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State of the art and advances in the impact assessment of dioxins and dioxin-like compounds

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Abstract Polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) are toxic and persistent organic pollutants that are able to enter the food chain, accumulate in the fat tissues of animals, and consequently pose a serious risk for human health. Consolidated tools for exposure assessment have been implemented during the last decades and widely used, both in the environmental monitoring and in modeling activities. Although the emissive trend and the concentrations in the environment have gradually decreased during the last 20 years, some situations are still underrated and not adequately controlled by the environmental legislation. On the other hand, a complete monitoring of all the pathways of exposure to PCDD/Fs and PCBs is technically and economically unfeasible. Therefore, this paper aims at providing an overview of the traditional approaches used to assess the impacts of PCDD/Fs and PCBs and presenting the novelties introduced during the last years. After an initial characterization of their toxicity and their effects on health, this paper focuses on activities and situations that can result in critical releases of PCDD/Fs and PCBs into the atmosphere and that can represent a hidden threat for the population. In the final part, this study presents the current methodologies for exposure assessment, summarizes the food chain models in a unified way, and puts the light on new methods that can help environmental scientists, risk assessors, and decision makers to estimate the risk related to exposure to PCDD/Fs in different contexts.

Keywords Polychlorinated dibenzo-*p*-dioxins · Polychlorinated dibenzofurans · Polychlorinated biphenyls · Persistent organic pollutants · Exposure assessment · Risk assessment

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Introduction

Polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) are semi-volatile chlorinated organic compounds, which are lipophilic, hydrophobic, and chemically stable (Lee and Nicholson 1994). These characteristics lead to a strong resistance to environmental degradation, which causes the tendency to magnification in the food chain and bioaccumulation in animal and human tissues. Their low solubility in water even decreases with increasing the degree of chlorination (Mackay et al. 2006). For these reasons, PCDD/Fs are classified as persistent organic pollutants (POPs).

PCDD/Fs are composed of 75 PCDD and 135 PCDF congeners. Among these, 17 congeners (7 for PCDDs and 10 for PCDFs) are particularly harmful to humans (IARC 2015). Their toxicity depends on the number and

position of the chlorine atoms on the aromatic ring. The most toxic compounds are the 2,3,7,8-tethrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) and the 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin (1,2,3,7,8-PeCDD) (Van den Berg et al. 2006). Among the 209 congeners that compose the group of PCBs, 12 co-planar congeners demonstrated toxic effects similar to those provoked by the 17 PCDD/F toxic congeners. For this reason, such compounds are usually referred to as "dioxin-like PCBs" (WHO 2014).

Several studies report that more than 90 % of the daily intake of PCDD/Fs results from ingestion of contaminated food (Sasamoto et al. 2006; Eduljee and Gair 1996). Thus, if specific situations of direct exposure by inhalation are excluded (Morra et al. 2006), the food chain is the primary pathway of exposure. PCDD/Fs, mainly emitted into the atmosphere, are partially adsorbed to particulate matter (PM), which reaches soil and vegetation through atmospheric deposition; PCDD/Fs can then contaminate cultivations (e.g., cereals and, less commonly, vegetables) and grass; hence, PCDD/Fs can directly enter the human body at this stage, through consumption of contaminated cereals and vegetables, or can contaminate the diet of cattle and livestock in general, through grass consumption and accidental soil ingestion; therefore, humans are exposed to PCDD/F intake by consumption of contaminated meat, eggs, fish, milk, and dairy products. The higher contribution from one kind of food rather than another depends on the diet of the single individual, although cereals, meat, fish, milk, and dairy products are major contributors (Bilau et al. 2009; De Mul et al. 2008; Fattore et al. 2006). Minor contributions are given by inhalation of contaminated air, accidental ingestion of soil, and dermal contact.

PCDD/Fs are not industrial products but are unwanted sub-products of thermal reactions or incomplete combustion in the presence of precursors containing chlorine (Fiedler 1996). In thermal processes, between 500 and 800 °C, PCDD/Fs are formed in the gas phase by pyrolytic rearrangement of precursors (e.g., chlorophenols and chlorobenzenes) (Stanmore 2004). As an alternative, at lower temperatures (200–400 °C), PCDD/Fs may undergo de novo formation, through oxidation of existing polycyclic compounds and subsequent chlorination, mediated by catalysts. Chlorine is usually made available in solid phase by chlorinated compounds or is present as atomic chlorine (Stanmore 2004).

During the last 20 years, different kinds of improvements have been introduced in the processes responsible for the generation of PCDD/Fs and in the technologies for air pollution control (APC). However, some situations are still uncontrolled and their contribution to releases of POPs may be underrated. On the other hand, a complete monitoring of all the pathways of exposure to PCDD/Fs and PCBs is technically and economically unfeasible. Therefore, this paper aims at providing an overview of the traditional approaches used to assess the impacts of PCDD/Fs and PCBs and presenting the novelties introduced during the last years. After a presentation of the activities and situations that can result in critical emissions and that can represent a hidden threat for the population, this paper presents the current methodologies for exposure assessment, with particular regards to food chain models, presented here in a unified way, and puts the light on new methods that can help environmental scientists, risk assessors, and decision makers to estimate the risk related to exposure to PCDD/Fs in different contexts.

Toxicity and risk estimation

Effects on human health

2,3,7,8-TCDD is the most studied congener, since it is an endocrine disruptor and a demonstrated carcinogen for humans, classified by the International Agency for Research of Cancer (IARC) in the Group 1 (IARC 2015). The remaining six toxic PCDD congeners are classified by the IARC as belonging to the Group 3, showing inadequate evidence of carcinogenicity in humans. The 12 dioxin-like PCBs were recently upgraded from Group 3 to Group 1, due to strong supporting evidence to their cancer potential, as well as all the 10 PCDF toxic congeners (IARC 2015).

Short-term (or acute) exposure to high concentrations of 2,3,7,8-TCDD alters the liver function and may cause skin lesions, like persistent chloracne and patchy darkening of the skin (WHO 2014). Long-term (or chronic) exposure to 2,3,7,8-TCDD may result in both non-carcinogenic and carcinogenic effects, such as lung cancer, soft-tissue sarcoma, non-Hodgkin lymphoma, digestive system cancer, multiple myeloma, and other malignant neoplasms (IARC 1997).

Determination of toxicity

The toxicity equivalence factor (TEF) is a parameter that defines the estimated toxicity of each congener with respect to an index chemical (USEPA 2010). With regards to dioxin-like compounds, the index chemical is 2,3,7,8-TCDD (Van den Berg et al. 1998). The main assumption behind the TEF definition is that the effects of each congeners are dose or concentration additive (Van den Berg et al. 1998). The weighted toxicity of each "*i*" congener, expressed as toxicity equivalent (TEQ_i), is obtained by multiplying the concentration of the chemical by its TEF. The overall toxicity of a mixture (TEQ) is expressed by summing up the contribution of each congener. The World Health Organization (WHO) suggested to re-evaluate TEFs about every 5 years and update them on the basis of new scientific information (USEPA 2010).

Prior to the TEF definition schemes proposed by the WHO, other schemes were provided by the US Environmental Protection Agency (USEPA) and the North Atlantic Treaty Organization (NATO). The latter is commonly referred to as International TEF (I-TEF) scheme. The evolution in the definition of the main TEF schemes is summarized in Table 1.

Risk estimation and related parameters

In this section, priority is given to long-term exposure and to chronic effects on health, which are usually hidden due to the low levels of concentrations that characterize this type of exposure.

Non-carcinogenic effects

The parameters that allow estimating the non-cancer risk are the USEPA's reference dose (RfD) or the reference concentration (RfC), depending on the route of exposure (oral or inhalation, respectively). RfD (or RfC) is defined as the daily dose (or mean concentration) of a specific substance that is likely to avoid an appreciable risk of adverse effects during a lifetime (USEPA 2015). The RfD is expressed in milligrams of substance per kilogram of body weight per day (mg kg^{-1} day^{-1}), while the RfC is expressed as mg m^{-3} of air contaminant.

The tolerable daily intake (TDI) was proposed by the WHO in 1990 for exposure by ingestion (WHO 1991). The TDI was introduced to ensure a level of exposure that should not lead to an appreciable health risk over a lifetime (WHO 2008). The TDI for PCDD/Fs and dioxin-like PCBs was progressively refined over the years: the first formulation, proposed by the WHO, defined a value of 10 $pg_{TEQ}\,kg^{-1}$ of body weight per day (WHO 1991); in 1998, the WHO updated this value to a range of 1–4 $pg_{WHO\text{-}TEQ}\,kg^{-1}$ day^{-1}, stressing the necessity of reaching a final value of 1 $pg_{WHO\text{-}TEQ}\,kg^{-1}$ day^{-1} (WHO 1998). The current guide value proposed by the European Community is 14 $pg_{WHO\text{-}TEQ}\,kg^{-1}$ per week (European Commission 2006a).

Carcinogenic effects

The USEPA's approach toward carcinogenic effects assumes a linear dose–response relationship with no threshold, since a damage to a single cell can be sufficient to cause cancer. The slope of this curve, called "cancer potency" or "slope factor" (SF), defines the cancer risk at low doses, expressed in probabilistic terms (USEPA 2005). Substance-specific SFs have been proposed by the USEPA both for ingestion (SF_{oral}) and for inhalation (SF_{inhal}). SFs are expressed in terms of $(mg\,kg^{-1}\,day^{-1})^{-1}$. A simplification of the concept of SF is given by the introduction of the unit risk (UR) approach: The UR expresses the cancer potency in terms of concentration of a substance in water or in air (USEPA 2005). Exposure occurs through ingestion (UR_{oral}) in the first case and through inhalation (UR_{inhal}) in the second case. The UR is defined as the upper-bound excess lifetime risk of cancer that can derive from continuous exposure to 1 $\mu g\,L^{-1}$ or 1 $\mu g\,m^{-3}$ of a carcinogenic substance, in water or in air, respectively (USEPA 2015). Similarly to RfC, the UR combines information on the exposure with information on the toxicity of an agent (Benjamin and Belluck 2001). The UR is expressed in terms of $(\mu g\,L^{-1})^{-1}$ or $(\mu g\,m^{-3})^{-1}$, for water ingestion or inhalation, respectively.

Congener-specific values for RfD and cancer risk parameters (SF_{oral}, SF_{inhal}, and UR_{inhal}) are provided by the USEPA and are reported in Table 2.

No congener-specific values for UR_{oral} are provided for dioxin and dioxin-like compounds. A general value of 1.0E-05 $(\mu g\,L^{-1})^{-1}$ is proposed for PCBs (USEPA 2014b). SFs for non-dioxin-like PCBs are also provided by the USEPA concerning the inhalation and ingestion routes, both equal to 2 $(mg\,kg^{-1}\,d^{-1})^{-1}$ (USEPA 2012). Since also non-dioxin-like PCBs can cause cancer, their total dose should be multiplied by the generic PCB SF and the consequent risk should be added to that induced by dioxin-like PCBs (USEPA 2000).

Table 1 Evolution of the TEFs of PCDD/Fs and dioxin-like PCBs

		USEPA 1987[a]	NATO 1989[b]	WHO 1994[c]	WHO 1998[d]	WHO 2005[e]
PCDDs						
2,3,7,8-TCDD		1	1		1	1
1,2,3,7,8-PeCDD		0.5	0.5		1	1
1,2,3,4,7,8-HxCDD		0.04	0.1		0.1	0.1
1,2,3,6,7,8-HxCDD		0.04	0.1		0.1	0.1
1,2,3,7,8,9-HxCDD		0.04	0.1		0.1	0.1
1,2,3,4,6,7,8-HpCDD		0.001	0.1		0.01	0.01
1,2,3,4,6,7,8,9-OCDD		0	0.001		0.0001	0.0003
PCDFs						
2,3,7,8-TCDF		0.1	0.1		0.1	0.1
1,2,3,7,8-PeCDF		0.1	0.05		0.05	0.03
2,3,4,7,8-PeCDF		0.1	0.5		0.5	0.3
1,2,3,4,7,8-HxCDF		0.01	0.1		0.1	0.1
1,2,3,6,7,8-HxCDF		0.01	0.1		0.1	0.1
2,3,4,6,7,8-HxCDF		0.01	0.1		0.1	0.1
1,2,3,7,8,9-HxCDF		0.01	0.1		0.1	0.1
1,2,3,4,6,7,8-HpCDF		0.001	0.1		0.01	0.01
1,2,3,4,7,8,9-HpCDF		0.001	0.1		0.01	0.01
1,2,3,4,6,7,8,9-OCDF		0	0.001		0.0001	0.0003
dioxin-like PCBs						
3,3′,4,4′-TCB	(PCB 77)			0.0005	0.0001	0.0001
3,4,4′,5-TCB	(PCB 81)			–	0.0001	0.0003
2,3,3′,4,4′-PeCB	(PCB 105)			0.0001	0.0001	0.00003
2,3,4,4′,5-PeCB	(PCB 114)			0.0005	0.0005	0.00003
2,3′,4,4′,5-PeCB	(PCB 118)			0.0001	0.0001	0.00003
2′,3,4,4′,5-PeCB	(PCB 123)			0.0001	0.0001	0.00003
3,3′,4,4′,5-PeCB	(PCB 126)			0.1	0.1	0.1
2,3,3′,4,4′,5-HxCB	(PCB 156)			0.0005	0.0005	0.00003
2,3,3′,4,4′,5′-HxCB	(PCB 157)			0.0005	0.0005	0.00003
2,3′,4,4′,5,5′-HxCB	(PCB 167)			0.00001	0.00001	0.00003
3,3′,4,4′,5,5′-HxCB	(PCB 169)			0.01	0.01	0.03
2,3,3′,4,4′,5,5′-HpCB	(PCB 189)			0.001	0.001	0.00003

[a] USEPA (1987)

[b] NATO/CCMS (1988)

[c] Ahlborg et al.(1994)

[d] Van den Berg et al. (1998)

[e] Van den Berg et al. (2006)

Cancer risk calculation

The cancer risk (R) is finally estimated by multiplying the suitable parameter (SF_{oral}, SF_{inhal}, UR_{oral}, or UR_{inhal}) by the average concentration (C) of the substance in the environmental medium and, when using SF_{oral} SF_{inhal}, by the exposure (E) (Benjamin and Belluck 2001):

$$R = C{\cdot}E{\cdot}SF = C{\cdot}UR \quad (1)$$

where C is expressed in mg kg^{-1}, μg L^{-1}, or μg m^{-3}, for

Table 2 Congener-specific values for RfD, SF_{oral}, SF_{inhal}, and UR_{inhal}

		RfD (mg kg^{-1} day^{-1})	SF_{oral} ((mg kg^{-1} day^{-1})$^{-1}$)	SF_{inhal} ((mg kg^{-1} day^{-1})$^{-1}$)	UR_{inhal} ((μg m^{-3})$^{-1}$)
PCDDs[a]					
2,3,7,8-TCDD		7.0E-10	1.3E+05	1.3E+05	3.8E+01
1,2,3,7,8-PeCDD		7.0E-10	1.3E+05	1.3E+05	3.8E+01
1,2,3,4,7,8-HxCDD		7.0E-11	1.3E+04	1.3E+04	3.8E+00
1,2,3,6,7,8-HxCDD		7.0E-11	6.2E+03	4.5E+03	1.3E+00
1,2,3,7,8,9-HxCDD		7.0E-11	6.2E+03	4.5E+03	1.3E+00
1,2,3,4,6,7,8-HpCDD		7.0E-12	1.3E+03	1.3E+03	3.8E-01
1,2,3,4,6,7,8,9-OCDD		2.0E-13	3.9E+01	3.8E+01	1.1E-02
PCDFs[a]					
2,3,7,8-TCDF		7.0E-11	1.3E+03	1.3E+04	3.8E+00
1,2,3,7,8-PeCDF		2.0E-11	3.9E+03	3.8E+03	1.1E+00
2,3,4,7,8-PeCDF		2.0E-10	3.9E+04	3.8E+04	1.1E+01
1,2,3,4,7,8-HxCDF		7.0E-11	1.3E+04	1.3E+04	3.8E+00
1,2,3,6,7,8-HxCDF		7.0E-11	1.3E+04	1.3E+04	3.8E+00
2,3,4,6,7,8-HxCDF		7.0E-11	1.3E+04	1.3E+04	3.8E+00
1,2,3,7,8,9-HxCDF		7.0E-11	1.3E+04	1.3E+04	3.8E+00
1,2,3,4,6,7,8-HpCDF		7.0E-12	1.3E+03	1.3E+03	3.8E-01
1,2,3,4,7,8,9-HpCDF		7.0E-12	1.3E+03	1.3E+03	3.8E-01
1,2,3,4,6,7,8,9-OCDF		2.0E-13	3.9E+01	3.8E+01	1.1E-02
PCBs[b]					
3,3′,4,4′-TCB	(PCB 77)	–	1.3E+01	3.8E-03	1.3E+01
3,4,4′,5-TCB	(PCB 81)	–	1.3E+01	3.8E-03	1.3E+01
2,3,3′,4,4′-PeCB	(PCB 105)	–	1.3E+01	3.8E-03	1.3E+01
2,3,4,4′,5-PeCB	(PCB 114)	–	6.5E+02	1.9E-02	6.5E+02
2,3′,4,4′,5-PeCB	(PCB 118)	–	1.3E+01	3.8E-03	1.3E+01
2′,3,4,4′,5-PeCB	(PCB 123)	–	1.3E+01	3.8E-03	1.3E+01
3,3′,4,4′,5-PeCB	(PCB 126)	–	1.3E+04	3.8E-00	1.3E+04
2,3,3′,4,4′,5-HxCB	(PCB 156)	–	6.5E+01	1.9E-02	6.5E+01
2,3,3′,4,4′,5′-HxCB	(PCB 157)	–	6.5E+01	1.9E-02	6.5E+01
2,3′,4,4′,5,5′-HxCB	(PCB 167)	–	1.3E+00	1.3E+00	3.8E-04
3,3′,4,4′,5,5′-HxCB	(PCB 169)	–	1.3E+03	1.3E+03	3.8E-01
2,3,3′,4,4′,5,5′-HpCB	(PCB 189)	–	1.3E+01	3.8E-03	1.3E+01

[a] USEPA (2014a)

[b] OEHHA (2009)

exposure by food ingestion, water ingestion, or inhalation, respectively. E is defined as follows:

$$E = \frac{IR \cdot ED \cdot EF}{BW \cdot AT} \quad (2)$$

where IR is the intake rate of contaminant (expressed in mg day^{-1}, L day^{-1}, or m^3 day^{-1}, for exposure by food ingestion, water ingestion, or inhalation, respectively); if referring to inhalation, IR is about 16 m^3 day^{-1} of air for adults (Stiefelman 2007), but the USEPA proposes a more precautionary value of 20 m^3 day^{-1} (USEPA 2014c); if referring to water consumption, IR can be assumed as 2 L day^{-1} for adults and 1 L day^{-1} for children (USEPA 2014c); ED is the exposure duration (expressed in years); EF is the exposure frequency (expressed in days year^{-1}); BW is the body weight

(expressed in kilograms); AT is the averaging time (expressed in years), normally assumed as 70 years (USEPA 2009). To account for the amplification of the risk related to exposure in the early-life stages, age-dependent adjustment factors (ADAFs) can be introduced for each period of life ($ADAF = 10$ for age <2 years; $ADAF = 3$ for 2 years ≤ age < 16 years; $ADAF = 1$ for age ≥16 years). For each period, the risk results in (USEPA 2009):

$$R = C \cdot \frac{IR \cdot ED \cdot EF}{BW \cdot AT} \cdot ADAF \cdot SF \quad (3)$$

The total R is obtained by sum of the single contributions during each period. The overall cancer risk is assumed to be given by the sum of the cancer risks (related to the same target organ) induced by each substance and through each route of exposure.

Due to the absence of a threshold dose for carcinogenic contaminants, the cancer risk is never zero but may be low enough to be considered acceptable. Conventionally, the cancer risk is considered acceptable if it is comprised between 10^{-6} and 10^{-4}, i.e., if it is able to induce no more than 1 excess case of cancer in 1,000,000 to 10,000 inhabitants (USEPA 1999a). The choice of the target value can depend on the population density of an area or on the economic feasibility of the efforts requested to reduce the impacts (USEPA 1999a).

Atmospheric fate and typical levels of exposure in the environment

Atmospheric fate

When emitted into the atmosphere, PCDD/Fs and PCBs in the gas phase are mainly subject to photolysis and reaction with ozone, nitrate radicals, and, especially, hydroxyl radicals (Atkinson 1996; Lohmann and Jones 1998). The photodegradation rate decreases when the degree of chlorination increases (Wu et al. 2005). Catalytic conversion, atmospheric transport, and, finally, deposition represent the main routes for particle-bound congeners (Atkinson 1996). Lowly chlorinated congeners are associated with larger particles: this lets us suppose that such congeners may not be transported at great distance from the source. Thus, their local impact may be higher with respect to congeners with higher degree of chlorination. Furthermore, the toxicity of lowly chlorinated congeners is higher, especially with regards to PCDD/Fs.

Higher concentration levels of PCDD/Fs and PCBs are normally observed in winter with respect to summer, both because of the lower height of the atmospheric boundary layer and because additional emission sources (e.g., domestic heating) are present in winter (Ding et al. 2012). After their emission into the atmosphere, the lower chlorinated compounds show higher tendency to remain in the gas phase; as the degree of chlorination grows, the affinity to the particle phase increases (Lohmann and Jones 1998). This behavior seems to be correlated with the octanol–air partition coefficient of each congener (Lohmann et al. 2007) and with their vapor pressure (Saral et al. 2015). Temperature also influences the partitioning: Low temperatures contribute to a higher particle-phase fraction (Lee et al. 2008); thus, a seasonality can be observed during the year (Saral et al. 2015; Ding et al. 2012). Depending on the degree of chlorination, the typical size of particles which the PCDD/Fs are bounded to is different: Lowly chlorinated congeners are prevalently associated with large particles (>2.1 μm), while highly chlorinated congeners are often associated with fine particles (<2.1 μm) (Oh et al. 2002).

Limit and guideline values in environmental media

The emissions of PCDD/Fs and dioxin-like compounds are regulated by many national legislations. In Europe, waste incinerators are obliged to comply with a concentration of 0.1 $ng_{I\text{-}TEQ}\,Nm^{-3}$ at the stack (European Commission 2000). The concentration of PCDD/Fs and dioxin-like compounds in foodstuffs is also regulated: At a European level, for instance, limit values for PCDD/Fs and dioxin-like PCBs in foodstuffs were first introduced in 2001 (European Commission 2001) and then re-evaluated twice in 2006 (European Commission 2006a; European Commission 2006b). In the last formulation (European Commission 2011), maximal PCDD/F and dioxin-like PCB concentrations are reported for different types of food.

Only guide values, with no regulatory purpose, were introduced for ambient air and sediment concentrations, and for atmospheric deposition. Limit values on soil concentrations exist in some countries: In Italy, for instance, PCDD/F limit values of 10 $ng_{WHO\text{-}TEQ}\,kg^{-1}$ on dry weight (dw) for residential and green areas and 100 $ng_{WHO\text{-}TEQ}\,kg^{-1}{}_{dw}$ for commercial and industrial areas have been enforced since 2006 (Official Journal of the Italian

Republic 2006). The same decree sets limit values for PCBs to 0.06 and 5 mg kg^{-1}_{dw} for residential and green areas and for commercial and industrial areas, respectively (Official Journal of the Italian Republic 2006).

Typical levels in the environment

The ambient air concentration gradually increases when moving from rural areas to urban and industrial districts (Abad et al. 2007). Concentrations in the range 5–45, 10–357, and 5–1196 $fg_{I\text{-}TEQ}\,m^{-3}$ were measured in Catalonia (Spain) during a 10-year study (1994–2004) in rural, urban, and industrial areas, respectively (Abad et al. 2007). Concentrations measured near a municipal solid waste (MSW) incinerator in Italy were comprised between 22 and 337 $fg_{I\text{-}TEQ}\,m^{-3}$ (Caserini et al. 2004). Concentrations between 10 and 138 $fg_{I\text{-}TEQ}\,m^{-3}$ were measured in the urban area of Trento (Italy) in 2006–2007 (Ragazzi et al. 2014a); monitoring campaigns carried out in other cities showed mean concentrations of 40–119 $fg_{I\text{-}TEQ}\,m^{-3}$ in Thessaloniki (Greece) (Kouimtzis et al. 2002), 26–220 $fg_{I\text{-}TEQ}\,m^{-3}$ in Manchester (Lohmann et al. 2000), 65 $fg_{I\text{-}TEQ}\,m^{-3}$ in Rome (Menichini et al. 2007), and 40–55 $fg_{I\text{-}TEQ}\,m^{-3}$ in Houston (Correa et al. 2004). Concentration levels are higher in developing countries: In China, for instance, mean PCDD/F concentrations of 57–1280 $fg_{I\text{-}TEQ}\,m^{-3}$ were measured in Guangzhou (Yu et al. 2006) and 18–644 $fg_{I\text{-}TEQ}\,m^{-3}$ in Beijing (Li et al. 2008), while concentrations of 156–1440 $fg_{I\text{-}TEQ}\,m^{-3}$ were measured near an MSW incinerator (Zhang et al. 2014).

Atmospheric deposition of PCDD/Fs varying from 0.75 to 3.73 $pg_{WHO\text{-}TEQ}\,m^{-2}\,day^{-1}$ was measured in the surroundings of an MSW incinerator in Italy (Vassura et al. 2011). In an Italian valley, characterized by the presence of a steel plant equipped with the best available technologies (BATs), the mean PCDD/F deposition measured during 1 year was 1.40 $pg_{WHO\text{-}TEQ}\,m^{-2}\,day^{-1}$ (Schiavon et al. 2013). In a similar context, in the vicinity of an Italian steel plant, the PCDD/F deposition measured during 1 year resulted in the range 0.91–3.17 $pg_{I\text{-}TEQ}\,m^{-2}\,day^{-1}$ (Onofrio et al. 2014).

Concentrations in soil are also variable depending on the land use: Industrial areas in Slovakia showed PCDD/F soil concentrations of 2.74–7.80 $ng_{WHO\text{-}TEQ}\,kg^{-1}_{dw}$, while a concentration of 0.66 $ng_{WHO\text{-}TEQ}\,kg^{-1}_{dw}$ was measured at a background site (Dömötörová et al. 2012). A monitoring campaign carried out in the Alpine valley of Trento (Italy) showed mean values ranging from 0.14 $ng_{I\text{-}TEQ}\,kg^{-1}_{dw}$ (background site) and 4.86 $ng_{I\text{-}TEQ}\,kg^{-1}_{dw}$ (industrial site), with intermediate values of 0.92–0.96 $ng_{I\text{-}TEQ}\,kg^{-1}_{dw}$ observed along a highway (Rada et al. 2015).

The emissions of PCDD/Fs and dioxin-like PCBs have generally decreased during the last 20 years, especially due to improvements in the industrial sector and after the adoption of the BATs (Argiriadis et al. 2014). Monitoring campaigns carried out in an urbanized valley in Italy showed a continuous decrease in the ambient air concentrations of urban background areas, from 110.4 $fg_{I\text{-}TEQ}\,m^{-3}$ in 2002 to 17.38–24.45 $fg_{I\text{-}TEQ}\,m^{-3}$ in 2009–2010 (Ragazzi et al. 2014a). A 70 % decline was observed between 1994 and 2004, during the monitoring campaign in Catalonia (Abad et al. 2007). The atmospheric deposition of PCDD/Fs and PCBs decreased from 12–32 to 5–10 $pg_{I\text{-}TEQ}\,m^{-2}\,day^{-1}$ between 1992 and 2011 in the Rhine-Ruhr region (Bruckmann et al. 2013). PCDD/F soil concentrations near a steel plant in an Alpine valley decreased from 4.10 $ng_{I\text{-}TEQ}\,kg^{-1}$ in 2010 to 2.14 $ng_{I\text{-}TEQ}\,kg^{-1}$ in 2012 (Rada et al. 2015).

In spite of their general reduction, critical PCDD/F and PCB levels are still observed near industrial facilities, especially in developing countries: very high concentrations in soil can be observed near abandoned industrial areas, like in the case of a pentachlorophenol factory in China, where average PCDD/F concentrations of 193 and 667 $ng_{WHO\text{-}TEQ}\,kg^{-1}$ were measured in soil and sediment samples, respectively (Li et al. 2012); in the vicinity of another Chinese pentachlorophenol factory, the average PCDD/F air concentration was 39.7 $pg_{I\text{-}TEQ}\,m^{-3}$ (Chang et al. 2011).

Given the dominant role of the food chain in the exposure to PCDD/Fs and PCBs, atmospheric deposition should be regarded with particular attention. Contrarily to ambient air concentrations, there is a lower number of monitoring activities concerning deposition fluxes. The introduction of limit values for PCDD/Fs and PCBs in food (European Commission 2006a) is a strategy to reduce human exposure through consumption of industrial products but may not be sufficient to limit the intake to the target value of 1 $pg_{WHO\text{-}TEQ}\,kg^{-1}\,day^{-1}$, as it actually occurs in various countries (Fattore et al. 2006). Furthermore, limit values for the PCDD/F and PCB content in foodstuffs may not preserve the population from the intake related to the consumption of locally produced food (Heimstad et al. 2015).

Emission sources and new criticalities

The presence of PCDD/Fs was historically associated to large-scale thermal processes, such as MSW, industrial and hazardous waste incineration, cement kilns, wood and coal burning for energy production, road traffic, non-ferrous metal smelting, and production of iron and steel (Schecter et al. 1995; Stanmore 2004; Fiedler 1996). During the last decade, important improvements have been introduced in the technologies for APC. The combination of improved conditions of combustion, the adoption of the BATs, and the imposition of strict emission limits have led to considerably reduce the impacts of MSW incinerators in the surroundings of their location (Schecter et al. 1995; Ragazzi et al. 2013). The reduction of the emissions from these processes let other sectors become important sources of PCDD/Fs, such as residential wood burning. According to the last comprehensive inventory on PCDD/F emission in Europe (Quaß et al. 2004), residential wood burning is the highest contributor of PCDD/Fs in the environment with 523–969 $g_{I\text{-}TEQ}\,year^{-1}$. This sector is followed by sintering plants (387–470 $g_{I\text{-}TEQ}\,year^{-1}$), preservation of wood (118–310 $g_{I\text{-}TEQ}\,year^{-1}$), fires (60–371 $g_{I\text{-}TEQ}\,year^{-1}$), residential coal burning (82–337 $g_{I\text{-}TEQ}\,year^{-1}$), MSW incineration (178–232 $g_{I\text{-}TEQ}\,year^{-1}$), steel plants equipped with electric arc furnace (141–172 $g_{I\text{-}TEQ}\,year^{-1}$), and domestic waste burning (116–187 $g_{I\text{-}TEQ}\,year^{-1}$) (Quaß et al. 2004).

Thanks to research studies carried out in the last decade, a new sector has been identified as a potential non-negligible emitter of PCDD/Fs: mechanical-biological treatments (MBTs) of MSW, such as composting, biostabilization and biodrying (Rada et al. 2006). Differently from thermo-chemical processes, PCDD/Fs are not produced in MBTs but are present in traces in the incoming waste (Rada et al. 2006). PCDD/Fs can be partially stripped by the process air, which becomes enriched in PCDD/Fs and releases them at the outlet (Liu et al. 2009). There is great variability in the emission factors reported in the literature, since PCDD/F emissions depend on the APC strategies adopted and on the PCDD/F content in the waste, specifically in the food waste, variable country by country (Ionescu et al. 2012).

At a European level, only Germany and Austria set PCDD/F concentration limit values in the emissions from MBTs. The limit is equal to that of MSW incinerators ($0.1\ ng_{I\text{-}TEQ}\,Nm^{-3}$) (Federal Ministry of Agriculture and Forestry, Environment and Water 2002; Federal Ministry for the Environment, Nature Conservation and Nuclear Safety 2001). However, considering MSW incineration and MBTs at the same level can lead to erroneous considerations: Indeed, potentially high impacts are expected in the surroundings of a MBT plant if the dispersion in the atmosphere is not optimized (Rada et al. 2007). The conventional APC systems adopted in MBTs are based on biofiltration, especially open biofilters. The latter are located at ground level and are characterized by low velocities of the outgoing treated airstream (in the order of cm s^{-1}). Such conditions negatively affect the dilution of the plume in the atmosphere (Rada et al. 2011; Ragazzi et al. 2014b).

Exposure assessment

Conventional methodologies

Monitoring

The human exposure and the health risk associated with emissions of toxic and persistent pollutants could be directly determined if the human habits of the single individual and the contamination levels in all the major environmental media were known. In this case, the contaminants should be continuously monitored at a single-individual level. In the case of POPs, if considering that oral exposure is dominant with respect to inhalation, the main object of the monitoring activity should be the food. Nevertheless, analyses on food (other than the regulatory inspections) would require time and high costs. Thus, the most convenient approach consists in the measurement of ambient air concentrations and atmospheric deposition to soil or in the analysis on the sediments of water basins.

The most common methodology adopted to sample air for PCDD/F and PCB determination is based on the use of passive samplers and, specifically, on the EPA TO-9A method (Ragazzi et al. 2014a; Castro-Jiménez et al. 2008), consisting in the use of a high-volume air sampler equipped with a quartz fiber filter and a polyurethane foam adsorbent to trap the gaseous phase (USEPA 1999b). Active sampling, following the same prescriptions for PM, can be also performed (Cortés et al. 2014). The analyses are usually carried out with gas chromatography and high-resolution mass spectrometry (GC-HRMS) after extraction of the sample

with a mixture acetone–exhane. However, in spite of a higher accuracy, such method implies higher costs for instrumentation and maintenance compared with passive sampling; higher costs may translate in a lower number of sampling points, and this would decrease the spatial resolution of the monitoring network; as a consequence, possible local situations of critical exposure might not be observed with this methodology.

Atmospheric deposition occurs through removal processes that take place in dry or wet conditions. The dry deposition, active in the absence of precipitation, deposits the pollutants on the surface of soil or vegetation by the combined action of turbulent diffusion, gravitational sedimentation, and inertial impaction. The wet deposition occurs in the presence of precipitation, through processes of incorporation of the pollutants in the clouds (rainout) and runoff pollution (washout). While in dry conditions the pollutants are deposited mainly in the vicinity of the emission source, in wet conditions, the pollutants can be transported over long distances (US Government 1984). The dry deposition (D_d) is related to the ambient air concentrations of the pollutants in gas phase ($C_{a,g}$) and in particle phase ($C_{a,p}$) through the respective deposition velocities (V_{dg} and V_p):

$$D_d = C_a V_d = C_{a,g} V_{dg} + C_{a,p} V_p \tag{4}$$

where V_d is the total dry deposition velocity (Lin et al. 2010). A mean value of 0.42 cm s^{-1} was provided for V_d by Shih et al. (2006), while a value of 0.010 cm s^{-1} was provided for V_{dg} by Sheu et al. (1996). A mean value of 0.44 cm s^{-1} for V_p was obtained by Lin et al. (2010). For diameters greater than the 10–20 μm, the speed is substantially coincident with that obtainable using the Stokes law, while, for the finest particles (<0.2–0.5 μm), the Brownian motion becomes more significant. For intermediate sizes (0.5–5 μm), the combined intervention of the two mechanisms is likely to determine the minimum speed. The wet deposition is composed by the contribution of the suspended particles removed by the precipitation and by the dissolution of the contaminant at the vapor phase. The wet deposition (D_w) is defined as

$$D_w = C_a \cdot S_{tot} \cdot P \tag{5}$$

where P is the precipitation and S_{tot} is the total scavenging ratio, representing the ratio between the concentration in the liquid phase produced by the precipitation and the concentration in air at ground level. S_{tot} is the sum of the contribution deriving from the concentration of the particle-bound contaminant in the raindrop (S_p) and the concentration of the contaminant in the dissolved phase in the raindrop (S_g) (Lin et al. 2010):

$$S_{tot} = S_p \Phi + S_g (1-\Phi) = \Phi \frac{C_{r,p}}{C_{a,p}} + (1-\Phi) \frac{C_{r,g}}{C_{a,g}} \tag{6}$$

where $C_{r,p}$ is the particle-phase concentration in the raindrop, $C_{r,g}$ is the dissolved-phase concentration in the raindrop, and Φ is the ratio between the air concentration of contaminant bound to particles and the total air concentration (Lin et al. 2010). The traditional methodology to monitor the bulk (wet and dry) deposition of organic compounds, still adopted in the deposition monitoring, follows the indications of the Bergerhoff® method (VDI 1996), consisting in a glass-made funnel-jar collection system, protected by a cylindrical container and a ring to avoid interferences by bird excreta (Guerzoni et al. 2004). Wet and dry deposition can be separately measured by adopting a double deposition sampling system equipped with a moveable cover, which can be automatically activated by a rain or moisture sensor (Argiriadis et al. 2014; Anderson and Downing 2006). If air sampling is the most direct method to assess exposure through inhalation, deposition sampling represents a low-cost alternative with respect to analyses on food if the target is the assessment of exposure through ingestion; this is due to the possibility of coupling deposition measurements with food chain models and statistics on food consumption to estimate the intake of PCDD/Fs and dioxin-like compounds.

Surface soil sampling is usually performed by precleaned stainless steel shovels (Li et al. 2012; Denys et al. 2012). The depth of the samples is conventionally 10 cm, both because of the low mobility of POPs in the soil and because of the importance of surface soil from the point of view of the food chain. Analogously to atmospheric deposition, the standard analytical methods to obtain the soil concentration of PCDD/Fs and PCBs are the EPA methods 1613 and 1668B (Rada et al. 2015; Liu et al. 2012; Zhang et al. 2009). In addition to being regulated by the environmental legislation to assess soil quality, the concentration of PCDD/Fs and dioxin-like compounds in soil can be directly used to estimate the accidental soil ingestion by humans and animals and the consequent accumulation in the food chain (Eqs. (7), (8), (14), (16), and (18)).

The standard analytical methods used in the measurement of soil concentration and deposition (EPA methods 1613 and 1668B) are still commonly adopted also for analyses on sediments (Argiriadis et al. 2014), even though an alternative method for extraction was recently adopted by Friedman and Lohmann (2014). What substantially differs from other media is the sampling method, which is conventionally made by manual core drilling, in accordance with the EPA standard operating procedure (SOP) 2016 (USEPA 1994a) or with the more recent SOP SRC-OGDEN-04 (Syracuse Research Corporation 2001). In addition to being a low-cost methodology, the analysis on sediments can provide indications on the contamination level of aquatic ecosystems and, hence, of fishes. However, further research is needed to estimate the interaction between contaminants in sediments and aquatic organisms.

Modeling

As an alternative, the exposure assessment can be based on the modeling approach with validation by on-field measurements. The modeling approach requires the identification of the dominant emission sources of the area of study. The first step consists in retrieving information on the type of emission source to be modeled (point, area, line, or volume source) and on the way the contaminants of interest are released into the environment (continuous or discontinuous release, presence of chimneys, temperature of the off-gas, outgoing velocity). The second step consists in retrieving emission data or, in the absence of measurements, emission factors representing the emission sources as more accurately as possible. The third step concerns the choice of the dispersion model, which should be suitable for the characteristics of the morphology of the area of study, for the source type of interest, for the desired resolution, for the extension of the domain, and for the kind of pollutant (chemically inert or reactive). Examples of dispersion models are AERMOD, CALPUFF, CALGRID, ADMS, and AUSTAL2000, even though several other models are available. The fourth step consists in retrieving the meteorological data required by the model pre-processor. Morphological data are also necessary at this stage to calculate wind fields. Additional information, such as the definition of the deposition velocities or the gas/particle partitioning, may usually be defined or modified in the model settings. The model can then be run and the results should be subsequently compared with on-field data; if necessary, the model should be re-run after calibration. If the approach stopped at this stage, no estimations of the level of exposure could be derived; however, it would be possible to obtain indications on the presence/absence of potential critical situations of exposure. Estimations on the oral intake are conventionally pursued by applying food chain models. The environmental compatibility of human activities can consequently be determined.

Estimation of the intake

The estimation of the exposure to a certain substance or a group of compounds must consider all the relevant routes of intake. The estimation of the total cancer risk is calculated by summing up the contribution of each route of exposure, taking into consideration that a route of exposure may be contaminated though different pathways.

The inhalation route is only affected by contamination of ambient air. Therefore, the estimation of the cancer risk through the definition of URs (Eq. (1)) or the comparison with the RfC for non-cancer risk is immediate. However, the determination of the cancer risk through the definition of SF (Eq. (3) and Eq. (4)) or the comparison with the RfD for non-cancer risk requires information on the exposure, e.g., indication of the inhalation *IR*, *ED*, *EF*, and *BW* of the reference population.

Determining the intake by ingestion is more complex, since diet is an indirect exposure route that is, on its turn, contaminated through different pathways. Fruit, cereals, and vegetables can be contaminated through two main processes: root uptake from soil and atmospheric deposition. Grass is subject to the same pathways of contamination, even though food chain models attribute the majority of the contamination to atmospheric deposition (Slob and Van Jaarsveld 1993). Since grass is the primary food for cattle and livestock, the POPs present in grass contaminate the diet of animals; accidental ingestion of soil contributes also to POP intake by animals; consequently, POPs accumulate in their fat tissues and pass to milk or eggs; meat, milk, dairy products, and eggs, as well as fruit, cereals, and vegetables (in minor contribution), work as carriers of POPs to the human body. Accidental soil ingestion, more probable in children than in adults, represents an additional route of exposure.

The traditional methodologies for intake estimation make use of food chain models developed during the last 25 years. The concentration of POPs in soil (C_s) can be estimated by the following reservoir model based on long-term deposition data (Lorber et al. 2000):

$$C_s = D_{LT}\frac{1-\exp(-K\cdot t_d)}{K\cdot M} \quad (7)$$

where D_{LT} is the mean total deposition flux (expressed in $pg_{WHO\text{-}TEQ}\,m^{-2}\,year^{-1}$), K is the mean annual soil dissipation rate (assumed equal to 0.02772 $year^{-1}$, corresponding to a PCDD/F half-life time of 25 years), t_d is the time since the deposition D_{LT} has been achieved, and M is the soil mixing mass (assumed equal to 112.5 kg m^{-2}) (Lorber et al. 2000). The intake rate of PCDD/Fs and dioxin-like PCBs through soil ingestion (IR_s) by humans can be calculated by the following expression:

$$IR_s = C_s\cdot I_s \quad (8)$$

where I_s is the average daily soil ingestion rate. I_s can be assumed equal to 85 mg day^{-1} for children (Thompson and Burmaster 1991) and to 50 mg day^{-1} for adults (USEPA 1997).

The intake of PCDD/Fs and dioxin-like PCBs through consumption of contaminated cereals, vegetables, and fruit can be estimated by first estimating the concentrations of the pollutants that enter the plant. The concentration of each congener i due to root uptake ($C_{p,r,i}$) can be estimated by the following expression (UK Environment Agency 2009):

$$C_{p,r,i} = C_{s,i}\cdot BCF_i \quad (9)$$

where $C_{s,i}$ is the soil concentration of the i congener and BCF_i is the soil-to-plant concentration factor of the i congener, depending on the type of vegetable (green vegetable, root vegetable, tuber, tree fruit, or herbaceous fruit) and retrievable from Harrad and Smith (1997). The BCF is the ratio between the concentration of a pollutant in a plant or animal tissue and its concentration in an environmental medium (Davies and Dobbs 1984).

The concentration deriving from the deposition can be estimated by summing up two contributions: the dry gaseous deposition ($C_{p,dg,i}$), which is to be calculated for each congener, and particulate deposition ($C_{p,p}$), defined by the following equations:

$$C_{p,dg,i} = \frac{B_{vpa,i}\cdot C_{a,i}}{\rho_a}\left(1-\frac{c\cdot S_t}{P_{l,i}+c\cdot S_t}\right) \quad (10)$$

$$C_{p,p} = \frac{D_{LT}\cdot R_p}{Y_p\cdot k_p}\left[1-\exp\left(-k_p\cdot T_p\right)\right] \quad (11)$$

where $B_{vpa,i}$ is the congener-specific air-to-leaves transfer factor, ρ_a is the air density, c is the Junge constant ($1.7\cdot 10^{-4}$ atm cm), S_t is the surface area of the particulates (which can be assumed as $3.5\cdot 10^{-6}$ cm^2 cm^{-3}), $P_{l,i}$ is the congener-specific saturation vapor pressure of sub-cooled liquid, R_p represents the interception rate of particles by the vegetation (which can be assumed as 0.51), k_p is the loss rate for the plant surface particles (which can be assumed as 126.6 $year^{-1}$), T_p is the exposure time of the vegetation exposure to the deposition per single harvest (0.12 years), and Y_p is the aerial biomass of grass (which can be assumed as 0.25 kg m^{-2}) (Lorber et al. 1994; Harrad and Smith 1997; Meneses et al. 2002). Congener-specific values of B_{vpa} are provided by Harrad and Smith (1997). The concentrations in fruit due to the uptake from air and the stem (C_{as}) can be calculated by a more complex model, whose detailed methodology is reported by Trapp (2007). The intake rate through consumption of vegetables and cereals (IR_v) by humans can be calculated by summing the product between the average daily consumption ($I_{v,j}$) of each of the four "j" categories of vegetables (green vegetable, root vegetable, tuber, and herbaceous fruit), the sum of the total concentrations $C_{p,r}$, $C_{p,dg}$, and $C_{p,p}$ in each of the four categories, estimated by Eq. (9), Eq. (10), and Eq. (11), and a coefficient representing the fraction of vegetable product coming from the contaminated area with respect to the total vegetable products consumed ($L_{v,j}$):

$$IR_v = \sum_{j=1}^{4}\left[L_{v,j}\cdot I_{v,j}\left(C_{p,r,j}+C_{p,dg,j}+C_{p,p,j}\right)\right] \quad (12)$$

With regards to fruit, the intake rate (IR_f) can be calculated by the fraction of fruit coming from the contaminated area with respect to the total fruit consumed (L_f), the average daily fruit consumption (I_f), and the sum of the total concentrations due to

the contribution of root uptake ($C_{p,r,f}$) and uptake from air and the stem:

$$IR_f = L_f I_f \left(C_{p,r,f} + C_{as}\right) \quad (13)$$

The intake of POPs through consumption of milk and dairy products can be estimated by calculating the concentration of POPs in milk fat and then considering the fraction of fat in each kind of product. The concentration in milk fat (C_{mf}) can be estimated by the following expression (Slob and Van Jaarsveld 1993):

$$C_{mf} = \frac{b_{mf}\left(A{\cdot}c_g{\cdot}D + I_{c,s}{\cdot}C_s\right)}{f_f{\cdot}P_m} \quad (14)$$

where A is the average area grazed by one cow per day (which can be assumed as 100 m^2 day^{-1}, approximately), c_g is the fraction of A covered with grass (assumed as 0.9), D is the mean daily deposition, $I_{c,s}$, is the average soil ingestion by the cow (about 225 g day^{-1}), f_f is the fraction of fat in cow's milk (approximately 4–5 %), P_m is the average daily milk production (about 20 kg day^{-1}) ,and b_{mf} is the bioavailability of POPs in milk fat, which is the fraction of contaminant present in milk fat with respect to the amount ingested by the animal (Slob and Van Jaarsveld 1993; Schiavon et al. 2013; Hoogenboom et al. 2006). Differently from the BCF, b_{mf} considers the absorption by the digestive tract and the metabolism (McLachlan 1997). Average values of b_{mf} are listed in Kerst et al. (2004). A mean value of 0.35 was established by the German guideline VDI 2310-46 for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, and 2,3,4,7,8-PeCDF, the most toxic congeners (VDI 2005). The calculation of C_{mf} be refined by summing up the congener-specific contributions after applying congener-specific b_{mf} values, like those provided by McLachlan (1997) as a result of previous studies. The POP intake rate though consumption of milk and dairy products (IR_{md}) can finally be estimated:

$$IR_{md} = C_{mf}\left(L_m{\cdot}I_m{\cdot}m_{f,m} + \sum_k L_{d,k}{\cdot}I_{d,k}{\cdot}m_{f,d,k}\right) \quad (15)$$

where L_m is the fraction of milk produced in the contaminated area over the total milk consumed, I_m is the average daily consumption of milk, $m_{f,\mathrm{m}}$ is the content of fat in milk, $L_{d,k}$ is the fraction of the "k" type of dairy product coming from the contaminated area over the total amount of the "k" dairy product consumed, $I_{d,k}$ is the average daily consumption of the "k" type of dairy product, and $m_{f,d,k}$ is its respective content of milk fat.

A food chain model to determine the concentration in meat was proposed by McLachlan (1997). The model calculates the concentration of POPs in beef fat (C_{bf}) through an integral over the time (t) till the life time of the animal (t_l):

$$C_{bf} = \frac{b_{bf}}{m_{f,b}} \int_{t=0}^{t_l} \left(A{\cdot}c_g{\cdot}D + I_{c,s}{\cdot}C_s\right) dt \quad (16)$$

where $m_{f,b}$ is the mass fraction of fat in beef and b_{bf} is the bioavailability in beef fat (McLachlan 1997). Congener-specific values of b_{bf} were proposed by Slob et al. (1995). The intake rate of POPs by beef consumption results in

$$IR_b = L_b{\cdot}C_{bf}{\cdot}m_{f,b}{\cdot}I_b \quad (17)$$

where L_b is the fraction of beef coming from animals grown in the contaminated area over the total amount of beef consumed and I_b is the average daily consumption of beef.

The concentration of POPs in eggs (C_e) was modeled by Harrad and Smith (1997) and is described by the following expression:

$$C_e = BCF_e{\cdot}m_{y,e}\left[\left(F_sC_s + F_gC_g + F_{fg}C_{fg}\right)\right] \quad (18)$$

where BCF_e is the concentration factor of the egg yolk, $m_{y,e}$ is the average mass fraction of yolk in the egg (assumed equal to 0.3), C_g is the POP concentration in grass, C_{fg} is the POP concentration in fodder or grain (which can be estimated as the sum of $C_{p,r}$, $C_{p,dg}$, $C_{p,p}$), F_s, F_g, and F_f are the fractions of the chicken's diet that is soil ingestion, grass, and fodder or grain, respectively. Values of F_s, F_g, and F_{fg} and congener-specific values of BCF_e are reported by Harrad and Smith (1997). Due to the complexity of species and of the aquatic ecosystem, to the authors' knowledge, no model is available to estimate the intake by fish consumption, even though very important contributions to the daily intake of PCDD/Fs and dioxin-like PCBs were demonstrated to derive from this route in several case studies (Godliauskienė et al. 2012; Marin et al. 2011; Sasamoto et al. 2006).

Novel options for exposure assessment

During the last decades, advances have been made in the development of tools for the exposure assessment. It

being understood that the classical methodology described in the "Conventional methodologies" section still represents an undeniable fundamental point in the exposure assessment, new approaches, integrative tools, or simplifying methodologies can help detecting the presence of critical situations of exposure, identifying the dominant emission sources, proposing design criteria for critical activities, and defining limit values to assure an acceptable risk level.

In particular, novel monitoring approaches have been proposed during the last decade. Researchers have investigated the potential of conifer needles as passive-sampling alternative to determine the spatial and temporal variability of mean concentrations of PCDD/Fs and PCBs by means of internal analytical procedures (Rappolder et al. 2007; Bertolotti et al. 2014; Klánová et al. 2009). The results of a monitoring campaign carried out in Germany on conifer needles of different ages were comparable with the time series recorded by the German Dioxin Database (Rappolder et al. 2007); as an added value, a wider area could be covered by this monitoring method. In another study, conifer needles were compared to passive samplers with polyurethane foam, which revealed to be less efficient than conifer needles in collecting particle-bound POPs (Klánová et al. 2009).

An additional and alternative method is represented by the biomonitoring performed with lichens exposed to contaminated air. Indeed, lichens are able to accumulate radionuclides, metals, and POPs (Conti and Cecchetti 2001). This method showed interesting results in three recent studies (Augusto et al. 2007; Augusto et al. 2009; Denys et al. 2012). A practical application of this methodology, concerning the area of Setúbal (Portugal), provided a risk map that would have required the adoption of several deposition samplers to cover the region investigated (Augusto et al. 2007). Another kind of biomonitor, consisting in an endemic Bromeliad species, was placed in funnels to measure atmospheric deposition of POPs: The deposition measured with this methodology was comparable with the results of conventional deposition measurements (de Souza Pereira et al. 2007a; de Souza Pereira et al. 2007b), although applications of this method are limited to one case study.

A proposal for the development of a novel kind of deposimeter was made in a recent study by Rada et al. (2014), who proposed the integration of a remotely controlled coverage and a webcam for video surveillance to quantify the contribution of a dominant and intermittent punctual source (e.g., industrial plant, energy production plant, and incinerator) with respect to periods of inactivity of the plant. Vassura et al. (2011) and Rada et al. (2014) also presented the potentialities of crossing the congener profiles measured in PCDD/F and PCB depositions with the typical fingerprints or measured congener profiles in the emissions of a specific activity present in the surroundings of the monitored area, in order to facilitate the estimation of the dominant source. In a similar experience, congener profiles and diagnostic ratios of POPs measured in the sediments of a pond were recently studied and successfully applied to investigate the dominant sources in an Alpine valley (Argiriadis et al. 2014). Analyses on sediments at different depths showed to be useful to understand the evolution of PCDD/F emissions in an area over the years: The results obtained by Argiriadis et al. (2014) were in agreement with the evolution of the emissive framework of the area investigated, where POP emissions decreased starting from 2009, when an important steel plant in that area adopted the BATs.

Alternative analytical strategies, based on the concept of biomonitoring, have been developed and tested. The most important one is the Chemically Activated Luciferase Gene Expression (CALUX) bioassay, based on a genetically modified cell line inducing luciferase when responding to dioxin-like compounds (Han et al. 2004); a new CALUX bioassay was recently used in place of the conventional GC-HRMS analyses (Croes et al. 2012). The classical approach considers the EPA methods 1613 and 1668B (USEPA 1994b; USEPA 2008) as the conventional analytical methodologies to determine PCDD/Fs and PCBs, respectively. The CALUX bioassay makes use of an alternative methodology based on a C18 filter (USEPA 1994b), to minimize the use of organic solvent and lower the toxicity toward the cells (Croes et al. 2011).

The opportunities offered by analyses on PCDD/F and PCB concentrations in the sewage sludge of wastewater treatment plants were recently investigated: In particular, it was demonstrated that the comparison of sewage sludge samples from uncontaminated and potentially contaminated regions is able to quickly highlight the presence of critical levels of exposure, since the POPs present in the wastewater (and thus in the diet of a population) are finally concentrated in the sewage sludge (Rada et al. 2013). This methodology was applied to a study area where a steel plant is present: In this specific case, a sensitivity analysis of this method

showed that, if the population was exposed to a concentration of POPs in food that is 100 times higher than that in an unexposed population, about an 8 times higher concentration would be observed in the sewage sludge of the wastewater treatment plant serving the population exposed (Rada et al. 2013).

Useful insights into the choice of the location of an activity or of the adequate APC technologies to be adopted by a stationary emission source can be obtained by coupling dispersion modeling with risk indicators: In two recent studies (Rada et al. 2011; Ragazzi et al. 2014b), the low capability of open biofilters (traditionally adopted in MBTs) in diluting the effluent released into the atmosphere was highlighted, as well as the consequent potential impacts on the surrounding region in terms of PCDD/F deposition.

Coupling-edited food chain models with data on the food consumption resulted in a recent proposal for a PCDD/F deposition limit value, specifically based on the typical diet of a target population (Schiavon et al. 2013): Consolidated food-chain models (Lorber et al. 2000; Slob and Van Jaarsveld 1993; Harrad and Smith 1997; Trapp 2007; Lorber et al. 1994) were edited to be run backward by starting from the food consumption of a population and from the TDI proposed by the WHO, determining the acceptable PCDD/F concentration in the food consumed, till deriving a region-specific acceptable value for PCDD/F deposition (Schiavon et al. 2013). This approach differs from that adopted in

Table 3 Benefits and possible limitations of the novel methodologies for exposure assessment here presented in comparison with the conventional monitoring approach

Novel methodologies		Comparison with conventional methodologies	
Environmental matrix/tools	References	Benefits	Limitations
Conifer needles	Rappolder et al. (2007); Bertolotti et al. (2014); Klánová et al. (2009)	Low-cost methodology improving the spatial resolution of the sampling; capability of estimating the temporal evolution of pollutant levels in the past	Possible underestimation of concentrations due to rain and wind removal
Lichens	Conti and Cecchetti (2001)	Low-cost methodology improving the spatial resolution of the sampling	Calibration through conventional methodologies is required
Diagnostic ratios and congener profiles	Argiriadis et al. (2014); Rada et al. (2014)	Quick estimation of the dominant source in the sampling area, not possible with the conventional monitoring approach alone; capability of estimating the temporal evolution of pollutant levels in the past (sediments only)	The estimation of past contamination levels requires the dating of sediment layers
CALUX bioassay	Croes et al. (2011); Croes et al. (2012)	Quick, sensitive and low-cost analytical methodology that can be used as a convenient alternative to GC-HRMS	Calibration through conventional methodologies is required
Sewage sludge	Rada et al. (2013)	Low-cost methodology to detect anomalous levels of exposure via diet	Possible interferences from external factors (e.g., non-residential population); acute episodes of exposure cannot be observed
Dispersion models and risk indicators	Rada et al. (2011); Ragazzi et al. (2014b)	Easy and spatially resolved assessment of local criticalities in terms of air quality and deposition; possibility of simulating scenarios and predict future trends	Calibration through conventional methodologies, emission factors, morphology, and meteorological data are required
Atmospheric deposition and food-chain models	Schiavon et al. (2013)	Capability of determining a safe deposition value to preserve the agricultural and livestock food chains from contamination	Information on the origin of food and statistics on the local diet are required

Belgium by De Fré et al. (2000) and Van Lieshout et al. (2001), since the acceptable deposition value is calculated by starting directly from the TDI and not by starting from a deposition value and obtaining the corresponding TDI by iterations.

Table 3 summarizes the benefits and the potential limitations of the novel methodologies here presented compared with the conventional monitoring approaches. While conventional methodologies generally provide only current and punctual information on the contamination levels of an area, the integration of the traditional approach with these novel methodologies allows extending the significance of the data collected by conventional monitoring methodologies over time and space. This is the case, for instance, of the analysis on sediment layers, of the adoption of dispersion models and of strategies based on the use of a widespread network of low-cost and natural sampling systems, such as conifer needles and lichens. Methods such as the CALUX bioassay and analysis on sewage sludge samples are low-cost analytical and monitoring options for a preliminary assessment, before adopting conventional and more accurate monitoring methods, if necessary. Methodologies like the use of food chain models, the determination of diagnostic ratios, and congener profiles even allow retrieving information that conventional methodologies alone do not.

Conclusions

In spite of the continuous advances in the environmental assessment, there is still a conspicuous number of issues to be solved. In particular, the advances in terms of monitoring, exposure assessment, and development of design criteria for specific activities have not been adequately accompanied by improvements in the environmental regulations: The introduction of legislative limit values for atmospheric deposition and ambient air concentration of PCDD/Fs and dioxin-like PCBs should be pursued to assure acceptable levels of exposure; the adoption of more efficient APC technologies should be regulated for the critical emission sources that have been recently highlighted (e.g., MBT plants and wood burning); this target should be accompanied by extending the emission limit values already established by some countries, at least with regards to MBTs (e.g., Germany and Austria); a correspondence between the IARC classification of the PCDD/F and dioxin-like PCB congeners and the respective TEFs should be pursued, since there is the risk that the effect of some congeners might be underestimated; in addition, the last revision of the TEFs dates back to 2005, while the IARC classification has been recently updated; finally, there is great uncertainty on the estimation of the locally produced food that is consumed by a target population; this last aspect has repercussions on the estimation of the daily intake and, therefore, on the overall exposure assessment; a synergistic approach between capillary networks of deposimeters and food traceability should be implemented to overcome this issue.

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