Environmental *versus* dietary exposure to POPs and metals: A probabilistic assessment of human health risks†

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The human health risks derived from the multipathway/multipollutant exposure to various chemicals were assessed in an area with significant petrochemical activity (Tarragona, Catalonia, Spain). Environmental exposure to several Persistent Organic Pollutants (POPs) (PCDD/Fs, PCBs, PCNs, and PAHs) and metals (As, Cd, Cr, Hg, Mn, Pb, and V) was determined and compared with the dietary intake of these pollutants. The mean environmental exposure to organic pollutants ranged from 6.36×10^{-6} ng WHO-TEQ kg^{-1} day^{-1} to 3.34 ng kg^{-1} day^{-1} for PCDD/Fs and PAHs, respectively. In turn, the minimum and maximum values of environmental exposure to metals corresponded to Cd (9.35×10^{-8} mg kg^{-1} day^{-1}) and Mn (8.72×10^{-5} mg kg^{-1} day^{-1}), respectively. Among the environmental exposure pathways, dermal absorption and soil ingestion were the most important pathways for POPs and metals, respectively. However, this exposure was notably lower than the dietary intake of these contaminants, with percentages of <2% for most of them. Considering cumulative effects, the current concentrations of micropollutants do not mean significant additional non-carcinogenic and carcinogenic human health risks. Notwithstanding, in order to consider the synergistic/antagonistic effects according to the target organ or mode-of-action, the development of alternative methodologies of risk assessment are necessary for a more accurate evaluation.

Introduction

Chemical and petrochemical industries are responsible of the emissions of inorganic gasses such as SO_2, NO_x, H_2S and CO, particulate matter, as well as a series of micropollutants. Among these, particular attention has been paid to heavy metals and Persistent Organic Pollutants (POPs) such as polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) and polychlorinated biphenyls (PCBs). Moreover, traffic has also been pointed out as an important emission source of some of these contaminants, to which polycyclic aromatic hydrocarbons (PAHs) and lead (Pb) can be included.[1–4]

Humans are exposed to environmental metals and POPs through various pathways (*i.e.*, inhalation and ingestion). However, it is well established that diet is the main route of entrance to the human body of these contaminants, accounting for more than 90% of total exposure.[5–7] Notwithstanding, most dietary studies are focused on single contaminants, rather than mixtures of pollutants. Therefore, integral/overall investigations to evaluate the human health risks derived from multipollutant and multipathway exposure are not particularly common.[8,9] One of the most important petrochemical complexes in southern Europe is located in Tarragona County (Catalonia, Spain). A broad spectrum of industrial facilities, including a big oil

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Environmental impact

Humans are exposed to pollutants through various pathways, diet being the most important contributor. Most studies are generally focused on single contaminants, rather than mixtures of pollutants. Therefore, overall investigations focused on evaluating human health risks derived from multipollutant and multipathway exposure are not particularly frequent. In this study, the cumulative environmental exposure to several Persistent Organic Pollutants and metals for the population of Tarragona (Catalonia, Spain) was determined and compared with the dietary intake of these pollutants. The human health risks following multipollutant/multipathway exposure to chemical pollution were assessed. The results should contribute to enhance the information regarding the health risks of environmental pollutants in more realistic scenarios.

refinery, a municipal solid waste incinerator, a hazardous waste incinerator, and a chlor-alkali plant, are operating in that zone, where traffic is also considerable. Due to local population concern, in 2002 we carried out a wide surveillance investigation to study the environmental concentrations of various micropollutants: metals, PCDD/Fs, PCBs and PAHs.[10] Polychlorinated naphthalenes (PCNs), a potential candidate to enter the list of POPs elaborated in the Stockholm Convention, were also included. Although the levels of these contaminants were relatively low, the local authorities decided to pursue with the surveillance program in order to evaluate the temporal trends.[11] In 2007, multimedia levels of the same chemicals analyzed in the baseline study were determined in the same zones of Tarragona County.[12]

The purpose of the present survey was to compare the environmental exposure to POPs and metals with their dietary intakes. Health risks for the local population were also assessed and the temporal trends with respect to the baseline survey were determined. To assess the uncertainty and variability, a Monte-Carlo simulation was applied.

Materials and methods

Concentration of POPs and metals

Environmental concentrations. As part of the environmental surveillance program, in the winter of 2007, 27 samples of soil and 8 samples of air were taken in various locations around the chemical/petrochemical industrial area of Tarragona County. Sampling details were recently reported.[12] In each sample, the levels of a number of organic compounds (PCDD/Fs, PCBs, PCNs, and PAHs) and metals (As, Cd, Cr, Hg, Mn, Pb, and V) were determined. Analyses of organic pollutants were carried out by high resolution gas chromatography/high resolution mass spectrometry (HRGC/HRMS), while metal analysis was carried out by means of ICP-MS (Perkin Elmer Elan 6000). Soils were treated with HNO_3, while quartz fiber filters were digested with a mixture of HNO_3 and HF in hermetic teflon bombs for 8 h at room temperature and 8 h at 80 °C. The analytical results in air and soil samples are summarized in Table 1.

Levels in foodstuffs. In spring of 2006, food samples were randomly acquired in local markets, big supermarkets, and grocery stores from various cities of Catalonia, encompassing several locations in Tarragona County. For sampling, 2 groups were differentiated: (a) retailed products (meat, vegetables and tubers, fruits, and eggs), and (b) brands/trademarks products (milk and dairy products, cereals, pulses, oils and fats, meat products and industrial bakery). Fish and seafood samples corresponding to 14 marine species had been also collected and analyzed in a previous survey.[13] Composite samples were made up by mixing individual subsamples. Only edible parts of the foodstuffs were used.[14–17] The concentrations of POPs (PCDD/Fs, PCBs, PCNs, and PAHs) and metals (As, Cd, Cr, Hg, Mn, Pb, and V) were determined in each sample by means of HRGC/HRMS and ICP-MS, respectively. Table 1 shows the pollutant concentrations in various food groups.

Table 1 Concentration of POPs and metals in environmental and food samples

	POPs[a]					Metals[b]						
	PCDD/Fs	PCBs	PCNs	PAHs	carcin. PAHs	As	Cd	Cr	Hg	Mn	Pb	V
Air[c]	8×10^{-6}	0.059	0.011	23.0	3.82	5.12×10^{-7}	1.54×10^{-7}	6.70×10^{-6}	$<0.2 \times 10^{-4}$	6.83×10^{-6}	2.35×10^{-6}	7.66×10^{-6}
Soil[c]	0.73	2950	79.2	197 181	72 270	5.34	0.18	15.30	<0.1	256.1	26.8	22.9
Foodstuffs[d]												
Meat	0.017	127.3	2.80	25 558	1789	<0.1	0.006	<0.25	<0.1	0.132	0.043	<0.25
Fish and seafood	0.131	16 264.7	47.1	8007	1642	4.457	0.034	<0.25	0.247	0.661	0.033	0.315
Pulses	0.014	66.7	10.2	7965	273	<0.1	0.117	<0.25	<0.1	3.216	0.047	<0.25
Cereals	0.024	87.8	7.99	20 444	667	0.045	0.009	0.239	<0.1	5.759	0.044	<0.25
Vegetables	0.009	24.0	2.29	1731	171	<0.1	<0.025	<0.25	<0.1	2.774	0.043	<0.25
Tubers	0.011	16.5	2.19	1214	216	<0.1	0.032	<0.25	<0.1	1.363	0.032	<0.25
Fruit	0.003	10.7	1.19	1048	148	<0.1	<0.025	<0.25	<0.1	0.530	0.028	<0.25
Milk	0.009	47.4	0.84	2585	407	<0.1	<0.025	<0.25	<0.1	0.029	0.064	<0.25
Dairy products	0.057	320.6	11.7	4076	538	<0.1	<0.025	<0.25	<0.1	<0.05	0.017	<0.25
Eggs	0.014	236.4	4.28	8555	891	<0.1	<0.025	0.220	<0.1	0.140	0.042	<0.25
Bakery products	0.018	129.0	15.31	11 218	1183	<0.1	<0.025	<0.25	<0.1	<0.05	0.012	<0.25
Oils and fats	0.223	673.5	21.5	23 476	3474	<0.1	<0.025	<0.25	<0.1	<0.05	0.080	<0.25

[a] Units: air, ng m^{-3}; soil and food, ng kg^{-1}. PCDD/Fs are expressed using WHO-TEQ. [b] Units: air, mg m^{-3}; soil and food, mg kg^{-1}. [c] Environmental levels, from Nadal *et al.*[12] [d] Food concentrations.[14–17]

Exposure assessment. Total exposure to POPs and metals for the population of Tarragona was calculated by summing exposure through 2 different pathways: (a) environmental (direct), and (b) dietary. To avoid an overestimation of exposure and risks,[18] calculations were carried out in a probabilistic framework. Probability density functions for each parameter, rather than deterministic point values, were used. The parameter distributions are given in the ESI, Table S1.† A Monte-Carlo simulation was applied by executing Crystal Ball 4.0® software. An iteration size of 10 000 was used.

Direct exposure. Environmental exposure to organic compounds (PCDD/Fs, PCBs, PCNs, and PAHs) was calculated by applying the VLIER-HUMAAN methodology.[19] Three exposure pathways were considered:

(a) Air inhalation, calculated as:

$$\mathrm{Inh} = \frac{V_r \cdot C_{air} \cdot f_r \cdot t_f}{BW}$$

(b) Dermal absorption of soil and dust, estimated as:

$$\mathrm{Der} = \frac{(\mathrm{Der}_o \cdot 0.01 \cdot f_m \cdot 24 \cdot SA_o \cdot t_{fso} \cdot C_{soil}) + (\mathrm{Der}_i \cdot 0.01 \cdot f_m \cdot f_{ri} \cdot 24 \cdot SA_i \cdot t_{fsi} \cdot C_{soil})}{BW}$$

(c) Ingestion of soil and dust, using the following equation:

$$\mathrm{Ing} = \frac{(I_{sp} \cdot [(24\text{-}t_s)/24] \cdot t_{fso} \cdot C_{soil}) + (I_{sp} \cdot [(24\text{-}t_s)/24] \cdot f_{ri} \cdot t_{fsi} \cdot C_{soil})}{BW}$$

The symbol meaning for each variable is given in the ESI (Table S1).†

In turn, the direct exposure to metals was calculated according to the US EPA methodology. Exposure to the same 3 pathways was estimated by applying the following mathematical formulas:

(a) Air inhalation:

$$\mathrm{Inh} = \frac{C_{air} \cdot EF \cdot V_r}{BW \cdot 365}$$

(b) Dermal absorption:

$$\mathrm{Der} = \frac{C_{soil} \cdot ABS \cdot SA \cdot AF \cdot EF \cdot 0.01}{BW \cdot 365}$$

(c) Soil ingestion:

$$\mathrm{Ing} = \frac{C_{soil} \cdot I_{sp} \cdot EF}{BW \cdot 365}$$

Dietary exposure. The daily intake of POPs and metals was also studied through a probabilistic approach. For this, consumption data of the analyzed foodstuffs were obtained from the ENCAT survey.[20] Raw data from the Frequency and Quantity questionnaires allowed computation of the individual average daily intake of each food group in a representative sample ($n = 2084$) of the general population of Catalonia. Daily consumptions were adjusted to gamma*binomial distributions, with the binomial term included to take into account the fraction of people that never consume some food groups (ESI,† Table S1). No significant correlations were observed between different food groups. However, correlations between consumption of some foods and body weight were apparent, and therefore, were incorporated into the Monte-Carlo simulations.

For each food group, the average pollutant concentration was calculated by summing the results of multiplying the pollutant concentration for each specific food item by the partial contribution of that item to the consumption of the food group. The contribution of each food to the food group was estimated from the data of the 24 h-recall questionnaires of ENCAT. The daily intake of a chemical by a food group was estimated by multiplying the average concentration by the daily consumption of the food group. Finally, the estimated total dietary intake of each chemical was obtained by summing the respective intakes from each food group and dividing by the body weight.

Risk characterization. Health risks derived from the total exposure to POPs and metals were assessed. Non-carcinogenic and carcinogenic risks were separately evaluated for each chemical through two pathways (oral and inhalation). The oral non-cancer risk, expressed as a Hazard Quotient (HQ), was determined by comparing oral exposure (sum of soil ingestion and dietary intake) with the oral Reference Dose (RfD_o). The carcinogenic risk was obtained by multiplying oral exposure and the oral Slope Factor (SF_o). To estimate health risks through inhalation, exposure concentration (EC) was previously calculated by applying the following equation:

$$EC = \frac{C_{air} \cdot EF \cdot ED \cdot ET}{AT}$$

The inhalation Hazard Quotient was calculated by dividing the exposure concentration by the inhalation reference concentration (RfC_i), while the inhalation cancer risk was obtained by multiplying EC by the US EPA Inhalation Unit Risk (IUR). Values of RfD_o, SF_o, RfC_i, and IUR for each pollutant are specified in ESI (Table S2).†

Results and discussion

The probabilistic environmental, dietary, and total exposure to POPs and metals for the adult population of Tarragona County are summarized in Table 2. All calculations were performed for adults as the largest group of people. However, as there exist other population groups especially sensitive to environmental pollution, the exposure was additionally evaluated for children (ESI,† Table S3). The mean direct exposure to organic pollutants for adults ranged from 6.36×10^{-6} ng WHO-TEQ kg^{-1} day^{-1} to 3.34 ng kg^{-1} day^{-1} for PCDD/Fs and PAHs, respectively. In turn, the minimum and maximum values of environmental exposure to metals corresponded to Cd (9.35×10^{-8} mg kg^{-1} day^{-1}) and Mn (8.72×10^{-5} mg kg^{-1} day^{-1}), respectively. It is known that diet is the main route of exposure to a number of chemicals, including POPs and metals.[5,21] In the present study, the contribution percentage of dietary intake was not less than 93%, with values above 97% for most pollutants. The only exceptions were the sum of 7 carcinogenic PAHs (benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene,

Table 2 Environmental (direct), dietary, and total exposure to POPs and metals for an adult population living in Tarragona County (Catalonia, Spain)

					Percentiles		
		Mean	Median	SD	75th	90th	95th
PCDD/Fs (ng WHO-TEQ kg^{-1} day^{-1})	Direct	6.36E−06	3.13E−06	1.05E−05	6.88E−06	1.41E−05	2.20E−05
	Dietary	4.37E−04	4.16E−04	1.41E−04	5.12E−04	6.18E−04	7.02E−04
	Total	4.41E−04	4.20E−04	1.41E−04	5.17E−04	6.27E−04	7.04E−04
PCBs (ng kg^{-1} day^{-1})	Direct	2.84E−02	1.25E−02	5.65E−02	2.61E−02	6.14E−02	1.01E−01
	Dietary	1.15E+01	1.03E+01	6.21E+00	1.45E+01	1.94E+01	2.31E+01
	Total	1.15E+01	1.02E+01	6.17E+00	1.43E+01	1.92E+01	2.30E+01
PCNs (ng kg^{-1} day^{-1})	Direct	1.41E−03	1.10E−03	1.46E−03	1.69E−03	2.56E−03	3.39E−03
	Dietary	1.15E−01	3.71E−02	1.09E−01	1.35E−01	1.64E−01	1.85E−01
	Total	1.16E−01	1.11E−01	3.72E−02	1.36E−01	1.64E−01	1.84E−01
PAHs (ng kg^{-1} day^{-1})	Direct	3.34E+00	2.48E+00	3.70E+00	3.94E+00	6.10E+00	8.22E+00
	Dietary	1.87E+02	1.79E+02	6.37E+01	2.22E+02	2.71E+02	3.07E+02
	Total	1.91E+02	1.81E+02	6.29E+01	2.25E+02	2.73E+02	3.07E+02
Carc. PAHs (ng kg^{-1} day^{-1})	Direct	8.51E−01	5.15E−01	1.26E+00	9.33E−01	1.72E+00	2.67E+00
	Dietary	1.40E+01	1.34E+01	4.38E+00	1.64E+01	1.97E+01	2.22E+01
	Total	1.48E+01	1.41E+01	4.58E+00	1.73E+01	2.07E+01	2.30E+01
As (mg kg^{-1} day^{-1})	Direct	1.91E−06	1.61E−06	1.28E−06	2.45E−06	3.43E−06	4.28E−06
	Dietary	6.06E−04	5.59E−04	2.44E−04	7.32E−04	9.24E−04	1.07E−03
	Total	6.07E−04	5.64E−04	2.47E−04	7.35E−04	9.25E−04	1.07E−03
Cd (mg kg^{-1} day^{-1})	Direct	9.35E−08	8.38E−08	4.89E−08	1.15E−07	1.56E−07	1.85E−07
	Dietary	2.64E−04	2.52E−04	7.90E−05	3.08E−04	3.67E−04	4.09E−04
	Total	2.65E−04	2.54E−04	8.03E−05	3.10E−04	3.72E−04	4.13E−04
Cr (mg kg^{-1} day^{-1})	Direct	6.56E−06	5.65E−06	3.95E−06	8.21E−06	1.15E−05	1.40E−05
	Dietary	2.81E−03	2.68E−03	9.16E−04	3.33E−03	3.96E−03	4.44E−03
	Total	2.81E−03	2.68E−03	9.13E−04	3.30E−03	4.02E−03	4.51E−03
Hg (mg kg^{-1} day^{-1})	Direct	2.06E−05	1.87E−05	1.14E−05	2.67E−05	3.56E−05	4.13E−05
	Dietary	2.76E−04	2.54E−04	1.20E−04	3.39E−04	4.34E−04	5.00E−04
	Total	2.96E−04	2.73E−04	1.19E−04	3.57E−04	4.56E−04	5.25E−04
Mn (mg kg^{-1} day^{-1})	Direct	8.72E−05	7.68E−05	5.17E−05	1.12E−04	1.54E−04	1.84E−04
	Dietary	2.77E−02	2.55E−02	1.15E−02	3.32E−02	4.27E−02	4.93E−02
	Total	2.78E−02	2.59E−02	1.15E−02	3.39E−02	4.26E−02	4.92E−02
Pb (mg kg^{-1} day^{-1})	Direct	9.50E−06	7.37E−06	7.76E−06	1.19E−05	1.84E−05	2.41E−05
	Dietary	4.13E−04	3.93E−04	1.35E−04	4.85E−04	5.85E−04	6.54E−04
	Total	4.23E−04	4.03E−04	1.37E−04	4.97E−04	6.02E−04	6.75E−04
V (mg kg^{-1} day^{-1})	Direct	9.27E−06	7.62E−06	6.57E−06	1.16E−05	1.69E−05	2.09E−05
	Dietary	2.73E−03	2.61E−03	8.88E−04	3.20E−03	3.89E−03	4.34E−03
	Total	2.75E−03	2.61E−03	9.00E−04	3.25E−03	3.92E−03	4.44E−03

indeno(1,2,3-c,d)pyrene, dibenzo(a,h)anthracene) and Hg, whose intakes were 5% and 7%, respectively.

The comparison of the environmental and dietary exposure to the individual congeners/compounds of PCDD/Fs, PCBs, PCNs and PAHs is depicted in Fig. 1. OCDD presented the highest mean environmental exposure to PCDD/Fs (3.23×10^{-4} ng kg^{-1} day^{-1}), while the maximum dietary intake corresponded to OCDF (5.15×10^{-3} ng kg^{-1} day^{-1}). Overall, OCDF was the congener showing the highest total exposure to PCDD/Fs. In terms of percentage, 1,2,3,7,8,9-HxCDF was the congener accounting for the highest contribution of direct exposure (10%). Concerning PCBs, the most-chlorinated congeners (No. 138, 153, and 180) showed the highest total exposure, with amounts of 4.05, 3.23 and 2.32 ng kg^{-1} day^{-1}, respectively. This exposure would basically be due to food consumption, which accounted for more than 99% for all the evaluated PCB congeners. On the other hand, the lowest-substituted homologues of PCNs (tetra- and penta-PCNs) showed the maximum exposure, while the percentage for the environmental pathway was more important for the high molecular weight compounds. Finally, the PAHs with a low or medium molecular weight presented the highest mean total exposure to these pollutants, with the maximum values corresponding to phenanthrene and naphthalene (60.0 and 32.4 ng kg^{-1} day^{-1}, respectively). Interestingly, for the heaviest compounds, marked as carcinogenic PAHs, the environmental exposure was the most important pathway, with a percentage higher than 12% (benzo(k)-fluoranthene). In fact, the direct exposure of benzo(a)pyrene and dibenzo(a,h)anthracene, the most toxic PAHs,[22] accounted for 8.6 and 5.2%, respectively. In Western Balkans, Bartoš *et al.*[23] reported that the majority of the human health risks derived from POP exposure at urban sites were associated with PAHs, while between 83 and 94% of the cumulative risk at such sites was assigned to chemicals sorbed to particles, and out of that, PAHs were responsible for 99%. These findings would be in the line of our results, as carcinogenic PAHs are mainly adsorbed to air particulate matter.

The percentage for each pathway (air inhalation, dermal absorption, and soil ingestion) for environmental exposure of the assessed chemicals is shown in Fig. 2. In general terms, dermal absorption was the most important route for organic substances, especially for PCDD/Fs and PCBs. However, the contribution of air inhalation to the exposure to PCNs and PAHs was also notable: 55% and 51% of the total direct exposure, respectively. In turn, soil and dust ingestion were the most significant routes of exposure to most metals, except Hg. It must be taken into

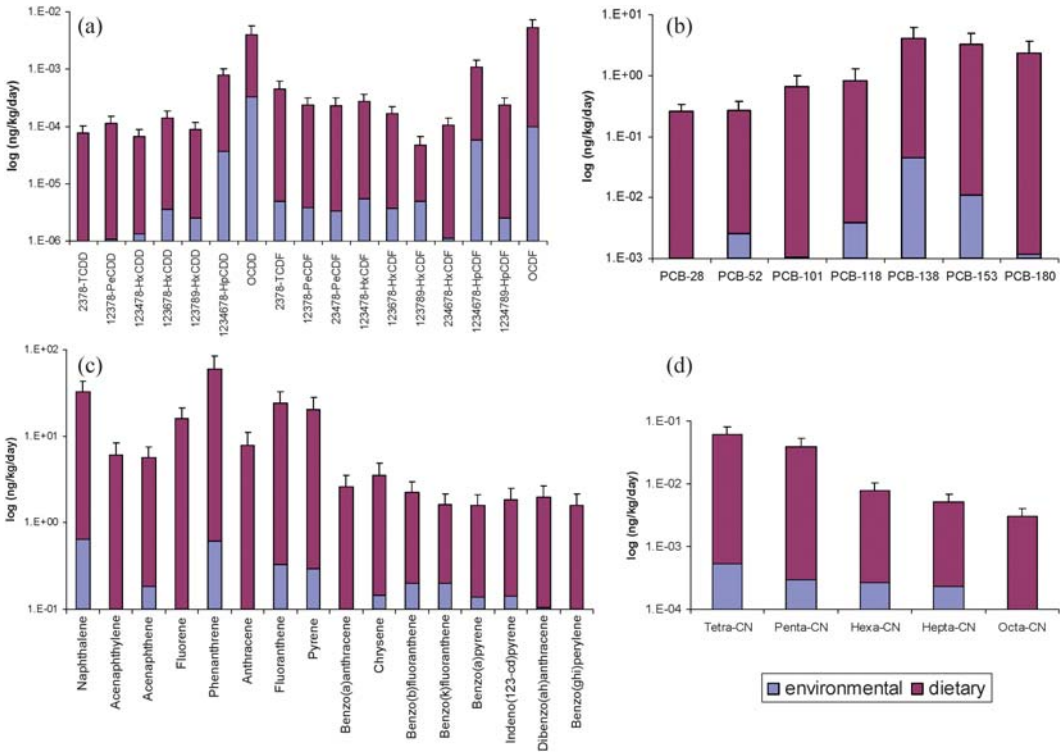

Fig. 1 Environmental and dietary exposure to (a) PCDD/Fs, (b) PCBs, (c) PAHs and (d) PCNs.

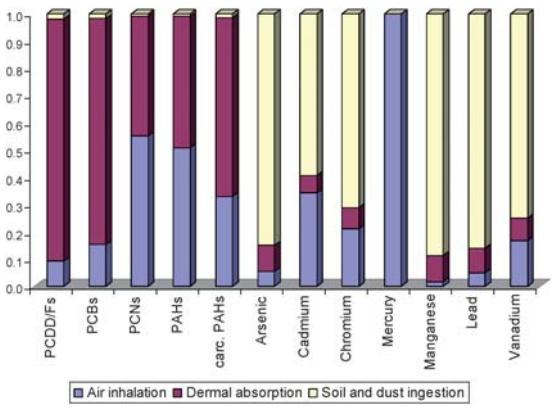

Fig. 2 Contribution percentage of each pathway to environmental exposure to POPs and metals.

account that environmental concentrations of Hg could not be detected in any sample, with concentrations <0.10 mg kg^{-1} in both soil and vegetation, and <0.02 μg m^{-3} in air.[12] Consequently, to assume that ND = 1/2LOD would probably mean an overestimation of Hg levels in the environment and the derived exposure. The dietary intake of POPs and metals according to each food group is summarized in Table 3. Fish and seafood was the group showing the highest contribution to the intake of PCDD/Fs (25%), PCBs (74%), As (76%), Hg and V. In fact, fish and seafood was the only group in which detectable concentrations of Hg and V could be detected: 0.25 and 0.31 mg kg^{-1}, respectively. Cereals showed the maximum percentage of exposure to PCNs (32%), PAHs (45%), Cr (87%), Mn (54%) and Pb (19%). Finally, the highest intakes of carcinogenic PAHs and Cd corresponded to consumption of meat (34%) and pulses (31%), respectively. As water consumption may also be an important pathway of exposure to the chemicals here evaluated, an additional assessment of the intake of POPs and metals through water was also assessed (Table 3). The Ebro River (Spain) is the supplying source of most of the water consumed by the population in Tarragona. The concentration of metals and PCBs were recently determined in the Catalan stretch of the river.[24,25] Moreover, data on PCDD/Fs, PCNs and PAHs were obtained from elsewhere.[26–28] In comparison to foodstuffs, the intake of chemicals through water consumption only meant a mean percentage of 3%, with a maximum value for Hg (8%).

The probabilistic values of Hazard Quotient (HQ) and cancer risk of exposure to organic pollutants and metals are summarized in Table 4. With respect to non-carcinogenic risks, none of the contaminants individually exceeded the threshold value of 1. The highest HQs through oral and inhalation uptake corresponded to PCCD/Fs and Hg, respectively. A higher influence of the oral route (than inhalation) was noted for naphthalene and As, while the contribution of inhalation was more notable for Mn. It must be noted that the non-carcinogenic risk resulting from integral exposure to the considered pollutants, assuming that human health effects are additive, would still be below the maximum recommended level. The Hazard Index following exposure to non-carcinogenic pollutants, which was calculated as the sum of HQs, was 0.10. Although the number of pollutants with potentially adverse health effects was higher for ingestion than inhalation (Table 4), the mean non-carcinogenic risks were increased for inhalation (0.011 and 0.092, respectively). In relation to cancer risks, only Cr and As exceeded the recommended threshold level of 10^{-6}, with levels of 1.52×10^{-5} and 1.05×10^{-6},

Table 3 Dietary intake of PCDD/Fs (pg WHO-TEQ day^{-1}), PCBs (ng day^{-1}), PCNs (ng day^{-1}), PAHs and carcinogenic PAHs (μg day^{-1}), and metals (μg day^{-1}) through different food groups for an adult (70 kg of body weight) living in Tarragona (Catalonia, Spain)[a]

	PCDD/Fs	PCBs	PCNs	PAHs	carc. PAHs	As	Cd	Cr	Hg	Mn	Pb	V
Meat	2.62	15.9	0.35	3.28	0.29	—	0.95	—	—	24.42	6.82	—
Fish and seafood	6.53	805.8	1.53	0.34	0.06	25.32	1.03	—	12.61	60.84	1.94	0.31
Pulses	0.43	1.0	0.15	0.22	0.01	—	3.00	—	—	77.18	1.20	—
Cereals	6.33	17.8	1.86	5.47	0.15	7.85	2.18	49.92	—	1203.62	8.35	—
Vegetables	1.28	2.0	0.23	0.34	0.03	—	—	—	—	625.98	7.16	—
Tubers	0.77	0.9	0.16	0.09	0.02	—	2.34	—	—	100.87	2.34	—
Fruit	0.65	1.1	0.17	0.20	0.03	—	—	—	—	126.55	5.92	—
Milk	1.47	5.5	0.12	0.40	0.07	—	—	—	—	6.30	6.80	—
Dairy products	1.81	16.8	0.26	0.14	0.02	—	—	—	—	—	2.13	—
Eggs	0.43	6.8	0.13	0.27	0.03	—	—	7.56	—	4.81	1.31	—
Bakery products	0.85	2.2	0.29	0.46	0.05	—	—	—	—	—	0.56	—
Oils and fats	2.50	16.0	0.56	0.85	0.09	—	0.31	—	—	—	0.59	—
Water[b]	1.43	0.24	0.20	0.36	0.03	0.74	0.10	1.10	1.01	6.24	1.41	—

[a] When no values are shown, it indicates that the chemical concentration in that food group was under the respective detection limit. [b] Intakes of pollutants through consumption of water were obtained from previous studies.[24–28]

respectively. However, because cancer risk is highly dependent on personal characteristics, values between 10^{-4} and 10^{-6} have been reported as assumable.[29,30] In Spain, the regulatory value of maximum risk has been set at 10^{-5}.[31]

Metal exposure brought about higher carcinogenic risks in comparison to organic compounds. If we consider health effects to be additive, cancer risks of 1.18×10^{-6} and 1.60×10^{-5} through ingestion and inhalation, respectively, would be

Table 4 Probabilistic non-carcinogenic and carcinogenic risks of total exposure to POPs and metals

						Percentiles		
			Mean	Median	SD	75th	90th	95th
Hazard Quotient	Oral	PCDD/Fs	3.09E−03	1.34E−03	6.31E−03	3.05E−03	6.81E−03	1.12E−02
		Acenaphthene	2.55E−07	1.42E−07	3.94E−07	3.01E−07	5.86E−07	8.65E−07
		Anthracene	1.37E−08	7.12E−09	2.17E−08	1.57E−08	3.10E−08	4.64E−08
		Fluoranthene	7.74E−07	3.28E−07	1.52E−06	8.03E−07	1.77E−06	2.88E−06
		Fluorene	1.57E−07	9.07E−08	2.21E−07	1.87E−07	3.48E−07	5.08E−07
		Naphthalene	3.83E−04	1.83E−04	6.62E−04	4.18E−04	8.62E−04	1.32E−03
		Pyrene	1.02E−06	4.41E−07	2.14E−06	1.07E−06	2.32E−06	3.70E−06
		As	5.43E−03	4.44E−03	4.08E−03	7.04E−03	1.03E−02	1.30E−02
		Cd	5.54E−05	4.63E−05	3.88E−05	7.12E−05	1.05E−04	1.30E−04
		Mn	5.58E−04	4.83E−04	3.52E−04	7.18E−04	1.00E−03	1.22E−03
		V	1.41E−03	1.08E−03	1.21E−03	1.82E−03	2.83E−03	3.67E−03
	Inhalation	As	3.23E−03	2.51E−03	2.74E−03	4.17E−03	6.38E−03	8.19E−03
		Hg	6.30E−02	5.72E−02	3.80E−02	8.59E−02	1.15E−01	1.35E−01
		Mn	2.59E−02	2.38E−02	1.39E−02	3.37E−02	4.43E−02	5.19E−02
		Naphthalene	2.18E−09	1.97E−09	1.25E−09	2.83E−09	3.81E−09	4.50E−09
Cancer risk	Oral	PCDD/Fs	1.23E−07	5.73E−08	2.31E−07	1.28E−07	2.76E−07	4.28E−07
		Benzo(a)anthracene	4.75E−10	1.95E−10	1.07E−09	4.85E−10	1.10E−09	1.71E−09
		Chrysene	7.04E−12	3.16E−12	1.44E−11	7.51E−12	1.60E−11	2.55E−11
		Benzo(k)fluoranthene	8.57E−11	4.27E−11	1.38E−10	9.51E−11	1.98E−10	3.05E−10
		Benzo(a)pyrene	3.64E−09	1.73E−09	6.96E−09	3.91E−09	8.36E−09	1.29E−08
		Indeno(1,2,3-c,d)pyrene	8.06E−10	3.56E−10	1.51E−09	8.73E−10	1.83E−09	2.94E−09
		Dibenzo(a,h)anthracene	1.96E−09	7.90E−10	4.14E−09	1.98E−09	4.44E−09	7.19E−09
		Benzo(b)fluoranthene	8.57E−10	4.27E−10	1.38E−09	9.51E−10	1.98E−09	3.05E−09
		PCBs	4.44E−10	1.76E−10	1.05E−09	4.45E−10	1.03E−09	1.67E−09
		As	1.05E−06	8.57E−07	7.87E−07	1.36E−06	1.99E−06	2.52E−06
	Inhalation	Chrysene	1.03E−09	8.44E−10	7.55E−10	1.34E−09	2.01E−09	2.49E−09
		Benzo(k)fluoranthene	1.88E−08	1.48E−08	1.54E−08	2.43E−08	3.74E−08	4.83E−08
		Benzo(a)pyrene	2.26E−07	1.63E−07	2.10E−07	2.89E−07	4.74E−07	6.23E−07
		Indeno(1,2,3-c,d)pyrene	4.08E−09	3.46E−09	2.74E−09	5.35E−09	7.47E−09	9.26E−09
		Benzo(g,h,i)perylene	6.55E−08	5.71E−08	4.13E−08	8.51E−08	1.19E−07	1.45E−07
		Dibenzo(a,h)anthracene	9.26E−09	7.10E−09	8.06E−09	1.18E−08	1.87E−08	2.46E−08
		Benzo(b)fluoranthene	1.88E−08	1.44E−08	1.60E−08	2.44E−08	3.77E−08	4.86E−08
		Naphthalene	2.51E−08	2.25E−08	1.46E−08	3.24E−08	4.39E−08	5.20E−08
		PCBs	6.39E−09	6.08E−09	3.13E−09	8.27E−09	1.05E−08	1.20E−08
		As	4.16E−07	3.23E−07	3.54E−07	5.37E−07	8.23E−07	1.06E−06
		Cd	5.28E−08	3.94E−08	4.76E−08	6.71E−08	1.07E−07	1.42E−07
		Cr	1.52E−05	1.19E−05	1.29E−05	1.94E−05	3.01E−05	3.96E−05

estimated. Overall, the multipathway and multipollutant cancer risk would be 1.72×10^{-5}, which is clearly lower than the assumable value of 10^{-4}.[29,30] It is well known that the combined presence of toxicants may result in an addition, potentiation, synergism, or antagonism of effects.[32] However, the extension of these cumulative effects is still unclear and difficult to assess. A preliminary approach would be to assess the risks in a simply cumulative way. However, further studies focused on developing more accurate alternative methodologies, for instance, considering different effects of the chemicals according to target organ or mode-of-action, are required.[33]

The current environmental surveillance program started in 2002. Furthermore, a human health risk assessment of exposure to PCDD/Fs and metals was carried out.[21,34] In the background survey (2002), the mean environmental exposure to PCDD/Fs ranged between 4.66×10^{-6} and 1.07×10^{-5} ng I-TEQ kg^{-1} day^{-1}. Air inhalation was the main exposure pathway, with a percentage of more than 59%. In contrast, in the current survey, inhalation of PCDD/Fs through air only accounted for 9%. The reason of this decline could be the decrease of the air immission levels of PCDD/Fs, which diminished from 65 to 8 fg WHO-TEQ m^{-3}. A significant reduction of 66% in the dietary intake of these pollutants was also observed in the present survey (2007) in comparison to the baseline study (2002). Dietary exposure to PCDD/Fs was reduced from 1.10 to 0.37 pg WHO-TEQ kg^{-1} day^{-1} in the period 2002–2007.[17] According to this trend, a global reduction of the non-carcinogenic and carcinogenic risks was found. The HQ decreased from 5.17×10^{-1} to 3.09×10^{-3}, while the cancer risk index diminished from 5.00×10^{-5} to 1.23×10^{-7}. On the other hand, in our first study, the predicted oral daily exposure to metals ranged from 3.91×10^{-8} to 1.41×10^{-4} (for Hg and Mn, respectively), while inhalation exposure was between 9.83×10^{-10} and 7.48×10^{-7} (for Hg and Pb, respectively). The current values of non-carcinogenic and carcinogenic risks due to chemical exposure are similar to those obtained in previous studies in a number of industrial areas.[34,35] Recently, Schuhmacher *et al.*[36] reported a reduction in the total cancer risk of exposure to PCDD/Fs and metals near a cement plant in Catalonia, after the 20% energy substitution from conventional fuel to sewage sludge (7.40×10^{-6} and 2.84×10^{-6} in 2003 and 2006, respectively). In turn, Kao *et al.*[29] assessed the site-specific health risks in the industrial area of Kaohsiung city (Taiwan). The average cancer risk of a resident living in the District, as a result of exposure to PCDD/Fs emitted by 17 sources, was 3.43×10^{-4}, with inhalation and ingestion being 3.76×10^{-8} and 3.42×10^{-4}, respectively.

One of the main advantages of probabilistic methods such as Monte-Carlo simulation is their capacity to deal with uncertainty and variability, in comparison to more traditional deterministic approaches. Data variability refers to true heterogeneity or diversity, while uncertainty is related to the lack of total knowledge.[21] In the present study both exposure and risk presented a high degree of uncertainty and variability, associated with standard deviations even higher than 50% of the mean value. This can be explained by the high variability of environmental pollutant concentrations found in soil and air samples collected in the area of study.[12] Moreover, food levels also presented an important variability, not only among different food groups but also among specific items of the same category.[14–17] As chemical analysis of micropollutants has continuously improved, the analytical uncertainty has decreased. However, the uncertainty associated to some exposure parameters is still high, being particularly notable for some variables linked to human physiology, such as intake fractions. Moreover, the variability of the same exposure parameters would also be important, as human receptors may present many differences (*e.g.*, body weight, age, *etc.*). Because of that, risk values of environmental and dietary exposure to micropollutants were presumed to present high variability/uncertainty, as has certainly been found.

In the present survey, we evaluated environmental exposure to POPs and metals for the local population of Tarragona County. Dermal absorption and soil ingestion were the most important pathways of environmental exposure for POPs and metals, respectively. However, this exposure is still considerably lower than the dietary intake of these contaminants, with percentages <2% in most cases. In summary, the current concentrations of micropollutants should not mean significant additional non-carcinogenic and carcinogenic risks for the population, considering a combined (environmental + dietary) exposure.

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