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PAPER

Occurrence and behavior of pharmaceuticals, steroid hormones, and endocrine-disrupting personal care products in wastewater and the recipient river water of the Pearl River Delta, South China†‡

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The occurrence and behavior of β-blockers, antiepileptic drug carbamazepine and its metabolites, X-ray contrast agent iopromide, natural and synthetic hormones, and several groups of hormone-like personal care products (PCPs), including antiseptics (triclocarban, triclosan, and 2-phenylphenol), parabens and bisphenol A, were investigated in municipal wastewater, sewage sludge, and urban river water of the Pearl River Delta, South China. The pharmaceuticals, natural hormones and PCPs were ubiquitously detected in the raw wastewater from a sewage treatment plant (STP). Only triclocarban and triclosan were detected at significant amounts in the dewatered sludge. Iopromide and the PCPs were greatly removed/transformed from the aqueous phase of the wastewater. The β-blockers were only moderately removed/transformed. Carbamazepine passed through the STP almost unchanged. Biodegradation was the dominant process for elimination/transformation of the pharmaceuticals, hormones, and most PCPs in the STP. However, sorption also played an important role in the fate of triclocarban with nearly 50% of the mass load entering the STP ended up and persisted in the dewatered sludge. The pharmaceuticals, estrone, and PCPs were also widely detected in the Pearl River at Guangzhou. Bisphenol A had the highest concentration. The pharmaceutical concentrations in the Pearl River were higher in March than in May, most likely due to less dilution by lower precipitation. The omnipresence and high levels of the pharmaceuticals and PCPs in the Pearl River may be associated with direct discharge of untreated wastewater and pose potential risks to the ecological system.

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1. Introduction

The ubiquitous presence of pharmaceuticals and personal care products (PPCPs) in the environment has become an increasing issue due to their potential ecotoxicological effects.[1,2] Among the huge family of PPCPs, steroid hormones and hormone-like personal care products (PCPs) have received particular attention owing to their potent endocrine disrupting properties that may

Environmental impact

The residues of pharmaceuticals, steroid hormones and hormone-like personal care products (PCPs) in the environment have raised increasing concerns due to their ecotoxicological potencies. In this work, we: (1) investigated the occurrence of four natural estrogens, one synthetic estrogen, one progestogen, several groups of endocrine disrupting PCPs, β-blockers, a X-ray contrast agent and antiepileptic drugs in municipal wastewater; (2) discussed in depth the behavior, transport, and fate of these analytes in a sewage treatment plant by determining their concentrations in dissolved and particulate phases as well as concentrations at outlets of the major treatment units, including dewatered sludge; and (3) studied the distribution of these PPCPs in the Pearl River at Guangzhou. The dilution effect of precipitation on the seasonal pattern of the pharmaceuticals was discussed. To the best of our knowledge, the distribution of β-blockers, iopromide and carbamazepine in surface water in China has not been reported previously.

interrupt endocrine functions of wildlife at environmentally relevant levels, and due to their bioaccumulation.[3–6] Pharmaceuticals are designed to treat diseases for humans and animals. The acute toxicity threshold concentrations of pharmaceuticals are generally much higher than reported for environmentally relevant concentrations,[7] however, their presence in the environment still raise concerns about the unwanted effects on non-target organisms, chronic and mixed effects due to their bioactivity.[8,9] Recently, several PPCPs, including estrone, estradiol, triclosan, triclocarban and carbamazepine were classified as high priority trace pollutants.[10,11]

Sewage treatment plants (STPs) are major point sources of PPCPs in the environment due to incomplete elimination.[12–18] Several studies have documented the occurrence, behavior, and fate of PPCPs in wastewater, usually focusing on determining concentrations of the dissolved portion.[7,12,13,16,18,19–22] Nevertheless, a certain amount of these chemicals may be bound to solid fractions (*i.e.*, suspended particulate matter and sewage sludge) due to their moderate hydrophobicity, and thus potentially enter the environment *via* sewage sludge.[23–28] Therefore, it is necessary to concurrently determine these compounds in both liquid and solid phases and perform the analyses separately in order to get an insight into their abundance and fate (*e.g.*, sorption or degradation). In addition, although there have been extensive related research worldwide,[7,12,13,16,17,20,21,27–31] data on the pharmaceuticals and endocrine disrupting chemicals in wastewater and natural waters in mainland China are so far scarce.[32–34]

The Pearl River Delta (PRD) is one of the most densely populated and industrialized areas in China, with a population of about 80 million. The production of domestic and industrial wastewater in recent years is around 4.5 and 2 billion cubic meters, respectively, of which about 60% of the domestic and 90% of the industrial wastewater are treated before discharge.[35] The remaining untreated wastewater and all treated wastewater are discharged into the Pearl River, which is the most important water source of the PRD and directly links to the South China Sea. Recent studies revealed the wide presence of estrogens and some PCPs in the urban section of the Pearl River at Guangzhou, the largest city of the PRD.[32,33]

In this context, the aims of this work are to investigate the occurrence, behavior and fate of pharmaceuticals, steroid hormones and endocrine disrupting PCPs in wastewater from a large scale STP located in Guangzhou and to screen the distribution of these compounds in the urban section of the Pearl River at Guangzhou. Ultra-high performance liquid chromatography–tandem mass spectrometry (UHPLC-MS/MS) was applied to determine these PPCPs. Concentrations were determined through the major units of the STP, including the dewatered sludge to get an insight into the effect of different mechanisms (*e.g.*, degradation and sorption) on the behavior and fate of the PPCPs. The dissolved and sorbed concentrations of the PPCPs in wastewater samples were determined separately to elucidate the transport path and more accurately evaluate the mass balance and phase associations of the chemicals. To the best of our knowledge, occurrence of these pharmaceuticals in surface water in mainland China has not been reported previously. This work fills the data gap about these pharmaceuticals in wastewater and river water of China and might be of importance in understanding the global distribution of PPCPs in the environment.

2. Materials and methods

2.1 Materials

Estrone (E1), 17α-estradiol (αE2), 17β-estradiol (E2), estriol (E3), 17α-ethynylestradiol (EE2), medroxyprogesterone (MedP), ethylparaben (EP), propylparaben (PP), butylparaben (BP), 2-phenylphenol (PHP), triclocarban (TCC), bisphenol A (BPA), metoprolol (MPL), propranolol (PPL), carbamazepine (CBZ) and dihydrocarbamazepine (10,11-CBZ) were purchased from Sigma-Aldrich (Oakville, ON, Canada). Methylparaben (MP) and triclosan (TCS) were bought from Fluka (Buchs, Switzerland). Iopromide (IPM) was bought from United States Pharmacopeia (Rockville, MD, USA). Deuterated compounds were supplied by C/D/N isotopes (Pointe-Claire, Quebec, Canada) except BPA-d_{16} and E2-d_3 that were bought from Sigma-Aldrich. EE2-$^{13}C_2$ was obtained from Cambridge Isotope Laboratories (Andover, MA, USA). The standards are of at least 97% purity. Some key physicochemical parameters of the investigated compounds are shown in Table 1.

2.2 Sample collection

The investigated STP is located in a densely populated area in Guangzhou and serves a population of about 2.5 million. It has three parallel treatment systems with a total capacity of 550 000 m^3 d^{-1}. The first and second treatment systems treat predominantly domestic wastewater (~90%) and use identical treatment processes consisting of a screen, a grit chamber, a bioreactor comprised successively of an anaerobic tank, an anoxic tank and an oxic tank followed by a secondary clarifier. The third treatment system has a bioreactor consisting successively of an anoxic tank, an anaerobic tank and an oxic tank and also receives a certain amount of industrial wastewater and landfill leachate. Chlorination is employed before final discharge of treated effluent. The hydraulic retention time is 11.5 h and sludge age is about 10 days. The daily production of dewatered sludge is 400 tons.

Sampling of wastewater was conducted on a weekday in May 2008. Wastewater was collected hourly from 8 : 00 to 12 : 00 am to build a 40 L composite sample. The influent, effluents from the anaerobic tank and secondary clarifier and the final effluent were sampled along the first treatment system. The influent and final effluent samples were also collected along the third treatment system in order to assess mass balance of the PPCPs within the STP. A dewatered sludge sample was also collected.

The Pearl River flows through Guangzhou city from west to east and merges into the South China Sea at the Pearl River Estuary (Fig. 1). Thirteen sampling sites were set along the Pearl River at Guangzhou. Samplings were performed twice, once in March and once in May 2008. Samples were always collected during ebbing period to prevent dilution from tidal influxes.

The water samples were stored into amber glass bottles without headspace. Sodium azide (0.5 g L^{-1}) was added immediately after sampling to suppress potential biodegradation. The sludge sample was wrapped with clean aluminium foil and sealed in a zip lock polyethylene bag. Samples were kept cold on ice during transport to the laboratory where the water samples were stored at 4 °C in darkness until treatment within 48 h from collection and the sludge sample was stored at −20 °C.

Table 1 Physicochemical parameters and method quantification limit (MQL) of the investigated compounds[d]

Compound	log K_{ow}[a]	Water solubility[a] (mg L^{-1}, 25 °C)	MQL (ng L^{-1})		Internal standard
			Surface water	Wastewater	
Iopromide (IPM)	−2.05	23.8	8.3	42	PPL-d_7
Metoprolol (MPL)	1.88	16900	0.3	1.2	PPL-d_7
Propranolol (PPL)	3.48	61.7	0.2	0.5	PPL-d_7
Carbamazepine (CBZ)	2.45	17.7	0.1	0.5	CBZ-d_{10}
Dihydrocarbamazepine (10,11-CBZ)	2.46	16.8	0.1	0.5	CBZ-d_{10}
Medroxyprogesterone (MedP)	3.50	2.95	0.2	0.4	E1-d_4
Estrone (E1)	3.13	30	0.1	0.2	E1-d_4
17α-estradiol (αE2)	3.94	3.9	0.1	0.2	E2-d_3
17β-estradiol (E2)	4.01[b]	3.6[b]	0.1	0.3	E2-d_3
Estriol (E3)	2.45	441	0.1	0.2	E2-d_3
17α-ethynylestradiol (EE2)	3.67	11.3	0.1	0.2	EE2-d_4
17α-ethynylestradiol-$^{13}C_2$ (EE2-$^{13}C_2$) [SS]			0.1	0.2	EE2-d_4
Methylparaben (MP)	1.96	2500	0.2	0.4	MP-d_4
Ethylparaben (EP)	2.47	850	0.2	0.4	MP-d_4
Propylparaben (PP)	3.04	500	0.1	0.3	PP-d_4
Butylparaben (BP)	3.57	207	0.1	0.3	PP-d_4
2-Phenylphenol (PHP)	3.09	700	0.1	0.2	PP-d_4
Bisphenol A (BPA)	3.32	120	0.2	0.5	BPA-d_{16}
Triclosan (TCS)	4.76	1.97–4.6[c]	0.1	0.2	TCS-d_3
Triclocarban (TCC)	4.9	0.65–1.55[c]	0.2	0.5	TCC-d_4

[a] http://www.syrres.com/esc/physdemo.htm. [b] Ref. 22. [c] Ref. 48. [d] SS = surrogate standard.

2.3 Sample preparation and analysis

Water samples were filtered through baked (450 °C, 4 h) 0.7 μm glass fiber filters (GF/F, Whatman, Maidstone, England). The filtrate and suspended particulate matter (SPM) retained on GFFs were analyzed separately. An aliquot of the filtrate (150 mL for the influent and effluent from the anaerobic tank and 400 mL for the other water samples) was spiked with the surrogate and internal standards (See Table 1) at 50 ng L^{-1} of each compound, added with sodium chloride at 0.1 mol L^{-1}, and pH adjusted to 7.0 with 10 mmol L^{-1} potassium acid phthalate buffer and sodium tetraborate buffer prior to enrichment by solid phase extraction (SPE) on an Oasis HLB cartridge (Waters, Milford, MA, USA). The cartridge was preconditioned successively with 3 × 2 mL ethyl acetate, methanol and ultrapure water. The samples were loaded at a flow rate of 5 mL min^{-1}. After sample passage, the cartridge was rinsed with 5 mL of 5% methanol solution and vacuum dried for 10 min. The analytes were then eluted with 3 × 2 mL of methanol. The eluate was evaporated to just dryness under a gentle stream of nitrogen and reconstitute into 0.4 mL of acetonitrile followed by filtration through a 0.22 μm syringe filter (Anpel, Shanghai, China). The pharmaceuticals, MedP, and TCC were directly determined by UHPLC-MS/MS. For determination of the estrogens and phenolic PCPs, 200 μL of the extract was further treated with derivatization by dansyl chloride according to the procedure detailed previously.[36] Briefly, 200 μL of sodium bicarbonate solution (100 mmol L^{-1}, pH adjusted to 10.5 with sodium hydroxide) and 200 μL of dansyl chloride (1 mg mL^{-1} in acetone) were added into the dried extract and then derivatized at 60 °C for 5 min. After cooling to room temperature, the derivatized sample was then treated by liquid–liquid extraction with *n*-hexane and further cleaned up on a silica gel column. The sample was finally reconstituted in 0.2 mL of acetonitrile prior to UHPLC-MS/MS analysis.

Preparation of the sludge sample and SPM followed a procedure detailed elsewhere.[36] Briefly, the lyophilized and homogenized sludge/SPM sample was spiked with the surrogate and internal standards and extracted by ultrasonic assisted extraction with acetonitrile–water (5 : 3, v/v). The extract was concentrated and diluted with ultrapure water prior to further SPE treatment as described above.

Chemical determination was performed on an Agilent HPLC 1200 system coupled to an Agilent 6410 triple quadrupole MS (Agilent, Palo Alto, CA, USA). The analysis was done with electrospray ionization in negative mode for TCC and in positive mode for the other analytes. Chromatographic separation was achieved on a Zorbax Eclipse XDB C18 rapid resolution high throughput column (2.1 mm × 50 mm, 1.8 μm particle size) fitted with a 4 mm C18 guard column (Phenomenex, Torrance, CA, USA). The chromatographic conditions, specific multiple reaction monitoring ion transitions and retention times of the analytes and internal standards as well as the other MS parameters have been provided in detail elsewhere.[36] Quantification was performed by internal standard method. The isotope-labeled internal standard for each analyte is shown in Table 1.

2.4 Quality assurance and quality control

Recovery tests were performed by spiking the analytes at several concentrations in various environmental matrices. A surrogate standard (EE2-$^{13}C_2$) was used in analysis of environmental samples to further monitor the method efficiency. Procedural blanks and instrumental blanks were set in each batch of

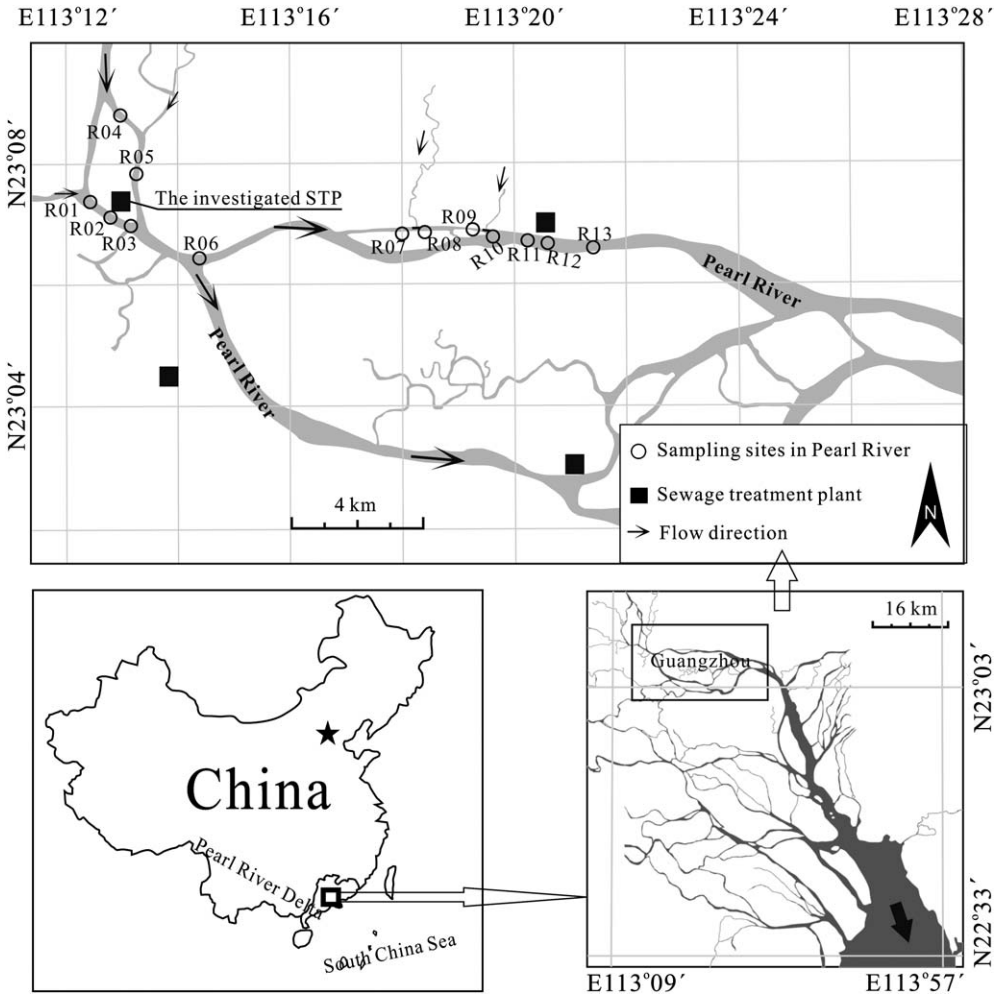

Fig. 1 Study area and sampling sites.

6 samples. Recoveries of the analytes were 40–111% in wastewater samples, 58–119% in river water samples and 65–124% in sludge samples. Surrogate recoveries were 73–118% in all the samples. Sample based method quantification limits of the analytes were 0.2–42 ng L^{-1} in wastewater, 0.1–8.3 ng L^{-1} in river water (Table 1) and 0.1–3 ng g^{-1} dry weight (dw) in sludge.[36] A trace amount of BPA was detected in procedural blanks and was appropriately subtracted from the reported concentrations of the samples. Relative standard deviations for replicate analyses of the environmental samples ranged from 1.3% to 17.9%. Detailed information about the QA/QC procedure was provided elsewhere.[36]

3. Results and discussion

3.1 Occurrence of the pharmaceuticals, hormones and personal care products in wastewater and sewage sludge

E3, EE2, and 10,11-CBZ were not quantifiably detected in any sample and therefore will be excluded from the following discussion.

IPM, CBZ, MPL and PPL were detected at 10 400, 51.6, 121.0 and 9.7 ng L^{-1}, respectively, in the influent of the first treatment system (Fig. 2a, Table S1‡), generally higher than those in the third system, which were 206, 45.8, 54.3 and 4.6 ng L^{-1} (Table S1‡), respectively, probably due to the difference in wastewater sources as described above. The IPM concentrations decreased sharply after treatment in the STP to 124 and 17 ng L^{-1} in the final effluent in the first and third systems, respectively. By contrast, the concentrations of MPL declined moderately to 88.3 ng L^{-1} in the final effluent in the first system but kept unchanged in the third system. The PPL concentrations showed moderate reduction, with 2.7–4.3 ng L^{-1} in the final effluent samples. The concentrations of CBZ remained unchanged in both systems. In the dewatered sludge, CBZ, MPL and PPL were detected only at trace levels (<10 ng g^{-1} dw).[36] These results were comparable or lower than those reported in other countries.[7,12,14,20,21,29,37–41]

E1, αE2 and MedP were detected in the influent samples from both systems, ranging from 5.0–66.8 ng L^{-1}, with the highest concentration observed for E1. E2 was only detected in the first system at 9.4 ng L^{-1} (Fig. 2a, Table S1‡). No hormone compounds were quantifiable in the final effluents and only E1 was detected at 22 ng g^{-1} dw in the dewatered sludge. These results fell into ranges reported in the literature.[13,15,18,19,29,39,42–45]

The PCPs were ubiquitously detected in the wastewater samples (Fig. 2b, Table S1‡). In the untreated wastewater samples, the highest concentration was found for BPA in the

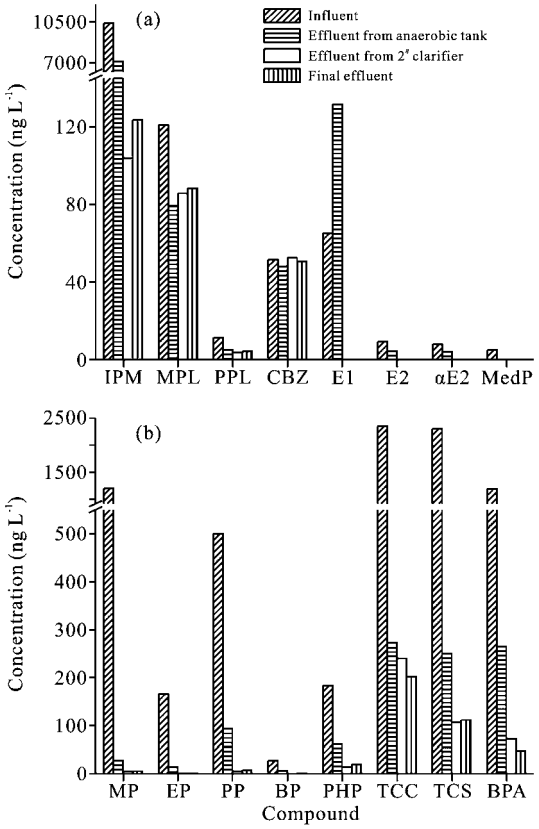

Fig. 2 Concentrations of the investigated pharmaceuticals, steroid hormones (a) and personal care products (b) in the wastewater of the first treatment system in a sewage treatment plant at Guangzhou. IPM = iopromide; MPL = metoprolol; PPL = propranolol; CBZ = carbamazepine; E1 = estrone; E2 = 17β-estradiol; αE2 = 17α-estradiol; MedP = medroxyprogesterone; MP = methylparaben; EP = ethylparaben; PP = propylparaben; BP = butylparaben; PHP = 2-phenylphenol; TCC = triclocarban; TCS = triclosan; BPA = bisphenol A.

third system (13 808 ng L^{-1}), probably due to the presence of industrial wastewater and landfill leachate in which very high concentrations of BPA was observed (data not shown). TCC (1217–2354 ng L^{-1}), TCS (712–2301 ng L^{-1}), and MP (1002–1194 ng L^{-1}) were also detected at low μg L^{-1} levels. Concentrations of the other PCPs were at tens to hundreds of ng L^{-1}. The concentrations decreased greatly after treatment in the STP and were at low ng L^{-1} in the final effluent for all the PCPs. In the dewatered sludge, TCC was the most abundant (5088 ng g^{-1} dw), followed by TCS (1188 ng g^{-1} dw). MP, PP, PHP and BPA were only detected at several to tens ng g^{-1} dw. EP and BP were not quantifiable. The results were comparable with those in wastewater and sludge of Canada, Australia and Switzerland.[16,26,27,46,47] However, concentrations of TCC and TCS in raw wastewater and sludge appeared lower than those in the U.S.[24,25,48]

3.2 Fate of the PPCPs in the sewage treatment plant

While it was difficult to coordinate sample collection with plant hydraulic residence time and minimize sample comparison uncertainties, Fig. 2 also roughly illustrates the transport and behavior of the investigated PPCPs along the first system in the STP. The pharmaceuticals were transported mainly in the aqueous phase, most likely due to their high water solubility and low octanol/water partition coefficients (Table 1). Their behavior appeared compound specific (Fig. 2a). The IPM concentration decreased moderately after anaerobic process and kept declining, with less than 5% remaining in the effluent from the secondary clarifier. Results about biodegradation of IPM are inconsistent in the literature. Ternes and Hirsch[14] reported that IPM was not readily biodegradable. However, appreciable degradation by cleavage of a side chain of iopromide was observed in laboratory tests.[49,50] Batt *et al.*[51] reported enhanced biodegradation of IPM in nitrifying activated sludge with a percent removal of 61% in a STP of the U.S. The concentrations of MPL and PPL were reduced by about 35% and 50%, respectively, which occurred primarily after the anaerobic process. Moderate to poor biodegradation of MPL and PPL has been reported previously.[18,21,41,52,53] However, PPL was also reported to be eliminated by 60–90% in Germany STPs.[12,54] On the other hand, CBZ passed through the STP unchanged. The persistence of CBZ has been widely reported.[17,38,55–57] The mass load ending up in the dewatered sludge accounted for 0.6, 2 and 11% of the inflow for CBZ, MPL and PPL, respectively (Fig. 3a), indicating that sorption was of little significance in the fate of pharmaceuticals.

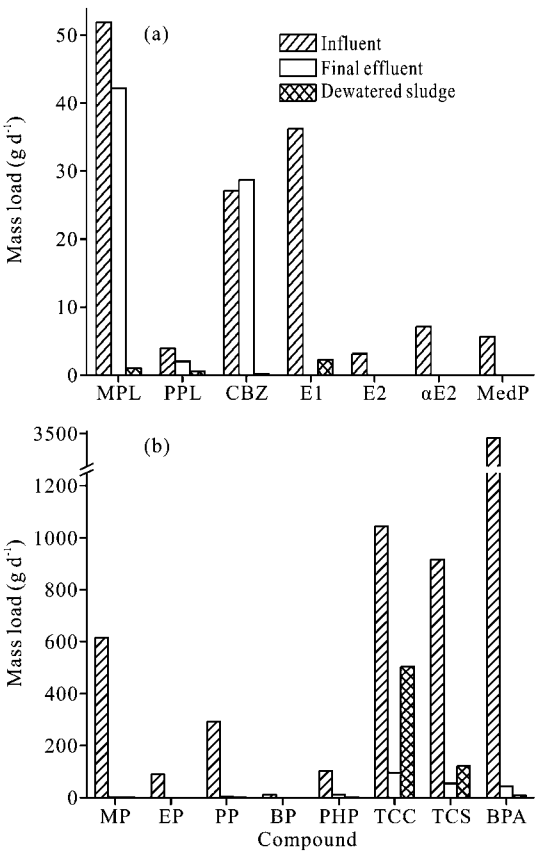

Fig. 3 Mass load of pharmaceuticals, steroid hormones (a) and personal care products (b) in a sewage treatment plant at Guangzhou. See Fig. 2 for full names of the abbreviated compounds.

Previous research also reported negligible adsorption of the pharmaceuticals to solids.[14,41,53,54]

After the anaerobic process, the concentration of E1 increased, probably due to cleavage of its conjugates to parent compound and/or conversion between E2, αE2 and E1.[58,59] Cleavage of estrogen conjugates (glucuronides and sulfates) into the parent compounds occurring in the first denitrification tank has been observed in a German STP.[42] The concentrations of the other hormones began to decrease from the outlet of the anaerobic tank. However, estrogens were found not to be degraded appreciably under anaerobic conditions.[42] No hormone compounds were quantifiable in the effluent from the secondary clarifier. In addition, only about 6% of E1 was found to be present in the dewatered sludge (Fig. 3a). A negligible mass load of estrogens absorbed onto sludge has been reported previously.[19,42,44] The results suggested that the hormones in the wastewater were efficiently removed/transformed by biodegradation. High biodegradation rate of estrogens, especially in aerobic condition has been extensively reported in the literature.[17,18,42,43] Clara *et al.*[37] reported elimination of natural estrogens was dependent on solid retention time (SRT), demonstrating efficient removal of estrogens in STPs with an SRT higher than 10 days. The SRT of the investigated STP is 10 days, which may be associated with the observed good removal/transformation of the estrogens.

Parabens and PHP were predominantly (>97%) present in the aqueous phase of the influent, probably attributed to their high water solubility (Table 1). On the other hand, 66–82% TCS and 82–86% TCC were sorbed onto the SPM in the raw wastewater, which may be due to their moderate lipophilicity with logK_{ow} of 4.8–4.9 (Table 1). Strong sorption of TCC to particulate matter in wastewater has been reported previously.[24] Concentrations of all the PCPs decreased significantly at the outlet of the anaerobic tank and kept decreasing in the following biological treatment (Fig. 2b, Table S1‡). Relative to the mass flows entering the STP, the mass loads were 0–1.6%, 1.3%, 11.8%, 6.0% and 9.1% in the final effluent and 0–0.4%, 0.3%, 3.3%, 13.2% and 48.4% in the dewatered sludge for parabens, BPA, PHP, TCS and TCC, respectively (Fig. 3b), indicating that biodegradation was the major mechanism for elimination/transformation of most of the PCPs except TCC for which sorption to sludge played an important role. Parabens and PHP have been observed to be largely removed in Swiss STPs.[47] Biodegradation of BPA was revealed previously.[44] Inconsistent results have been obtained for fate of TCS in wastewater. Singer *et al.*[46] reported the result in a Sweden STP similar to this work, in which only 15% of TCS was sorbed onto sludge and 6% remained in the treated effluent. Removal rates of TCS in five Australian STPs were 72–93% and biological degradation was believed to be the predominant removal mechanism while adsorption onto sludge also played a significant role.[27] About 30% of TCS in wastewater was sorbed to the sludge, while about 5% was emitted *via* the effluents in a Germany STP.[23] On the other hand, Heidler and Halden[25] observed that about half of the TCS mass remained in sludge and only <50% was biotransformed or lost due to other mechanisms in a STP in the U. S. Persistence and strong sorption of TCC in wastewater has been revealed, with 76 ± 30% of TCC accumulated in sludge.[24] Chlorinated disinfection led to no significant losses for all the PPCPs. Recalcitrance of these PPCPs to chlorination has been reported previously.[24,60]

3.3 Occurrence of the PPCPs in the Pearl River

IPM, CBZ and MPL were omnipresent in the urban section (R01–R13) of the Pearl River at Guangzhou with a median concentration of 89.7, 9.1, and 24.8 ng L^{-1}, respectively, whereas PPL was not quantifiable. These results were comparable or lower than those reported worldwide.[12,14,20,21,29,40,52,56,61] E1 was the most frequently detected hormone compound, with a median and maximum concentration of 5.6 and 21.3 ng L^{-1}, respectively. E2 and αE2 were only occasionally detected with a maximum concentration of 4.8 and 6.5 ng L^{-1}, respectively. The PCPs were ubiquitously detected. BPA was the most abundant, followed by PHP, TCS and TCC. Parabens were at low ng L^{-1} levels in the Pearl River (Fig. 4a, Table S2‡) although MP and PP were detected at μg L^{-1} levels in wastewater, probably due to their liable biodegradation as discussed above. However, the PCP concentrations were much higher than those reported in rivers in North America and Europe despite comparable concentrations in STP effluents,[47,56,62] which may be ascribed to partial treatment of wastewater (about 60% for domestic and 90% for industrial wastewater) in the Pearl River Delta. Omnipresence of the investigated PPCPs even at sites upstream of STP outfalls, *e.g.*, R1 and R4 (Fig. 1) also suggests the presence of direct wastewater discharge. Elevated concentrations of the PPCPs at sites immediately downstream of STP outfalls, *i.e.*, R3 and R13 (Fig. 1) indicates that STPs are also important point sources of these chemicals in the Pearl River. However, more work is needed to accurately apportion sources of the PPCPs in the Pearl River, including contributions from STPs upstream of Guangzhou and from direct wastewater discharge.

The median concentrations of the pharmaceuticals (*i.e.*, IPM, CBZ and MPL) were obviously higher in March than in May (Fig. 4b, Table S2‡); whereas no statistical seasonal differences were found in the concentrations of the PCPs. Seasonal comparison cannot be made for the hormones due to low detection frequency and concentrations. Higher concentrations of β-blockers and other pharmaceuticals were observed in rivers in winter than in other seasons in Sweden, which was ascribed to weaker biodegradation owing to cold temperature.[21,61] Relatively lower TCS concentrations were observed in surface waters in summer in Switzerland and Australia, which was believed to be caused by stronger photodegradation and faster biodegradation due to the higher temperatures, respectively.[27,46] However, neither temperature nor sunshine duration in March and May is likely to result in a significant difference in biodegradation or photodegradation (Table 2). Therefore, a smaller dilution by lower precipitation (Table 2) appears a likely significant factor for the higher pharmaceutical concentrations in March in the Pearl River. A dilution effect by water flow on the occurrence of PPCP contaminants was also reported for the stream waters in the U.S.[63] However, the cause of the seasonal distribution pattern of the PCPs in the Pearl River cannot be unambiguously elucidated based on the limited data in this work, probably due to a mixed effect of dilution by rainfall, seasonally different consumption of these compounds and other unidentified factors. Nevertheless, more data,

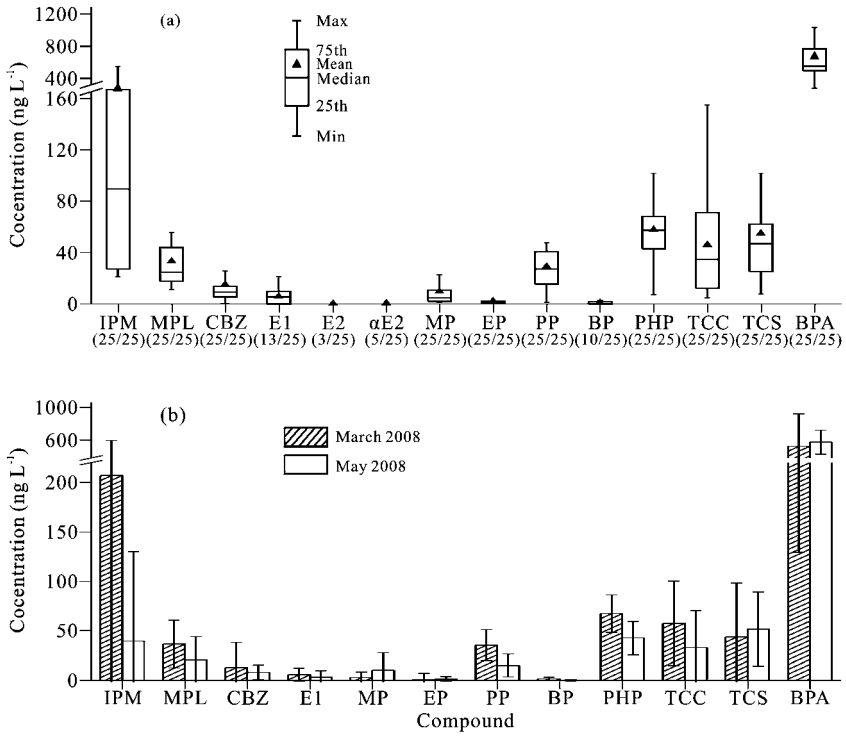

Fig. 4 Distribution (a) and seasonal patterns (b) of the PPCPs in the Pearl River at Guangzhou, South China. Numbers in the parentheses are quantifiable samples *versus* total analyzed samples. See Fig. 2 for full names of the abbreviated compounds.

Table 2 Precipitation, sunshine duration and average temperature during the sampling months[a]

	Precipitation (mm)	*T* (°C)	Sunshine (h)
March 2008	70.9	20.1	100.9
May 2008	285.2	25.6	69.1

[a] http://www.stats.gov.cn.

including detailed flow information, are needed to discuss in detail the seasonal effect on distribution, loads, mass balance and fate of the PPCPs in the Pearl River.

4. Conclusion

Occurrence of several groups of pharmaceuticals, hormones and hormone-like personal care products were investigated in wastewater and urban river water of the Pearl River Delta, South China. Concentrations were determined in the influent and effluents from the major units as well as the sludge in a large scale STP to gain insight into the mechanisms impacting the behavior and fate of these chemicals.

The X-ray contrast agent iopromide, antiepileptic drug carbamazepine, β-blockers, natural estrogens, medroxyprogesterone, parabens, 2-phenylphenol, triclosan, triclocarban and bisphenol A were widely detected in the raw wastewater of the Pearl River Delta, South China. No hormones were quantifiable in the treated effluent. Iopromide and the PCPs were significantly removed/transformed from the aqueous phase of the wastewater. β-blockers were only moderately removed. Carbamazepine passed through the STP almost unchanged. Biodegradation was the dominant process for elimination/transformation of the PPCPs in the STP. However, sorption played an important role in the fate of triclocarban with nearly 50% of the mass load entering the STP finally ending up in the dewatered sludge. The high level of TCC in the dewatered sludge may be of concern because it can enter and persist in the terrestrial environment *via* disposal of sludge and consequently pose risks to the health of ecological system.

The pharmaceuticals, estrone, and PCPs were also ubiquitous in the Pearl River at Guangzhou. BPA was the most abundant compound. The omnipresence and high levels of the PPCPs in the Pearl River may be associated with direct discharge of untreated wastewater and may become an environmental issue.

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References

1 C. G. Daughton and T. A. Ternes, *Environ. Health Perspect.*, 1999, **107**, 907–938.
2 K. Kümmerer, *J. Environ. Manage.*, 2009, **90**, 2354–2366.
3 E. Silver, N. Rajapakse and A. Kortenkamp, *Environ. Sci. Technol.*, 2002, **36**, 1751–1756.

4 S. Jobling, D. Casey, T. Rodgers-Gray, J. Oehlmann, U. Schulte-Oehlmann, S. Pawlowski, T. Baunbeck, A. P. Turner and C. R. Tyler, *Aquat. Toxicol.*, 2004, **66**, 207–222.
5 J. V. Brian, C. A. Harris, M. Scholze, T. Backhaus, P. Booy, M. Lamoree, G. Pojana, N. Jonkers, T. Runnalls, A. Bonfa, A. Marcomini and J. P. Sumpter, *Environ. Health Perspect.*, 2005, **113**, 721–728.
6 T. A. Roepke, M. J. Snyder and G. N. Cherr, *Aquat. Toxicol.*, 2005, **71**, 155–173.
7 D. B. Huggett, I. A. Khan, C. M. Foran and D. Schlenk, *Environ. Pollut.*, 2003, **121**, 199–205.
8 M. Oetken, G. Nentwig, D. Loffler, T. Ternes and J. Oehlmann, *Arch. Environ. Contam. Toxicol.*, 2005, **49**, 353–361.
9 S. F. Owen, E. Giltrow, D. B. Huggett, T. H. Hutchinson, J. Saye, M. J. Winter and J. P. Sumpter, *Aquat. Toxicol.*, 2007, **82**, 145–162.
10 P. H. Howard and D. C. G. Muir, *Environ. Sci. Technol.*, 2010, **44**, 2277–2285.
11 K. E. Murray, S. M. Thomas and A. A. Bodour, *Environ. Pollut.*, 2010, **158**, 3462–3471.
12 T. A. Ternes, *Water Res.*, 1998, **32**, 3245–3260.
13 C. Baronti, R. Curini, G. D'Ascenzo, A. D. Corcia, A. Gentili and R. Samperi, *Environ. Sci. Technol.*, 2000, **34**, 5059–5066.
14 T. A. Ternes and R. Hirsch, *Environ. Sci. Technol.*, 2000, **34**, 2741–2748.
15 M. Carballa, F. Omil, J. M. Lema, M. Llompart, C. García-Jares, I. Rodríguez, M. Gómez and T. Ternes, *Water Res.*, 2004, **38**, 2918–2926.
16 H. Lee, T. E. Peart and M. L. Svoboda, *J. Chromatogr., A*, 2005, **1094**, 122–129.
17 N. Nakada, T. Tanishima, H. Shinohara, K. Kiri and H. Takada, *Water Res.*, 2006, **40**, 3297–3303.
18 V. Gabet-Giraud, C. Miège, J. M. Choubert, S. M. Ruel and M. Coquery, *Sci. Total Environ.*, 2010, **408**, 4257–4269.
19 O. Braga, G. A. Smythe, A. I. Schäfer and A. J. Feitz, *Environ. Sci. Technol.*, 2005, **39**, 3351–3358.
20 N. M. Vieno, T. Tuhkanen and L. Kronberg, *J. Chromatogr., A*, 2006, **1134**, 101–111.
21 N. M. Vieno, H. Harkki, T. Tuhkanen and L. Kronberg, *Water Res.*, 2007, **41**, 1001–1012.
22 R. A. Trenholm, B. J. Vanderford, J. C. Holady, D. J. Rexing and S. A. Snyder, *Chemosphere*, 2006, **65**, 1990–1998.
23 K. Bester, *Water Res.*, 2003, **37**, 3891–3196.
24 J. Heidler, A. Sapkota and R. U. Halden, *Environ. Sci. Technol.*, 2006, **40**, 3634–3639.
25 J. Heidler and R. U. Halden, *Chemosphere*, 2007, **66**, 362–369.
26 S. Chu and C. D. Metcalfe, *J. Chromatogr., A*, 2007, **1164**, 212–218.
27 G. Ying and R. S. Kookana, *Environ. Int.*, 2007, **33**, 199–205.
28 A. S. Stasinakis, G. Gatidou, D. Mamais, N. S. Thomaidis and T. D. Lekkas, *Water Res.*, 2008, **42**, 1796–1804.
29 S. D. Kim, J. Cho, I. S. Kim, B. J. Vanderford and S. A. Snyder, *Water Res.*, 2007, **41**, 1013–1021.
30 Z. Moldovan, G. Schmutzer, F. Tusa, R. Calin and A. C. Alder, *J. Environ. Monit.*, 2007, **9**, 986–993.
31 F. Sacher, M. Ehmann, S. Gabriel, C. Graf and H. J. Brauch, *J. Environ. Monit.*, 2008, **10**, 664–670.
32 X. Peng, Y. Yu, C. Tang, J. Tan, Q. Huang and Z. Wang, *Sci. Total Environ.*, 2008, **397**, 158–166.
33 J. Zhao, G. Ying, L. Wang, J. Yang, X. Yang, L. Yang and X. Li, *Sci. Total Environ.*, 2009, **407**, 962–974.
34 W. Xue, C. Wu, K. Xiao, X. Huang, H. Zhou, H. Tsuno and H. Tanaka, *Water Res.*, 2010, **44**, 5999–6010.
35 http://www.gdstats.gov.cn/tjnj/table/19/c19_1.htm.
36 Y. Yu, Q. Huang, J. Cui, K. Zhang, C. Tang and X. Peng, *Anal. Bioanal. Chem.*, 2011, **399**, 891–902.
37 M. Clara, N. Kreuzingera, B. Strenn, O. Gans, E. Martinez and H. Kroiss, *Water Res.*, 2005, **39**, 97–106.
38 X. Miao, J. Yang and C. D. Metcalfe, *Environ. Sci. Technol.*, 2005, **39**, 7469–7475.
39 S. Castiglioni, R. Bagnati, R. Fanelli, F. Pomati, D. Calamari and E. Zuccato, *Environ. Sci. Technol.*, 2006, **40**, 357–363.
40 A. C. Alder, C. Schaffner, M. Majewsky, J. Klasmeier and K. Fenner, *Water Res.*, 2010, **44**, 936–948.
41 M. Scheurer, M. Ramil, C. D. Metcalfe, S. Groh and T. A. Ternes, *Anal. Bioanal. Chem.*, 2010, **396**, 845–856.
42 H. Andersen, H. Siegrist, B. Halling-Sørensen and T. A. Ternes, *Environ. Sci. Technol.*, 2003, **37**, 4021–4026.
43 A. Joss, H. Andersen, T. Ternes, P. Richle and H. Siegrist, *Environ. Sci. Technol.*, 2004, **38**, 3047–3055.
44 J. Y. Hu, X. Chen, G. Tao and K. Kekred, *Environ. Sci. Technol.*, 2007, **41**, 4097–4102.
45 V. Kumar, N. Nakada, M. Yasojima, N. Yamashita, A. C. Johnson and H. Tanaka, *Chemosphere*, 2009, **77**, 1440–1446.
46 H. Singer, S. Müller, C. Tixier and L. Pillonel, *Environ. Sci. Technol.*, 2002, **36**, 4998–5004.
47 N. Jonkers, H. E. Kohler, A. Dammshäuser and W. Giger, *Environ. Pollut.*, 2009, **157**, 714–723.
48 R. U. Halden and D. H. Paull, *Environ. Sci. Technol.*, 2005, **39**, 1420–1426.
49 W. Kalsch, *Sci. Total Environ.*, 1999, **225**, 143–153.
50 T. Steger-Hartmann, R. Länge, H. Schweinfurth, M. Tschampel and I. Rehmann, *Water Res.*, 2002, **36**, 266–274.
51 A. L. Batt, S. Kim and D. S. Aga, *Environ. Sci. Technol.*, 2006, **40**, 7367–7373.
52 D. Bendz, N. A. Paxéus, T. R. Ginn and F. J. Loge, *J. Hazard. Mater.*, 2005, **122**, 195–204.
53 M. Ramil, T. E. Aelf, G. Fink, M. Scheurer and T. A. Ternes, *Environ. Sci. Technol.*, 2010, **44**, 962–970.
54 A. Wick, G. Fink, A. Joss, H. Siegrist and T. A. Ternes, *Water Res.*, 2009, **43**, 1060–1074.
55 R. Andreozzi, R. Marotta, G. Pinto and A. Pollio, *Water Res.*, 2002, **36**, 2869–2877.
56 B. Kasprzyk-Hordern, R. M. Dinsdale and A. J. Guwy, *Water Res.*, 2008, **42**, 3498–3518.
57 T. Kosjek, H. R. Andersen, B. Kompere, A. Ledin and E. Heath, *Environ. Sci. Technol.*, 2009, **43**, 6256–6261.
58 C. P. Czajka and K. L. Londry, *Sci. Total Environ.*, 2006, **367**, 932–941.
59 R. Gomes, M. Scrimshaw and J. Lester, *Environ. Sci. Technol.*, 2009, **43**, 3612–3618.
60 M. M. Huber, S. Korhonen, T. A. Ternes and U. V. Gunten, *Water Res.*, 2005, **39**, 3607–3617.
61 A. Daneshvar, J. Svanfelt, L. Kronberg, M. Prévost and G. A. Weyhenmeyer, *Chemosphere*, 2010, **80**, 301–309.
62 W. Hua, E. R. Bennett and R. J. Letcher, *Environ. Int.*, 2005, **31**, 621–630.
63 D. W. Kolpin, M. Skopec, M. T. Meyer, E. T. Furlong and S. D. Zaugg, *Sci. Total Environ.*, 2004, **328**, 119–130.

