Environmental Risk Assessment of Personal Care Products

Babu Rajendran Ramaswamy

Abstract Extensive usage and continuous release of personal care products (PCPs) lead to ubiquitous contamination of aquatic environment. As PCPs are mainly intended for external use on the human body, they are not subjected to metabolic alterations; therefore, large quantities enter the environment as such. Being biologically active and persistent, they are expected to pose a wide range of risks to aquatic habitat. Although studies on environmental concentration and toxicity endpoints are available for many PCPs, environmental risk assessment (ERA) was scantily reported. It was observed that most of the ERAs were based on hazard/risk quotient approach and not following three-tier approach due to lack of sufficient toxicological data (i.e., long-term toxicity at environmentally relevant (ppt–ppb) concentrations). From the ERA reports, it was understood that disinfectants, triclosan and triclocarban, cause high risk to aquatic organisms. In case of preservatives (parabens), the risk was low. Some fragrances (synthetic musks) and UV filters were also shown to be toxic in the aquatic habitat; however, majority of them are categorized as less risky. Other than the risk to macro forms, the antibacterial PCPs are likely to affect the community structure of nontarget (nonpathogenic) bacteria and may aid in developing (multidrug) resistance among pathogenic and nonpathogenic species. Therefore, for better risk assessment, environmentally relevant studies on nontarget organisms are to be given due importance, and it may include interactions of chemical mixture, degradation products, and bioavailability criterion as well.

Keywords Antimicrobials, Bacterial resistance, Environmental risk assessment, Hazard quotient, Personal care products

B.R. Ramaswamy (✉)
Department of Environmental Biotechnology School of Environmental Sciences, Bharathidasan University, Tiruchirappalli, Tamil Nadu, India
e-mail: ramaswamybr@gmail.com

M.S. Díaz-Cruz and D. Barceló (eds.), *Personal Care Products in the Aquatic Environment*, Hdb Env Chem (2015) 36: 139–164, DOI 10.1007/698_2014_297,

Contents

1 Introduction

Chemical pollution by pesticides, biocides, pharmaceuticals and personal care products, industrial chemicals, etc., poses a greater (cumulative) threat to environment. Personal care products are a varied group of compounds comprising preservatives (e.g., parabens), disinfectants (e.g., triclosan), fragrances (e.g., musks), UV filters/stabilizers (e.g., methylbenzylidene camphor, benzotriazoles), and insect repellants (e.g., DEET). Millions of consumers use cosmetic/personal care products and their ingredients on a daily basis to improve the quality of life. The unavoidable growth in the use of cosmetics/PCPs burdens the environment with their residues. The global production of personal care products is expected to reach 333 billion dollars by 2015 [1].

Although PCPs provide various benefits to the quality of life of the consumer, viz., soap, shower gels, toothpaste are to maintain hygiene and dental care, deodorants prevent body odor, and sunscreens protect human skin against adverse effects of UV light, they are generally excreted and emitted through the sewerage/wastewater system after use and ultimately released into nearby terrestrial or aquatic systems (Fig. 1).

Chemicals used in personal care products are biologically active compounds that are designed to interact with specific pathways and processes in humans and animals. A number of personal care products have been identified in environmental matrices and drinking waters [3–7], and their concentrations in environmental matrices are mostly in the range of ng–μg level. Many PCPs are environmentally persistent and bioactive and have the bioaccumulation potential. Thus, humans and terrestrial/aquatic ecosystems are greatly exposed to unknown cocktail of chemicals of parent as well as transformed products. Environmental (chemical) risk assessments of transformed products are rather complex than parent

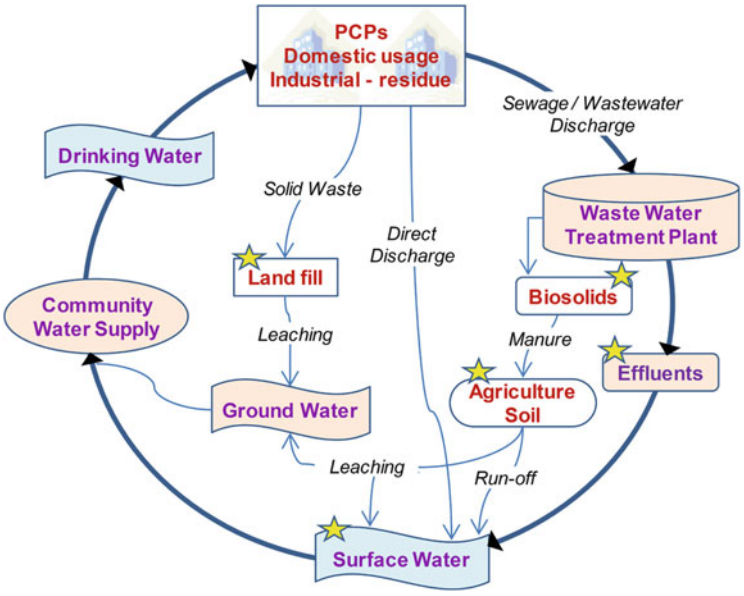

Fig. 1 Life cycle of PCPs in the environment with *star (mark)* showing the risk assessment (Adapted with modification from [2])

compounds due to scarce or nonavailability of toxicity data. The safety of a chemical in use is obviously based on a hypothetical zero-risk situation; however, that does not exist/or possible in a real-world situation. This peculiar, albeit unrealistic, aspect poses a major challenge for the risk assessment of chemicals and their ingredients/metabolites.

There have been a number of publications since the past few decades reporting on toxicity, fate, and transport of endocrine disrupting chemicals; nevertheless, information on residue levels and environmental risk assessment (ERA) of PCPs is scarce or nil until the end of the last century, and researchers started showing interest on analytical methods, bioaccumulation, and risk evaluation of PCPs only in the recent past.

Apart from the health risk to macroorganisms, the impact of PCPs on microbial community is still a question with few key outcomes. As we are aware, the prevalence of antibiotic-resistant bacteria in hospital, industrial [8], as well as domestic wastewater [9] environment is not uncommon; nevertheless, increasing use of antimicrobial compounds leads to similar problem of resistance in bacteria from sewage and surface water, drinking water, etc. [10–12]. Bacterial resistance for PCPs such as parabens in aquatic system is a growing environmental problem [11]. Moreover, a number of pollutants (i.e., pesticides, pharmaceuticals, illicit drugs, etc.) are continuously released into the environment, and their long-term effects on the receiving ecosystems are relatively unknown. Furthermore, interactions (synergistic/antagonistic) among the co-occurring compounds can also take

place, complicating environmental assessment [13]. Considering the importance of PCPs' emerging threat, this paper summarizes their risk assessment in the environment.

2 Pathways of Exposure and Uptake

The entry of PCPs into the aquatic environment includes direct disposal of domestic sewage and wastewater from hospitals and manufacturing industries, also they enter through wastewater treatment plant (WWTPs) effluents, leakage from septic tanks or leaching of landfill sites, and surface water runoff. The effluent and sludge from WWTPs and biosolids as manure shall be the prime source of PCPs in agriculture soil. The exposure of PCPs by organisms in the environment varies depending on the usage and resulting residual concentration/dilution in receiving waters, WWTP efficacy, and other possible exposure pathways.

The uptake of PCPs in aquatic ecosystem is mainly via contaminated water and secondarily by sediment. Some of the PCPs (e.g., triclosan) are ionizable substances, and the uptake of such ionizable substances depends on environmental conditions such as pH and soil/sediment characteristics. Mostly, the studies consider the bioavailability and uptake based on the properties such as octanol–water partition coefficient, bioconcentration/biomagnification factor, etc. [14]. However, no clear data on PCP uptake through food chain exists, so much research needs to be imparted to understand the real scale of PCPs bioavailability ([5, 14, 15] and references therein).

3 Methods of Risk Assessment

According to European commission [16], ERA is defined as an attempt to address the concern for the potential impact of individual substances on the environment by examining both exposures resulting from discharges and/or releases of chemicals and the effects of such emissions on the structure and function of the ecosystem.

Risk assessment identifies potential hazardous consequences of anthropogenic chemicals and determines the probability to occur in a specific environment (i.e., exposure assessment) and their severity (i.e., toxicity) [16]. Methods for assessing the ecological risks of anthropogenic pollutants are ample, and the most followed is the hazard quotient (HQ) approach [6, 16–18]. The quantitative approach to ERA includes three main components, viz., exposure assessment (predicted environmental concentration in different compartments such as water, soil/sediment, etc.), effect assessment (predicted no-effect concentration from dose–response relationship), and the risk characterization (calculating HQ). The hazard quotient or risk quotient (RQ) is calculated as the ratio between the predicted environmental concentration (PEC) or measured environmental concentration (MEC) and the

predicted no-effect concentration (PNEC) in organisms [17]. The HQ/RQ values <0.1, 0.1–1, and >1 indicate low, medium, and high risks, respectively, of the individual compound [4].

The PEC for PCPs can be calculated based on multiple factors like type of substance, sales, population density, and usage statistics, and it may vary for each country and/or region. Nowadays, developed countries like the USA started using computational models (e.g., E-FAST) to predict the flux of PCPs in waterways [19]; nevertheless, it is quite difficult to calculate for developing countries where substantial statistics on production, sale, exact population, effluent load, etc., are hard to collect. In such condition, the relative MEC of specific compound is used instead of PEC. For calculating PNEC, most of the studies rely on either short-term acute toxicity (e.g., LC50, EC50, etc.) or long-term (sub-)chronic toxicity outcomes (e.g., no observed effective concentration (NOEC), lowest observed effective concentration (LOEC), etc.). Often, NOEC is calculated for individual organisms based on their toxicity endpoints; however, single NOEC representing multiple organisms (based on acute/chronic toxicity results) can be calculated by software such as ecological structure activity relationships (ECOSAR) of United States Environmental Protection Agency (USEPA). Indeed, for proper assessment, cumulative effect (chronic toxicity: growth rate, fecundity, abnormalities, etc.) is always preferred over one-time acute toxicity assay, because chronic data provides much better idea for the "true" risk of chemicals or chemical group and significantly lowers the use of uncertainty in risk assessment [20].

In risk calculation, an uncertainty/safety assessment factor (e.g., 10, 100, 1,000, etc.) is applied to acute or chronic toxicity endpoints to arrive at the PNEC. This application of uncertainty factor is based on the nature/form of toxicological data for different classes of organisms in each level of hierarchy/food chain. Usually, a safety factor of 1,000 is applied for acute toxicity endpoints, whereas safety factor of 10 is applied for chronic toxicity [17]. In general, among PNECs the lowest value for a specific taxonomic group was used to estimate the maximum risk posed by the chemical of concern [20].

The conventional PNEC calculated for a compound or stressor may not represent wider species assemblage or population (natural community). Therefore, to determine PNEC which is protective for most species/population/community, species sensitivity distribution (SSD) approach is followed, which represents the cumulative probability distributions of toxicity values from multiple species. Therefore, SSD is used in many instances [15, 21, 22], rather than conventional (single species) approach ([23] and references therein).

Jjemba [24] proposed an ecotoxicity potential (EP) to assess the extent of the risk of pharmaceutical and personal care products (PPCPs) based on fate (i.e., degradability), exposure factor (i.e., bioavailability), and effect factor (i.e., susceptibility) of the substance of concern.

$$\mathrm{EP} = T/V(\mathrm{NOEC})$$

where T and V are the overall residence time and concentration of a substance in the environment, respectively. It is obvious that the lower the degradability (or the

higher the persistence) and/or the higher the bioavailability of a chemical to nontarget organisms, the higher the magnitude of ecotoxicity potential.

Conventional HQ predicts risk based on MEC or PEC obtained from limited area and may not necessarily reflect a risk for larger ecosystem (e.g., entire river stretch). To fill the gap, environmental exposure models are developed to more precisely determine (weigh) the nominal exposure, for large area, over a period of time. Apart from PEC and MEC, exposure assessment models use variables such as the pathways of contaminant, form of the chemical(s) released, and its fate in different environmental compartments. Models like P*h*ATE™ (Pharmaceutical Assessment and Transport Evaluation) and GREAT-ER (Geo-referenced Regional Exposure Assessment Tool for European Rivers) can be adopted for exposure assessments [25].

Apart from toxicity studies, computational approaches are gaining importance to replace/append the present risk prediction techniques (e.g., HQ), and one such approach is QSAR (quantitative structure activity relationship). Garcia et al. [26] performed the QSAR study using EPI Suite™ interface, to understand the possible adverse effects of 96 PPCPs and metabolites with negligible experimental data and established a ranking of concern based on persistence (P), bioaccumulation (B), and toxicity (T) (extensive) of those PPCPs in Spanish aquatic environments. Their findings revealed that higher number of metabolites has got ranking equal to or greater than their parent compounds. Further, P, B, and T indexes are recommended recently by the Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) regulation to estimate the potential negative impact of chemicals on the environment [26].

Regarding PCPs, most of the studies either report the environmental concentration or its toxicological profile; however, only few studies were performed for risk assessment. In the present review, literature-based risk assessments of PCPs pertaining to HQ were primarily collected and grouped in Tables 1 and 2. The worst-case scenario reported for organisms in each of the study was taken for discussion. Further, the main purpose of the review was to collectively present the available ERAs of PCPs.

4 Classification and Risk of PCPs

Regarding classification, each country adopts their own way of classification, e.g., sunscreens are cosmetics in the EU, whereas in the USA they are OTC drugs. Hair dyes are cosmetics in the EU but quasi-drugs in Japan, and their safety would be subjected to drug regulations necessitating drug-like safety dossiers [38]. Moreover, the PCPs can be grouped into categories based on their application (Fig. 2) such as antimicrobials (disinfecting agents and preservatives), insect repellants, fragrances (musks), UV filters/stabilizers, and siloxanes.

Table 1 Aquatic risk assessment of disinfectants and preservatives based on PNEC and MEC

Compound	Matrix	Country	Organism	Toxicity endpoint	PNEC (μg/L)	MEC/PEC (μg/L)	Maximum HQ/RQ/RCR	Risk	Reference
Disinfectants									
Triclosan	River water	Eight countries	*Pseudokirchneriella subcapitata*	NOEC[a]	0.053	1.023	>10	High	Tamura et al. [27]
			Danio rerio, *Ceriodaphnia dubia*		2.6–3	1.023	>0.1	Medium	
	River water	India	*D. magna*, *P. promelas*, *Lepomis macrochirus*, *O. mykiss*, *Oryzias latipes*, *D. rerio*	EC50[b]/LC50[b]/NOEC[b]	0.22–3.4	5.16	1.51–23.4	High	Ramaswamy et al. [6]
	River water/sediment	China	Aquatic organism	NOEC[a]	0.05	0.478	9.55–28.47	High	Zhao et al. [28]
	Lake water, WWTP effluent	USA	Aquatic organism	ECOSAR[a]	NA	0.041–0.85	1.2–11.8	High	Blair et al. [7]
	WWTP effluent	Greece	Invertebrates, fishes, algae	NOEC[a]/LC50[b]/EC50	NA	0.452	>0.1 to >100	Medium–high	Kosma et al. [29]
Triclocarban	River water	USA, China	*P. subcapitata*, *C. dubia*, *D. rerio*	NOEC[a]	0.19–2.4	5.6	>1 to >10	High	Tamura et al. [27]
	River water, sediment	China	Aquatic organism	NOEC[a]	0.058	0.338	5.83–24.54	High	Zhao et al. [28]
	Lake water, WWTP effluent	USA	Aquatic organism	ECOSAR[c]	NA	0.015–0.98	0.5 to >10	Medium–high	Blair et al. [7]

(continued)

Table 1 (continued)

Compound	Matrix	Country	Organism	Toxicity endpoint	PNEC (μg/L)	MEC/ PEC (μg/L)	Maximum HQ/RQ/ RCR	Risk	Reference
Resorcinol	River water	China	*P. subcapitata*, *C. dubia*	NOEC[a]	17–6,700	0.0531	>0.001 to >0.1	Low–medium	Tamura et al. [27]
p-Thymol	River Water	Japan	*P. subcapitata*, *C. dubia*, *D. rerio*	NOEC[a]	107–250	0.715	>0.01	Low	
Phenoxyethanol	River Water	Japan	*P. subcapitata*, *C. dubia*, *D. rerio*	NOEC[a]	580–13,000	14	>0.01	Low	
Preservatives									
Methyl, Ethyl, Isopropyl, Propyl, isobutyl, Butyl, Benzyl parabens	Surface water/ WWTP	Belgium, Canada, UK	*D. magna*/*P. promelas*	NOEC	NA	NA	0.00023–0.0000078	Unlikely	Dobbins et al. [30]
Methyl, *i*-butyl, benzylbutyl parabens	River Water	Japan	*O. latipes*	NOEC[a]/ LC50[b]/ EC50	NA	0.002–0.676	0.00032–0.0042	Low	Yamamoto et al. [31]
Ethyl, *n*-propyl, *i*-propyl, *n*-butyl parabens			*D. magna*	LC50[b]/ EC50	NA	0.046–0.207	0.017–0.00087	Low	
Methyl, Ethyl, Propyl, Butyl parabens	River water	India	*P. promelas*, *D. magna*	LOEC[a]	20–2,500	0.0432–11.3	0.000008–0.001	Low	Ramaswamy et al. [6]

ERA environmental risk assessment, *RQ* risk quotient, *RCR* risk characterization ratio, *HQ* hazard quotient, *PNEC* predicted no-effect concentration, *MEC* measured environmental concentration, *PEC* predicted environmental concentration

[a]Chronic

[b]Acute

[c]Predicted

Table 2 Aquatic risk assessment of synthetic musks and UV filters based on PNEC and MEC

Compound	Matrix	Country	Organism	Toxicity Endpoint	PNEC (μg/L)	MEC/PEC (μg/L)	Maximum HQ/RQ/RCR	Risk	Reference
Synthetic musks									
Toxalide	River Water	South Korea	Fish	NOEC	43.45	NA	>0.01	Low	Lee et al. [32]
Galaxolide, Musk ketone					0.646–6.8	NA	≥0.1	Medium	
Total musks					NA	NA	≥1	High	
Toxalide	NA	NA	Aquatic organisms	NOEC	3.5	0.3	0.086	Low	Balk and Ford [33]
			Fish-eating predators		10[a]	0.12[a]	0.012	Low	
			Sediment organisms		11[b]	0.48[b]	0.44	Medium	
			Soil organisms		0.32[b]	0.029[b]	0.091	Low	
			Worm-eating predators		10[a]	0.065[a]	0.007	No	
Galaxolide	NA	NA	Aquatic organisms	NOEC	6.8	0.5	0.074	Low	Balk and Ford [33]
			Fish-eating predators		100[a]	0.12[a]	0.001	No	
			Sediment organisms		25[b]	0.16[b]	0.064	Low	
			Soil organisms		0.32[b]	0.032[b]	0.1	Medium	
			Worm-eating predators		100[a]	0.099[a]	0.001	No	

(continued)

Table 2 (continued)

Compound	Matrix	Country	Organism	Toxicity Endpoint	PNEC (μg/L)	MEC/PEC (μg/L)	Maximum HQ/RQ/RCR	Risk	Reference
UV filters									
BP1	Surface water	Hong Kong	*P. promelas*	Vitellogenin induction[c]	4,919/2,668	15.5	>0.01	Low	Tsui et al. [34]
BP3			*O. latipes*	Egg production[c]	16	54.1	≥1	High	
			D. rerio	Transcriptional activity[c]	84	54.1	≥0.1	Medium	
			D. magna	EC50[c]/LC50	1,670/1,900	54.1	>0.01	Low	
			D. subspicatus	IC10[c]	560	54.1	≥0.1	Medium	
			Acropora sp.	Bleaching rate[c]	2,376	54.1	>0.01	Low	
BP4			*D. rerio*, *D. magna*	Transcriptional activity[c] LC50[c]	3,000–0,000	49.7	>0.01	Low	
EHMC			*O. latipes*, *P. promelas*, *D. rerio*	Transcriptional activity[c]	2.2–9,873	50.5	>0.01 to ≥1	Low–high	
			D. magna, *Desmodesmus subspicatus*	EC50[c]/LC50/IC10[c]	570/290/240	50.5	≥0.1	Medium	
			Acropora sp.	Bleaching rate[c]	1,999	54.1	>0.01	Low	
4MBC			*O. latipes*	Transcriptional activity[c]	9,922	20.7	>0.01	Low	
			D. magna, *D. subspicatus*	EC50[c]/LC50/IC10[c]	800/560/210	20.7	>0.01 to ≥0.1	Low–medium	
			Acropora sp.	Bleaching rate[c]	1,053	20.7	>0.01	Low	

BP1	NA	NA	*O. mykiss*	LOEC[c]	49.2	0.125	0.003	No	Fent et al. [35]
BP2			*O. mykiss*	LOEC[c]	12	0.125	0.01	Low	
BP3, BP4	NA	NA	*D. magna*	LOEC[c]/EC50[d]	6–50	0.44–0.849	0.02–0.07	Low	Fent et al. [36]
EHMC			*D. magna*, *O. latipes*	EC50[d]/LOEC[c]	0.29–9.9	0.39	0.04–1.35	Low–high	
E-PABA	NA	NA	*O. mykiss*	LOEC[c]	43.9	0.125	0.003	No	Fent et al. [35]
3BC	NA	NA	*D. magna*, *O. mykiss*	LOEC[c]	0.03	0.009–0.082	0.3–2.73	Medium–high	Fent et al. [35, 36]
4MBC	NA	NA	*D. magna*, *O. latipes*	EC50[d]/LOEC[c]	0.56–9.9	0.799	0.08–1.43	Low–high	Fent et al. [36]
EHMC	NA	NA	*Paracentrotus lividus*	EC10[d]	0.488	0.052	0.11	Medium	Paredes et al. [37]
BP3			*Isochrysis galbana*		0.037	0.068	1.86	High	
4MBC			*I. galbana*		0.054	0.084	1.57	High	

ERA environmental risk assessment, *RQ* risk quotient, *RCR* risk characterization ratio, *HQ* hazard quotient, *PNEC* predicted no-effect concentration, *MEC* measured environmental concentration, *PEC* predicted environmental concentration

[a]μg/g fw

[b]μg/g dw

[c]Chronic

[d]Acute

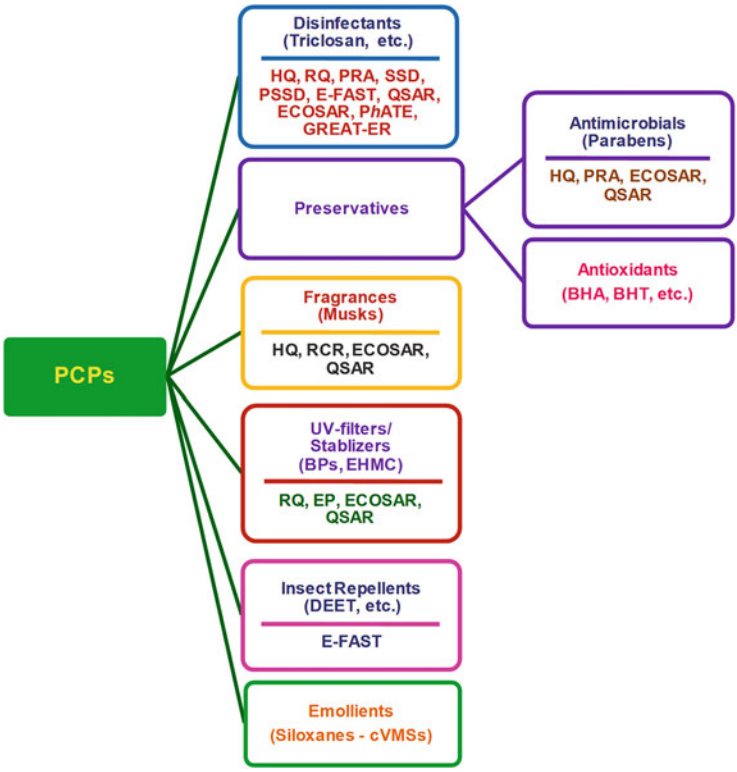

Fig. 2 Major classes of PCPs with examples in *parentheses* and available ERA

4.1 Disinfecting Agents

Disinfecting agents are antimicrobial compounds that are added as ingredients in sanitizers, disinfectants, and sterilants to control, prevent, or destroy harmful microorganisms (i.e., bacteria, viruses, or fungi). Since no single disinfectant is adequate for all situations, multiple disinfecting compounds are added in the formulations of PCPs [39].

Triclosan (TCS) (5-chloro-2-(2,4-dichlorophenoxy)phenol) and triclocarban (TCC) (3,4,4′-trichlorocarbanilide) are broadly used as antimicrobial and antifungal agents in household products of daily use (e.g., soaps, deodorants, skin creams, toothpaste and plastics, antimicrobial sprays, etc.). Due to extensive and inadvertent usage, residues of triclosan are ubiquitously found in surface water and sediment, WWTP influent/effluent, and fish ([6, 40] and references therein). Occasionally, fraction of TCS can occur as negative phenolate ion in environment due to its pKa (~8) and pH of the environment, which is considered to cause lesser toxicity than neutral (parent) form [41]. Further, Price et al. [41] opined that the risks of TCS

calculated based on PEC/PNEC ratio will be an overestimate, so aquatic toxicity evaluation based on speciation is warranted.

Both TCC and TCS, having a log Kow of 4.2–4.76, are highly expected to get adsorbed onto solids and sediments and thus available for bioaccumulation [42–44]. Bioaccumulation studies showed that higher pH in environment can favor TCS bioaccumulation whereas lower pH could favor methyl-TCS to accumulate more [5].

Ecological risk assessment based on acute and (sub-)chronic toxicity tests was mostly available only for five antimicrobial agents in which TCS in river water from various countries (Switzerland, Japan, the USA, Slovenia, Spain, the UK, China, and India) showed high risk based on HQ for algae and most of the fishes and medium risk for crustacean (*C. dubia*) [6, 27]. Zhao et al. [28] reported high risk of TCS in Pearl River (Liuxi, Shijing, and Zhujiang rivers) water and sediment from China with maximum HQ observed as 23.4 and 28.7, respectively. Aside from rivers, Michigan lake and STP effluent in the USA were also found to contain the TCS at high risk level based on ECOSAR PNEC [7]. In addition to surface water samples, Kosma et al. [29] reported that TCS in WWTP effluents discharged into the rivers in Greece (Kalamas, Arachthos, Acheloos, Grevenitis, and Aliakmonas) may pose high risk to algae (HQ >100), fish (HQ >1), and invertebrates (HQ >1) in outfall locations.

Similar to TCS, TCC was also found at alarming level in river water (the USA and China), showing HQ >10 [27]. Zhao et al. [28] also indicated higher risk of TCC in water (HQ = 5.8) and sediment (HQ = 24.54) from the tributaries of Pearl River in China. In Michigan lake water (in the USA), medium risk was reported for TCC; however, effluent entering into the lake showed high risk (HQ >10) [7]. From Table 1, it is prominent that most of the HQs obtained for TCS (15 results out of 18) and TCC (6 results out of 7) were >1, pointing their risk in the aquatic environment is more likely. Among other disinfectants, resorcinol showed low (algae, *P. subcapitata*) and medium risk (*C. dubia*) for river water in Japan, whereas *p*-thymol and phenoxyethanol were found with low risk for daphnia, algae, and fish. This indicates that the risk from *p*-thymol and phenoxyethanol in Japanese rivers is minimal, unlike TCS and TCC [27].

Apart from risk assessment based on individual MEC, Reiss et al. [45] performed probabilistic exposure estimation based on transport and fate of TCS in wastewater effluents in the USA by using a model. The study compared the estimated exposure concentration with PNEC of most sensitive species of algae, plant, fish, and invertebrates and reported that some sensitive algae and plants may be at risk at effluent outfall with meager dilution. Further, the risk at downstream of the river is considered less because of dissipation of triclosan. While HQ is mostly derived from individual PNEC, some of the studies have generated common PNEC by SSD. Capdevielle et al. [15] constructed SSD based on chronic toxicity values for 14 aquatic species including fish, invertebrates, macrophytes, and algae and predicted lower risk of TCS to pelagic species immediately downstream of wastewater treatment plant discharge points in rivers of Europe (GREAT-ER model based on Calder river) and the USA (P*h*ATE™ model based on 11 catchment

areas) by using a common PNEC of 1,550 ng/l. Further, Lyndall et al. [22] reported that 95th percentiles of measured and predicted TCS levels for water, sediment, and biota are consistently below the fifth percentile of the respective SSD, indicating no adverse effect of TCS.

The application of biosolids and wastewater containing TCC, TCS, and drugs to plant (soybean) showed higher accumulation of antimicrobials (at root tissue and beans) rather than drugs [46]; further it was reported that antimicrobials are not metabolized and thus accumulated whereas drugs can be eliminated/transformed by plants' metabolism. So similar bioconcentration condition may favor the bioaccumulation of antimicrobials in aquatic food chain also. While there are ample reports on fate and risks of parental compounds, investigation on risk assessment of their derivatives/metabolites is scantily found. For instance, methyltriclosan, having greater hydrophobicity and bioaccumulation potential than triclosan, is less studied for its toxicity. Therefore, the environmental risk assessment may not be complete unless data on major derivatives/metabolites are also available.

4.2 Antimicrobial Preservatives

Among preservatives, parabens (alkyl esters of *p*-hydroxybenzoic acid) are widely used as bacteriostatic and fungistatic agents in cosmetic (creams, skin lotions, shampoos, soaps, toothpaste, etc.), pharmaceutical, and food industries [3, 31]. There are seven different types of parabens currently in use (benzyl, butyl, ethyl, isobutyl, isopropyl, methyl, and propyl). Although reports on environmental occurrence of parabens are ample ([3] and references therein), environmental risk assessment was scantily carried out [6, 30, 31].

Probabilistic risk assessment (PRA) of parabens in *D. magna* and fathead minnow was performed by Dobbins et al. [30] based on acute and chronic toxicity data. The observed HQs based on NOEC were much lower ($7.8 \times 10^{-6} - 2.3 \times 10^{-4}$) than 1 (Table 1), which indicates no/little risk of parabens to fathead minnow and *D. magna* in surface waters of developed countries such as Belgium, Canada, and the UK [30]. Further, Yamamoto et al. [31] carried out an elaborate risk assessment for seven parabens in Tokushima and Osaka rivers in Japan. Unlike other studies, the NOEC values obtained from vitellogenin expression of fish were used, and the HQ showed no risk to aquatic organisms (algae, daphnia, and medaka) with the highest HQ obtained for *n*-propylparaben (0.01). Nevertheless, the sum of HQs of individual parabens showed low risk (HQ = 0.017) to those riverine organisms, and the PNEC based on *n*-butylparaben equivalence-based approach also showed low risk, with a maximum HQ of 0.018. They suggested that chronic tests at early life stages of fish are important for less erroneous risk assessment. Among developing countries, Ramaswamy et al. [6] evaluated the risk of four parabens in major rivers (Kaveri, Vellar, and Tamiraparani) of southern India. The lowest and highest HQs were observed for ethylparaben (8×10^{-6}) and butylparaben (0.001) to fish, respectively.

However, the calculated HQs for crustacean (*D. magna*) and fish (*P. promelas*) in all the rivers for all the parabens were below low risk criteria of 0.01.

4.3 Antioxidant Preservatives

Antioxidants are chemical substances used to prolong the shelf life of food items. Due to less stability of natural antioxidants, synthetic phenolic antioxidants (SPAs) like butylated hydroxytoluene (BHT) and butylated hydroxyanisole (BHA) are often preferred for their fat-soluble nature. The level of BHT was higher than triclosan and parabens in the rinse-off and leave-on cosmetics, respectively [47]. Their undisputed usage has resulted in trace quantities in food and environmental samples [26, 47]. Although BHA and BHT were classified as noncarcinogenic by the USEPA and safe food additives by the FDA and the EU, they possess estrogenic properties [48, 49]. Further, there are no environmental risk assessments available due to lack of toxicity data.

4.4 Insect Repellents

DEET (*N*,*N*-diethyl-meta-toluamide or *N*,*N*-diethyl-3-methylbenzamide), a broad-spectrum repellent and the most common active ingredient in insect repellents, is efficacious against mosquitoes and other insects of medical and veterinary importance. Till date, only few studies have reported acute toxicity in invertebrates, fish, and algae with EC50/LC50 in the range of 71.3–388 mg/l [5, 50]. Costanzo et al. [50] measured DEET residues in surface waters from Australia, Germany, the Netherlands, and the USA at safer level (75,000 times lower than EC50/LC50) for aquatic organisms such as algae (*Chlorella prothecoides*), water flea (*D. magna*), scud (*Gammarus fasciatus*), and fishes (*Pimephales promelas*, *Gambusia affinis*, *Oncorhynchus mykiss*). Further, Aronson et al. [19] estimated the flux of DEET in US rivers by iSTREEM (in-STREam Exposure Model) and E-FAST (Exposure and Fate Assessment Screening Tool) and predicted that DEET level was not expected to reach the lowest NOEC (521 mg/l) observed for algae, crustaceans, and fishes, indicating no risk of DEET in riverine habitat. Another insect repellent, 4-dichlorobenzene showed short-term exposure toxicity among invertebrates, fishes, and algae at lower concentration (1–60 mg/l) than DEET ([5] and references therein). Although newer repellents such as icaridin (1-piperidinecarboxylic acid 2-(2-hydroxyethyl) 1-methylpropyl ester) [51] and *m*-toluamide (*N*,*N*-diethyl-m-methylbenzamide) [52] are reported in the environment, their toxicity and risk assessment studies are not yet available.

4.5 Fragrances

Fragrances, the most widely used PCPs, seem to be omnipresent in the environment [3, 5]. Synthetic musks (SMs), being the most commonly used fragrances, are present in a wide range of products comprising deodorants, soaps, and detergents. Commonly used nitro musks are musk xylene (MX) and musk ketone (MK), whereas musk ambrette (MA), musk moskene (MM), and musk tibetene (MT) are used less frequently. In the case of polycyclic musks, celestolide (ABDI), galaxolide (HHCB), and toxalide (AHTN) are used most frequently, and traseolide (ATII), phantolide (AHMI), and cashmeran (DPMI) are used less often [3].

Although they are water-soluble compounds, due to high octanol–water partition coefficient of MK (log Kow = 3.8) and polycyclic musks (log Kow of 5.4–5.9), potential accumulation is expected in aquatic organisms. Rather than biomagnifications, direct impact on organisms is often understood by deriving HQ. In Nakdong River, South Korea, Lee et al. [32] reported low risk of toxalide and medium risk of galaxolide and musk ketone to fish. Combined risk of total SMs (Table 2) clearly indicates higher risk than individual, with higher contribution from MK. Apart from species-specific PNEC, Balk and Ford [33] used common PNEC to determine the risk of musks (AHTN and HHCB) in various environmental matrices, and the obtained HQ was always <1 (either no or low or medium risk). The risk characterized for AHTN based on NOEC for aquatic organism and fish-eating predators showed low risk (0.01–0.08), whereas medium risk (HQ = 0.44) was ascertained for sediment-dwelling organisms. For, HHCB, aquatic and sediment-bound organisms showed low risk, whereas no risk was determined for fish-eating predators. Interestingly, no risk was observed for worm-eating predators from HHCB (HQ = 0.001), even though medium risk (HQ = 0.1) was anticipated for soil organisms. Earlier, Tas et al. [53] also performed environmental risk assessment to understand the safety level of MK and MX in the Netherlands and found HQ ≤0.1 for both aquatic and sediment-dwelling organisms, while much lower HQ (0.01) was observed for fish-eating predators. Nevertheless, higher HQs were predicted for soil organisms with 0.5 for MK and 1.3 for MX, indicating medium to high risk, respectively and elevated HQ obtained for soil organisms implies the need for consideration of sludge being applied as fertilizer. Based on collective toxicity data and MECs, Brausch and Rand [5] suggested that probable risk for aquatic wildlife is more certain due to AHTN than other musks. However, chronic toxicity data on algae and benthic invertebrates are still lacking for effective aquatic risk assessment [5]. Apart from SMs, fragrances such as acetophenone, camphor, D-limonene, ethyl citrate, indole, isoborneol, isoquinolone, and skatole were also reported in surface waters. However, no acute/chronic toxicity data is available to evaluate their environmental risk [5].

4.6 UV Filters and Stabilizers

UV filters and stabilizers are found mainly in cosmetics and to some extent included in other personal care products, pharmaceuticals, food packaging, plastics, textiles, and vehicle maintenance products. Among organic and inorganic (ZnO, TiO_2) variants, the organic forms are mainly used. Currently, 27 UV filters were designated for use in cosmetics, plastics, etc., and they are used in combinations (up to eight compounds) ([5] and references therein). The common feature of organic UV filters is the presence of an aromatic moiety with a side chain having different degrees of unsaturation and forming benzophenones (BPs), 4-methyl-benzylidine-camphor (4MBC), 3-benzylidine-camphor (3BC), homosalate (HMS), 2-ethyl-hexyl-4-trimethoxycinnamate (EHMC), ethyl-PABA (E-PABA), etc. After usage (showering, wash-off, laundering, automobile servicing, etc.), these chemicals enter the aquatic system indirectly (major input) from wastewater treatment plants and directly due to recreational activities such as bathing and swimming in lakes, rivers, and coastal waters (beaches).

In the environment, they may stay for longer duration because of high lipophilicity (log Kow 4–8) and poor biodegradability and eventually accumulate in sediments and biota as well [5, 54]. Like many xenobiotics, sunscreens do cause effects on aquatic animals [3, 55]. Danovaro et al. [56] reported that UV filters (commercial sunscreens, MBC, ethylhexylmethoxycinnamate, octocrylene, BP3, etc.) at very low concentrations cause rapid and complete bleaching of corals. The BCF for 4-MBC in roach, *Rutilus rutilus*, was calculated (9,700–23,000) ten times lower than methyltriclosan having similar log Kow (5) [55]. Due to potential bioaccumulation and toxicity, use of sunscreen products is now banned in some of the famous tourist destinations including marine ecoparks in Mexico ([56] and references therein).

Several studies have reported degradation of UV filters by photolysis ([57] and references therein), and the ecotoxicological data on parental compounds and their degradation products is scarce. Even though little information is available on their toxicity, environmental concentrations suggest low potential risk [58]. However, Gago-Ferrero et al. [58] presumed long-term risk associated with its pseudo-persistency in the environment. According to Diaz-Cruz and Barcelo [59], most of the UV filters and their metabolites are found to elicit hormonal (estrogenic and androgenic) activities based on bioassays (Fig. 3). Five compounds (including four BPs) showed high estrogenic activity, whereas only two showed high androgenic activity, and this indicates that UV filters possess endocrine disrupting potential.

Based on risk assessment of UV filters (Table 1), among benzophenones, BP1 and BP4 were found at levels to cause low risk (HQ >0.01) to fish and daphnia, respectively. Another benzophenone (BP3) showed medium, low, medium, ow risks to fishes, crustaceans, algae, and corals, respectively, indicating the variable toxicity expected in aquatic community. For the same organisms, 4-methyl-benzylidine-camphor showed low risk (HQ >0.01), except for algae with medium risk (HQ ≥ 0.1). Similarly, EHMC also pose low to high risks over a range of organisms; particularly, high risk was assumed for fishes. As reported by Fent

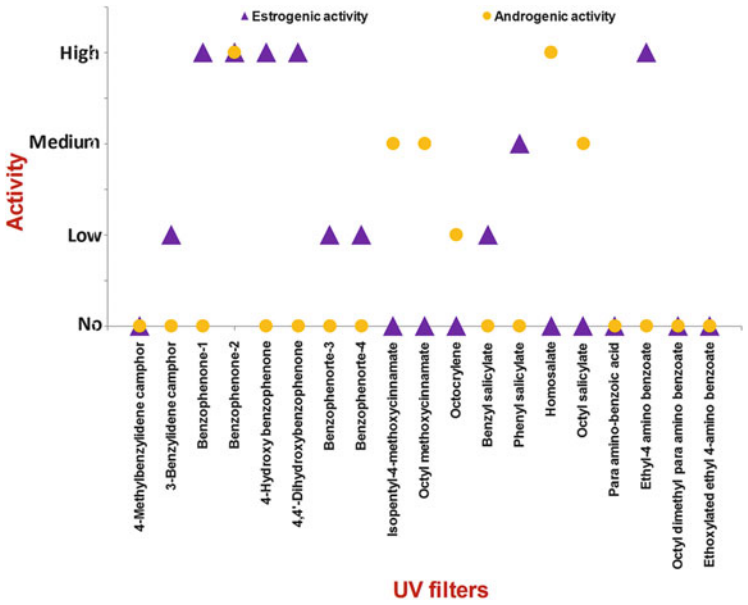

Fig. 3 Endocrine disrupting potentials of UV filters based on hormonal bioassays (*no*: activity not detected; *low*: submaximal dose–response curves with <30% efficacy; *medium*: submaximal dose–response curves with ≥30% efficacy; *max*: response curves with ≥80% efficacy) (From [59])

et al. [35, 36], 3BC can cause serious risk to *O. mykiss* (HQ = 2.73) and *D. magna* (HQ = 1.43), and EHMC too pose a risk to *D. magna* (HQ = 1.35). Among other compounds, BP1 and E-PABA showed no risk, and BP2-4 showed low risk to aquatic species. Fent et al. [36] suggested the consideration of additive interaction of UV filters in mixtures for risk assessment; they investigated the acute (48 h) and chronic (21 day) toxicities on *D. magna* and found that acute toxicity increased with lipophilicity. In case of sea urchin (*Paracentrotus lividus*) and microalgae (*Isochrysis galbana*), medium (EHMC) to high risk (BP3 and 4MCB) was observed by Paredes et al. [37], and they opined that RQ is dependent on the selection of assessment factor which is still a debatable topic indeed.

Apart from the above compounds, 2-hydroxyphenyl derivatives of benzotriazoles (BTZs) are also one of the major groups of UV stabilizers reported in surface waters and biota ([60, 61] and references therein). Regarding the toxicological status, few studies are available based on acute studies which suggested BTZs and their derivatives are nontoxic with NOEC at few μg/l level for freshwater and marine organisms [60] and suggested for more chronic toxicity data for the organisms in different food chain for deriving any conclusion relevant to environmental risk assessments.

4.7 Siloxanes

Siloxanes used in many PCPs and industrial coatings are now ubiquitously reported in freshwater and marine environment [62–64]. Mostly, cyclic volatile methylsiloxanes (cVMSs), commonly called as cyclosiloxanes, are widely added as carrier solvents and emollients in cosmetics and other PCP formulations. Therefore, now the concern is about their potential toxicity, transport, and fate in the environment [65]. So far, cVMSs have received very little attention in ecotoxicological research, i.e., hazards and risks to aquatic biota. Wang et al. [63] reviewed the toxicological properties of octamethylcyclotetrasiloxane (D4), decamethylcyclopentasiloxane (D5), and dodecamethylcyclohexasiloxane (D6) with their respective log Kow (4.45, 5.20, and 5.86) and suggested strong tendency of cVMSs to bind organic matter in soil and sediment. Further, the BCF for D4, D5, and D6 were reported in the range of 1,875–10,000, 3,362–13,300, and 1,600, respectively, with most of the studies confirming the bioaccumulative (>2,000) and very bioaccumulative (>5,000) criterion ([63] and references therein). Further, Wang et al. [63] observed the most sensitive fish toxicity (acute/chronic) values for cVMSs in the range of 4.4–69 μg/l; however, to our knowledge, no ERA has been performed.

4.8 Antibacterial Resistance

Apart from the toxicity of antimicrobials to macro life forms, the more affected are the nontargeted microbes in the environment. It may hamper the bacterial diversity in environment, thereby affecting the community structure. Ricart et al. [66] demonstrated that environmental concentration of TCS can eliminate 85% of bacterial population at 500 μg/l level. Moreover, the biocidal effect [67] can trigger antibacterial resistance among the bacterial community. Evidences are growing on the prevalence of multidrug-resistant bacteria in the environment, drinking water, and patients, especially in developing countries such as India [68, 69], and the antibiotic-resistant genes (ARGs) have been isolated from the surface water, sewage, and in hospital environment [10]. Such conditions lead to the emergence/transmission of antibiotic resistance among bacteria in the environment [68]. Although the contribution of antibacterials in antibiotic resistance or multidrug resistance is largely unknown, the scientific committee on emerging and newly identified health risks by the EU [70] pointed that antibacterial resistance may develop rapidly when exposed to preservative(s). Therefore, uncontrolled and continuous use of antimicrobial/preservative compounds (triclosan, triclocarban, parabens, etc.) may lead to resistance in bacteria. Recent studies confirmed antibacterial resistance of PCPs (parabens, triclosan) from wastewater and surface water [11, 71]. Selvaraj et al. [11] reported bacterial resistance in common pathogens in effluents of sewage treatment plants in India for parabens and suggested the

possible transfer of resistant genes to other pathogenic bacteria in natural waters because of the release of untreated wastewaters directly into the environment. Moreover, the resistant strains have the potency to modify PCPs into toxic compounds [72] which may further affect the organisms.

5 Present Risk and Future Prospective of PCPs in the Environment

On comparing the risk levels of major PCPs (Fig. 4), it is understood that most of the antimicrobials and UV filters showed medium to high risk whereas synthetic musks pose high risk only on total concentrations. Further, it clearly demonstrates that all the compounds within a group do not elicit similar toxicity but elicit cumulative risk. Apart from these three classes as shown in Fig. 4, reports on ERAs for antioxidants, fragrances, and siloxanes are lacking to be represented.

In most of the studies, ERAs were performed based on individual compound and not for mixtures present in the environment; therefore, it is critical to assess and to understand their activity in mixture (combinations). Further, for more appropriate environmental risk assessment of PCPs, it appears essential to consider not only mixtures of parent compounds but also degradation products (metabolites, photodegradates, and chlorination by-products). This may pose an undefined

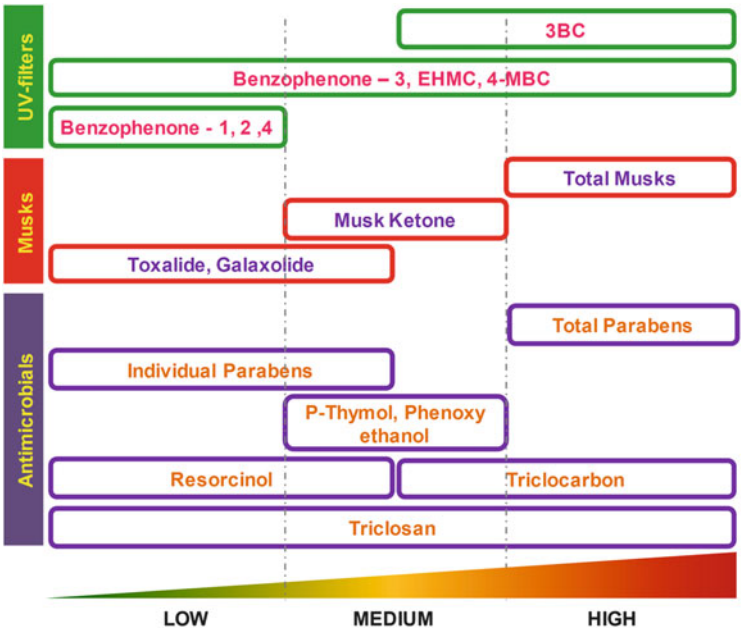

Fig. 4 Aquatic health risk of PCPs based on literature data [6, 7, 27–37]

ecotoxicological risk to resident organisms as well as a great challenge to ecotoxicologists. Moreover, testing chemical mixtures for toxicity is not an easy task due to the possible presence of thousands of organic and inorganic compounds (pollutants) in the environment; however, integrated dose–response relationships may be promising. In addition, the effect of individual components in the mixture can be extrapolated to understand/predict the cumulative effect via in silico approach, which can be further validated selectively through bioassays.

6 Conclusive Remarks

The impact of anthropogenic pollutants on the environment is severe and being given priority to understand well in this century. Despite their occurrence at submicrogram level in environment, the risk ascertained is quite high in many parts of the world. Developed countries are reporting high removal efficacy of WWTPs for few PCPs; however, higher risk is anticipated in developing countries where no proper treatment facilities are available. The exponential growth of population depletes freshwater resources and results in water shortage in this twenty-first century and in future. To combat water scarcity, reuse of wastewater is often advocated; such reuse has raised many questions with the occurrence of PCPs and other emerging chemicals residues.

Current environmental risk assessment procedures are limited in their proven ability to evaluate the combined effects of multiple xenoestrogens. Hence, ERA for mixtures (various forms of chemicals and their environmental derivatives) based on potential synergistic and/or antagonistic effects should be considered. As of the present situation, wider chronic toxicity studies should be imparted for many PCPs. Further, the effect of PCPs in the base of food chain may lead to adverse consequences through food chain magnification and ultimately on ecosystem. However, at present such scenario is entirely speculative and more appropriate studies to probe for this outcome have not yet been conducted holistically. Apart from the risk to aquatic organisms, some PCPs such as triclosan, parabens, etc., entering the aquatic environment may reduce the bacterial diversity and also act as buffers for the emergence of multidrug-resistant bacteria such as “superbug.” These concerns also need to be addressed for the safety of future generation.

References

1. GIA (2012) Global personal care products market to reach $333bn by 2015 says GIA. In: cosmeticsdesign-asia.com. http://www.cosmeticsdesign-asia.com/Market-Trends/Global-personal-care-products-market-to-reach-333bn-by-2015-says-GIA. Accessed 14 June 2014
2. Bedoux G, Roig B, Thomas O, Dupont V, Le Bot B (2012) Occurrence and toxicity of antimicrobial triclosan and by-products in the environment. Environ Sci Pollut Res Int 19:1044–1065

3. Daughton CG, Ternes TA (1999) Pharmaceuticals and personal care products in the environment: agents of subtle change? Environ Health Perspect 107:907–938
4. Hernando MD, Mezcua M, Fernandez-Alba AR, Barcelo D, Fern AR, Barcelo D (2006) Environmental risk assessment of pharmaceutical residues in wastewater effluents, surface waters and sediments. Talanta 69:334–342
5. Brausch JM, Rand GM (2011) A review of personal care products in the aquatic environment: environmental concentrations and toxicity. Chemosphere 82:1518–1532
6. Ramaswamy BR, Shanmugam G, Velu G, Rengarajan B, Larsson DGJ (2011) GC–MS analysis and ecotoxicological risk assessment of triclosan, carbamazepine and parabens in Indian rivers. J Hazard Mater 186:1586–1593
7. Blair BD, Crago JP, Hedman CJ, Klaper RD (2013) Pharmaceuticals and personal care products found in the Great Lakes above concentrations of environmental concern. Chemosphere 93:2116–2123
8. Marathe NP, Regina VR, Walujkar SA, Charan SS, Moore ERB, Larsson DGJ, Shouche YS (2013) A treatment plant receiving waste water from multiple bulk drug manufacturers is a reservoir for highly multi-drug resistant integron-bearing bacteria. PLoS One 8:e77310
9. Young TA, Heidler J, Matos-Perez CR, Sapkota A, Toler T, Gibson KE, Schwab KJ, Halden RU (2008) Ab initio and in situ comparison of caffeine, triclosan, and triclocarban as indicators of sewage-derived microbes in surface waters. Environ Sci Tech 42:3335–3340
10. Zhang XX, Zhang T, Fang HHP (2009) Antibiotic resistance genes in water environment. Appl Microbiol Biotechnol 82:397–414
11. Selvaraj KK, Sivakumar S, Sampath S, Shanmugam G, Sundaresan U, Ramaswamy BR (2013) Paraben resistance in bacteria from sewage treatment plant effluents in India. Water Sci Technol 68:2067–2073
12. Mao D, Luo Y, Mathieu J, Wang Q, Feng L, Mu Q, Feng C, Alvarez PJJ (2014) Persistence of extracellular DNA in river sediment facilitates antibiotic resistance gene propagation. Environ Sci Tech 48:71–78
13. Ginebreda A, Kuzmanovic M, Guasch H, de Alda ML, Lopez-Doval JC, Munoz I, Ricart M, Romaní AM, Sabater S, Barcelo D (2014) Assessment of multi-chemical pollution in aquatic ecosystems using toxic units: compound prioritization, mixture characterization and relationships with biological descriptors. Sci Total Environ 468–469:715–723
14. Hirsch (2013) Pharmaceuticals and personal care products. http://serc.carleton.edu/NAGTWorkshops/health/case_studies/pharmaceutical.html. Accessed 15 June 2014
15. Capdevielle M, Egmond ARV, Whelan M, Versteeg D, Hofmann-kamensky M, Inauen J, Cunningham V, Woltering D (2008) Consideration of exposure and species sensitivity of triclosan in the freshwater environment. Integr Environ Assess Manag 4:15–23
16. European Commission (2003) European union risk assessment report, Trichlorobenzene. http://echa.europa.eu/documents/%2010162/44180838-c246-4d42-9732-45e2af411e52. Accessed 15 June 2014
17. USEPA (1998) Guidelines for ecological risk assessment. http://www.epa.gov/raf/publications/pdfs/ECOTXTBX.PDF. Accessed 15 June 2014
18. Shanmugam G, Sampath S, Selvaraj KK, Larsson DGJ, Ramaswamy BR (2014) Non-steroidal anti-inflammatory drugs in Indian rivers. Environ Sci Pollut Res 21:921–931
19. Aronson D, Weeks J, Meylan B, Guiney PD, Howard PH (2012) Environmental release, environmental concentrations, and ecological risk of N, N-Diethyl-m-toluamide (DEET). Integr Environ Assess Manag 8:135–166
20. Mutiyar PK, Mittal AK (2013) Occurrences and fate of an antibiotic amoxicillin in extended aeration-based sewage treatment plant in Delhi, India: a case study of emerging pollutant. Desalin Water Treat 51:6158–6164
21. Kummerer K (2001) Pharmaceuticals in the environment. Springer, Berlin/Heidelberg
22. Lyndall J, Fuchsman P, Bock M, Barber T, Lauren D, Leigh K, Perruchon E, Capdevielle M (2010) Probabilistic risk evaluation for triclosan in surface water, sediments, and aquatic biota tissues. Integr Environ Assess Manag 6:419–440

23. Versteeg DJ, Belanger SE, Carr GJ (1999) Understanding single-species and model ecosystem sensitivity: data-based comparison. Environ Toxicol Chem 18:1329–1346
24. Jjemba PK (2006) Excretion and ecotoxicity of pharmaceutical and personal care products in the environment. Ecotoxicol Environ Saf 63:113–130
25. Cunningham VL (2001) Environmental exposure modeling: application of PhATE™ and GREAT-ER to human pharmaceuticals in the environment. In: Kummerer K (ed) Pharmaceuticals in the environment, 3rd edn. Springer, Heidelberg, pp 133–146
26. García ODS, Pinto GP, García-Encina PA, Mata RI (2013) Ranking of concern, based on environmental indexes, for pharmaceutical and personal care products: an application to the Spanish case. J Environ Manage 129:384–397
27. Tamura I, Kagota K, Yasuda Y, Yoneda S, Morita J, Nakada N, Kameda Y, Kimura K, Tatarazako N, Yamamoto H (2012) Ecotoxicity and screening level ecotoxicological risk assessment of five antimicrobial agents: triclosan, triclocarban, resorcinol, phenoxyethanol and p-thymol. J Appl Toxicol 33:1222–1229
28. Zhao J-L, Ying G-G, Liu Y-S, Chen F, Yang J-F, Wang L (2010) Occurrence and risks of triclosan and triclocarban in the Pearl River system, South China: from source to the receiving environment. J Hazard Mater 179:215–222
29. Kosma CI, Lambropoulou DA, Albanis TA (2014) Investigation of PPCPs in wastewater treatment plants in Greece: occurrence, removal and environmental risk assessment. Sci Total Environ 466–467:421–438
30. Dobbins LL, Usenko S, Brain RA, Brooks BW (2009) Probabilistic ecological hazard assessment of parabens using *Daphnia magna* and *Pimephales promelas*. Environ Toxicol Chem 28:2744–2753
31. Yamamoto H, Tamura I, Hirata Y, Kato J, Kagota K, Katsuki S, Yamamoto A, Kagami Y, Tatarazako N (2011) Aquatic toxicity and ecological risk assessment of seven parabens: individual and additive approach. Sci Total Environ 410–411:102–111
32. Lee I-S, Kim U-J, Oh J-E, Choi M, Hwang D-W (2014) Comprehensive monitoring of synthetic musk compounds from freshwater to coastal environments in Korea: with consideration of ecological concerns and bioaccumulation. Sci Total Environ 470–471:1502–1508
33. Balk F, Ford RA (1999) Environmental risk assessment for the polycyclic musks, AHTN and HHCB. II. Effect assessment and risk characterisation. Toxicol Lett 111:81–94
34. Tsui MMP, Leung HW, Lam PKS, Murphy MB (2014) Seasonal occurrence, removal efficiencies and preliminary risk assessment of multiple classes of organic UV filters in wastewater treatment plants. Water Res 53:58–67
35. Fent K, Kunz PY, Gomez E (2008) UV filters in the aquatic environment induce hormonal effects and affect fertility and reproduction in fish. CHIMIA Int J Chem 62:368–375
36. Fent K, Kunz PY, Zenker A, Rapp M (2010) A tentative environmental risk assessment of the UV-filters 3-(4-methylbenzylidene-camphor), 2-ethyl-hexyl-4-trimethoxycinnamate, benzophenone-3, benzophenone-4 and 3-benzylidene camphor. Mar Environ Res 69(Suppl): S4–S6
37. Paredes E, Perez S, Rodil R, Quintana JB, Beiras R (2014) Ecotoxicological evaluation of four UV filters using marine organisms from different trophic *levels Isochrysis galbana*, *Mytilus galloprovincialis*, *Paracentrotus lividus*, and *Siriella armata*. Chemosphere 104:44–50
38. Nohynek GJ, Antignac E, Re T, Toutain H (2010) Safety assessment of personal care products/cosmetics and their ingredients. Toxicol Appl Pharmacol 243:239–259
39. Dvorak G (2005) Disinfection 101. In: center for food security and public health 2160 veterinary medicine. http://www.coastalwaters2001.ecu.edu/cs-dhs/agromedicine/upload/Disinfection101Feb2005.pdf. Accessed 16 June 2014
40. Shanmugam G, Ramasamy K, Selvaraj KK, Sampath S, Ramaswamy BR (2014b) Triclosan in fresh water fish *Gibelion catla* from the Kaveri river, India and its consumption risk assessment. Environ Forensics 15:207–212

41. Price OR, Williams RJ, Egmond RV, Wilkinson MJ, Whelan MJ (2010) Predicting accurate and ecologically relevant regional scale concentrations of triclosan in rivers for use in higher-tier aquatic risk assessments. Environ Int 36:521–526
42. USEPA (2008) Ecological hazard and environmental revised risk assessment – Triclosan. http://www.oehha.org/prop65/public_meetings/052909coms/triclosan/ciba11.pdf. Accessed 15 June 2014
43. USEPA (2008) Ecological hazard and environmental revised risk assessment – Triclocarbon. http://www.epa.gov/hpvis/rbp/101-20-2_Triclocarban_Web_April%202009.pdf. Accessed 15 June 2014
44. Schebb NH, Flores I, Kurobe T, Franze B, Ranganathan A, Hammock BD, Teh SJ (2011) Bioconcentration, metabolism and excretion of triclocarban in larval Qurt medaka (*Oryzias latipes*). Aquat Toxicol 105:448–454
45. Reiss R, Mackay N, Habig C, Griffin J (2002) An ecological risk assessment for triclosan in lotic systems following discharge from wastewater treatment plants in the United States. Environ Toxicol Chem 21:2483–2492
46. Wu C, Spongberg AL, Witter JD, Fang M, Czajkowski KP (2010) Uptake of pharmaceutical and personal care products by soybean plants from soils applied with biosolids and irrigated with contaminated water. Environ Sci Tech 44:6157–6161
47. Sanchez-Prado L, Alvarez-Rivera G, Lamas JP, Lores M, Garcia-Jares C, Llompart M (2011) Analysis of multi-class preservatives in leave-on and rinse-off cosmetics by matrix solid-phase dispersion. Anal Bioanal Chem 401:3293–3304
48. Soto AM, Sonnenschein C, Chung KL, Fernandez MF, Olea N, Serrano FO (1995) The E-SCREEN assay as a tool to identify estrogens: an update on estrogenic environmental pollutants. Environ Health Perspect 103:113–122
49. Rodil R, Quintana JB, Basaglia G, Pietrogrande MC, Cela R (2010) Determination of synthetic phenolic antioxidants and their metabolites in water samples by downscaled solid-phase extraction, silylation and gas chromatography–mass spectrometry. J Chromatogr A 1217:6428–6435
50. Costanzo SD, Watkinson AJ, Murby EJ, Kolpin DW, Sandstrom MW (2007) Is there a risk associated with the insect repellent DEET (N, N-diethyl-m-toluamide) commonly found in aquatic environments? Sci Total Environ 384:214–220
51. Rodil R, Moeder M (2008) Stir bar sorptive extraction coupled to thermodesorption–gas chromatography–mass spectrometry for the determination of insect repelling substances in water samples. J Chromatogr A 1178:9–16
52. Mottaleb MA, Usenko S, O'Donnell JG, Ramirez AJ, Brooks BW, Chambliss CK (2009) Gas chromatography–mass spectrometry screening methods for select UV filters, synthetic musks, alkylphenols, an antimicrobial agent, and an insect repellent in fish. J Chromatogr A 1216:815–823
53. Tas JW, Balk F, Ford RA, van de Plassche EJ (1997) Environmental risk assessment of musk ketone and musk xylene in the Netherlands in accordance with the EU-TGD. Chemosphere 35:2973–3002
54. Giokas DL, Salvador A, Chisvert A (2007) UV filters: from sunscreens to human body and the environment. TrAC Trends Anal Chem 26:360–374
55. Balmer ME, Buser HR, Muller MD, Poiger T (2005) Occurrence of some organic UV filters in wastewater, in surface waters, and in fish from Swiss Lakes. Environ Sci Tech 39:953–962
56. Danovaro R, Bongiorni L, Corinaldesi C, Giovannelli D, Damiani E, Astolfi P, Greci L, Pusceddu A (2008) Sunscreens cause coral bleaching by promoting viral infections. Environ Health Perspect 116:441–447
57. Díaz-Cruz MS, Llorca M, Barcelo D (2008) Organic UV filters and their photodegradates, metabolites and disinfection by-products in the aquatic environment. TrAC Trends Anal Chem 27:873–887
58. Gago-Ferrero P, Díaz-Cruz MS, Barceló D (2012) An overview of UV-absorbing compounds (organic UV filters) in aquatic biota. Anal Bioanal Chem 404:2597–2610

59. Diaz-Cruz MS, Barcelo D (2009) Chemical analysis and ecotoxicological effects of organic UV-absorbing compounds in aquatic ecosystems. TrAC Trends Anal Chem 28:708–717
60. Janna H, Scrimshaw MD, Williams RJ, Churchley J, Sumpter JP (2011) From dishwasher to tap? Xenobiotic substances benzotriazole and tolyltriazole in the environment. Environ Sci Tech 45:3858–3864
61. Kim J-W, Isobe T, Ramaswamy BR, Chang K-H, Amano A, Miller TM, Siringan FP, Tanabe S (2011) Contamination and bioaccumulation of benzotriazole ultraviolet stabilizers in fish from Manila Bay, the Philippines using an ultra-fast liquid chromatography–tandem mass spectrometry. Chemosphere 85:751–758
62. Zhang Z, Qi H, Ren N, Li Y, Gao D, Kannan K (2011) Survey of cyclic and linear siloxanes in sediment from the songhua river and in sewage sludge from wastewater treatment plants, northeastern china. Arch Environ Contam Toxicol 60:204–211
63. Wang D-G, Norwood W, Alaee M, Byer JD, Brimble S (2013) Review of recent advances in research on the toxicity, detection, occurrence and fate of cyclic volatile methyl siloxanes in the environment. Chemosphere 93:711–725
64. Hong WJ, Jia H, Liu C, Zhang Z, Sun Y, Li YF (2014) Distribution, source, fate and bioaccumulation of methyl siloxanes in marine environment. Environ Pollut 191:175–181
65. Richardson SD, Ternes TA (2014) Water analysis: emerging contaminants and current issues. Anal Chem 86:2813–2848
66. Ricart M, Guasch H, Alberch M, Barcelo D, Bonnineau C, Geiszinger A, Farre ML, Ferrer J, Ricciardi F, Romaní AM, Morin S, Proia L, Sala L, Sureda D, Sabater S (2010) Triclosan persistence through wastewater treatment plants and its potential toxic effects on river biofilms. Aquat Toxicol 100:346–353
67. McMurry LM, Oethinger M, Levy SB (1998) Triclosan targets lipid synthesis. Nature 394:531–532
68. Kristiansson E, Fick J, Janzon A, Grabic R, Rutgersson C, Weijdegard B, Soderstrom H, Larsson DGJ (2011) Pyrosequencing of antibiotic-contaminated river sediments reveals high levels of resistance and gene transfer elements. PLoS One 6:e17038
69. Walsh TR, Weeks J, Livermore DM, Toleman MA (2011) Dissemination of NDM-1 positive bacteria in the New Delhi environment and its implications for human health: an environmental point prevalence study. Lancet Infect Dis 11:355–362
70. European Commission (2009) Scientific Committee on Emerging and Newly Identified Health Risks, assessment of the antibiotic resistance. Effects of biocides. http://ec.europa.eu/health/scientific_committees/consultations/public_consultations/scenihr_cons_09_en.htm. Accessed 30 June 2014
71. Middleton JH, Salierno JD (2013) Antibiotic resistance in triclosan tolerant fecal coliforms isolated from surface waters near wastewater treatment plant outflows (Morris County, NJ, USA). Ecotoxicol Environ Saf 88:79–88
72. Amin A, Chauhan S, Dare M, Bansal AK (2010) Degradation of parabens by *Pseudomonas beteli* and *Burkholderia latens*. Eur J Pharm Biopharm 75:206–212