	Clara et al., 2005a
Influent Concentration (ng/L)	Ibuprofen	2448.00	Clara et al., 2005a
Influent Concentration (ng/L)	Ibuprofen	2300.00	Clara et al., 2005a
Influent Concentration (ng/L)	Ibuprofen	1200.00	Clara et al., 2005a
Influent Concentration (ng/L)	Ibuprofen	34000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Ibuprofen	84000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Ibuprofen	168000.00	Gómez et al., 2007
Influent Concentration (ng/L)	Ibuprofen	984.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Ibuprofen	6328.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Ibuprofen	2700.00	Khan and Ongerth, 2005
Influent Concentration (ng/L)	Ibuprofen	40.00	Kimura et al., 2007
Influent Concentration (ng/L)	Ibuprofen	8840.00	Lishman et al., 2006
Influent Concentration (ng/L)	Ibuprofen	8450.00	Lishman et al., 2006
Influent Concentration (ng/L)	Ibuprofen	1050.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	694.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	706.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	746.00	Nakada et al., 2006

Influent Concentration (ng/L)	Ibuprofen	545.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	806.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	513.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	479.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	748.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	1130.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	756.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	636.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	381.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	650.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	407.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	452.00	Nakada et al., 2006
Influent Concentration (ng/L)	Ibuprofen	5700.00	Quintana et al., 2005
Influent Concentration (ng/L)	Ibuprofen	14600.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Ibuprofen	31300.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Ibuprofen	33764.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Ibuprofen	27979.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Ibuprofen	7741.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Ibuprofen	0.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	2687.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	0.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	4113.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	4113.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	2687.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	0.00	Rosal et al., 2010
Influent Concentration (ng/L)	Ibuprofen	115000.00	Santos et al., 2009

Influent Concentration (ng/L)	Ibuprofen	603000.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	0.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	294000.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	84400.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	0.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	105000.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	319000.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	0.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	12130.00	Santos et al., 2007,
Influent Concentration (ng/L)	Ibuprofen	373110.00	Santos et al., 2007,
Influent Concentration (ng/L)	Ibuprofen	3730.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	69700.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	353000.00	Santos et al., 2009
Influent Concentration (ng/L)	Ibuprofen	10800.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Ibuprofen	9500.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Ibuprofen	14700.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Ibuprofen	178.00	Thomas et al., 2007
Influent Concentration (ng/L)	Ibuprofen	6900.00	Zorita et al., 2009
MBR Removal	Ibuprofen	0.99	Clara et al, 2005b
MBR Removal	Ibuprofen	0.99	Clara et al, 2005b
MBR Removal	Ibuprofen	0.97	Clara et al, 2005b
MBR Removal	Ibuprofen	0.98	Clara et al., 2005a
MBR Removal	Ibuprofen	0.99	Kim et al., 2007
MBR Removal	Ibuprofen	0.98	Kim et al., 2007
MBR Removal	Ibuprofen	0.95	Kimura et al, 2007
MBR Removal	Ibuprofen	0.98	Kimura et al, 2007
MBR Removal	Ibuprofen	0.97	Quintana et al., 2005
MBR Removal	Ibuprofen	0.99	Radjenovic et al., 2009
MBR Removal	Ibuprofen	1.00	Radjenovic et al., 2009

MBR Removal	Ibuprofen	1.00	Snyder et al., 2007
MBR Removal	Ibuprofen	0.00	Snyder et al., 2007
Microfiltration Removal	Ibuprofen	0.55	Snyder et al., 2007
Nanofiltration Removal	Ibuprofen	1.00	Kim et al., 2007
Primary + CAS Removal	Ibuprofen	0.96	Bendz et al., 2005
Primary + CAS Removal	Ibuprofen	1.00	Blair et al., 2013
Primary + CAS Removal	Ibuprofen	1.00	Blair et al., 2013
Primary + CAS Removal	Ibuprofen	0.84	Blair et al., 2013
Primary + CAS Removal	Ibuprofen	1.00	Blair et al., 2013
Primary + CAS Removal	Ibuprofen	0.17	Blair et al., 2013
Primary + CAS Removal	Ibuprofen	1.00	Buser et al., 1999
Primary + CAS Removal	Ibuprofen	0.99	Buser et al., 1999
Primary + CAS Removal	Ibuprofen	0.96	Buser et al., 1999
Primary + CAS Removal	Ibuprofen	0.63	Carballa et al., 2004
Primary + CAS Removal	Ibuprofen	0.63	Carballa et al., 2004
Primary + CAS Removal	Ibuprofen	0.67	Carballa et al., 2004
Primary + CAS Removal	Ibuprofen	0.00	Clara et al, 2005b
Primary + CAS Removal	Ibuprofen	0.92	Clara et al, 2005b
Primary + CAS Removal	Ibuprofen	0.98	Clara et al, 2005b
Primary + CAS Removal	Ibuprofen	0.99	Clara et al, 2005b
Primary + CAS Removal	Ibuprofen	0.99	Clara et al, 2005b
Primary + CAS Removal	Ibuprofen	0.99	Clara et al, 2005b
Primary + CAS Removal	Ibuprofen	0.99	Clara et al., 2005a
Primary + CAS Removal	Ibuprofen	0.00	Clara et al., 2005a
Primary + CAS Removal	Ibuprofen	0.98	Clara et al., 2005a
Primary + CAS Removal	Ibuprofen	0.92	Gómez et al., 2007
Primary + CAS Removal	Ibuprofen	0.94	Kasprzyk-Hordern et al., 2009
Primary + CAS Removal	Ibuprofen	0.97	Khan and Ongerth, 2005
Primary + CAS Removal	Ibuprofen	0.98	Kimura et al, 2007
Primary + CAS Removal	Ibuprofen	1.00	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.83	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.88	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.80	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.99	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.85	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.98	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.84	Lee et al., 2003

Primary + CAS Removal	Ibuprofen	0.94	Lee et al., 2003
Primary + CAS Removal	Ibuprofen	0.95	Lishman et al., 2006
Primary + CAS Removal	Ibuprofen	0.92	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.90	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.96	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.84	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.97	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.96	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.94	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.96	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.99	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	1.00	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.99	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.93	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.97	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	1.00	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.99	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.99	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.99	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.00	Nakada et al., 2006
Primary + CAS Removal	Ibuprofen	0.88	Santos et al., 2007
Primary + CAS Removal	Ibuprofen	0.90	Santos et al., 2007
Primary + CAS Removal	Ibuprofen	0.92	Santos et al., 2007
Primary + CAS Removal	Ibuprofen	0.99	Santos et al., 2007
Primary + CAS Removal	Ibuprofen	0.94	Santos et al., 2009
Primary + CAS Removal	Ibuprofen	0.92	Santos et al., 2009
Primary + CAS Removal	Ibuprofen	0.90	Santos et al., 2009
Primary + CAS Removal	Ibuprofen	0.93	Santos et al., 2009
Primary + CAS Removal	Ibuprofen	0.82	Suarez et al., 2005
Primary + CAS Removal	Ibuprofen	0.65	Thomas et al., 2007
Primary + CAS Removal	Ibuprofen	0.72	Yu and Chu, 2009
Primary + CAS Removal	Ibuprofen	0.76	Yu and Chu, 2009
Primary + CAS Removal	Ibuprofen	0.87	Yu et al., 2006
Primary + CAS Removal	Ibuprofen	0.99	Zorita et al., 2009
Ultrafiltration Removal	Ibuprofen	0.08	Snyder et al., 2007
Influent Concentration (ng/L)	Naproxen	3650.00	Bendz et al., 2005
Influent Concentration (ng/L)	Naproxen	4200.00	Blair et al., 2013

Influent Concentration (ng/L)	Naproxen	9400.00	Blair et al., 2013
Influent Concentration (ng/L)	Naproxen	2900.00	Blair et al., 2013
Influent Concentration (ng/L)	Naproxen	1800.00	Blair et al., 2013
Influent Concentration (ng/L)	Naproxen	780.00	Blair et al., 2013
Influent Concentration (ng/L)	Naproxen	3100.00	Blair et al., 2013
Influent Concentration (ng/L)	Naproxen	1790.00	Carballa et al., 2004
Influent Concentration (ng/L)	Naproxen	4600.00	Carballa et al., 2004
Influent Concentration (ng/L)	Naproxen	3450.00	Carballa et al., 2004
Influent Concentration (ng/L)	Naproxen	620.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Naproxen	3504.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Naproxen	6500.00	Khan and Ongerth, 2005
Influent Concentration (ng/L)	Naproxen	276.00	Kimura et al., 2007
Influent Concentration (ng/L)	Naproxen	5580.00	Lishman et al., 2006
Influent Concentration (ng/L)	Naproxen	38.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	100.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	230.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	162.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	103.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	230.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	80.60	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	173.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	138.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	97.60	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	61.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	145.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	164.00	Nakada et al., 2006

Influent Concentration (ng/L)	Naproxen	116.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	68.30	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	89.40	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	72.60	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	54.60	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	38.00	Nakada et al., 2006
Influent Concentration (ng/L)	Naproxen	130.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Naproxen	670.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Naproxen	463.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Naproxen	1196.00	Rosal et al., 2010
Influent Concentration (ng/L)	Naproxen	5228.00	Rosal et al., 2010
Influent Concentration (ng/L)	Naproxen	2363.00	Rosal et al., 2010
Influent Concentration (ng/L)	Naproxen	2020.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	2050.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	1600.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	1100.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	7230.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	26640.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	27400.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	9100.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	4040.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	11140.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	5180.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	5070.00	Santos et al., 2007
Influent Concentration (ng/L)	Naproxen	10000.00	Suárez et al., 2005
Influent Concentration (ng/L)	Naproxen	10300.00	Thomas and Foster, 2005

Influent Concentration (ng/L)	Naproxen	12800.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Naproxen	11700.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Naproxen	3200.00	Yu et al., 2006
Influent Concentration (ng/L)	Naproxen	4900.00	Zorita et al., 2009
MBR Removal	Naproxen	0.36	Kim et al., 2007
MBR Removal	Naproxen	0.41	Kim et al., 2007
MBR Removal	Naproxen	0.96	Kimura et al, 2007
MBR Removal	Naproxen	0.96	Kimura et al, 2007
MBR Removal	Naproxen	0.72	Quintana et al., 2005
MBR Removal	Naproxen	0.91	Radjenovic et al., 2009
MBR Removal	Naproxen	0.92	Radjenovic et al., 2009
MBR Removal	Naproxen	0.96	Snyder et al., 2007
MBR Removal	Naproxen	1.00	Snyder et al., 2007
Microfiltration Removal	Naproxen	0.00	Snyder et al., 2007
Nanofiltration Removal	Naproxen	0.44	Chon et al., 2012
Nanofiltration Removal	Naproxen	1.00	Chon et al., 2012
Nanofiltration Removal	Naproxen	1.00	Chon et al., 2012
Nanofiltration Removal	Naproxen	1.00	Kim et al., 2007
Primary + CAS Removal	Naproxen	0.93	Bendz et al., 2005
Primary + CAS Removal	Naproxen	0.88	Blair et al., 2013
Primary + CAS Removal	Naproxen	0.94	Blair et al., 2013
Primary + CAS Removal	Naproxen	0.96	Blair et al., 2013
Primary + CAS Removal	Naproxen	0.98	Blair et al., 2013
Primary + CAS Removal	Naproxen	0.00	Blair et al., 2013
Primary + CAS Removal	Naproxen	0.55	Carballa et al., 2004
Primary + CAS Removal	Naproxen	0.43	Carballa et al., 2004
Primary + CAS Removal	Naproxen	0.46	Carballa et al., 2004
Primary + CAS Removal	Naproxen	0.58	Hollender et al., 2009
Primary + CAS Removal	Naproxen	0.86	Kasprzyk-Hordern et al., 2009
Primary + CAS Removal	Naproxen	0.95	Khan and Ongerth, 2005
Primary + CAS Removal	Naproxen	0.64	Kimura et al, 2007
Primary + CAS Removal	Naproxen	0.51	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.92	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.62	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.49	Lee et al., 2003

Primary + CAS Removal	Naproxen	0.42	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.72	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.84	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.68	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.75	Lee et al., 2003
Primary + CAS Removal	Naproxen	0.92	Lishman et al., 2006
Primary + CAS Removal	Naproxen	0.59	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.65	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.00	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.95	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.72	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.34	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.65	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.63	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.20	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.23	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.49	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.37	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.15	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.23	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.47	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.68	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.00	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.89	Nakada et al., 2006
Primary + CAS Removal	Naproxen	0.89	Santos et al., 2007
Primary + CAS Removal	Naproxen	0.35	Santos et al., 2007
Primary + CAS Removal	Naproxen	0.65	Santos et al., 2007
Primary + CAS Removal	Naproxen	0.61	Santos et al., 2007
Primary + CAS Removal	Naproxen	0.43	Santos et al., 2009
Primary + CAS Removal	Naproxen	0.80	Santos et al., 2009
Primary + CAS Removal	Naproxen	0.54	Santos et al., 2009
Primary + CAS Removal	Naproxen	0.61	Santos et al., 2009
Primary + CAS Removal	Naproxen	0.68	Suárez et al., 2005
Primary + CAS Removal	Naproxen	0.88	Yu et al., 2006
Primary + CAS Removal	Naproxen	0.93	Zorita et al., 2009
Ultrafiltration Removal	Naproxen	0.13	Snyder et al., 2007
Ultrafiltration Removal	Naproxen	0.24	Snyder et al., 2006
Influent Concentration	Sulfamethoxazole	20.00	Bendz et al., 2005

(ng/L)			
Influent Concentration (ng/L)	Sulfamethoxazole	360.00	Benotti et al., 2007
Influent Concentration (ng/L)	Sulfamethoxazole	210.00	Blair et al., 2013
Influent Concentration (ng/L)	Sulfamethoxazole	1200.00	Blair et al., 2013
Influent Concentration (ng/L)	Sulfamethoxazole	110.00	Blair et al., 2013
Influent Concentration (ng/L)	Sulfamethoxazole	120.00	Blair et al., 2013
Influent Concentration (ng/L)	Sulfamethoxazole	54.00	Blair et al., 2013
Influent Concentration (ng/L)	Sulfamethoxazole	170.00	Blair et al., 2013
Influent Concentration (ng/L)	Sulfamethoxazole	390.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	310.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	1000.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	400.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Carballa et al., 2004
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Carballa et al., 2004
Influent Concentration (ng/L)	Sulfamethoxazole	580.00	Carballa et al., 2004
Influent Concentration (ng/L)	Sulfamethoxazole	381.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	316.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	984.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	300.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	611.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	660.00	Choi et al., 2008

Influent Concentration (ng/L)	Sulfamethoxazole	156.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	221.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	849.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	263.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	652.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	877.00	Choi et al., 2008
Influent Concentration (ng/L)	Sulfamethoxazole	145.00	Clara et al., 2005a
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Clara et al., 2005a
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Clara et al., 2005a
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Clara et al., 2005a
Influent Concentration (ng/L)	Sulfamethoxazole	78.00	Clara et al., 2005a
Influent Concentration (ng/L)	Sulfamethoxazole	25.00	Clara et al., 2005a
Influent Concentration (ng/L)	Sulfamethoxazole	159.00	Ghosh et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	184.00	Ghosh et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	177.00	Ghosh et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	230.00	Gobel et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	430.00	Gobel et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	570.00	Gobel et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	1250.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	170.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	560.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	130.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	350.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	450.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	250.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Sulfamethoxazole	20.00	Kasprzyk-Hordern et al., 2009

Influent Concentration (ng/L)	Sulfamethoxazole	274.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	674.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	144.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	231.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	337.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	302.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	0.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Sulfamethoxazole	250.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	1300.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	93.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	279.00	Rosal et al., 2010
Influent Concentration (ng/L)	Sulfamethoxazole	162.00	Rosal et al., 2010
Influent Concentration (ng/L)	Sulfamethoxazole	530.00	Rosal et al., 2010
Influent Concentration (ng/L)	Sulfamethoxazole	261.00	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Sulfamethoxazole	158.70	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Sulfamethoxazole	13.50	Spongberg and Witter, 2008
Influent Concentration (ng/L)	Sulfamethoxazole	125.00	Thomas et al., 2007
Influent Concentration (ng/L)	Sulfamethoxazole	360.00	Watkinson et al., 2007
Influent Concentration (ng/L)	Sulfamethoxazole	250.00	Watkinson et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	16.00	Xu et al., 2007
Influent Concentration (ng/L)	Sulfamethoxazole	118.00	Xu et al., 2007
Influent Concentration (ng/L)	Sulfamethoxazole	10.00	Xu et al., 2007

Influent Concentration (ng/L)	Sulfamethoxazole	25.00	Xu et al., 2007
Influent Concentration (ng/L)	Sulfamethoxazole	49.00	Zhou et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	110.00	Zhou et al., 2009
Influent Concentration (ng/L)	Sulfamethoxazole	181.00	Zhou et al., 2009
MBR Removal	Sulfamethoxazole	0.64	Kim et al., 2007
MBR Removal	Sulfamethoxazole	0.70	Kim et al., 2007
MBR Removal	Sulfamethoxazole	0.81	Radjenovic et al., 2009
MBR Removal	Sulfamethoxazole	0.78	Radjenovic et al., 2009
MBR Removal	Sulfamethoxazole	0.00	Snyder et al., 2007
MBR Removal	Sulfamethoxazole	0.57	Snyder et al., 2007
Microfiltration Removal	Sulfamethoxazole	0.00	Snyder et al., 2007
Microfiltration Removal	Sulfamethoxazole		Watkinson et al., 2007
Nanofiltration Removal	Sulfamethoxazole	0.38	Chon et al., 2012
Nanofiltration Removal	Sulfamethoxazole	0.46	Chon et al., 2012
Nanofiltration Removal	Sulfamethoxazole	0.73	Chon et al., 2012
Nanofiltration Removal	Sulfamethoxazole	0.99	Kim et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.75	Batt et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.77	Batt et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.36	Batt et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.36	Batt et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.00	Bendz et al., 2005
Primary + CAS Removal	Sulfamethoxazole	0.61	Benotti et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.00	Blair et al., 2013
Primary + CAS Removal	Sulfamethoxazole	0.75	Blair et al., 2013
Primary + CAS Removal	Sulfamethoxazole	0.39	Blair et al., 2013
Primary + CAS Removal	Sulfamethoxazole	0.37	Blair et al., 2013
Primary + CAS Removal	Sulfamethoxazole	0.66	Blair et al., 2013
Primary + CAS Removal	Sulfamethoxazole	0.57	Carballa et al., 2004
Primary + CAS Removal	Sulfamethoxazole	0.83	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.43	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.80	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.92	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.70	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.58	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.80	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.33	Choi et al., 2008

Primary + CAS Removal	Sulfamethoxazole	0.63	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.17	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.72	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.44	Choi et al., 2008
Primary + CAS Removal	Sulfamethoxazole	0.35	Gobel et al., 2005
Primary + CAS Removal	Sulfamethoxazole	0.00	Gobel et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.09	Gobel et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.00	Gobel et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.00	Gobel et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.60	Gobel et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.83	Kasprzyk-Hordern et al., 2009
Primary + CAS Removal	Sulfamethoxazole	0.62	Nakada et al., 2006
Primary + CAS Removal	Sulfamethoxazole	0.00	Spongberg and Witter, 2008
Primary + CAS Removal	Sulfamethoxazole	0.00	Spongberg and Witter, 2008
Primary + CAS Removal	Sulfamethoxazole	0.00	Spongberg and Witter, 2008
Primary + CAS Removal	Sulfamethoxazole	0.61	Thomas et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.80	Watkinson et al., 2009
Primary + CAS Removal	Sulfamethoxazole	0.00	Xu et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.00	Xu et al., 2007
Primary + CAS Removal	Sulfamethoxazole	0.53	Zhou et al., 2009
Primary + CAS Removal	Sulfamethoxazole	0.66	Zhou et al., 2009
Primary + CAS Removal	Sulfamethoxazole	0.82	Zhou et al., 2009
Ultrafiltration Removal	Sulfamethoxazole	0.05	Snyder et al., 2007
Ultrafiltration Removal	Sulfamethoxazole	0.14	Snyder et al., 2006
Influent Concentration (ng/L)	Triclosan	380.00	Bendz et al., 2005
Influent Concentration (ng/L)	Triclosan	4300.00	Blair et al., 2013
Influent Concentration (ng/L)	Triclosan	9100.00	Blair et al., 2013
Influent Concentration (ng/L)	Triclosan	89.00	Blair et al., 2013
Influent Concentration (ng/L)	Triclosan	130.00	Blair et al., 2013
Influent Concentration (ng/L)	Triclosan	610.00	Blair et al., 2013
Influent Concentration (ng/L)	Triclosan	680.00	Blair et al., 2013
Influent Concentration (ng/L)	Triclosan	390.00	Gómez et al., 2007

Influent Concentration (ng/L)	Triclosan	4200.00	Gómez et al., 2007
Influent Concentration (ng/L)	Triclosan	1800.00	Gómez et al., 2007
Influent Concentration (ng/L)	Triclosan	1930.00	Lishman et al., 2006
Influent Concentration (ng/L)	Triclosan	5210.00	McAvoy et al., 2002
Influent Concentration (ng/L)	Triclosan	3830.00	McAvoy et al., 2002
Influent Concentration (ng/L)	Triclosan	16600.00	McAvoy et al., 2002
Influent Concentration (ng/L)	Triclosan	15400.00	McAvoy et al., 2002
Influent Concentration (ng/L)	Triclosan	10700.00	McAvoy et al., 2002
Influent Concentration (ng/L)	Triclosan	618.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	620.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	978.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	518.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	803.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	1020.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	541.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	352.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	387.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	404.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	434.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	219.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	262.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	346.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	381.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	296.00	Nakada et al., 2006
Influent Concentration (ng/L)	Triclosan	860.00	Rosal et al., 2010
Influent Concentration (ng/L)	Triclosan	0.00	Rosal et al., 2010

Influent Concentration (ng/L)	Triclosan	2417.00	Rosal et al., 2010
Influent Concentration (ng/L)	Triclosan	450.00	Ruel et al., 2010
Influent Concentration (ng/L)	Triclosan	3000.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Triclosan	3600.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Triclosan	3300.00	Thomas and Foster, 2005
Influent Concentration (ng/L)	Triclosan	800.00	Yu et al., 2006
MBR Removal	Triclosan	0.66	Kim et al., 2007
MBR Removal	Triclosan	0.73	Kim et al., 2007
MBR Removal	Triclosan	0.96	Snyder et al., 2007
MBR Removal	Triclosan	0.76	Snyder et al., 2007
Microfiltration Removal	Triclosan	0.00	Snyder et al., 2007
Nanofiltration Removal	Triclosan	0.99	Kim et al., 2007
Primary + CAS Removal	Triclosan	0.58	Bendz et al., 2005
Primary + CAS Removal	Triclosan	0.97	Blair et al., 2013
Primary + CAS Removal	Triclosan	0.99	Blair et al., 2013
Primary + CAS Removal	Triclosan	0.73	Blair et al., 2013
Primary + CAS Removal	Triclosan	0.75	Blair et al., 2013
Primary + CAS Removal	Triclosan	0.49	Blair et al., 2013
Primary + CAS Removal	Triclosan	0.89	Gómez et al., 2007
Primary + CAS Removal	Triclosan	0.60	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.99	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.73	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.69	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.84	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.83	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.84	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.47	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.94	Lee et al., 2003
Primary + CAS Removal	Triclosan	0.94	Lishman et al., 2006
Primary + CAS Removal	Triclosan	0.46	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.57	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.62	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.00	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.79	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.76	Nakada et al., 2006

Primary + CAS Removal	Triclosan	0.52	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.70	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.93	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.98	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.86	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.77	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.66	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.59	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.91	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.75	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.50	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.70	Nakada et al., 2006
Primary + CAS Removal	Triclosan	0.72	Yu and Chu, 2009
Primary + CAS Removal	Triclosan	0.79	Yu and Chu, 2009
Primary + CAS Removal	Triclosan	0.69	Yu et al., 2006
Ultrafiltration Removal	Triclosan	0.88	Snyder et al., 2007
Ultrafiltration Removal	Triclosan	0.00	Snyder et al., 2006
Influent Concentration (ng/L)	Trimethoprim	80.00	Bendz et al., 2005
Influent Concentration (ng/L)	Trimethoprim	300.00	Benotti et al., 2007
Influent Concentration (ng/L)	Trimethoprim	220.00	Blair et al., 2013
Influent Concentration (ng/L)	Trimethoprim	590.00	Blair et al., 2013
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Influent Concentration (ng/L)	Trimethoprim	180.00	Brown et al., 2006
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Influent Concentration (ng/L)	Trimethoprim	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Trimethoprim	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Trimethoprim	0.00	Brown et al., 2006

Influent Concentration (ng/L)	Trimethoprim	0.00	Brown et al., 2006
Influent Concentration (ng/L)	Trimethoprim	1000.00	Brown et al., 2006
Influent Concentration (ng/L)	Trimethoprim	0.00	Brown et al., 2006
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Influent Concentration (ng/L)	Trimethoprim	135.00	Choi et al., 2008
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Influent Concentration (ng/L)	Trimethoprim	81.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	496.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	84.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	19.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	97.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	0.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	125.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	401.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	104.00	Choi et al., 2008
Influent Concentration (ng/L)	Trimethoprim	26.00	Ghosh et al., 2009
Influent Concentration (ng/L)	Trimethoprim	106.00	Ghosh et al., 2009
Influent Concentration (ng/L)	Trimethoprim	89.00	Ghosh et al., 2009
Influent Concentration (ng/L)	Trimethoprim	210.00	Göbel et al., 2005
Influent Concentration (ng/L)	Trimethoprim	440.00	Gobel et al., 2005
Influent Concentration (ng/L)	Trimethoprim	290.00	Gobel et al., 2005
Influent Concentration (ng/L)	Trimethoprim	120.00	Gulkowska et al., 2008
Influent Concentration (ng/L)	Trimethoprim	120.00	Gulkowska et al., 2008
Influent Concentration (ng/L)	Trimethoprim	320.00	Gulkowska et al., 2008
Influent Concentration (ng/L)	Trimethoprim	210.00	Gulkowska et al., 2008
Influent Concentration (ng/L)	Trimethoprim	200.00	Gulkowska et al., 2008

Influent Concentration (ng/L)	Trimethoprim	1100.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	210.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	580.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	140.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	1300.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	300.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	50.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	700.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	120.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	350.00	Karthikeyan and Meyer, 2006
Influent Concentration (ng/L)	Trimethoprim	1514.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Trimethoprim	4673.00	Kasprzyk-Hordern et al., 2009
Influent Concentration (ng/L)	Trimethoprim	140.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	364.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	208.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	172.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	99.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	651.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	1300.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	548.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	946.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	251.00	Lindberg et al., 2005
Influent Concentration (ng/L)	Trimethoprim	150.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Trimethoprim	430.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Trimethoprim	204.00	Radjenovic et al., 2009
Influent Concentration (ng/L)	Trimethoprim	213.00	Roberts and Thomas, 2006

Influent Concentration (ng/L)	Trimethoprim	263.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Trimethoprim	213.00	Roberts and Thomas, 2006
Influent Concentration (ng/L)	Trimethoprim	78.00	Rosal et al., 2006
Influent Concentration (ng/L)	Trimethoprim	197.00	Rosal et al., 2006
Influent Concentration (ng/L)	Trimethoprim	104.00	Rosal et al., 2006
Influent Concentration (ng/L)	Trimethoprim	835.00	Thomas et al., 2007
Influent Concentration (ng/L)	Trimethoprim	340.00	Watkinson et al., 2007
Influent Concentration (ng/L)	Trimethoprim	430.00	Watkinson et al., 2009
MBR Removal	Trimethoprim	0.00	Kim et al., 2007
MBR Removal	Trimethoprim	0.00	Kim et al., 2007
MBR Removal	Trimethoprim	0.67	Radjenovic et al., 2009
MBR Removal	Trimethoprim	0.48	Radjenovic et al., 2009
MBR Removal	Trimethoprim	0.76	Snyder et al., 2007
MBR Removal	Trimethoprim	0.90	Snyder et al., 2007
Microfiltration Removal	Trimethoprim	0.00	Snyder et al., 2007
Nanofiltration Removal	Trimethoprim	0.95	Kim et al., 2007
Primary + CAS Removal	Trimethoprim	0.68	Batt et al., 2007
Primary + CAS Removal	Trimethoprim	0.96	Batt et al., 2007
Primary + CAS Removal	Trimethoprim	0.75	Batt et al., 2007
Primary + CAS Removal	Trimethoprim	0.72	Batt et al., 2007
Primary + CAS Removal	Trimethoprim	0.49	Bendz et al., 2005
Primary + CAS Removal	Trimethoprim	0.60	Benotti et al., 2007
Primary + CAS Removal	Trimethoprim	0.00	Blair et al., 2013
Primary + CAS Removal	Trimethoprim	0.56	Blair et al., 2013
Primary + CAS Removal	Trimethoprim	0.42	Blair et al., 2013
Primary + CAS Removal	Trimethoprim	0.00	Blair et al., 2013
Primary + CAS Removal	Trimethoprim	0.24	Blair et al., 2013
Primary + CAS Removal	Trimethoprim	1.00	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	0.36	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	1.00	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	0.84	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	0.76	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	1.00	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	1.00	Choi et al., 2008

Primary + CAS Removal	Trimethoprim	0.00	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	0.75	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	0.73	Choi et al., 2008
Primary + CAS Removal	Trimethoprim	0.00	Ghosh et al., 2009
Primary + CAS Removal	Trimethoprim	0.35	Ghosh et al., 2009
Primary + CAS Removal	Trimethoprim	0.03	Gobel et al., 2007
Primary + CAS Removal	Trimethoprim	0.00	Gobel et al., 2007
Primary + CAS Removal	Trimethoprim	0.14	Gobel et al., 2007
Primary + CAS Removal	Trimethoprim	0.20	Gobel et al., 2007
Primary + CAS Removal	Trimethoprim	0.00	Gobel et al., 2007
Primary + CAS Removal	Trimethoprim	0.70	Kasprzyk-Hordern et al., 2009
Primary + CAS Removal	Trimethoprim	0.70	Nakada et al., 2006
Primary + CAS Removal	Trimethoprim	0.26	Thomas et al., 2007
Primary + CAS Removal	Trimethoprim	0.98	Watkinson et al., 2009
Ultrafiltration Removal	Trimethoprim	0.18	Snyder et al., 2007
Ultrafiltration Removal	Trimethoprim	0.21	Snyder et al., 2006

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Example Code for Ibuprofen

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2700
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14600
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8450
1050
694
5700
33764
2687
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12130
3730
31300
10800
9500
6900
3590
8840
2687
0
4113
14700
84000
6328
115000
5700
2640
69700
373110
353000
603000
3000
11000
1300
```

840
670
4800
168000
0
706
746
545
806
513
479
748
1130
756
636
381
650
407
452
27979
7741
1480
2679
2448
2300
1200
0
294000
84400
0
105000
319000
0
178
];

CASRemovalwo=[
0.940746055
0.92
0.903238095
0.93373913
0.87
0.895164018

0.88
0.919313525
0.992370464
0.954556213
0.91547619
0.999292035
0.990441176
0.960294118
0.64612326
0.95821727
0.93766347
0.830985915
0.883468835
0.798913043
0.989189189
0.850404313
0.983118971
0.838709677
0.935457006
0.987173913
0
0.988343949
0.960761905
0.940634006
0.991997319
0.990963173
0.923945409
0.971929825
0.991837161
0.966788991
0.843362832
0.958068783
0.928144654
0.992473262
0.896307692
0.955036855
0.996880531
0.995223097
0.97965412
0
0.918088737
0.98
0.986486486
0.992534528

```
0.991830065
0.669090909
0.72
0.76
0.631578947
0.632575758
0.993945815
0.998580889
0.999
0.999
0.838461538
0.999
0.166666667
0.82
0.970769231
0
0.98
];


MBRmedianremvolwo=[
0.982896928
0.946083418
0.982197355
0.968421053
0.990225564
0.983082707
0
0.999943141
0.985135135
0.991787981
0.971813725
0.992
0.995
   ];

Ultramedianremovalwo=[
0.076923077
0.076923077
   ];
Nanoremovalwo=[
0.99981203
0.99981203
   ];
```

```
MicroMedianremovalwo=[
0.55
0.55
  ];

xaxisnumbers=[1,2,3,4,5];
Secondaryin=bootstrp(10000,@mean,Secondaryinwo);
CASRemoval=bootstrp(10000,@mean,CASRemovalwo);
MBRmedianremvol=bootstrp(10000,@mean,MBRmedianremvolwo);
Ultramedianremoval=bootstrp(10000,@mean,Ultramedianremovalwo)
Nanoremoval=bootstrp(10000,@mean,Nanoremovalwo);
MicroMedianremoval=bootstrp(10000,@mean,MicroMedianremovalwo);

figure('color',[1 1 1]);
hold on;
for i=1:10000;
SecondaryEffluentmedian(i)=Secondaryin(i)*(1-CASRemoval(i));
MBReffmedian(i)=SecondaryEffluentmedian(i)*(1-MBRmedianremvol(i));
Ultraeffmedian(i)=SecondaryEffluentmedian(i)*(1-Ultramedianremoval(i));
Nanomedian(i)=SecondaryEffluentmedian(i)*(1-Nanoremoval(i))
Microeffmedian(i)=SecondaryEffluentmedian(i)*(1-MicroMedianremoval(i));
end;

d=[SecondaryEffluentmedian;MBReffmedian;Ultraeffmedian;Nanomedian;Microeffmedi
an];
d2=d'; %tranpose matrix
a=boxplot(d2,'Label',{'WWTP','MBR','UF','NF','MF'},'color','k');

set(a(7,:),'Visible','off'); %Turn outliers off
ylabel('Effluent Concentration (ng/L)');
set(gca,'yScale','log');
dpi=600;
set(gca,'box','off')
% Turn offscientific notation
%numericticks=get(gca,'yTick'); % get the values of the ticks
%fullticks = num2str(numericticks,'%7.0f'); % save as strings
%set(gca,'yTickLabel',fullticks); % set the text labels to force
%ylim([1 10000]) %Set y axis limits


%
%
%
```

```
title('Ibuprofen (CAS WWTP)',  'FontWeight','bold');
SecondaryEffluentmax=max(SecondaryEffluentmedian);
MBReffmax=max(MBReffmedian);
Ultraeffmax=max(Ultraeffmedian);
Nanoeffmax=max(Nanomedian);
Microeffmax=max(Microeffmedian);


maxconc=[SecondaryEffluentmax, MBReffmax, Ultraeffmax, Nanoeffmax,
Microeffmax];
scatter (xaxisnumbers,maxconc,'+','k');
hold off;
figure('color',[1 1 1]);
hist(Secondaryin,50);
%set(gca,'yScale','log');
ylabel('Count');
xlabel('Concentration (ng/L)');
title('Histogram - CAS Influent',  'FontWeight','bold');

%Start RQ Plot

hold on;
figure('color',[1 1 1]);
x = [0 6];
y = [1 1];
plot(x,y,'--','color','k')

hold on;
h = [0 6];
t = [.1 .1];
plot(h,t,'--','color','k')
hold on
for i=1:10000;
RQCAS(i)=SecondaryEffluentmedian(i)/PNEC;
RQMBR(i)=MBReffmedian(i)/PNEC;
RQUltra(i)=Ultraeffmedian(i)/PNEC;
RQnano(i)=Nanomedian(i)/PNEC;
RQMicro(i)=Microeffmedian(i)/PNEC;

end
hold on;
b=[RQCAS;RQMBR;RQUltra;RQnano;RQMicro];
b2=b';
```

```
z=boxplot(b2,'Label',{'WWTP','MBR','UF','NF','MF'},'color','k','whisker',1.5);

set(z(7,:),'Visible','off');
ylabel('Effluent Risk Quotient');
set(gca,'yScale','log');
%
%
%
title('Ibuprofen (CAS WWTP)',  'FontWeight','bold');
dpi=600;
RQSecondaryEffluentmax=max(RQCAS);
RQMBReffmax=max(RQMBR);
RQUltraeffmax=max(RQUltra);
RQNanoeffmax=max(RQnano);
RQMicroeffmax=max(RQMicro);

maxRQ=[RQSecondaryEffluentmax, RQMBReffmax, RQUltraeffmax, RQNanoeffmax,
RQMicroeffmax];
scatter (xaxisnumbers,maxRQ,'+','k');

set(gca,'box','off')
hold off;

RQCAS1max=max(RQCAS);
RQMBR1max=max(RQMBR);
RQUltra1max=max(RQUltra);
RQNano1max=max(RQnano);
RQMicro1max=max(RQMicro);
SecondaryEffluent1max=max(SecondaryEffluentmedian);
MBReff1max=max(MBReffmedian);
Ultraeff1max=max(Ultraeffmedian);
Nanoeff1max=max(Nanomedian);
Microeff1max=max(Microeffmedian);


RQCAS1=median(RQCAS);
RQMBR1=median(RQMBR);
RQUltra1=median(RQUltra);
RQNano1=median(RQnano);
RQMicro1=median(RQMicro);
SecondaryEffluent1=median(SecondaryEffluentmedian);
MBReff1=median(MBReffmedian);
Ultraeff1=median(Ultraeffmedian);
```

```
Nanoeff1=median(Nanomedian);
Microeff1=median(Microeffmedian);

display(RQCAS1max);
display(RQCAS1);
display(RQMBR1max);
display(RQMBR1);
display(RQUltra1max);
display(RQUltra1);
display(RQNano1max);
display(RQNano1);
display(RQMicro1max);
display(RQMicro1);
display(SecondaryEffluent1max);
display(SecondaryEffluent1);
display(MBReff1max);
display(MBReff1);
display(Ultraeff1max);
display(Ultraeff1);
display(Nanoeff1max);
display(Nanoeff1);
display(Microeff1max);
display(Microeff1);

output=[RQCAS1max;
 RQUltra1max;
 RQMBR1max;
 RQMicro1max;
 RQNano1max;
RQCAS1;
RQUltra1;
RQMBR1;
RQMicro1;
RQNano1;
SecondaryEffluent1max;
Ultraeff1max;
MBReff1max;
Microeff1max;
Nanoeff1max;
SecondaryEffluent1;
Ultraeff1;
MBReff1;
Microeff1;
Nanoeff1]
```

CURRICULUM VITAE

Benjamin D. Blair

EDUCATION

Master of Science in Business Education – Online Education Emphasis, University of Wisconsin at Whitewater, 2005 – 2006

Bachelor of Business Administration in Finance, University of Wisconsin at Whitewater, 2000-2004

ARTICLES

Blair, B., Crago, J., Hedman, C., Klaper, R., Pharmaceuticals and Personal Care Products Found in the Great Lakes above Concentrations of Environmental Concern, Chemosphere, 2013; 93:2116-2123

Blair, B., Crago, J., Hedman, C., Treguer, R., Magruder, C., Royer, S., Klaper, R., Evaluation of a Model for the Removal of Pharmaceuticals, Personal Care Products, and Hormones from Wastewater, Science of the Total Environment, 2013;444C:515-521

Treguer, R., Blair, B., Klaper, R., Royer, S., Magruder, C., Evaluation of Actiflo® Carb Process for the Combined Removal of Trace Organic Compounds and Phosphorous during Wastewater Tertiary Treatment, Proceedings of the Water Environment Federation's Annual Technical Exhibition and Conference, 2012

TEACHING EXPERIENCE

Assistant Professor, Business Finance course (Online), Herzing University, Department of Management and Accounting, 2009

Assistant Professor, Business Decision Making course (Online), Herzing University, Department of Accounting, 2009

Lecturer, Business Finance course (Instructed both online and in-class), University of Wisconsin - Whitewater, College of Business and Economics – Department of Finance and Business Law, 2006 – 2007, 2009

Lecturer, Personal Financial Planning course, University of Wisconsin - Whitewater, College of Business and Economics –Department of Finance and Business Law, 2006 – 2007

Lecturer, Consumer Education course, University of Wisconsin - Whitewater, College of Business and Economics – Department of Information Technology and Business Education, 2006 – 2007

PRESENTATIONS (PRESENTER UNDERLINED)

Blair, B., Klaper, R., Kehl, J., Assessing the Change in Net Environmental Benefit from Water Quality Trading. Presented at the American Water Research Association Conference, March 25, 2013

Treguer, R., Blair, B., Klaper, R., Royer, S., Magruder, C., Evaluation of Actiflo® Carb Process for the Combined Removal of Trace Organic Compounds and Phosphorous during Wastewater Tertiary Treatment. Presented at the Water Environment Federation's Annual Technical Exhibition and Conference October 3, 2012

Blair, B., Hedman, C., Crago, J., Magruder, C., Royer, S., Treguer, R., Klaper, R., Patterns of Removal of Pharmaceutical and Personal Care Products from Wastewater Treatment and their Fate in Lake Michigan: Choosing Model Chemicals for Monitoring. Presented at the National Society of Toxicology and Chemistry Conference, November 16, 2011

Klaper, R., Blair, B., Hedman, C., Crago, J., Magruder, C., Royer, S., Treguer, R., Removal of Pharmaceutical and Personal Care Products from a Milwaukee, WI, Wastewater Treatment Facility. Presented at the Midwest Society of Toxicology and Chemistry Conference, March 24, 2011

POSTERS (PRESENTER UNDERLINED)

Blair, B., Crago, J., Hedman, C., Klaper, R., Pharmaceuticals and Personal Care Products Found in Lake Michigan above Concentrations of Environmental Concern. Poster presented at the International Association for Great Lakes Research Conference, June 5, 2013

Blair, B., Hedman, C., Crago, J., Magruder, C., Royer, S., Treguer, R., Klaper, R., Using Estimated Biodegradation Rates to Predict the Removal of Emerging Contaminants during Wastewater Treatment. Poster presented at the American Water Research Association Conference, June 26, 2012

RESEARCH EXPERIENCE

Visiting Scholar, University of Colorado – Denver, School of Public Affairs, 2013 – Present

Supervisors: Chris Weible, Ph.D., Tanya Heikkila, Ph.D.

-Explore how the characteristics of coalitions and their interactions have influenced changes in hydraulic fracturing policy or regulations and what actions

might lead to common ground and policy change in the future across different venues and scales

Research Assistant, University of Wisconsin – Milwaukee, 2010 – Present
Supervisor: Rebecca Klaper, Ph.D.
-Oversaw data collection and analysis of pharmaceutical and personal care products in wastewater and the environment
- Model policy implications of emerging contaminants and assess environmental impacts of nutrient reduction policies

Research Assistant, University of Wisconsin - Whitewater, College of Business and Economics Online, 2005 - 2006
Supervisor: Robert Schramm, Ph.D.
-Duties included oversight of technology used in the College of Business and Economics Online and assisting with the creation of online courses

WORK EXPERIENCE

Herzing University, Milwaukee, WI, March 2009 – September 2009
Curriculum Manager in Online Education Division / Assistant Professor

- Supervised the content development of 70 online courses using Blackboard
- Instructed Business Finance and Business Decision Making

University of Wisconsin – Whitewater, Whitewater, WI, August 2006 – May 2009
Lecturer

- Instructed and developed the online course for Business Finance
- Instructed Personal Financial Planning, Consumer Education, and Business Finance

HUSCO International, Waukesha, WI, May 2008 – September 2008
Engineer Intern – Summer

- General design and testing of hydraulic valves and components
- Created technical manuals and literature for customers and internal use

University of Wisconsin – Whitewater, Whitewater, WI, August 2005 – January 2007
Technology Coordinator / Associate Information Processing Consultant / Graduate Assistant

- Assisted faculty with technology and educational training for online education using D2L
- Created and managed content for online courses in the College of Business and Economics