

# Contaminants of Emerging Concern in the San Francisco Estuary: Alkylphenol Ethoxylates

## Final Report

By

Susan Klosterhaus

Rachel M. Allen

Jay Davis

The Regional Monitoring Program  
for Water Quality in the San Francisco Estuary



**SAN FRANCISCO ESTUARY INSTITUTE**

4911 Central Avenue, Richmond, CA 94804

p: 510-746-7334 (SFEI), f: 510-746-7300, [www.sfei.org](http://www.sfei.org)

This report should be cited as:  
Klosterhaus, S., R.M. Allen, and J. Davis. 2012. Contaminants of Emerging Concern in the San Francisco Estuary: Alkylphenol Ethoxylates. A Report of the Regional Monitoring Program for Water Quality in the San Francisco Estuary. SFEI Contribution #657. Final Report. San Francisco Estuary Institute, Richmond, CA.

## I. Use and Production

Alkylphenol ethoxylates (APEs; Figure 1) are nonionic surfactants that have been used since the 1940s as detergents and emulsifiers in domestic, industrial, and institutional applications, including paper production, leather and textile processing, and cleaning products. APEs have also been used as an adjuvant in some pesticide formulations (Siemerling et al., 2008). In 1997 (the most recent estimate available), it was estimated that approximately 80% of the APEs in use were nonylphenol ethoxylates (NPEs) and 20% were octylphenol ethoxylates (OPEs) (Chiu et al., 2010). The majority of use was in industrial applications (~55%), followed by industrial and institutional cleaners and detergents (~30%) and household and personal care products (~15%).

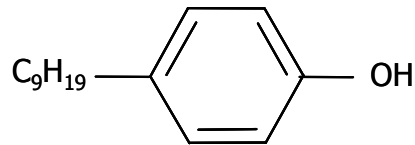
The use of APEs has been restricted in many countries due to concerns about the environmental effects of APE breakdown products, particularly nonylphenol (NP). Switzerland banned the use of NPEs in domestic laundry detergents in 1986. Several other countries in the European Union (EU) also banned or restricted the use of NPEs, NP, and/or APEs in the 1990s and early 2000s. In 2000, Canada added NP and NPEs to their Toxic Substances List and required users of these compounds to develop and implement Pollution Prevention Plans to reduce concentrations in the environment. In 2005, the EU restricted the use of NP and NPEs in a variety of products.

There are currently no regulatory restrictions for the use of APEs in the United States (US), though some US companies have voluntarily phased out their use in their products. Procter and Gamble eliminated the intentional use of NP and NPEs in consumer products for the majority of their usage volume in the 1990s. In 2006, Wal-Mart announced that it would phase-out products that contain NPEs and JohnsonDiversey/SC Johnson reportedly stopped using APEs. The Textile Rental Services Association, which represents 98% of the industrial laundry facilities in the US, recently committed to voluntarily phase out the use of NPEs in industrial liquid detergents by the end of 2013 and industrial powder detergents by the end of 2014. In 2010, the USEPA released a Chemical Action Plan for NP and NPEs, which outlines steps EPA intends to take to address the risks associated with these chemicals (US EPA, 2010). These steps include the support and encouragement of the ongoing voluntary phase-out of NPEs in industrial laundry detergents and the consideration of future regulatory action. In June 2010 the Industrial/Institutional laundry industry pledged to eliminate the use of NPE surfactants. The current deadline for complete phase-out in all liquid detergents is December 31, 2013, and in all powder detergents, December 31, 2014 (US EPA, 2012).

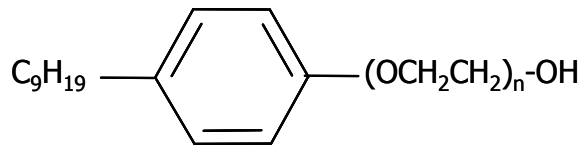
The US demand for NP, which is used to produce NPEs, was estimated to be 380 million pounds in 2010 (USEPA, 2010). This is within the range for the reported production volume reported under the EPA Inventory Update Rule (IUR) in 2006, which ranged from approximately 100 to 500 million pounds. US and Canadian consumption of NPE surfactants has been estimated between 300 and 400 million pounds per year (USEPA, 2010). US production volumes for branched NP did not change between 1986 and 2006 (100-500 million pounds), but the reported production volumes for linear NP, which are not used in detergents, have decreased during that time (100-500 million pounds in 1986 to <500,000 pounds in 2006). Because information on production and use of NP and NPEs is limited, it is not clear if the overall

consumption of NPE surfactants in the US has decreased as a result of the NPE phase-outs. Production volumes were not available for OP or OPEs.

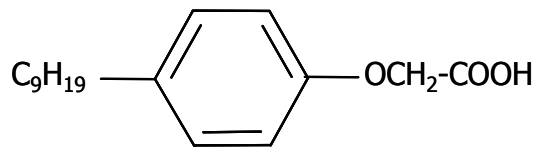
**Figure 1: Chemical structures of nonylphenol and related products**



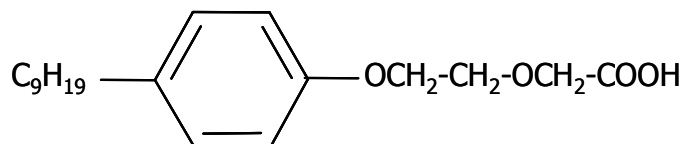
Nonylphenol (NP)



Nonylphenol ethoxylates (NPE)



Nonylphenoxy carboxylate (NP1EC)



Nonylphenoxy ethoxy carboxylate (NP2EC)

A variety of substitutes are available for NPEs, though information on the extent of their use in the US is not readily available (Toxicology Environmental Consulting Ltd., 2002; USEPA, 2010). Alcohol ethoxylates are the most common replacement and are considered to be environmentally safer because they are readily biodegradable and their biodegradation intermediates are less toxic than the parent compounds. The USEPA has recently completed a draft alternatives assessment for NPEs (<http://www.epa.gov/dfe/pubs/projects/npe/index.htm>).

## II. Fate in Wastewater Treatment Plants (WWTPs)

In WWTPs, APEs are biodegraded to form NP, octylphenol (OP), mono-, di-, and tri-ethoxylates (e.g. NP1Es, NP2Es, NP3Es), and alkylphenoxy carboxylates (APECs). Most studies of the fate of APEs in WWTPs have focused on NPEs and NP, due to their extensive use, frequency of detection in WWTPs, and toxicity. Removal efficiencies depend on the WWTP location and type of treatment, and have been reported to vary between 50 and 99% for APEs in general (Staples et al., 2001), 47 to 99% for NPEs (Ying, 2006), and 9 to 94% for NP specifically (Soares et al., 2008). In US WWTPs, removal efficiencies are often higher and have been estimated to range from 93 to 99% for NPEs and 72 to 97% for OPEs (Ying et al., 2002; Loyo-Rosales et al., 2007). Degradation of APEs during wastewater treatment complicates removal efficiency estimates because the breakdown products may be present in higher concentrations in the effluent than influent (Ahel et al., 1994; Loyo-Rosales et al., 2007). Studies have indicated that removal of NP from wastewater can be enhanced by adding activated carbon filters, UV treatment, and ozonation to existing treatment processes (Soares et al., 2008). To more efficiently remove APECs from wastewater, Chiu et al. (2010) suggest improving biological treatment processes and optimizing their operating conditions (e.g., solid retention time) as part of tertiary treatment.

Sorption to sludge, as a consequence of high affinities for organic matter, and biological degradation are the major removal mechanisms for APEs during wastewater treatment. The fate of APEs has been most extensively studied in Europe, where NP concentrations in sludge have been extremely variable (0.02 to 2530  $\mu\text{g/g}$  dry weight; review in Soares et al., 2008). Few studies have been conducted in the US. In 2001, La Guardia et al., (2001) investigated concentrations of APE degradation products (NP, OP, NP1E, NP2E) in biosolids from 11 US WWTPs, including anaerobically digested sludge samples from five California WWTPs. Concentrations were lowest in composted biosolids (6 to 176  $\mu\text{g/g}$  dry weight, sum total) and highest in the anaerobically digested samples (721 to 981  $\mu\text{g/g}$  dry weight). In the majority of samples, NP was the most abundant compound detected (>84% of the total), followed by NP1E and NP2E, and then OP. Anaerobically digested sludge samples from New York WWTPs contained NP concentrations (range 1100 to 1800, mean 1500  $\mu\text{g/g}$ ) that were approximately

twice as high as those in the California WWTPs from the La Guardia et al. (2001) study (Pryor et al., 2002), suggesting concentrations may also be widely variable in the US.

Despite high removal efficiencies under some treatment conditions, APEs and their degradation products are commonly detected in WWTP effluent. Studies of wastewater effluents in Europe, Asia, and Canada have reported concentrations of NP ranging from below detection to 343  $\mu\text{g/L}$ , with most ranging from 0.2 to 2  $\mu\text{g/L}$  (Soares et al., 2008, Ying et al., 2002). In the US, NP concentrations in municipal effluent have generally ranged from 0.2 to 37  $\mu\text{g/L}$  (Hale et al 2000; Ying et al. 2002; Loyo-Rosales et al. 2007; Lubliner et al., 2010). Longer chain APEs, such as NP1E and NP2E, have been analyzed in a few studies and are often present in effluents at concentrations higher than their respective alkylphenols (e.g., NP) due to their higher water solubility (Loyo-Rosales et al., 2007; Glassmeyer et al., 2005). OP and OPEs, which are used in lower volumes and are less frequently analyzed, are typically present in concentrations one order of magnitude lower than NP and NPEs (Loyo-Rosales et al., 2007). APECs, which have been investigated less frequently, dominate the metabolite profile in effluents when analyzed (Loyo-Rosales et al., 2007; Chiu et al., 2010). NP has also been detected in wastewater effluents from industrial facilities and shipyards that have received little or no pre-discharge treatment (Hale et al., 2000).

### **III. Fate and Occurrence in the Environment**

#### **Surface Waters**

Discharges from WWTPs have been identified as the major source of APEs and their degradation products to aquatic environments, though atmospheric deposition (Fries and Puttmann, 2004), urban runoff (King County, 2007; Bjorkland et al., 2009), and direct discharge, such as application of pesticides to aquatic vegetation (Siemering et al., 2008) may also be important pathways. Studies suggest that once in the water column, these compounds can be removed via particle sorption and settling (Ferguson et al., 2001) and undergo photolysis (Ahel et al., 1994), volatilization (Dachs et al., 1999), and microbial degradation (Montgomery-Brown and Reinhard, 2003). It is well established that long chained APEs are readily biodegradable, and that the majority of this degradation occurs during wastewater treatment, but the ultimate environmental fate of the degradation products is not well understood and the evidence for further degradation of these compounds in aquatic environments is not consistent (Montgomery-Brown and Reinhard, 2003). Studies have reported a wide range of degradation rates, which are dependent on the test system and experimental conditions and are complicated by the formation of various metabolites (Ying et al., 2002). The half-lives for NPEs and other degradation products in water have been estimated as varying from 4 to 58 days.

Since the 1980s, numerous studies have analyzed APEs and their degradation products in surface waters around the world (Chiu et al., 2010; Loyo-Rosales et al., 2010; Bennie, 1999; Soares et al. 2008). Most of these studies have been conducted in Europe and Asia, with only a small number of studies reporting APE concentrations in the US and Canada. Differences in the compounds analyzed and the analytical methods used in each study complicate comparisons of occurrence data among locations. For surface waters in particular, studies report either dissolved

phase or whole water (i.e., dissolved plus particulate) concentrations, further complicating comparisons.

NP, NP1E, and NP2E were the compounds most frequently analyzed and detected in surface waters, with concentrations in the US generally ranging from below detection to 3 µg/L. The exception was a 1999-2000 US stream survey which detected maximum concentrations of 40, 20, and 9 µg/L for NP, NP1E, and NP2E, respectively, though this study targeted wastewater-receiving streams in highly urbanized areas (Kolpin et al., 2002). Concentrations of OP, OP1E, and OP2E are generally at least an order of magnitude lower and have ranged from below detection to 0.3 µg/L in US surface waters. The US stream survey by Kolpin et al. (2002) was again an exception, with maximum concentrations of 2 and 1 µg/L for OP1E and OP2E, respectively (OP was not analyzed). It should be noted that these studies have typically targeted surface waters expected to be highly impacted by wastewater effluent. NP was recently detected at a maximum concentration of 0.3 µg/L in marine waters of Puget Sound, which may be more representative of urban estuarine conditions (King County, 2007).

Due to their relatively high water solubility, APECs are the dominant degradation product in surface waters when measured, reflecting the pattern also observed in effluents. In the few US studies that have analyzed APECs in surface waters, NP1EC and NP2EC concentrations ranged from below detection to 31 µg/L.

Three recent studies have analyzed APEs and related compounds in California surface waters. In the effluent-dominated Santa Ana River, concentrations of APs (individual NP and OP data not reported) and APECs ranged from 0.05 to 0.4 µg/L and 1 to 14.8 µg/L, respectively, though APEs were not detected (Lin et al., 2006). In Southern California in 2006 and 2007, the Southern California Coastal Water Research Project (SCCWRP) detected NP and OP in bottom water near ocean wastewater outfalls at concentrations in the low ng/L range (unpublished data). Lastly, NP was recently detected in Morro Bay by Dr. Lars Tomanek's research group at California Polytechnic State University at concentrations ranging from 0.1 to 0.9 µg/L, with the majority of samples at or near the detection limit (0.1 µg/L). However, NP concentrations in water samples from a Morro Bay tributary receiving municipal wastewater effluent were 1.3 and 3.4 µg/L (all unpublished data).

## **Surface Sediments**

Compared to APEs and APECs, which are water soluble, APs and other APE degradation products are relatively hydrophobic ( $\log K_{ow}$  3.9-4.5) and therefore primarily adsorb to organic matter and mineral components in suspended and bed sediments. Researchers generally agree that the majority of APE biodegradation occurs during wastewater treatment, that formation of alkylphenols occurs only under anaerobic conditions, and that the degradation products are stable and persistent compared to their precursors (Ying, 2006). However, there is disagreement regarding the extent of biodegradation that may occur in the environment (Montgomery-Brown and Reinhard, 2003). It has been reported that aerobic conditions facilitate further biotransformation of APE degradation products more readily than anaerobic conditions, but this is an area that is not well understood (Ying, 2006). A half-life of more than 60 years has been estimated for total NPEs, including NP, in marine sediments and it has been suggested that once

these compounds are incorporated into marine sediments, degradation does not occur very rapidly, if at all (Shang et al., 1999; David et al., 2009)

Similar to studies of occurrence in surface waters, the majority of studies of APE occurrence in sediments have been conducted in Asia and Europe and most have been conducted in freshwater environments. In US marine sediments, concentrations of NP, NP1E, and NP2E have ranged from 7 to 13,700, 12 to 13,300, and 3 to 3,600 ng/g dry weight, respectively, with the highest concentrations reported in the highly sewage-impacted Jamaica Bay, NY (reviewed in David et al., 2009). Estuarine marshes in Savannah, GA contained among the lowest concentrations of NP (< 0.3-18 ng/g dry weight; Senthil Kumar et al., 2008). In US rivers, maximum concentrations of NP and NPEs were observed in sediments collected near an oil company in Detroit (60,000 ng/g), a federal aerospace research facility (14,000 ng/g), and sewage treatment plants (12,400 ng/g); however, most concentrations were below 1000 ng/g (Naylor et al., 1992; Hale et al., 2000; Kannan et al., 2001; Rice et al., 2003). Similar to surface waters, concentrations of OP and OPEs were generally detected at concentrations at least an order of magnitude lower, which reflects the higher volume use of NPEs.

A few studies have investigated APEs concentrations in sediments collected in California coastal waters. In the Southern California Bight, sediments were collected in 2003 near ocean wastewater outfalls and a reference site (Schlenk et al., 2005). The Orange County Sanitation District sediments had the highest NP (3,200 ng/g) and NPE concentrations (330–3,900 ng/g), while concentrations at the other sites were consistently lower (NP 122 to 198 ng/g and NPEs 2 to 92 ng/g). In a similar study conducted by SCCWRP in 2006 and 2007, NP was detected in most samples of sediment and ranged from ~80 to 800 ng/g dry weight (unpublished data). NP was also recently detected in sediments collected from Morro Bay by the Tomanek research group at California Polytechnic State University. Concentrations ranged from below detection to 158 ng/g dry weight, with most concentrations between 40 and 60 ng/g dry weight (unpublished data).

## **Wildlife and Humans**

APE degradation products have been observed to accumulate in wildlife in marine and freshwater environments around the world (Rice et al., 2003; Soares et al., 2008; David et al., 2009). Most studies have been conducted in Europe and Asia with only a very small number conducted in the US. These studies have primarily focused on NP and OP because of toxicity concerns and their lipophilic and persistent nature, though accumulation of NP1E, NP2E, OP1E, OP2E, and APECs has also been reported. Among the highest concentrations in wildlife ever reported were in a freshwater river receiving secondary wastewater effluent in Switzerland (Ahel et al., 1993). In this study, concentrations in algae (maximum 38000, 80000, and 28000 ng/g dry weight for NP, NP1E, and NP2E, respectively) were higher than those in fish (maximum 2000, 7000, and 3000 ng/g dry weight for NP, NP1E, and NP2E, respectively). These results are consistent with other studies indicating that these compounds do not biomagnify in foodwebs. Studies have indicated that NP and OP can be rapidly metabolized in fish to polar byproducts so lower concentrations compared to other degradation products and other types of organisms have been observed (Arukwe et al., 2000; Ferrara et al., 2008; Rice et al., 2003). In US rivers, NP and NPEs have been frequently detected in fish at concentrations as high as ~2000 ng/g wet weight



(Rice et al., 2003 and references therein). Regarding accumulation in invertebrates, marine bivalves worldwide have typically contained NP concentrations ranging from 2 to 5000 ng/g dry weight, though the majority were less than 700 ng/g (David et al., 2009). A study of benthic invertebrates in Lake Ontario indicated that in addition to NP, significant accumulation of APECs may also occur (Mayer et al., 2007). In the only available study conducted in a US estuary, oysters in Savannah, GA contained concentrations of 2 to 20 and 2 to 10 ng/g dry weight for NP and OP, respectively (Senthil Kumar et al., 2008). NP and OP have also been detected in human urine samples as high as approximately 2000 ng/L (Calafat et al., 2005; Calafat et al., 2008). To our knowledge, APEs in bird eggs have not been reported in the peer-reviewed literature.

Two studies have investigated APEs concentrations in wildlife collected from California coastal waters. SCCWRP analyzed benthic fish livers collected near ocean wastewater discharges in Southern California in 2006 and 2007 and detected NP and OP in most samples (~80-100 ng/g wet weight). NP was also recently detected in wildlife collected from Morro Bay and other west coast estuaries by the Tomanek research group at California Polytechnic State University (unpublished data). Average concentrations in Morro Bay oysters and mussels were 204 and 290 ng/g wet weight, respectively. Average concentrations in small fish from all the estuaries studied ranged from 105 to 220 ng/g wet weight. Average NP concentrations in liver samples from Morro Bay fish, seabird, and marine mammals were much higher, ranging from ~300 to 5000 ng/g wet weight.

#### **IV. Toxicity**

Exposure to APE degradation products can result in acute and/or chronic toxicity to aquatic organisms, though the potential for estrogenic effects at low  $\mu\text{g/L}$  concentrations is the primary concern (reviewed in Servos, 1999). Studies have demonstrated that APEs compete for the binding site of the natural estrogen receptor in vertebrates and can elicit a variety of responses, including induction of vitellogenin, an egg yolk precursor protein normally only expressed in females. Estrogenic responses that have been observed in fish and invertebrates exposed to APE degradation products include decreased growth and weight, suppressed gonad development, altered sex hormone concentrations, altered sex ratios, development of mixed secondary sex characteristics, and larval deformities. OP is the most estrogenic APE degradation product but has been estimated to be 100 to 1,000 times less potent than  $17\beta$ -estradiol, the natural vertebrate estrogen, and is thus considered weakly estrogenic (Montgomery-Brown and Reinhard, 2003). NP is approximately 25% less estrogenic than OP, with NP1E and NP2E only slightly less estrogenic than NP. Because of the similarity in mode of action, it is considered that estrogenic effects of APEs and their degradation products are at least additive, and can act in conjunction with natural estrogens and other synthetic compounds (Sumpter and Jobling, 1995; Brian et al., 2007). It has also more recently been established that NP has androgenic activity in vertebrates (Lee et al., 2003).

Acute, chronic, and endocrine disruption effects on aquatic organisms have generally not been observed at water concentrations below 1  $\mu\text{g/L}$  for any of the APE degradation products and most effects have been observed at much higher concentrations (~10-3000  $\mu\text{g/L}$ ; Servos,

1999; Langston et al., 2005). An exception to this was the observation of reduced settlement in barnacle larvae at 0.06 µg/L of NP (Billinghamurst et al., 1998). Induction of vitellogenin in fish in a long-term exposure to 1 µg/L of NP has also been reported (Ackermann et al., 2002). Among marine organisms, gastropods and bivalves appear to be the most sensitive to the effects of NP and OP, followed by fish, other invertebrates, and then algae (Staples et al., 2004; Van Miller and Staples, 2005). In general, decreasing ethoxylate chain length has been correlated with increasing toxicity. The relative toxicities for NPEs have been estimated to be 1, 0.5, 0.005 and 0.005 for NP, NP1EO and NP2EO, NP3-17E, and NP1EC and NP2EC, respectively (Servos et al., 2003).

Toxicity concerns have led to the development of water and sediment quality guidelines for the protection of aquatic life. In 2005, the USEPA established water quality criteria of 7.0 µg/L (acute) and 1.7 µg/L (chronic) for NP in saltwater and 28 µg/L (acute) and 6.6 µg/L (chronic) for NP in freshwater (USEPA, 2005). In 2001, Environment Canada developed interim guidelines of 1.4 and 1.0 µg/g dry weight for NP and its ethoxylates in freshwater and marine/estuarine sediments, respectively (Environment Canada, 2007). In 2002, they developed guidelines of 1.0 and 0.7 µg/L for NP and its ethoxylates in freshwater and marine waters, respectively. To account for exposure to mixtures of these compounds, Environment Canada developed a toxic equivalency (TEQ) approach so that the environmental concentrations of NP/OP, NPEs/OPEs and NPECs/OPECs can be expressed in terms of NP equivalents, and the sum of these concentrations can then be compared against the guidelines. The European Union has established Environmental Quality Standards of 0.3 µg/L (annual average) and 2.0 µg/L (maximum allowable concentration) for NP in all surface waters, and 0.1 µg/L and 0.01 µg/L (annual average only) for OP in inland and other surface waters, respectively (European Parliament and Council, 2008).

## **V. Potential Impact in San Francisco Bay**

### **Bay Data**

As part of a 2002 and 2003 pilot study, the RMP analyzed NP in sediment, water, and bivalve samples collected as part of the annual Status and Trends monitoring. NP was not detected in sediments (detection limit 0.5 ng/g dry weight). Concentrations in surface waters ranged from 0.005 to 0.073 µg/L, with the highest concentrations observed in Central Bay. NP was rarely detected in bivalves and when detected concentrations were highly variable. In 2002, the highest concentration was observed in mussels at the reference site near Bodega Bay (150 ng/g dry weight). In 2003, NP was detected at three stations in concentrations ranging from 316 to 917 ng/g dry weight with the highest values detected in resident clams from the two river stations located on the San Joaquin and Sacramento rivers.

In 2002 and 2004, Double-crested cormorant eggs were collected from three locations in the Bay (Wheeler Island, Richmond Bridge, and the Don Edwards National Wildlife Refuge) as part of a RMP pilot study. Composites (10 eggs/composite; 2 composites/site) were analyzed for a variety of chemical contaminants, including NP (Davis et al., 2006). One composite each from the Richmond Bridge and Don Edwards sites from 2002 were the only samples with detectable

NP concentrations (method reporting limit 10 ng/g wet weight). Concentrations of NP were 60 and 120 ng/g wet weight in the Richmond Bridge and Don Edwards composites, respectively.

In 2009, Dr. Lars Tomanek's research group analyzed NP in arrow gobies collected from West Coast estuaries, including fish from four locations in San Francisco Bay collected in 2006 (Diehl et al. 2012). The Bay sites were Point Isabel Regional Shoreline (near Berkeley, CA), Candlestick Point State Recreation Area (near San Francisco, CA), Martin Luther King Jr. Regional Shoreline (near inner Oakland Harbor), and Bird Island (near Foster City, CA). Concentrations of NP in the gobies ranged from 50-420 ng/g wet weight, with the highest concentrations observed in gobies at Bird Island (two composites; 200 and 420 ng/g wet weight).

In January 2010, whole water, sediment, and resident mussel samples were collected from five nearshore sites in the Bay (Central, South, and Lower South Bays) and analyzed for NP, OP, NP1EO, and NP2EO as part of a pilot study investigating contaminants of emerging concern.

- NP was detected in surface water from three sites at concentrations ranging from 0.035 to 0.073  $\mu\text{g/L}$  (detection limit 0.01  $\mu\text{g/L}$ ), similar to those collected from the Bay in 2002 and 2003. OP, NP1EO, and NP2EO were not detected in water samples (detection limits ranged from 0.001 to 0.023  $\mu\text{g/L}$ ).
- NP was consistently detected in sediments at concentrations ranging from 20 to 90 ng/g dry weight (mean  $50 \pm 30$  ng/g), with the highest concentration detected in the Lower South Bay. Higher concentrations in the 2010 samples compared to the 2002/2003 analysis may be the result of an increase in concentration over time or analytical method differences. Concentrations of NP1E and NP2E in sediments ranged from 4 to 40 ng/g (mean  $20 \pm 10$  ng/g) and <1 to 20 ng/g (mean  $9 \pm 7$  ng/g), respectively. OP was not detected in sediment samples (detection limits <0.3 ng/g).
- NP was detected in mussel tissue at two sites (both  $\sim 90$  ng/g wet weight in the Central and South Bays). At the Central Bay site where NP was detected (San Leandro Bay), concentrations of NP1EO and NP2EO in mussels were 40 and 200 ng/g, respectively. NP1EO and NP2EO were not detected in mussels at any other sites (maximum detection limits 30 and 3 ng/g, respectively) and OP was not detected at any site (maximum detection limit 0.2 ng/g). Laboratory blank contamination prevented low level quantification of these compounds at most sites.

A sediment porewater concentration for NP was estimated using the maximum sediment concentration detected in the Bay, an average Bay sediment organic carbon content ( $\sim 1.5\%$ ), and the organic carbon normalized sediment-water partitioning coefficients ( $K_{oc}$ ), recognizing that a number of factors such as fluctuating pH and salinity, and the organic carbon content and quality of sediments may influence these estimates and result in some variation and uncertainty. Using a  $K_{oc}$  of 141,254 (Sekela et al., 1999), the estimated maximum porewater concentration was 0.04  $\mu\text{g/L}$  for NP.

## **Potential Impact**

Concentrations of NP and its ethoxylates in San Francisco Bay are generally well below concentrations expected to elicit toxic effects in aquatic organisms. The maximum NP surface water concentration detected in the 2010 RMP pilot study (0.07 µg/L) was more than 20 times lower than the USEPA water quality criteria for NP (1.7 µg/L), more than 25 times lower than the maximum allowable concentration permitted by the EU (2.0 µg/L), and four times lower than the annual average concentration permitted in EU surface waters (0.3 µg/L). Using the TEQ approach developed by Environment Canada, the maximum concentrations of NP and its ethoxylates in Bay waters and sediments (0.07 µg/L and 0.12 µg/g dry weight, respectively) were an order of magnitude below the water and sediment quality guidelines developed by that agency to be protective of aquatic life. Maximum concentrations of NP in Bay surface waters were, however, comparable to the concentration observed to impact barnacle settlement in a laboratory study (0.06 µg/L) (Billinghurst et al., 1998). Concentrations of NP in Bay cormorant eggs were two to three orders of magnitude below concentrations observed to adversely impact Japanese quail embryos (Razia et al., 2006).

Though the concentrations observed to date at the nearshore sites studied generally indicate that APEs are not likely to impact Bay wildlife, water and sediment closer to wastewater or stormwater outfalls in the Bay may contain higher concentrations of APEs which may increase the likelihood of impacts. Among the nearshore sites sampled in 2010, only two (Cooley Landing and Foster City) were within 5 km of a WWTP outfall. Studies suggest that effects from APEs and their degradation products may be additive; thus organisms living in wastewater receiving waters may be the most susceptible to impacts, particularly since they are continuously exposed to the many estrogenic substances that have been identified in wastewater effluent. Few studies have investigated the potential for sub-lethal impacts on populations due to long-term exposures to low concentrations of APEs and their degradation products and these would be needed to thoroughly assess their risk to wildlife in the Bay.

## **VI. Key Information Gaps**

- More information is needed on the potential effects on Bay wildlife due to long-term exposure to APEs concentrations that are typically found in aquatic environments. As estrogenic compounds, APEs may contribute to the combined impact of the potential presence of several estrogenic compounds in the Bay.
- More information is needed on the concentrations of APEs and their degradation products in sediment and biota near Bay Area treatment plant outfalls, where exposures are anticipated to be highest.

## References

Ackermann GE, Schwaiger J, Negele RD, Fent K. 2002. Effects of long-term nonylphenol exposure on gonadal development and biomarkers of estrogenicity in juvenile rainbow trout (*Oncorhynchus mykiss*). *Aquatic Toxicol* 60:203–221.

Ahel M, McEvoy J, Giger W. 1993. Bioaccumulation of the lipophilic metabolites of nonionic surfactants in freshwater organisms. *Environ Poll* 79:243-248.

Ahel M, Giger W, Schaffner C. 1994. Behaviour of alkylphenol polyethoxylate surfactants in the aquatic environment-I. Occurrence and transformation in sewage treatment. *Water Res* 28:1131-42.

Ahel M, Scully FE, Hoigne J, Giger W. 1994. Photochemical degradation of nonylphenol and nonylphenol polyethoxylates in natural waters. *Chemosphere* 28:1361-1368.

Arukwe AR, Thibaut R, Ingebrigtsen K, Celius T, Goksøyr A, Cravedi J. 2000. In vivo and in vitro metabolism and organ distribution of nonylphenol in Atlantic salmon (*Salmo salar*). *Aquat Toxicol* 49:289–304.

Bennie DT. 1999. Review of the environmental occurrence of alkylphenols and alkylphenol ethoxylates. *Water Qual Res J Can* 34:79-122.

Billinghurst Z, Clare AS, Fileman T, Mcevoy J, Readman J, Depledge MH. 1998. Inhibition of barnacle settlement by the environmental oestrogen 4-nonylphenol and the natural oestrogen 17 beta oestradiol. *Mar Poll Bull* 36:833–839.

Bjorkland K, Palm Cousins A, Strömvall A, Malmqvist P. 2009. Phthalates and nonylphenols in urban runoff: Occurrence, distribution and area emission factors. *Sci Tot Env* 407:4665-4672.

Brian JV, Harris CA, Scholze M, Kortenkamp A, Booy P, Lamoree M, Pojana G, Jonkers N, Marcomini A, Sumpter JP. 2007. Evidence of Estrogenic Mixture Effects on the Reproductive Performance of Fish. *Enviro. Sci Technol* 41:337-344.

Calafat AM, Kuklennyik Z, Reidy JA, Caudill SP, Ekong J, Needham LL. 2005. Urinary concentrations of bisphenol A and 4-nonylphenol in a human reference population. *Environ Health Perspect* 113:391-395.

Calafat AM, Ye X, Wong L, Reidy JA, Needham LL. 2008. Exposure of the U.S. population to bisphenol A and 4-tertiary-octylphenol: 2003–2004. *Environ Health Perspect* 116:39-44.

Chiu TY, Paterakis N, Cartmell E, Scrimshaw MD, Lester JN. 2010. A Critical Review of the Formation of Mono- and Dicarboxylated Metabolic Intermediates of Alkylphenol Polyethoxylates during Wastewater Treatment and Their Environmental Significance. *Critical Rev Environ Sci Technol* 40:199-238.

Dachs J, Van Ry DA, Eisenreich SJ. 1999. Occurrence of estrogenic nonylphenols in the urban and coastal atmosphere of the lower Hudson River estuary. *Environ Sci Technol* 33:2676-9.

David A, Fenet H, Gomez E. 2009. Alkylphenols in marine environments: Distribution monitoring strategies and detection considerations. *Mar Poll Bull* 58:953-960.

Davis JA, Hunt JA, Ross JRM, Melwani AR, Sedlak M, Adelsbach T, Crane D, Phillips L. 2006. Pollutant Concentrations in Eggs of Double-crested Cormorants from San Francisco Bay in 2002 and 2004: A Regional Monitoring Program Pilot Study. SFEI Contribution #434. San Francisco Estuary Institute, Oakland, CA.

Diehl J, Johnson SE, Xia K, West A, Tomanek L. 2012. The distribution of 4-nonylphenol in marine organisms of North American Pacific Coast estuaries. *Chemosphere* 87:490-497.

Environment Canada 2007. Canadian Water Quality Guidelines for the Protection of Aquatic Life. Summary Table, Update 7.1. <http://ceqg-rcqe.ccme.ca/>

European Parliament and Council. 2008. Directive 2008/105/EC. Official Journal of the European Union 348:84-97.

Ferguson PL, Iden CR, Brownawell BJ. 2001. Distribution and fate of neutral alkylphenol ethoxylate metabolites in a sewage-impacted urban estuary. *Environ Sci Technol* 35:2428-2435.

Ferrara F, Ademollo N, Delise M, Fabietti F, Funari E. 2008. Alkylphenols and their ethoxylates in seafood from the Tyrrhenian Sea. *Chemosphere* 72:1279-1285.

Glassmeyer ST, Furlong ET, Kolpin DW, Cahill JD, Zaugg SD, Werner SL, Meyer MT, Kryak DD. 2005. Transport of chemical and microbial compounds from known wastewater discharges: Potential for use as indicators of human fecal contamination. *Environ. Sci. Technol.* 39:5157-5169.

Hale RC, Smith CL, de Fur PO, Harvey E, Bush EO, La Guardia MJ, Vadas GG. 2000. Nonylphenols in sediments and effluents associated with diverse wastewater outfalls. *Environ Toxicol Chem* 19:946-52.

King County. 2007. Survey of endocrine disruptors in King Country surface waters. Prepared by Richard Jack and Deb Lester, Water and Land Resources Division. Seattle, Washington.

Kannan K, Kober JL, Kang Y, Masunaga S, Nakanishi J, Ostaszewski A, Giesy JP.

2001. Polychlorinated naphthalenes, biphenyls, dibenzo-p-dioxins, and dibenzofurans as well as polycyclic aromatic hydrocarbons and alkylphenols in sediment from the Detroit and Rouge Rivers, Michigan, USA. *Environ Toxicol Chem* 20:1878–89.

Kolpin DW, Furlong ET, Meyer MT, Thurman EM, Zaugg SD, Barber LB, Buxton HT. 2002. Pharmaceuticals, Hormones and Other Organic Wastewater Contaminants in US Streams, 1999-2000: A National Reconnaissance. *Environ Sci Technol* 36:1202-1211.

Langston WJ, Burt GR, Chesman BS, Vane CH. 2005. Partitioning, bioavailability and effects of oestrogens and xeno-oestrogens in the aquatic environment *J. Mar. Biol. Ass. U.K.*, 85:1-31.

Lubliner B Redding M, Ragsdale D. 2010. Pharmaceuticals and Personal Care Products in Municipal Wastewater and Their Removal by Nutrient Treatment Technologies. Washington State Department of Ecology, Olympia, WA. Publication Number 10-03-004. [www.ecy.wa.gov/biblio/1003004.html](http://www.ecy.wa.gov/biblio/1003004.html).

La Guardia MJ, Hale RC, Harvey E, Mainor TM. 2001. Alkylphenol ethoxylate degradation products in land-applied sewage sludge (biosolids). *Environ Sci Technol* 35:4798–804.

Lee HJ, Chattopadhyay S, Gong EY, Ahn RS, Lee K. 2003. Antiandrogenic effects of bisphenol A and nonylphenol on the function of androgen receptor. *Toxicol Sci* 75:40–6.

Loyo-Rosales JE, Rice CP, Torrents A. 2007. Fate of Octyl- and Nonylphenol Ethoxylates and Some Carboxylated Derivatives in Three American Wastewater Treatment Plants. *Environ Sci Technol* 41:6815-6821.

Lin AYC, Plumlee MH, Reinhard M. 2006. Natural attenuation of pharmaceuticals and alkylphenol polyethoxylate metabolites during river transport: Photochemical and biological transformation. *Environ Toxicol Chem* 25:1458-1464.

Mayer T, Bennie D, Rosa F, Rekas G, Palabrica V, Schachtschneider J. 2007. Occurrence of alkylphenolic substances in a Great Lakes coastal marsh, Cootes Paradise, ON, Canada. *Environ Poll* 147:683-690.

Montgomery-Brown J, Reinhard M. 2003. Occurrence and behavior of alkylphenol polyethoxylates in the environment. *Environ Eng Sci* 20:471-486.

Naylor CG, Mieure JP, Adams WJ, Weeks JA, Castaldi FJ, Ogle LD, Romano RR. 1992. Alkylphenol ethoxylates in the environment. *J Am Oil Chem Soc* 69:695–703.

Pryor SW, Hay AG, Walker LP. 2002. Nonylphenol in anaerobically digested sewage sludge from New York state. *Environ Sci Technol* 36:3678–82.

Razia S, Maegawa Y, Tamotsu S, Oishi T. 2006. Histological changes in immune and endocrine organs of quail embryos: exposure to estrogen and nonylphenol. *Ecotox Environ Safety* 65:364-71.

- Rice CP, Schmitz-Afonso I, Loyo-Rosales JE, Link E, Thoma R, Fay L, Altfater D, Camp MJ. 2003. Alkylphenol and alkylphenol-ethoxylates in carp, water, and sediment from the Cuyahoga River, Ohio. *Environ Sci Technol* 37:3747–54.
- Schlenk D, Sapozhnikova Y, Irwin MA, Xie L, Hwang W, Reddy S, Brownawell BJ, Armstrong J, Kelly M, Montagne DE, Kolodziej EP, Sedlak D, Snyder S. 2005. In vivo bioassay-guided fractionation of marine sediment extracts from the Southern California Bight, USA, for estrogenic activity. *Environ Toxicol Chem* 24:2820–2826.
- Sekela M, Brewer R, Moyle G, Tuominen T. 1999. Occurrence of an environmental estrogen (4-nonylphenol) in sewage treatment plant effluent and the aquatic receiving environment. *Water Sci Technol* 39:217–220.
- Senthil Kumar K, Sajwan KS, Richardson JP, Kannan K. 2008. Contamination profiles of heavy metals, organochlorine pesticides, polycyclic aromatic hydrocarbons and alkylphenols in sediment and oyster collected from marsh/estuarine Savannah GA, USA. *Mar Poll Bull* 56:136–149.
- Servos MR. 1999. Review of the aquatic toxicity, estrogenic responses and bioaccumulation of alkylphenols and alkylphenol polyethoxylates. *Water Qual Res J Canada* 34:123-177.
- Servos MR, Maguire RJ, Bennie DT, Lee H, Cureton PM, Davidson N, Sutcliffe R, Rawn DFK. 2003. An ecological risk assessment of nonylphenol and its ethoxylates in the aquatic environment. *Hum Ecol Risk Assess* 9:569–87.
- Shang DY, MacDonald RW, Ikonomou MG. 1999. Persistence of nonylphenol ethoxylate surfactants and their primary degradation products in sediments from near a municipal outfall in the Strait of Georgia, British Columbia, Canada. *Environ Sci Technol* 33:1366–1372.
- Siemering GS, Hayworth JD, Greenfield BK. 2008. Assessment of Potential Aquatic Herbicide Impacts to California Aquatic Ecosystems. *Arch Environ Contam Toxicol* 55:415-31.
- Soares A, Guieysse B, Jefferson B, Cartmell E, Lester JN. 2008. Nonylphenol in the environment: A critical review on occurrence, fate, toxicity and treatment in wastewaters. *Env Int* 34:1033-1049.
- Staples CA, Naylor CG, Williams JB, Gledhill WE. 2001. Ultimate biodegradation of alkylphenol ethoxylate surfactants and their biodegradation intermediates. *Environ Toxicol Chem* 20: 2450–2455.
- Staples C, Mihaich E, Carbone J, Woodburn K, Klecka G. 2004. A weight of evidence analysis of the chronic ecotoxicity of nonylphenol ethoxylates, nonylphenol ether carboxylates, and nonylphenol. *Hum Ecol Risk Assess* 10:999–1017.
- Sumpter JP and Jobling S. 1995. Vitellogenesis as a biomarker for estrogenic contamination



of the aquatic environment. *Environ Health Perspect* 103:173–8.

Toxecology Environmental Consulting Ltd. 2002. Alternatives to Nonylphenol Ethoxylates: Review of Toxicity, Biodegradation, & Technical-Economic Aspects. Prepared for Environment Canada.

USEPA. 2005. Aquatic Life Ambient Water Quality Criteria Nonylphenol FINAL. U.S. Environmental Protection Agency Office of Water & Office of Science and Technology. Washington, DC.

USEPA. 2010. Nonylphenol (NP) and Nonylphenol Ethoxylates (NPEs) Action Plan. RIN 2070-ZA09.

USEPA. 2012. Design for the Environment Alternatives Assessment for Nonylphenol Ethoxylates. <http://www.epa.gov/dfe/pubs/projects/npe/index.htm>

Ying G, Williams B, Kookana R. 2002. Environmental fate of alkylphenols and alkylphenol ethoxylates – a review. *Env Int* 28:215–226.

Ying G. 2006. Fate, behavior and effects of surfactants and their degradation products in the environment. *Env Int* 32:417–431.

Van Miller JP, Staples CA. 2005. Review of the potential environmental and human health-related hazards and risks from long-term exposure to p-tert-octyl phenol. *Hum Ecol Risk Assess* 11:319-351.