The use of common swift *(Apus apus)*, an aerial feeder bird, as a bioindicator of persistent organic microcontaminants

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Summary. In this study we investigated the accumulation of polychlorobiphenyls (PCBs), polychlorodibenzo-*p*-dioxins (PCDDs), polychlorodibenzofurans (PCDFs), and the chlorinated pesticides 1,1-dichloro-2,2-*bis*(4-chlorophenyl)-ethene (DDE), 1,1,1-trichloro-2,2-*bis*(4-chlorophenyl)-ethane (DDT), and hexachlorobenzene (HCB) in the breast muscle, liver, lung, heart and brain tissues of adult common swifts (*Apus apus*, a long-lived aerial feeder bird). Individuals were collected in an urban area located in Rome during the breeding period. As shown by lipid-base normalized data, in general analytes had a significant minimum concentration in the brain. PCDD and PCDF concentration values in such tissue were approximately one order of magnitude lower than those found in breast muscle, heart, and lung tissues, and as much as two orders of magnitude below the relatively high levels found in the liver. PCB levels followed the same accumulation patterns. Of all analytes, HCB exhibited the most uniform distribution pattern over the five matrices assayed. DDE and DDT were by far the most and the least concentrated pesticide. In the urban environment of Rome, an air-to-swift bioconcentration factor (lipid based) in the order of 5×10^6 (2×10^5, fresh tissue base) was estimated for PCDDs and PCDFs. Our study suggests that airborne arthropod feeders, such us the common swift, are suitable biomonitors for air quality assessment.

Key words: organochlorine contaminants, POPs, air contamination, bioindicators, common swift, PCDDs and PCDFs.

Riassunto *(Il rondone comune "*Apus apus, Aves*", un insettivoro aereo specializzato, come biondicatore di contaminanti organici persistenti)*. È stata quantificata la presenza nel rondone comune *(Apus apus)* di policlorobifenili (PCBs), policlodibenzodiossine (PCDDs), policlorodibenzofurani (PCDFs), e dei pesticidi clorurati 1,1-dicloro-2,2-*bis* (4-clorofenil)-etene (DDE), 1,1,1-tricloro-2,2-*bis* (4-clorofenil)-etano (DDT), e esaclorobenzene (HCB). Esemplari di rondoni adulti sono stati raccolti a Roma durante il periodo riproduttivo. Come mostrato dai dati normalizzati sul grasso corporeo, in generale gli analiti hanno una concentrazione minima significativa nel cervello. Le concentrazioni di PCDD e PCDF in questo tessuto risultavano approssimativamente un ordine di grandezza inferiori a quelle trovate in muscolo pettorale, cuore e tessuto polmonare, e fino a due ordini di grandezza inferiori ai livelli relativamente alti trovati nel fegato. I livelli di PCB misurati nel cervello avevano le stesse caratteristiche di distribuzione. Di tutti gli analiti, l'HCB esibiva la distribuzione più uniforme di contaminazione tra tutte le matrici analizzate mentre DDE) e DDT erano di gran lunga i pesticidi più e meno concentrati. Nell'ambiente urbano di Roma è stato stimato un fattore di bioconcentrazione aria-rondone per PCDDs e PCDFs dell'ordine di 5×10^6 (2×10^5, peso fresco).

Parole chiave: contaminanti organoclorurati, contaminanti organici persistenti, contaminazione dell'aria, bioindicatori, rondone comune, policlorodibenzo-p-diossine, policlorodibenzofurani.

INTRODUCTION

The ubiquitous polychlorobiphenyls (PCBs), polychlorodibenzo-*p*-dioxins (PCDDs), polychlorodibenzofurans (PCDFs), and chlorinated pesticides such as 1,1-dichloro-2,2-*bis* (4-chlorophenyl)-ethene (DDE), 1,1,1-trichloro-2,2-*bis*(4-chlorophenyl)-ethane (DDT), and hexachlorobenzene (HCB) are lipophilic organic microcontaminants of the environment characterized by a remarkable environmental persistence, potential for bioconcentration, and a range of possible adverse effects on wildlife populations including carcinogenicity and interference with the endocrine system [1, 2]. Furthermore, in humans several of these compounds exhibit a strong toxic action, including carcinogenity, even at very low exposure levels [3-8].

Many authors have suggested the use of birds as

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monitors of pollutants in terrestrial and especially aquatic environments, and fish-eating birds or raptors high on the foodchain have been primarily used [9]. Some studies have demonstrated that insectivorous passerine birds are suitable monitors for air, heavy metal, and organochlorine pollution [10, 11]. However, to our knowledge no effort has been attempted to find bioindicators specific for the urban environment and air pollution, while the number of species/populations adopting synanthropic habits has markedly increased in the last decades [12, 13]. Among animals, birds probably represent the most successfully adapted to synanthropic life, showing a rapid radiation on co-adaptive use of human by-products. Many species with different breeding and feeding styles continuously use human settlements and man-made landscape and share with human populations a somehow similar pattern of exposure to xenobiotics.

The long-lived (up to 12 years) common swift (*Apus apus*, order *Apodiformes*) breeds in colonies, preferentially in town and village buildings, and spends most of its active time flying. It feeds exclusively on airborne arthropods, and mostly in the range from approximately ground level to 100 m above open ground [14]. The species is a sexually monomorphic, long-distance migratory bird (weight: 38-42 g), whose western Palearctic populations move regularly from sub-Saharan Africa over to Europe for breeding [15]. At our latitudes (Rome, Italy, 41° 44' N, 12° 24' E), they arrive in early spring to spend approximately four months (April-July) from colony establishment to fledging of the offspring. Very little is known of those periods when swifts are presumably in central and southern Africa [15]. The chief asset of the common swift as bioindicator is its peculiar aerial niche occupied in the urban and peri-urban environment, together with its abundance and wide distribution permitting sampling in almost any urban area across much of Europe.

The present study is a follow-up of a preliminary investigation [16]. We aim: *(a)* to evaluate whether the common swift has a potential for use as a bioindicator of the chemicals of interest; *(b)* to assess how exposure, body burden, and distribution in different organs are related. Moreover, in the same population monitored, patterns of parental behaviour deviating from the expectation (bi-parental care vs maternal care) have been reported [17], leading to hypothesize the occurrence of endocrine disruption phenomena [18-20]. The assessment of the above mentioned contaminants may serve as a first step to indicate (or not) causal links explaining the observed variation. Results are described and discussed together with an exposure model tentatively developed for the common swift. As there is a substantial lack of relevant data concerning airborne-feeding birds and their possible exposure pattern(s) to the microcontaminants dealt with here, this study, while serving to fill in information gaps, is also intended to generate hypotheses and provide suggestions for further research.

MATERIALS AND METHODS

Sample collection

Forty fatally injured grounded adult swifts were collected in Rome between April 29 and July 14 1996, during the breeding period [16]. The injured birds were transported to the laboratory and euthanized with ethyl ether once it was ascertained they had no chance of survival. The plumage and skin of each body were carefully removed upon delivery to the laboratory, and so were the brain, breast muscle, heart, liver, and lungs. Instruments were carefully cleaned with hexane after each specimen's dissection. Individual specimens were wrapped in aluminum foil, identified, and stored at -80 °C awaiting further treatments.

Analysis

Fifteen randomly-selected specimens from the entire specimen batch available were allowed to thaw out in the laboratory; then, five tissue-specific samples were obtained by pooling the specimens of a type. Samples were combined with a 1:1 (v/v) n-hexane-acetone mixture, spiked with ^{13}C-labeled standards (CIL, Cambridge Isotope Laboratories), and homogenized and extracted by means of a mechanical homogenizer to remove the lipid component. The crude organic extract was subjected to a number of cleanup steps including liquid-liquid partition, treatment with concentrated sulfuric acid, and chromatographic filtration on an activated alumina column. The two main eluates obtained from alumina cleanup were used for determination of PCBs, DDE, DDT, and HCB *(Fraction 1)*, and PCDDs and PCDFs *(Fraction 2)* by HRGC-HRMS(SIM). GLP and QA/QC protocols were applied throughout; confirmatory determinations were eventually carried out. Uncertainty of the GC-MS measurements reported herein was estimated as $<CV> \approx 10\%$ ($CV < 30\%$). The analytical procedure was adopted from the USEPA Method No 1613 [21]. In the text the prefixes T_4, P_5, H_6, H_7, and O_8 have been used to indicate chlorosubstitution levels of four through eight.

RESULTS AND DISCUSSION

Analytical outcomes

A synopsis of the relevant findings obtained is presented in *Table 1*, where PCB and PCDD and PCDF amounts are expressed as cumulative data. The fresh tissue samples utilized for analytical assessments weighed between 4.24 and 11.7 g. The lipid amounts extracted ranged from 89.0 to 574 mg, between 2.10 and 5.82% of the original matrices. The lowest lipid content was found in lung tissue, whereas the highest was measured in the brain, as expected.

All the analytes appear to have a consistent minimum concentration in the brain, as shown specifically by lipid-base normalized data (however, DDT concentration in the liver could not be measured). In particular, PCDD and PCDF con-

Table 1 | *Concentration levels [a] of the chlorinated microcontaminants assessed in five selected tissue types of the common swift (sampling from April 29 to July 14, 1996). Analyte values are expressed per unit fresh tissue weight and per unit extracted lipid weight*

Analyte	Analyte levels per tissue type				
	Brain	Breast muscle	Heart	Liver	Lungs
Data normalized on fresh tissue base (ng/g, except where noticed)					
PCBs[b]	64.2	371	155	225	100
PCDDs+PCDFs[c] (pg/g)	11.6	71.6	117	947	144
PCDDs+PCDFs[c] (pg I-TE/g)	1.13	8.27	6.92	25.8	4.27
PCDDs+PCDFs[c] (pg WHO-TE/g)	1.39	10.3	8.63	32.2	4.79
DDE	112	379	263	251	117
DDT	0.388	0.928	1.09	<0.034	0.377
HCB	5.45	8.21	6.65	7.76	4.02
Data normalized on lipid base (ng/g, except where noticed)					
PCBs[b]	1100	7560	4060	6680	4760
PCDDs+PCDFs[c] (pg/g)	199	1460	3060	27,900	6830
PCDDs+PCDFs[c] (pg I-TE/g)	19.4	168	181	763	203
PCDDs+PCDFs[c] (pg WHO-TE/g)	23.9	210	226	954	228
DDE	1930	7720	6900	7410	5560
DDT	6.67	18.9	28.7	<1.0	17.9
HCB	93.6	167	174	229	191
Ancillary information					
Matrix size (g)	5.98	11.7	6.12	10.6	4.24
Extracted lipid (g)	0.348	0.574	0.234	0.357	0.0890
Extracted lipid (%)	5.82	4.91	3.82	3.37	2.10

(a) Values corrected for analytical recovery and rounded off to three figures. Individual figures of the original data sets preceded by ≤ or < (signs respectively indicating an upper limit estimate or below limit of quantification, S/N ≈ 3; N ≈ 4 σ_n), were entered as half their nominal value to calculate PCB and PCDD and PCDF cumulative data.

(b) As per PCB congeners reported in Table 2.

(c) As per PCDD and PCDF congeners reported in Table 3.

centration values in the brain (199 pg/g and 19.4 pg I-TE/g or 23.9 pg WHO-TE/g where I-TE and WHO-TE indicate conversion of congener-specific analytical data to 2,3,7,8-T_4CDD toxicity equivalents as per the I-TEF [22] and WHO-TEF [23] systems are approximately one order of magnitude lower than the pertinent levels found in breast muscle, heart, and lung tissues, and up to two orders of magnitude below the relatively high levels found in the liver (27,900 pg/g and 763 pg I-TE/g or 954 pg WHO-TE/g). In terms of concentration drop magnitude, PCBs come next with an assessed value in the brain of 1100 ng/g, some four to eight times smaller than the concentration figures estimated for the remaining tissues, all of which fall in the comparatively narrow range of 4060-7560 ng/g. Aside from the aforecited unassessed DDT level in the liver, pesticide tissue distributions seem to be characterized by concentration levels not as variable as those detected for PCBs and PCDDs and PCDFs, with reduced differences between pertinent maxima and minima whose ratios are approximately within a factor of 4. Of the entire group of analytes, HCB exhibits the most uniform distribution pattern (93.6-229 ng/g), whereas DDE and DDT are, respectively, by far the most and the least concentrated pesticide in the five tissues examined (specifically, [DDT] << [HCB] << [DDE]).

When compared with the published literature, the PCB levels in the upper range *(Table 1)* appear to be in reasonable agreement with similar data obtained from areas under general anthropogenic impact [24, 25], an observation which also applies to the chlorinated pesticides quantified.

Exposure analysis and model

Pesticide contamination levels and distribution patterns may reflect the fact that the organisms exposed had the chance to reach or approach what will be here referred to as a "steady-state-like condition". This situation would apply particularly well to the exposure period(s) of approximately seven months a year that swifts spend in Africa, where DDT is still used [26]. However, exposure to DDT and its kin compounds and metabolites – including the prominent DDE – is liable to occur also in those countries (such as Italy) that banned their use in the open long ago, but where the chemicals are still environmentally present as historical residues, not counting chemical accidents, other forms of local releases, and the long-range transport. Indeed, it

may be presumed that, moving from Africa to Italy, birds would be exposed to lesser amounts of the pesticides of interest, this entailing a relatively slow re-adjusting to a somewhat lower level of the previously reached steady-state-like condition. DDT absence in the liver may be explained by the fact that most of the substance metabolic fate is met there [3], the remaining organs examined merely acting as storing and/or supplying matrices. Toxic and stable DDE is an important metabolite of DDT and an end product itself in some metabolic systems [3, 4]: the DDE-DDT pattern detected *(Table 1)* – for which a high $[DDE] \times [DDT]^{-1}$ ratio is estimated (>>200) – could indicate the occurrence of an old exposure [27], thus supporting the hypothesis that birds were sampled after a steady-state-like condition for the pesticides was reached.

HCB is not only a widely used pesticide (fumigant), but also a common intermediate or by-product of important chloro-organic industrial productions [8]. Due to its comparatively greater volatility, HCB is a worldwide atmospheric contaminant exhibiting substantially uniform distribution levels.

Urban areas are important sources of PCDDs and PCDFs, as proven by different studies around the world [7] including those for the Italian cities of Rome and Florence [28, 29]. Therefore, a significant uptake of the aforcited microcontaminants could be associated with the comparatively short period(s) the swifts are in Italy, thus determining a chemical distribution in the swifts collected that did not have enough time to develop into the features of a steady-state-like condition. The relatively high levels of these substances found in the liver – a canonical primary target organ [5] – together with the remarkable unevenness of concentration levels in the tissues examined may be thought of as supporting evidence of such a hypothesis. In addition, it may be observed that the long PCDD and PCDF half-lives (hls) in living organisms – actually, the longest hls known for xenobiotic chemicals – would favor a comparatively rapid building-up of tissue levels (and, conversely, a comparatively slow depletion), thereby stressing inter-tissue concentration differences before the steady-state-like condition is reached [7]. Finally, let us assume that the swifts assayed were exposed to PCDDs and PCDFs primarily in the urban environment of Rome. This assumption is based on the fact that swifts show a high nest/colony fidelity and during the whole breeding period adults have to return to their nests every 2-4 h for the parental activities [17, 30]. Then, the indicative PCDD and PCDF concentrations for the swifts (body burden) and the correlated media (ambient air) may respectively be taken as 10 pg I-TE/g, fresh tissue base, derived from a coarse weighting of tissue contributions *(Table 1)*, and 5×10^{-5} pg I-TE/g (approximately, 0.07 pg I-TE/m^3) [28, 29]. The swift is a pure aerial feeder, therefore an air-to-swift bioconcentration factor (BCF) can be taken into consideration. The above-indicated figures would yield an estimate of BCF in the order of 2×10^5 or possibly greater (the air concentration values taken as a reference were obtained mostly during the winter period, when home heating was an additional source of contamination with respect to the spring-summer period). If the lipid base concentrations are considered *(Table 1)*, the BCF estimate is in the order of 5×10^6.

PCB distribution pattern seems to fall between the two extreme cases described above: it is somewhat more uneven than that of pesticides, but does not show the variability of up to two orders of magnitude observed for PCDD and PCDF concentration levels. Therefore, owing to the worldwide environmental diffusion of PCBs, their distribution levels and pattern in the common swift tissues analyzed may reflect substantially continuous exposures the year-round – possibly, to some extent greater in Italy [28, 29] than in Africa's wintering areas – and may also reflect the presence in the organisms examined of a steady-state-like condition nearly reached. In particular, if moving from Africa to Italy would have the birds exposed to greater environmental PCB concentrations, a re-adjusting to a somewhat higher level of the previously reached steady-state-like condition, with a comparatively rapid building-up of PCB tissue levels, should be expected. This process could stress inter-tissue concentration differences while the organisms are shifting toward a steady-state-like condition of a somehow higher level. Similar to the pesticides, the industrially-produced PCBs are still environmentally present as historical residues from a wide variety of commercial uses that, for decades since the 1940s, determined their largely uncontrolled release into the environment; today, their use is restricted to closed-cycle applications (*e.g.*, as dielectric fluids in heavy-duty electrical appliances) and their production is banned almost anywhere [6]. Besides chemical accidents, (improper) treatment and disposal of PCB-containing waste, and other forms of local releases however uncommon, the widespread environmental presence of the aforecited chemicals is also fueled by long-range atmospheric transport.

Finally, it may be observed that the general analyte concentration drop found in the brain *(Table 1)* is accounted for by the well-known hindering action that hematoencephalic (or blood-brain) barrier exerts on xenobiotics or, in general, exogenous substances [31].

Analyte congener- and compound-specific profiles

In *Table 2*, H_6CB[138+163], H_7CB[170], H_7CB [180], and H_7CB[187] appear to supply firm contributions larger than 20% of base congener H_6CB[153] (relative abundance, 100%). This five-congener cluster, where H_7CB[180] may be seen to compete as the base congener, is a recurrent and remarkably stable primary feature typical of all PCB distributions assessed in the swift samples, and is typical of animals sharing high trophic levels. PCB profiles reported in *Table 2* and detected in the five tissues examined show a degree of high reciprocal similarity – a fact

Table 2 | *PCB analytical profile descriptors obtained from original tissue- and congener-specific data sets available from the assay of common swift brain, breast muscle, heart, liver, and lung specimens.[a] From the mean descriptor data set, a mean profile is obtained*

Analyte	Profile descriptors[b]					Analyte	Profile descriptors[b]				
	Mean	n	cv%	Min	Max		Mean	n	cv%	Min	Max
T_4CB [66+80]	1.89	4	16.2	1.61	≤10.9[c]	H_6CB [167]	4.67	4	9.88	≤3.15	5.30
T_4CB [74]	1.95	4	37.4	1.24	≤12.7	H_7CB [170]	26.7	4	6.88	≤18.2	29.1
P_5CB [87]	1.26	5	41.6	0.713	2.13	H_7CB [171]	1.57	3	15.7	1.30	≤2.05
P_5CB [95]	2.52	4	55.6	≤1.03	4.54	H_7CB [174]	1.19	3	28.0	≤0.759	1.57
P_5CB [99]	3.60	4	34.0	≤1.90	5.40	H_7CB [177]	8.56	2	—	≤6.36	9.29
P_5CB [101]	3.33	5	53.4	1.75	6.25	H_7CB [178]	4.62	5	7.38	4.16	5.11
P_5CB [105]	2.55	4	64.1	0.365	≤5.77	H_7CB [180]	92.2	5	14.1	70.9	106
P_5CB [118]	18.3	5	20.9	16.2	25.1	H_7CB [183]	9.41	4	7.80	≤7.99	10.1
H_6CB [138+163]	52.3	3	13.4	≤35.7	59.5	H_7CB [187]	32.9	5	8.68	29.3	37.0
H_6CB [146]	10.9	3	9.12	≤9.45	11.8	O_8CB [194]	13.9	5	19.8	9.67	17.1
H_6CB [149]	3.59	5	33.1	2.71	5.63	O_8CB [195]	3.38	4	20.8	≤2.36	4.27
H_6CB [151]	1.16	5	30.2	0.839	1.74	O_8CB [201]	11.9	5	15.6	9.54	14.1
H_6CB [153]	100	4	5.89	≤86.0	105	O_8CB [202]	2.15	5	22.8	1.62	2.82
H_6CB [156]	9.21	5	5.03	8.82	9.93	O_8CB [203+196]	8.33	5	19.1	5.98	10.1
H_6CB [157]	1.98	3	22.9	≤0.791	2.37						

(a) Sampling campaign, from April 29 to July 14, 1996.

(b) Mean, minimum, and maximum descriptor values are normalized on the base congener (H_6CB [153]) mean estimate. N provides the number of original entries utilized for congener data processing.

(c) The sign ≤ indicates an upper limit estimate. Data of this type were not entered in calculations, a fact that accounts for N < 5.

suggestive that a steady-state-like condition has nearly come about. In particular, each value is associated with its coefficient (CV) and range of variation. Profile stability is borne out by the magnitude of CV estimates, most of which (frequency, 15/29) are less than 20%. In any case, the less chlorinated homologs are known to have a metabolic reactivity greater than those with a higher degree of chlorosubstitution [1]; therefore, the perceivable differences that characterize the occurrence in the different tissues of several T_3CB, T_4CB, and P_5CB congeners could be tentatively ascribed to exposure or metabolic factors, or a combination thereof.

As seen for PCBs, tissue-specific pesticide profiles were so similar to each other that they could be made into a single data set of mean compound-specific values. As stated before, this is additional evidence that a steady-state-like condition may have taken place. Again, profile stability is indicated by the magnitude of DDE, DDT, and HCB CV estimates, respectively 1.03, 21.1, and 31.2%. The comparatively large HCB CV value is at least in part attributable to the chemical's greater volatility, a factor that may influence analytical repeatability.

From the tissue- and congener-specific data of *Table 3*, it may readily be observed that PCDFs consistently provide only a minor and quite variable contribution (2.8-23%) to the overall cumulative analytical amounts; their contribution to cumulative ite values is larger and not as spread (17-40%) but still visibly smaller than the PCDD complement. From the table, it may be seen that PCDD and PCDF analytical profiles are fingerprinted by the recurrent presence of O_8CDD – the base congener, accounting for some 30-70% of the pertinent cumulative amounts – accompanied by 1,2,3,4,6,7,8-H_7CDD and 1,2,3,6,7,8-H_6CDD, two congeners that eventually compete as the next most important constituent. The congener 2,3,4,7,8-P_5CDF appears as the most prominent of PCDFs, although in two cases only (brain and breast muscle tissues) does it come within the five principal constituent group.

As stated previously, average PCB and pesticide profiles could be built by merging the original tissue-specific findings, owing to the good inter-tissue similarity of individual profiles. For PCDDs and PCDFs, such an operation cannot be performed due to their irregular distribution, at both congener and cumulative (analytical) levels, in the five tissues examined. This aspect is clearly borne out by the outcomes (*Table 4*, "five-tissue" part) of the analysis of original congener data shown in *Table 3*. The lowest CV values, 46.8 and 55.4%, are respectively associated with 2,3,7,8-T_4CDD and 1,2,3,7,8,9-H_6CDF. In all the remaining cases, CVs are well above 60% and often (frequency, 6/17) exceed 100%. The important fingerprinting congeners O_8CDD and 1,2,3,4,6,7,8-H_7CDD exhibit the largest CVs, 160 and 167%, respectively. Anyway, the tissue-specific congener accumulation seem to follow the pertinent increase in partition coefficient values. As absolute analytical figures (pg/g, fresh tissue base) were used for this evaluation, CV magnitude may be taken as an indicator of inter-tissue distribution variability of individual PCDDs and PCDFs (*i.e.*, the larger the CV value, the more irregular the distribution). On this basis, it may be pointed out that the whole PCDF congener group and, in particular, the PCDD and

Table 3 | *PCDD and PCDF concentration levels[a] (pg/g, fresh tissue base) in five selected tissue types of the common swift (sampling from April 29 to July 14, 1996)*

Analyte		Analyte levels per tissue type				
		Brain	Breast muscle	Heart	Liver	Lungs
2,3,7,8-T_4CDD	(D1)	0.249	0.707	0.590	1.03	0.496
1,2,3,7,8-P_5CDD	(D2)	≤1.05[b]	4.15	3.54	13.8	1.24
1,2,3,4,7,8-H_6CDD	(D3)	0.408	5.67	7.28	39.3	5.21
1,2,3,6,7,8-H_6CDD	(D4)	0.840	13.7	15.3	52.0	7.31
1,2,3,7,8,9-H_6CDD	(D5)	0.262	4.23	4.69	23.1	3.61
1,2,3,4,6,7,8-H_7CDD	(D6)	0.892	9.46	13.1	150	15.2
O_8CDD	(D7)	5.74	20.5	64.2	641	104
2,3,7,8-T_4CDF	(F1)	<0.40[b]	<0.075	<0.33	0.112	0.178
1,2,3,7,8-P_5CDF	(F2)	0.250	0.320	<0.29	0.865	0.296
2,3,4,7,8-P_5CDF	(F3)	0.681	4.39	2.26	5.69	1.81
1,2,3,4,7,8-H_6CDF	(F4)	0.174	2.56	1.35	5.33	0.853
1,2,3,6,7,8-H_6CDF	(F5)	0.0929	1.39	0.860	3.24	0.638
1,2,3,7,8,9-H_6CDF	(F6)	0.267	1.95	1.34	2.10	1.03
2,3,4,6,7,8-H_6CDF	(F7)	0.211	1.95	1.16	2.95	0.877
1,2,3,4,6,7,8-H_7CDF	(F8)	<1.0	0.493	0.535	5.44	0.523
1,2,3,4,7,8,9-H_7CDF	(F9)	<0.17	<0.16	<0.36	0.364	<0.37
O_8CDF	(F10)	<0.38	<0.056	<0.33	<0.19	<0.47
Totals (pg/g)		11.6	71.6	117	947	144

(a) Values corrected for analytical recovery and rounded off to three figures.

(b) Figures preceded by ≤ or < (signs respectively indicating an upper limit estimate or below limit of quantification, S/N ≈ 3; N ≈ 4 σ_n) were entered as half their nominal value to calculate cumulative data.

PCDF congeners with lower chlorosubstitution, seem to have a less irregular distribution than that of, respectively, PCDDs and those congeners with a higher number of chlorine. The ratios [max] × [min]$^{-1}$ exhibit a pattern that is by and large in agreement with that of CV estimates, an additional evidence in support of the aforecited observations. Ratio values range from less than five to over two orders of magnitude (3.46-168), reflecting the differences of congener concentrations in liver and brain tissues, consistently associated with respectively maximum and minimum estimates. Exclusion from the data analysis of concentration figures concerning the brain (*Table 4*, "four-tissue" part) results in a substantial reduction of all CV estimates in spite of the concurrent degree-of-freedom reduction (N, 5 × 4 or 4 × 3). The values of the ratio [max] × [min]$^{-1}$ also appear significantly decreased, more visibly for PCDDs than for PCDFs. In either case, however, elimination of the lowest concentration figures from the general data set still leaves congener distribution patterns characterized by remarkable amounts of spread (CV, from 31.5 to 146%; [max] × [min]$^{-1}$, 2.03-31.3). In other words, even if the effect of the hematoencephalic barrier is accounted for, distributions of individual PCDDs and PCDFs over the remaining four tissues are still very uneven, more so for congeners with a higher number of chlorine, a fact that may be reasonably explained if exposure to these chemicals is comparatively recent, and therefore a steady-state-like condition has not been reached as yet. The observations reported above hold for PCDD and PCDF cumulative levels as well, as may be deduced from *Table 4*. Lastly, the above discussion was based on PCDD and PCDF outcomes normalized on fresh sample weights: the use of lipid-base normalized data would have the effect of increasing somewhat the inter-tissue distribution variability, leading however to evaluations similar to those already described.

CONCLUSIVE REMARKS

In general terms, the use of the common swift as a bioindicator is suggested especially to detect those chemicals that are known or expected to have a rather uniform distribution worldwide such as DDT, DDE, HCB and PCBs, even when at relatively low concentration levels. With regard to substances with an uneven distribution in the environment and long metabolic hls, the swift should be used with caution as a "chemical memory" related to previous exposures that may interfere with the actual measurements. As exposure patterns and hls are key factors in determining tissue levels and distribution, they are also determinative for us to establish whether the common swift may be suitable for use as a "sentinel species" for air quality assessment.

It may be pointed out that the measured DDE and PCB concentrations in the swifts studied appear, re-

Table 4 | *Selected examples of PCDD and PCDF inter-tissue variability descriptors as derived from the analytical outcomes reported in Table 3*[a]

Analyte	Based on data sets from five tissues				Based on data sets from four tissues[b]			
	Mean[c]	(cv%)	N	[Max]/[Min]	Mean[c]	(cv%)	N	[Max]/[Min]
D1	0.615	46.8	5	4.14	0.706	33.1	4	2.08
D2	5.68	97.7	4	11.1	5.68	97.7	4	11.1
D3	11.6	136	5	96.4	14.4	116	4	7.55
D4	17.8	112	5	61.9	22.1	91.7	4	7.12
D5	7.19	126	5	88.3	8.92	106	4	6.41
D6	37.8	167	5	168	47.0	146	4	15.9
D7	167	160	5	112	207	140	4	31.3
F1	-	-	2	-	-	-	2	-
F2	0.433	66.9	4	3.46	0.494	65.1	3	2.92
F3	2.96	68.5	5	8.35	3.54	51.6	4	3.15
F4	2.05	98.7	5	30.6	2.52	79.3	4	6.24
F5	1.24	97.0	5	34.8	1.53	77.0	4	5.07
F6	1.34	55.4	5	7.87	1.61	31.5	4	2.03
F7	1.43	73.8	5	14.0	1.74	53.6	4	3.37
F8	1.75	141	4	11.0	1.75	141	4	11.0
F9	-	-	1	-	-	-	1	-
F10	-	-	0	-	-	-	0	-
Σ[PCDD+PCDF]	258	150	5	81.8	320	131	4	13.2

(a) Values appearing in the table are generally rounded off to three figures; constituents associated with figures preceded by ≤ or < (cfr. Table 3, Note b) were not taken into account. Cfr. Table 3 for labels D1–D7 and F1–F10.

(b) Brain figures omitted.

(c) Values in pg/g, fresh tissue base.

spectively, two and one order of magnitude lower than those reported as yielding acute effects in other birds [32]. Therefore, focusing future research on the role of the observed tissue concentrations in terms of sublethal effects at population level would appear to be appropriate, in particular to characterize the potential for endocrine disruption. The chemicals dealt with in this work are known to have what especially on those behavioral domains known to be modulated by steroid hormones, such as mating, aggression, parental care and homeostatic activities (*e.g.*, drinking, eating) [9].

The outcomes of our study prove that the common swift, may be utilized as a bioindicator of environmental contamination due to the chemical levels built-up in their tissues. As mentioned in the Introduction, behavioral studies hints also for different types of observations [33]. In line with that, investigations carried out on a swift colony in Rome revealed unexpected sexually dimorphic patterns of parental care [17, 34]. Although the authors have offered a functional explanation for that, we have now provided evidence that the observed (outlying) behavioral pattern could be a consequence of altered endocrine regulation induced by the chemicals of interest. In the light of the above, based on variation of sex typical features and if its causal association with chemical body burden (exposure) will be proven, this species might also find a future use for monitoring the exposure to – and, therefore, the environmental occurrence of – endocrine disrupting agents.

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