Fate of Triclosan and Evidence for Reductive Dechlorination of Triclocarban in Estuarine Sediments

TODD R. MILLER,[†] JOCHEN HEIDLER,[†]
STEVEN N. CHILLRUD,[‡]
AMELIA DELAQUIL,[†] JERRY C. RITCHIE,[§]
JANA N. MIHALIC,[†] RICHARD BOPP,[‖]
AND ROLF U. HALDEN*,[†,⊥]

Center for Water and Health, Department of Environmental Health Sciences, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland 21205, USA, Lamont-Doherty Earth Observatory, Columbia University, Palisades, New York 10964, U.S. Department of Agriculture, Agricultural Research Service (USDA-ARS), Hydrology and Remote Sensing Laboratory, Beltsville, Maryland 20705, Department of Earth and Environmental Sciences, Rensselaer Polytechnic Institute, Troy, New York 12180, and Center for Environmental Biotechnology, The Biodesign Institute at Arizona State University, Tempe, Arizona 85287

The biocides triclosan and triclocarban are wastewater contaminants whose occurrence and fate in estuarine sediments remain unexplored. We examined contaminant profiles in ^{137}Cs/^{7}Be-dated sediment cores taken near wastewater treatment plants in the Chesapeake Bay watershed (CB), Maryland and Jamaica Bay (JB), New York. In JB, biocide occurrences tracked the time course of biocide usage and wastewater treatment strategies employed, first appearing in the 1950s (triclocarban) and 1960s (triclosan), and peaking in the late 1960s and 1970s (24 ± 0.54 and 0.8 ± 0.4 mg/kg dry weight, respectively). In CB, where the time of sediment accumulation was not as well constrained by ^{137}Cs depth profiles, triclocarban was only measurable in ^{137}Cs-bearing sediments, peaking at 3.6 ± 0.6 mg/kg midway through the core and exceeding 1 mg/kg in recent deposits. In contrast, triclosan concentrations were low or not detectable in the CB core. Analysis of CB sediment by tandem mass spectrometry produced the first evidence for complete sequential dechlorination of triclocarban to the transformation products dichloro-, monochloro-, and unsubstituted carbanilide, which were detected at maxima of 15.5 ± 1.8, 4.1 ± 2.4, and 0.5 ± 0.1 mg/kg, respectively. Concentrations of all carbanilide congeners combined were correlated with heavy metals ($R^2 > 0.64$, $P < 0.01$), thereby identifying wastewater as the principal pathway of contamination. Environmental persistence over the past 40 years was observed for triclosan and triclocarban in JB, and for triclocarban's diphenylurea backbone in CB sediments.

* Corresponding author phone: 480-727-0893; fax: 480-727-0889; e-mail: halden@asu.edu.
† Johns Hopkins Bloomberg School of Public Health.
‡ Columbia University.
§ USDA-ARS.
‖ Rensselaer Polytechnic Institute.
⊥ The Biodesign Institute at Arizona State University.

Introduction

Use of detergents and personal care products containing triclosan (TCS; 5-chloro-2-[2,4-dichlorophenoxy]-phenol) and triclocarban (TCC; 3,4,4′-trichlorocarbanilide) is steadily increasing (*1*). Current, combined inputs of both biocides into the U.S. environment are known to exceed 600 000 kg/yr and may be as high as 10 000 000 kg/yr (Supporting Information Table S1) (*1*). Conventional activated sludge wastewater treatment plants (WWTPs) effectively reduce biocide levels in wastewater from μg/L to ng/L levels due to effective sequestration of chemicals in sludge and incomplete biodegradation (*2–5*). Reclamation of wastewater and agricultural recycling of municipal sludge represent two important dispersal mechanisms for these biocides that are widely distributed in environmental habitats (*6*).

Contamination of aquatic environments with TCS and TCC is of potential concern since laboratory studies indicate both biocides may be harmful to aquatic organisms. For example, in cell-based and *in vitro* enzymatic assays TCS activates the pregnane X receptor, inhibits human estrogen and thyroid hormone sulfotransferases, impairs mitochondrial function, and induces other phenotypes associated with endocrine disruption (*7–11*). In tadpoles, exposure to environmentally relevant concentrations of TCS (150 ng/L) causes changes in thyroid hormone receptor gene expression, a reduction in body weight, increased hind limb development, and a decrease in swimming activity (*12, 13*). At high concentrations (>600 μg/L) TCS causes death in fish and increases vitellogelin production in fish eggs, suggesting estrogenic activity (*14, 15*). Antimicrobial effects of TCS are in part due to inhibition of type II fatty acid metabolism in bacteria. Many plants, algae, and protozoans also rely on a bacteria-like type II fatty acid pathway and are sensitive to low μg/L levels of TCS (*15–18*). Comparatively less is known about the toxicity of TCC. This chemical has been shown to cause abnormal development in clams and to reduce larval survival at low levels (30 μg/L) (*19*). In addition, TCC can act as an agonist toward the aryl hydrocarbon receptor that mediates toxic effects caused by structurally diverse compounds, including the known carcinogen 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (*20*). Furthermore, TCS and TCC potentially may disrupt critical ecological processes performed by beneficial microorganisms in nature, because both compounds are active toward a wide spectrum of microbial species (*21, 22*).

Additional concerns center on the environmental behavior of TCS and TCC and on the toxicity of their transformation products. Both biocides are sparingly water soluble at circumneutral pH and display strong sorptive behavior due to their significant lipophilicity (*23*). Predicted half-lives for TCS and TCC in water are approximately 60 days, and empiric half-lives reported for TCS in receiving streams range from several hours to weeks, depending on the specific environmental conditions (*3, 24*). Both biocides may be removed from the water column through various physical, chemical, and biological processes, including sedimentation followed by uptake and/or transformation by biota, and phototransformation. For example, TCS undergoes partial photochemical degradation in the water column, thereby producing dichlorodioxins and carcinogenic chlorophenols, two problematic and potentially persistent degradates (*25*). Likewise, aerobic bacterial degradation of TCC results in the formation of chloroanilines, compounds that are cytotoxic and carcinogenic (*26, 27*). In addition, recent studies show that TCC, TCS, and its biotransformation product methyl-TCS
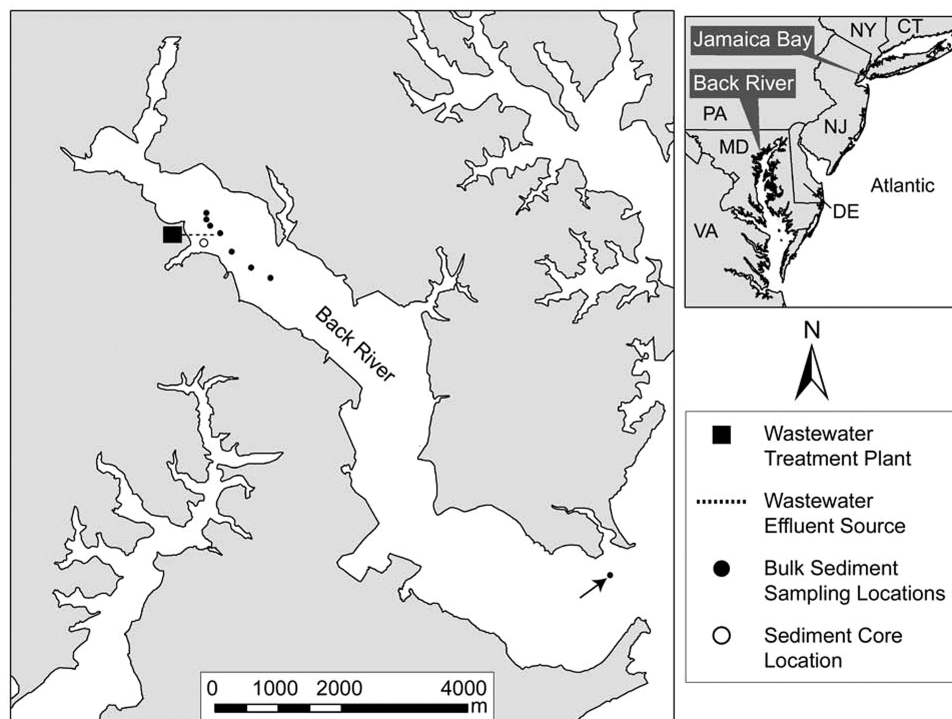
FIGURE 1. Map of the U.S. East Coast showing sampling locations in a tributary to the Chesapeake Bay, MD, and in a deep estuarine basin in Jamaica Bay, NY. A close-up view of MD shows 8 sites of sampling for surface sediment (0–10 cm depth) taken along a cross section beginning 450 m inland to 5700 m (arrow) seaward relative to the wastewater outfall.

accumulate in algae 3 orders of magnitude above ambient water concentrations, thereby effectively raising the exposure concentration for algae and aquatic biota feeding upon them (28). In the water column, they may also adsorb to abiotic particulate matter, which also is ingested by planktivorous fish, clams, oysters, crustaceans, and other filter feeding organisms. Indeed, fish have been shown to contain the primary metabolite of triclosan, methyl- triclosan, at ppm levels (μg/g lipid weight) (29). In their final sink, aquatic sediments, both biocides are predicted to exhibit half-lives of greater than 500 days (30). However, few empirical measurements support this claim, and lower chlorinated derivatives of TCS and TCC that could provide evidence for anaerobic biotransformation have not yet been reported (2).

The scarcity of data on the fate of TCS and TCC in sediment is unfortunate, as the record of chemical deposition in sediment may provide a rich history of chemical production, geographic usage, environmental loading, and in situ degradation. One study reported TCS in a dated sediment core taken from Lake Greifensee, Switzerland, where TCS concentrations fluctuated over time according to usage and efficiency of wastewater treatment regimes (2). The highest amount of TCS was detected in >30 year old sediment, suggesting that TCS has a very slow rate of in situ degradation in freshwater sediment if a constant source term is assumed (2). Increasing concentrations of TCS were observed in recently deposited top sediment, thereby reflecting greater biocide usage in the past decade (2).

In this study, we investigated TCS and TCC in sediment from two estuarine environments. We describe the distribution of both biocides near a wastewater outfall in the Chesapeake Bay watershed (CB) in Maryland, and we compare their environmental fate in dated sediment cores from CB and Jamaica Bay (JB), New York. To the best of our knowledge, this is the first study to examine the environmental fate of either biocide in estuarine sediments.

Experimental Section

Chemicals. Solvents, reagent-grade water, nonlabeled TCS, TCC, and nonchlorinated carbanilide (NCC) were of the highest purity available from Sigma Aldrich (Milwaukee, WI). The synthesis of monochlorocarbanilide (MCC) is presented in the Supporting Information. The origin and purity of dichlorocarbanilide (DCC), $^{13}C_6$- TCC, and $^{13}C_6$- TCS has been reported in detail elsewhere (5, 31).

Description of Study Sites and Sample Collection. Sediment samples were obtained from two study sites on the U.S. East Coast (Figure 1). The CB site is on the Back River, a residential tributary of the Chesapeake Bay located near Baltimore, Maryland. This location receives effluent from a WWTP serving 1.3 million people. A full description of the plant has been provided previously (32). In July of 2005, samples of the top 10 cm of sediment were collected from eight CB locations in Back River, MD beginning 450 m inland from a WWTP outfall and extending 5700 m seaward (Figure 1). Each sample was mixed to homogeneity prior to analysis. Subsequently, several push cores were collected 130 m downstream of the WWTP outfall in July of 2006 using methods previously described (33). The cores were divided into 2–4 cm sections, dried for several days at 35 °C, ground to a fine powder, and stored at room temperature until further analysis. One of these cores, CB1, was chosen for full radionuclide and biocide analyses.

Jamaica Bay is an urbanized estuary located on the southwestern shore of Long Island that receives much of its freshwater as effluent from JB WWTPs. Eastern JB was dredged extensively in 1939, creating an unusually deep area in a section called Grassy Bay, which serves as a settling basin for fine-grained particles. Archived sediment was obtained from sections of JB16, a gravity core collected on October 4, 1996 from Grassy Bay and stored as dried sediment at room temperature in airtight, enamel-lined aluminum cans. Further information on JB WWTPs, the sampling
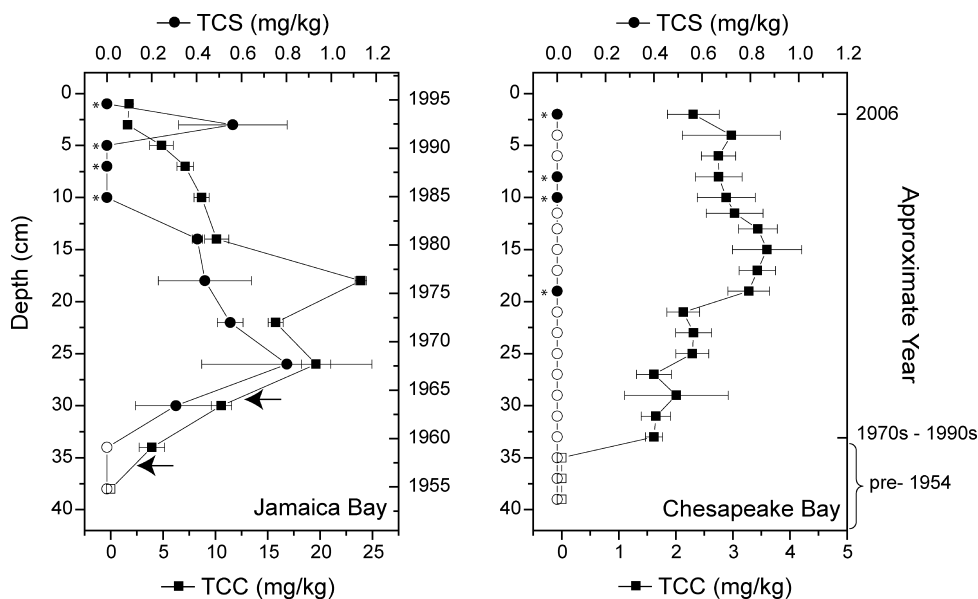
FIGURE 2. Depth-time profile of TCS and TCC in sediment from Jamaica Bay and Chesapeake Bay estuarine sampling locations. Open symbols indicate values below the detection limit and filled symbols with an asterisk indicate that the analyte was detected, but its concentration was below the LOQ. Note that the TCS scale is on the upper *x*-axis and the TCC scale is on the lower *x*-axis. Arrows on the JB plot indicate patent issuance dates for TCS (1964) and TCC (1957), that ushered in chemical mass production. Error bars indicate the standard deviation of analyte concentrations in two or more replicate extractions of the same sediment section.

location in Grassy Bay, and additional dating information relevant for core JB16 may be found elsewhere (*34*).

Sediment Dating. The radioisotopes ^{7}Be and ^{137}Cs were measured in CB1 sediment cores by γ-spectrometry as previously accomplished (*33, 35*). Values for ^{137}Cs were decay corrected to the time of sampling. Interpretation of radionuclide data are described in the Supporting Information.

Sample Preparation and Analysis. Dried sediments from JB and CB were analyzed along with blanks and quality control samples by isotope dilution liquid chromatography negative electrospray ionization mass spectrometry (LC-ESI-MS) using previously reported procedures (*36*) Details of the sediment extraction method are provided in the Supporting Information. Extraction efficiency of target analytes and percent recovery of surrogate standards are presented in Supporting Information Tables S2 and S3 along with monitored target and reference ions. When low analyte concentrations in sediment necessitated better sensitivity for accurate quantitation, the various carbanilides were measured in sediment extracts using liquid chromatography tandem mass spectrometry as previously described (*5*). The limit of quantitation (LOQ) and detection limit (DL) for TCC, DCC, MCC, and NCC was 0.0005 and 0.0001 mg/kg, respectively, and for TCS was 0.05 and 0.01 mg/kg, respectively. All biocide concentrations in sediment are reported on a dry weight basis.

Metals Analysis. Heavy metals (Cu, Pb, Zn) in JB16 sediment were analyzed by atomic absorption spectroscopy and inductively coupled plasma mass spectrometry using procedures reported previously (*37*). A similar procedure was used to measure metals in CB sediment (Supporting Information).

Results

Distribution of Biocides in Back River Sediment. Both biocides were detected in homogenized surface sediment samples taken near the WWTP on Back River, a tributary of CB (Figure 1). Concentrations of TCC did not significantly vary within 400 m upstream or 1000 m downstream of the outfall and ranged from 0.7–1.6 mg/kg dry weight, with an average of 1.0 ± 0.48 mg/kg ($n = 21$). In contrast, TCS was below the LOQ (0.05 mg/kg) in 70% of the samples and showed a maximum of 0.08 ± 0.01 mg/kg and an average of

0.07 ± 0.01 mg/kg ($n = 6$), which is 14 times lower than that of TCC. In a single sediment sample taken 5700 m away in a seaward direction, TCC remained detectable at 0.11 ± 0.16 mg/kg, whereas TCS was not found at an estimated detection limit of 0.01 mg/kg.

Sediment Dating. The age−depth relationship in core JB16 and earlier co-located cores have been presented and discussed previously (*33, 34*). The radionuclide measurements on the CB1 core allow much less detailed age determinations of its depth sections. The well-conserved morphology of loose, lightly colored organic flocculates in the core top of CB1, as seen through the core liner, together with the measured activity of ^{7}Be in the core top (0–2 cm; 46 $\pm$ 33 Bq/kg (1 sigma counting error) suggest that surface sediment in the CB1 core probably included particles that had accumulated within several months of time of sampling (July 2006); however, the counting statistics did not reach the 95% confidence level. The top 35 cm of CB1 contains measurable activities of ^{137}Cs that are very similar (12.6 ± 2.1 Bq/kg) without any prominent subsurface peak (Supporting Information Table S4 and Figure S1). Intense sediment mixing over the top 30+ cm is ruled out by the contaminant measurements that show sharp gradients as a function of depth (Figures 2 and 3 and Supporting Information Table S5). Sediments below 35 cm do not contain measurable levels of ^{137}Cs, indicating that these sediments accumulated prior to the early 1950s. Overall, the radionuclide profile is most consistent with a major erosional/dredging event having occurred sometime between the 1970s and the 1990s, removing sediments accumulated since at least the early 1950s. Recent accumulation occurring over the last several years to the last few decades would account for the ^{137}Cs activity in the upper sections of the core. In this interpretation, the 34–36 cm depth section of CB1 would represent a mixture of old sediment (deposited some time prior to the early 1950s) and the first sediment that accumulated after the erosional/dredging event.

Depth Profile of Biocides in Chesapeake Bay and Jamaica Bay Sediment. In the JB16 sediment core TCS was detected in all sediment sections deposited since commercial production of the compound began in the mid 1960s (Figure 2). TCS levels peaked at between 0.6 and 0.8 mg/kg in samples

A. Jamaica Bay, NY

Chemical (μmoles/kg)

B. Chesapeake Bay, MD

Chemical (μmoles/kg)
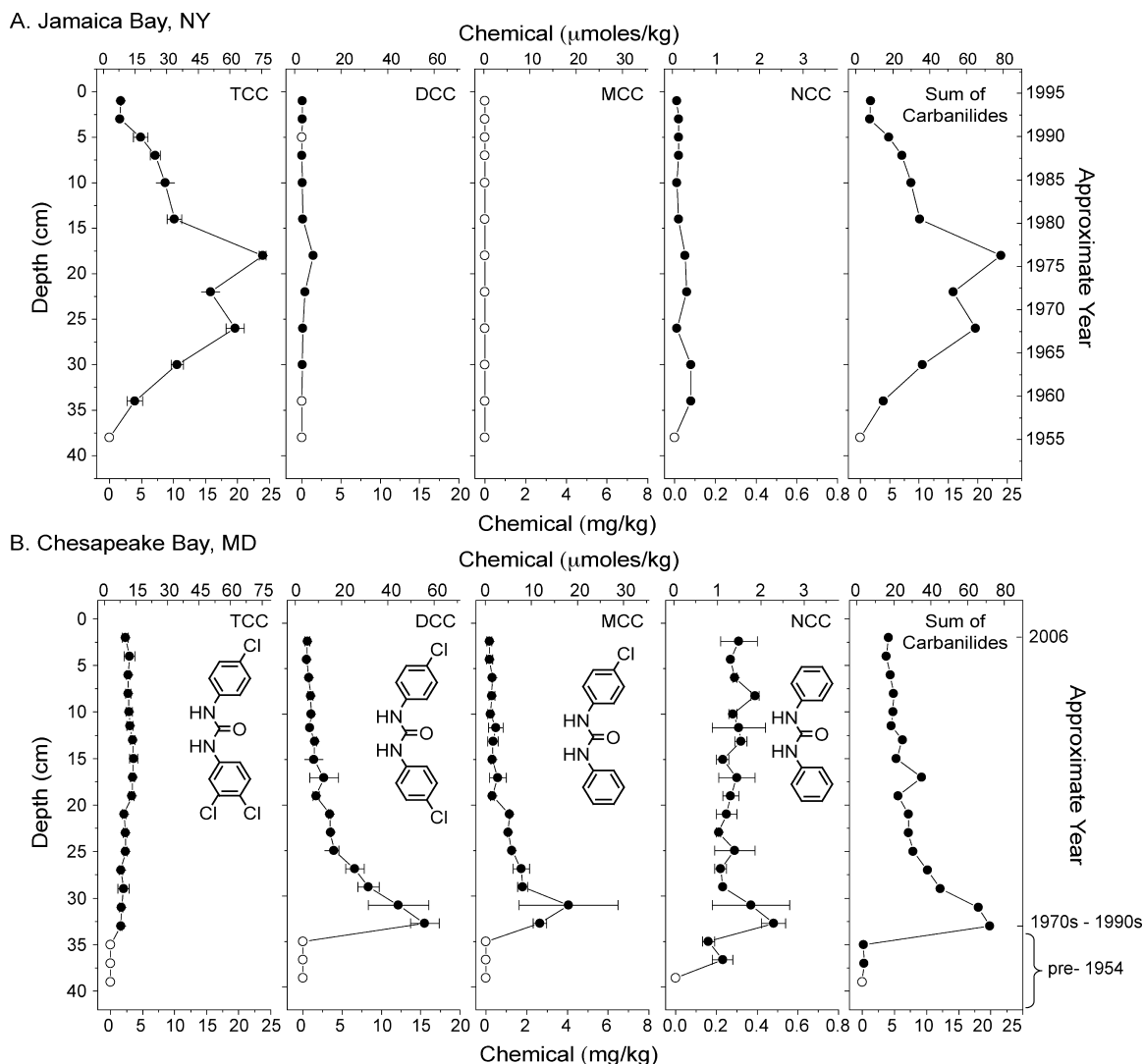
Chemical (mg/kg)

FIGURE 3. Depth-time profile of TCC and its postulated products of complete sequential dechlorination in sediment cores taken from the Chesapeake Bay, MD and Jamaica Bay, NY. Open symbols indicate values below the detection limit. In JB16, the dechlorination products DCC, MCC, and NCC were measured in a single extract from each sediment section, and for all other data points error bars are the average and standard deviation of measurements of two or more sediment extracts from each sediment section. For comparison, the units on the upper axis are in molarity, and that of the bottom axis are in mg/kg. TCC appears to be recalcitrant in Jamaica Bay sediment, whereas Chesapeake Bay sediment shows evidence of TCC dechlorination in deep sediment layers.

deposited between the mid 1960s and late 1970s. The significant decline to less than 0.05 mg/kg in four of the five samples deposited since the mid 1980s may be a reflection of the introduction of full activated sludge treatment to the JB WWTPs in 1978 (33). The 2–4 cm sample, with a mean TCS level of 0.6 mg/kg may reflect the influence of major combined sewer overflow events. Combined sewer overflow has been a persistent problem for wastewater treatment in the JB serwershed (33, 38). TCC was detected in all but the deepest core section, consistent with its earlier commercial production compared to TCS (Figure 2). Peak levels of TCC, on the order of 24 mg/kg, were about 25 times greater than those of TCS but occurred over a similar time period, the mid 1960s to the late 1970s. The more recent decrease in levels, again, are likely associated with the WWTP upgrade in 1978, but the decrease was not as dramatic as for TCS (Figure 2). Concentrations of TCC in JB sediment remained above 1 mg/kg in the top sediment layer.

At the location of sediment core CB1 (Figure 1), TCS was at or below the LOQ (0.05 mg/kg) when detectable at all (Figure 2). In contrast, TCC was found consistently throughout the top 33 cm of the CB1 core, ranging from about 1.5 to 3.5 mg/kg. As discussed above (see Sediment Dating

section), our best estimate is that this layer accumulated between 1980 or 1990 and 2006. The levels of TCC are comparable to those from the sections of JB16 deposited between about 1990 and 1996.

Sediment Deposition Rate of Biocides Compared to Heavy Metals. Wastewater effluent is known to contain elevated concentrations of heavy metals that are subsequently deposited in sediment of effluent receiving surface waters (34, 39). In JB16 and CB1 cores, the overall average concentration of Cu (298 ± 58 and 209 ± 88 mg/kg, respectively) was similar to that of Pb (305 ± 72 and 215 ± 88 mg/kg, respectively) and Zn (300 ± 120 and 1441 ± 260 mg/kg, respectively), although Zn was slightly higher in CB1. The depth−time profile of the metals was nearly identical to one another at each location (Supporting Information Table S5). In the JB16 core, the depth−time profile of both TCS and TCC paralleled those of the metals (Supporting Information Figure S2 and Table S5), and a significant association was observed between TCC and Cu (Spearman coefficient of determination and probability; $R^2 = 0.67$, $P = 0.002$) and TCC and Pb ($R^2 = 0.65$, $P = 0.003$), consistent with WWTP effluent being the major pathway of contaminants to JB sediment. In contrast, an inverse relationship between Cu

and TCC, and Pb and TCC in the CB1 core was evident, but not statistically significant ($R^2 = 0.3$, $P = 0.08$ and $R^2 = 0.21$, $P = 0.15$, respectively).

Evidence for Dechlorination of TCC in the Chesapeake Bay Watershed. Significant quantities of dichloro-, monochloro-, and to a lesser extent nonchlorinated carbanilide (DCC, MCC, and NCC, respectively) were observed at lower depths in the CB1 sediment core (Figure 3B). At a depth of 26–34 cm, the three different dechlorination products achieved maximum concentrations. DCC was most abundant (15.5 ± 1.8 mg/kg), followed by MCC (4.1 ± 2.4 mg/kg) and then NCC (0.5 ± 0.1 mg/kg). After these maxima, upper sediment layers contained decreasing concentrations of all dechlorination products. The sum of all carbanilide congeners examined reached a maximum of 19.8 mg/kg at a depth of 33 cm. A previously described manufacturing impurity of TCC, 3,3′,4,4′-tetrachlorocarbanilide (TetCC) was not detected in the CB1 sediment core (<0.001 mg/kg; data not shown). There was a significant inverse association between TCC and DCC ($R^2 = 0.55$, $P < 0.0001$) and between TCC and MCC ($R^2 = 0.48$, $P < 0.0001$), indicating that, as levels of these dechlorination products increased, levels of TCC decreased.

In contrast to findings in the Chesapeake Bay watershed, TCC dechlorination products were rarely detected in JB sediment (Figure 3A). DCC was repeatedly detected at levels below 0.5 mg/kg, whereas MCC and NCC were found infrequently and only at nonquantifiable levels. At an estimated detection limit of 0.001 mg/kg, TetCC was not observable in any sediment section of the JB16 core (not shown).

TCC was not significantly correlated with heavy metals in CB1 sediment due to its dechlorination; however, the sum of all carbanilides in both JB16 and CB1 sediment was significantly correlated with Cu ($R^2 = 0.64$, $P = 0.003$; and $R^2 = 0.77$, and $P = 0.0004$; respectively), Pb ($R^2 = 0.17$, $P = 0.001$; and $R^2 = 0.67$, and $P = 0.002$; respectively) and Zn ($R^2 = 0.66$, $P = 0.002$; and $R^2 = 0.68$, and $P = 0.002$; respectively) (Figure S3). These observations suggest that despite dechlorination of TCC, the carbanilide backbone has resisted degradation in sediment at both locations.

Discussion

TCC is More Persistent than TCS. TCC was more abundant than TCS in sediment, regardless of sample type, sediment depth, sampling site, and sediment age. This could be due to slightly greater annual production volumes reported for TCC over the last few decades (Supporting Information Table S1). However, this does not take into account usage patterns. More likely, the data are in agreement with observations at the CB sewage treatment plant, where TCC was observed to persist to a greater extent than TCS (76 ± 30 vs $50 \pm 19\%$, respectively) following aerobic and anaerobic treatment strategies (5, 32). Whereas the ratio of TCC to TCS concentrations in chlorinated effluent from the CB facility previously was observed to be less than 2:1 (5, 32), it was found to be greater than 14:1 in surface sediment (Figure 2). Physicochemical properties and differences in the transformation potential of TCS and TCC may account for the variance in persistence. At pH 7.0 the organic carbon–normalized sorption coefficient of TCC is slightly greater than that of TCS (log $K_{OC} = 4.50$ for TCC vs 4.13 for TCS); and in wastewater, at pH < 7.0, a slightly greater fraction of the mass of TCC adsorbs to particulate matter and sludge (5, 32). In environmentally relevant pH conditions, only TCS (but not TCC) exhibits ionic properties ($pK_A = 7.8$ for TCS vs 12.7 for TCC), and at pH 8 the calculated log K_{OC} of TCS decreases to 3.78, whereas that of TCC remains unchanged. This is important in estuarine environments, as the pH may fluctuate above the pK_A of TCS with saltwater influx during high tide

and during summer algal blooms (pH > 9.0), thereby effectively increasing the fraction of the mass of TCS in the dissolved phase (40, 41). A greater proportion of TCS in the dissolved phase, particularly in its deprotonated form, may result in an overall lower TCS deposition rate due to decreased sedimentation and higher rates of biological and photochemical degradation (2, 42).

Geographic Variability in the Environmental Fate of Both Biocides. The two biocides were more persistent in JB sediment when compared to those from the CB location (Figure 2). Conclusions concerning the persistence of both organic chemicals were drawn based on the relationship between levels of biocides and heavy metals in JB sediment. Here, treated effluent discharged from the local WWTP is the principal source of heavy metals (34). Heavy metal behavior is essentially conservative, and total concentrations are unaffected by biological, chemical, and photodegradative processes. The fact that the profiles of TCC and TCS parallels those of the heavy metals in core JB16 indicates that the biocides are not degraded to any significant extent in this environment. Treated effluent as the principal source of biocides in sediment at this study location is further evidenced by the fact that concentrations of TCS and TCC increased in JB16 sediment until the 1970s and then decreased thereafter (Figure 2), coinciding with upgrading of the local WWTP to secondary treatment by activated sludge processing. This resulted in a marked decrease in the concentration of total suspended solids in JB wastewater effluent from 55 to 20 mg/L (33). Because sorption of biocides to activated sludge is extensive (2, 5), concentrations of both analytes in effluent would be expected to substantially decrease after the treatment plant upgrade, consistent with the sediment core profiles of both TCC and TCS in JB16.

The geographically variable persistence of both biocides in JB and CB may be related to the distinct physicochemical and associated biological characteristics of the two sites. The JB core was taken from an area in JB known as Grassy Bay, which essentially acts as a large catchment basin for wastewater effluent (33). This inland bay is poorly flushed by tidal currents and experiences seasonal water-column stratification and bottom water anoxia (43). In contrast, bottom waters at the CB location are well oxygenated year round (40). In addition, in the deeper and more stratified Grassy Bay, phototransformation of the biocides may be more limited. Similarly, bottom water anoxia may limit any aerobic biological degradation of the two antimicrobials during sedimentation (44).

TCC Dechlorination. Significant quantities of DCC, MCC, and NCC were detected in aged, deep sediment from CB, suggesting ongoing dechlorination of TCC in situ. Although DCC and TetCC are known to occur as impurities at levels of about 0.2% by weight in technical grade TCC (31), the quantities are far too low to account for the amounts of DCC found in estuarine sediments in this study. In addition, DCC is known to occur in CB surface waters at a TCC:DCC ratio of 70:1, whereas in CB sediment this ratio is 1:5 or less. Because preferential partitioning would shift the ratio in the other direction, the elevated levels of DCC found in sediment must have been produced in situ (Figure 3) (31). Furthermore, a significant negative correlation was observed between TCC and each dechlorination product along the sediment core, providing further evidence for TCC dechlorination.

Although elucidation of the mechanism of TCC dechlorination was beyond the scope of this study, the involvement of dehalorespiring microorganisms is one possibility. Consistent with reductive dechlorination being a strictly anaerobic process, no significant quantities of dechlorination products were observed in partially oxygenated surface sediments (Figure 3). The highest concentrations of TCC transformation products were found in deep sediment likely

to be anoxic, suggesting that TCC transformation may require long incubation times similar to other organochlorines (*45*). In CB sediment, the greater abundance of DCC and MCC relative to NCC suggests that TCC dechlorination is still actively occurring, albeit at a very slow rate. Assuming that all observed dechlorination products arose from TCC, the amount of TCC dechlorinated can be calculated by dividing the molar concentration of TCC by the sum of all carbanilides using data presented in Figure 3. In upper sediment layers, the extent of transformation was less than 40%, whereas the largest amount of transformation occurred at a depth of about 33 cm, where approximately 94% of the deposited TCC had been transformed to lower chlorinated derivatives.

In JB sediment, there was less TCC dechlorination activity, as evidenced by lower levels of the expected transformation products and higher concentrations of TCC in aged sediment (Figure 3). Trending of TCC and TCS with levels of sewage derived heavy metals suggest that both antimicrobials can persist for decades in sediments of Jamaica Bay. Lack of dechlorination in JB sediment is somewhat puzzling since deep sediment (>1 cm) at both locations is expected to lack oxygen (*46*). However, dechlorination of similar organic contaminants has been shown to be geographically variable (*47*). This may be related to a variety of site-specific factors including microbial species composition, competition for electron donors, and chemical inhibition (reviewed in (*48*)). Whether or not any of these factors play a role in lack of TCC dechlorination in JB sediment is unknown.

Implications for Aquatic Toxicity. Although both study sites are heavily impacted by wastewaters, they represent habitats for key aquatic organisms. Observations made in this study have implications for the sustainable use of these natural resources for fishing, crabbing, and boating. High levels of TCC in CB and JB sediment may harm aquatic organisms. At present, a biota–sediment accumulation factor has not been established for TCC, making it difficult to determine how sediment concentrations relate to exposure levels. However, the pore water concentration of TCC may be estimated. Given that CB and JB sediment have an average organic carbon content of approximately 5 and 6%, respectively (*34, 49*), average and maximum pore water concentrations for TCC of 1.77 ± 0.67 and $3.96\ \mu g/L$ or 4.05 ± 3.38 and $10.78\ \mu g/L$ were calculated for the CB and JB locations, respectively. These levels approach or exceed the minimal inhibitory values reported for some aquatic organisms including invertebrates (*Ceriodaphnia* sp.; no observed effect concentration (NOEC) of $1.46\ \mu g/L$), shellfish larvae (*Mercenaria mercenaria*; $LC_{50} = 30\ \mu g/L$), and fish species (*Pimephales promelas*; $NOEC = 5\ \mu g/L$) (*50*). Using a predicted no effect concentration (PNEC) that is one-tenth of the NOEC value (*51*), estimated pore water concentrations of TCC in sediment likely exceed the PNEC for *Ceriodaphnia* and *P. promelas*. Yet, assays to assess NOEC concentrations involve exposure of these organisms to dissolved biocide mass for no more than a few days for acute toxicity and for less than a month to estimate chronic toxicity. In addition, these assays also do not control for ingestion of biocide-laden particulate matter. From our results we have to conclude that particulate matter may serve as a delivery mechanism for high doses of TCC. This may be of importance to filter feeding benthic organisms, including worms, shellfish, crabs, and important herbivorous fish species such as Atlantic Menhaden (*Brevoortia tyrannus*).

This study explored the 40-year history of TCS and TCC deposition in estuarine sediment at two locations on the U.S. East Coast. Data show that (i) TCC, and to a lesser extent TCS, are persistent organic contaminants of estuarine sediment; (ii) TCC is more persistent and more abundant than TCS; (iii) in aged sediment, TCC can undergo slow anaerobic dechlorination but that the process shows geographic variability; (iv) anaerobic transformation processes can alter the chlorine substitution pattern but do not reduce the overall quantity of carbanilide species present, and (v) TCC contamination of estuarine sediment in some locations extends into the ppm range, representing potentially unhealthy levels for aquatic organisms. Further studies into the aquatic toxicity of these persistent antimicrobial additives are needed to more accurately judge their actual threat to aquatic ecosystems.

Supporting Information Available

Additional detail regarding experimental procedures and results may be found in the Supporting Information that accompanies this article. This information is available free of charge via the Internet at http://pubs.acs.org.

Literature Cited

(1) TSCA Chemical Substances Inventory; U.S. Environmental Protection Agency: Washington D.C., 2003.

(2) Singer, H.; Muller, S.; Tixier, C.; Pillonel, L. Triclosan: Occurrence and fate of a widely used biocide in the aquatic environment: field measurements in wastewater treatment plants, surface waters, and lake sediments. *Environ. Sci. Technol.* **2002**, *36* (23), 4998–5004.

(3) Bester, K. Fate of triclosan and triclosan-methyl in sewage treatment plants and surface waters. *Arch. Environ. Contam. Toxicol.* **2005**, *49* (1), 9–17.

(4) McAvoy, D. C.; Schatowitz, B.; Jacob, M.; Hauk, A.; Eckhoff, W. S. Measurement of triclosan in wastewater treatment systems. *Environ. Toxicol. Chem.* **2002**, *21* (7), 1323–1329.

(5) Heidler, J.; Sapkota, A.; Halden, R. U. Partitioning, persistence, and accumulation in digested sludge of the topical antiseptic triclocarban during wastewater treatment. *Environ. Sci. Technol.* **2006**, *40* (11), 3634–3639.

(6) Kolpin, D. W.; Furlong, E. T.; Meyer, M. T.; Thurman, E. M.; Zaugg, S. D.; Barber, L. B.; Buxton, H. T. Pharmaceuticals, hormones, and other organic wastewater contaminants in U.S. streams, 1999–2000: A national reconnaissance. *Environ. Sci. Technol.* **2002**, *36*, 1202–1211.

(7) Jinno, H.; Hanioka, N.; Onodera, S.; Nishimura, T.; Ando, M. Irgasan(R) DP 300 (5-chloro-2-(2,4-dichlorophenoxy)phenol) induces cytochrome P450s and inhibits haem biosynthesis in rat hepatocytes cultured on Matrigel. *Xenobiotica* **1997**, *27* (7), 681–692.

(8) Kester, M. H. A.; Bulduk, S.; van Toor, H.; Tibboel, D.; Meinl, W.; Glatt, H.; Falany, C. N.; Coughtrie, M. W. H.; Schuur, A. G.; Brouwer, A.; Visser, T. J. Potent inhibition of estrogen sulfotransferase by hydroxylated metabolites of polyhalogenated aromatic hydrocarbons reveals alternative mechanism for estrogenic activity of endocrine disrupters. *J. Clin. Endocrinol. Metab.* **2002**, *87* (3), 1142–1150.

(9) Schuur, A. G.; Legger, F. F.; van Meeteren, M. E.; Moonen, M. J. H.; van Leeuwen-Bol, I.; Bergman, A.; Visser, T. J.; Brouwer, A. In vitro inhibition of thyroid hormone sulfation by hydroxylated metabolites of halogenated aromatic hydrocarbons. *Chem. Res. Toxicol.* **1998**, *11* (9), 1075–1081.

(10) Jacobs, M. N.; Nolan, G. T.; Hood, S. R. Lignans, bacteriocides and organochlorine compounds activate the human pregnane X receptor (PXR). *Toxicol. Appl. Pharmacol.* **2005**, *209* (2), 123–133.

(11) Newton, A. P. N.; Cadena, S. M. S. C.; Rocha, M. E. M.; Carnieri, E. G. S.; de Oliveira, M. B. M. Effect of triclosan (TRN) on energy-linked functions of rat liver mitochondria. *Toxicol. Lett.* **2005**, *160* (1), 49–59.

(12) Veldhoen, N.; Skirrow, R. C.; Osachoff, H.; Wigmore, H.; Clapson, D. J.; Gunderson, M. P.; Van Aggelen, G.; Helbing, C. C. The bactericidal agent triclosan modulates thyroid hormone-associated gene expression and disrupts postembryonic anuran development. *Aquat. Toxicol.* **2006**, *80* (3), 217–227.

(13) Fraker, S. L.; Smith, G. R. Direct and interactive effects of ecologically relevant concentrations of organic wastewater contaminants on *Rana pipiens* tadpoles. *Environ. Toxicol.* **2004**, *19* (3), 250–256.

(14) Ishibashi, H.; Matsumura, N.; Hirano, M.; Matsuoka, M.; Shiratsuchi, H.; Ishibashi, Y.; Takao, Y.; Arizono, K. Effects of triclosan on the early life stages and reproduction of medaka *Oryzias latipes* and induction of hepatic vitellogenin. *Aquat. Toxicol.* **2004**, *67* (2), 167–179.

(15) Orvos, D. R.; Versteeg, D. J.; Inauen, J.; Capdevielle, M.; Rothenstein, A.; Cunningham, V. Aquatic toxicity of triclosan. *Environ. Toxicol. Chem.* **2002**, *21* (7), 1338–1349.

(16) Tatarazako, N.; Ishibashi, H.; Teshima, K.; Kishi, K.; Arizono, K. Effects of triclosan on various aquatic organisms. *Environ. Sci.* **2004**, *11* (2), 133–140.

(17) Lund, E. D.; Soudant, P.; Chu, F. L. E.; Harvey, E.; Bolton, S.; Flowers, A. Effects of triclosan on growth, viability and fatty acid synthesis of the oyster protozoan parasite *Perkinsus marinus*. *Dis. Aquat. Org.* **2005**, *67* (3), 217–224.

(18) White, S. W.; Zheng, J.; Zhang, Y. M.; Rock, C. O. The structural biology of type II fatty acid biosynthesis. *Annu. Rev. Biochem.* **2005**, *74*, 791–831.

(19) Davis, H. C.; Hidu, H. Effects of pesticides on embryonic development of clams and oysters and on survival and growth of the larvae. *Fish. Bull.* **1969**, *67* (2), 393–404.

(20) Zhao, B.; Baston, D. S.; Hammock, B.; Denison, M. S. Interaction of diuron and related substituted phenylureas with the Ah receptor pathway. *J. Biochem. Mol. Toxicol.* **2006**, *20* (3), 103–113.

(21) Neumegen, R. A.; Fernandez-Alba, A. R.; Chisti, Y. Toxicities of triclosan, phenol, and copper sulfate in activated sludge. *Environ. Toxicol.* **2005**, *20* (2), 160–164.

(22) Dokianakis, S. N.; Kornaros, M. E.; Lyberatos, G. On the effect of pharmaceuticals on bacterial nitrite oxidation. *Water Sci. Technol.* **2004**, *50* (5), 341–346.

(23) Duan, M. S.; Zhao, N.; Ossurardottir, I. B.; Thorsteinsson, T.; Loftsson, T. Cyclodextrin solubilization of the antibacterial agents triclosan and triclocarban: Formation of aggregates and higher-order complexes. *Int. J. Pharm.* **2005**, *297* (1–2), 213–222.

(24) Sabaliunas, D.; Webb, S. F.; Hauk, A.; Jacob, M.; Eckhoff, W. S. Environmental fate of triclosan in the River Aire Basin, UK. *Water Res.* **2003**, *37* (13), 3145–3154.

(25) Sanchez-Prado, L.; Llompart, M.; Lores, M.; García-Jares, C.; Bayona, J. M.; Cela, R. Monitoring the photochemical degradation of triclosan in wastewater by UV light and sunlight using solid-phase microextraction. *Chemosphere* **2006**, *65* (8), 1338–1347.

(26) Chhabra, R. S.; Huff, J. E.; Haseman, J. K.; Elwell, M. R.; Peters, A. C. Carcinogenicity of *p*-chloroaniline in rats and mice. *Food Chem. Toxicol.* **1991**, *29* (2), 119–24.

(27) Gledhill, W. E. Biodegradation of 3,4,4′-trichlorocarbanilide, TCC, in sewage and activated-sludge. *Water Res.* **1975**, *9* (7), 649–654.

(28) Coogan, M. A.; Edziyie, R. E.; La Point, T. W.; Venables, B. J. Algal bioaccumulation of triclocarban, triclosan, and methyl-triclosan in a North Texas wastewater treatment plant receiving stream. *Chemosphere* **2007**, *67* (10), 1911–1918.

(29) Buser, H. R.; Balmer, M. E.; Muller, M. D.; Poiger, T. Occurrence of some organic UV filters in wastewater, in surface waters, and in fish from Swiss Lakes. *Environ. Sci. Technol.* **2005**, *39* (4), 953–962.

(30) Halden, R. U.; Paull, D. H. Co-occurrence of triclocarban and triclosan in U.S. water resources. *Environ. Sci. Technol.* **2005**, *39*, 1420–1426.

(31) Sapkota, A.; Heidler, J.; Halden, R. U. Detection of triclocarban and two co-contaminating chlorocarbanilides in US aquatic environments using isotope dilution liquid chromatography tandem mass spectrometry. *Environ. Res.* **2007**, *103* (1), 21–29.

(32) Heidler, J.; Halden, R. U. Mass balance assessment of triclosan removal during conventional sewage treatment. *Chemosphere* **2006**, *66* (2), 362–369.

(33) Ferguson, P. L.; Bopp, R. F.; Chillrud, S. N.; Aller, R. C.; Brownawell, B. J. Biogeochemistry of nonylphenol ethoxylates in urban estuarine sediments. *Environ. Sci. Technol.* **2003**, *37* (16), 3499–3506.

(34) Bopp, R. F.; Simpson, H. J.; Chillrud, S. N.; Robinson, D. W. Sediment-derived chronologies of persistent contaminants in Jamaica Bay, NY. *Estuaries* **1993**, *16* (3), 608–616.

(35) Ritchie, J. C.; Finney, V. L.; Oster, K. J.; Ritchie, C. A. Sediment deposition in the flood plain of Stemple Creek Watershed, northern California. *Geomorphology* **2004**, *61* (3–4), 347–360.

(36) Halden, R. U.; Paull, D. H. Analysis of triclocarban in aquatic samples by liquid chromatography electrospray ionization mass spectrometry. *Environ. Sci. Technol.* **2004**, *38*, 4849–4855.

(37) Chillrud, S. N.; Hemming, S.; Shuster, E. L.; Simpson, H. J.; Bopp, R. F.; Ross, J. M.; Pederson, D. C.; Chaky, D. A.; Tolley, L. R.; Estabrooks, F. Stable lead isotopes, contaminant metals and radionuclides in upper Hudson River sediment cores: implications for improved time stratigraphy and transport processes. *Chem. Geol.* **2003**, *199* (1–2), 53–70.

(38) Ferguson, P. L.; Iden, C. R.; Brownawell, B. J. Analysis of nonylphenol and nonylphenol ethoxylates in environmental samples by mixed-mode high-performance liquid chromatography-electrospray mass spectrometry. *J. Chromatogr. A* **2001**, *938* (1–2), 79–91.

(39) Boller, M. Tracking heavy metals reveals sustainability deficits of urban drainage systems. *Water Sci. Technol.* **1997**, *35* (9), 77–87.

(40) Maryland Department of Natural Resources, Fixed Monthly Water Quality Monitoring Program. Available at: http://mddnr. chesapeakebay.net/bay_cond/bay_cond.cfm?param=bdo& station=WT41 (accessed Nov 16, 2007).

(41) Wong, G. T. F. Alkalinity and ph in the southern Chesapeake Bay and the James River Estuary. *Limnol. Oceanogr.* **1979**, *24* (5), 970–977.

(42) Tixier, C.; Singer, H. P.; Canonica, S.; Muller, S. R. Phototransformation of triclosan in surface waters: a relevant elimination process for this widely used biocide — Laboratory studies, field measurements, and modeling. *Environ. Sci. Technol.* **2002**, *36* (16), 3482–3489.

(43) Bopp, R. F.; Chillrud, S. N.; Shuster, E. L.; Simpson, H. J.; Estabrooks, F. D. Trends in chlorinated hydrocarbon levels in Hudson River basin sediments. *Environ. Health Perspect.* **1998**, *106* (Suppl 4), 1075–1081.

(44) Ying, G.-G.; Yu, X.-Y.; Kookana, R. S. Biological degradation of triclocarban and triclosan in a soil under aerobic and anaerobic conditions and comparison with environmental fate modelling. *Environ. Pollut.* **2007**, *150*, 300–305.

(45) Cho, Y. C.; Sokol, R. C.; Rhee, G. Y. Kinetics of polychlorinated biphenyl dechlorination by Hudson River, New York, USA, sediment microorganisms. *Environ. Toxicol. Chem.* **2002**, *21* (4), 715–719.

(46) Kristensen, E. Organic matter diagenesis at the oxic/anoxic interface in coastal marine sediments, with emphasis on the role of burrowing animals. *Hydrobiologia* **2000**, *426* (1), 1–24.

(47) Sokol, R. C.; Kwon, O. S.; Bethoney, C. M.; Rhee, G. Y. Reductive dechlorination of polychlorinated-biphenyls in St. Lawrence River sediments and variations in dechlorination characteristics. *Environ. Sci. Technol.* **1994**, *28* (12), 2054–2064.

(48) Wiegel, J.; Wu, Q. Microbial reductive dehalogenation of polychlorinated biphenyls. *FEMS Microbiol. Ecol.* **2000**, *32* (1), 1–15.

(49) Ashley, J. T. F.; Baker, J. E. Hydrophobic organic contaminants in surficial sediments of baltimore harbor: inventories and sources. *Environ. Toxicol. Chem.* **1999**, *18* (5), 838–849.

(50) TCC Consortium. High Production Volume (HPV) Chemical Challenge Program Data Availability and Screening Level Assessment for Triclocarban CAS#: 101–20–2; U.S. Environmental Protection Agency: Washington D.C., 2002; pp 1–40.

(51) Recommendations for and Documentation of Biological Values for Use in Risk Assessment, PB-179874, 600/6–87/008; U.S. Environmental Protection Agency: Cincinnati, OH, 1988.

ES702882G