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There are various exposure pathways of toxic substances to general population: direct pathways are soil ingestion, dust inhalation, dermal contact; indirect ingestion through the food chain is one of the most important pathways for the entry of PHEs into the human body.

In order to avoid possible consequences to humans and the environment, it is necessary to investigate the source, origin, pathways, distribution in all the environmental compartments, and to ascertain if metal bioaccumulation is likely to occur, affecting human health.

Risk Assessment procedures include two components, the Environmental Risk Assessment and the Human Health Risk Assessment. The former has been used mainly for comparative and priority setting purposes with reference to contaminated sites. The latter refers to the possible consequences of human exposure to contaminant sources. The ecological risk is generally considered a second priority in comparison to human health risk.

Estimate of exposure levels is a central step in Ecological Risk Assessment to evaluate ecotoxicity risks posed by PHEs. For example, agricultural soils contaminated with metals result in elevated uptake and transfer of metals to vegetables; consequently, severe health hazard can be caused by the consumption of metal-contaminated vegetables. Bioaccumulation of heavy metals in edible parts of vegetables is thus responsible for major health concern.

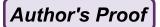
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Biological monitoring is a promising method of assessing environmental and human health risk by analysing PHEs concentration in environmental matrixes (e.g. plants, animals), or in human tissues (hairs, nails), or in a biological matrix (blood, urine). Concerning human health, biological monitoring is usually described as the measurement of a particular chemical substance, or a metabolite of that substance, in a suitable biological matrix (e.g. blood, urine, serum, and tissues such as hairs, nails, sweats), that act as an effective biomarker, allowing identification of potential hazards.

Examples of how the risk assessment process may be carried out are given with reference to exposure levels and exposure-response relationships for the contaminants of concern.

Keywords	
(separated by "-"))

Rassessment - PHEs - Bioavailability - Bioaccessibility - Bioaccumulation factor



Chapter 10 **Risk Assessment of PHEs**

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Claudio Bini 3

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45 1 Introduction

Environmental contamination is a concern whose importance has been recently perceived by public opinion, and constitutes one of the great emergencies of twenty-first century (Bini 2009). Modern society is paying increasing attention to its effects on the human health, and is acquiring more and more consciousness of the health risk connected to exposure to chemicals and toxic products (e.g. heavy metals, radionuclides, asbestos, hydrocarbons, dioxins). Yet, a serious concern exists about the presence of chemicals in the environment and their bioaccumulation in foodstuff (Martorel et al. 2010).

Many organic substances (pesticides, fertilisers, PCB, PAH, etc.) contribute to contaminate ecosystems and are very poisonous to living organisms and to human health. Correspondingly, inorganic compounds (e.g. heavy metals, volatiles, anions) from different non-point and point sources have a potential to contaminate soil and water. Moreover, potentially harmful elements (PHEs), when present at high concentration in the environment, are critical or toxic to living organisms (Salomons 1995). Environmental contamination rises from double driving forces: natural or anthropogenic. Indeed, supergenic alteration processes may lead to the release of potentially toxic elements, particularly heavy metals, in the environment. Possible "natural" accumulation of elements into the ecosystems may be related to heavy metal-bearing rocks (e.g. Ni and Cr in serpentine: Angelone et al. 1993) or to mineralized areas (e.g. Pb and Zn from mixed sulfide mines), while anthropogenic accumulation is almost related to industrial activities (e.g. Cd in metallurgy, Cr in varnish and leather factories: Bini et al. 2008), agriculture and urban sewage sludge (e.g. Zn and Cu from fertilizers: De Luisa et al. 1996; Cd, Pb, Cr from sludge:

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Petruzzelli 1989). Metals can be dispersed in the environment and/or accumulated 69 in plants and animals, and taken in by human beings through the food chain (Lim 70 et al. 2008). The local physico-chemical, climatic, biologic, geologic conditions 71 control the ultimate fate of a toxic element, that is, if it will precipitate as an 72 insoluble phase or will be adsorbed on the surface of some other phase, or will be released, transported and eventually taken up by plants (Brummer 1986; Langmuir 74 1997).

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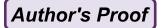
Actually, the identification of the sources responsible for soil contamination is 76 an important issue, since high loads of heavy metals applied to soils with sludge, or 77 discharged and stored in soils, may cause soil quality degradation, surface and 78 groundwater pollution, accumulation in plants, phytotoxicity and possible adverse effects on human health. Just one - or more - heavy metal(s) could determine 80 irreversible damage to the environment and/or to resident population (Chon 81 et al. 2011).

Indeed, all trace elements, including those essential to living organisms (e.g. Cu, 83 Mo, Ni, Se, Zn) are toxic if taken up in concentrations markedly larger than the 84 nutrient requirements. In particular Ag, As, Be, Cd, Ce, Ge, Hg, Pb, Tl are suitable examples of potentially harmful elements (PHEs) that have no proven essential 86 functions, and are known to have adverse physiological effects at relatively low 87 concentrations (Abrahams 2002).

Examples of toxicity by heavy metals are known since the Antiquity (Nriagu 89 1983). For instance, one of the supposed causes for the Roman Empire collapse is 90 the increasing lead toxicity from Pb-bearing potteries and wine containers, as it was 91 found in Roman findings and bones. Lead (plumbism) and Hg (mercurialism) 92 poisoning cases were frequently recorded in workers employed in mining industry 93 and even in hat factories in Tuscany (Dall'Aglio et al. 1966). At present, diseases 94 and toxicity related to microelement contamination (Cd, Cr, Cu, Ni, Pb, Tl, Zn,) of 95 air, water and soil from industrial activities are well established (Thornton 1993; 96 Abrahams 2002). For example, the most notable cause of Tl poisoning occurred 97 adjacent to a cement plant in Germany (Abrahams 2002).

Environmental and human health risk assessment, therefore, is assuming more 99 and more importance in the solution of problems connected with land restoration. 100 Indeed, the risk assessment criteria are applied to identify and classify the various 101 sites on the basis of intervention priority, to establish objectives and standard of 102 decontamination, to select the more appropriate and site-specific technology (Pizzol 103 et al. 2009).

The risk arising from metals depends on their bioavailability, which in turn 105 depends on the form in which they occur (Adriano et al. 1995). This is the reason 106 why the risk to human health cannot be assessed on the basis of the total concentration of the toxic metal. Background values correspond to the total content of 108 metals in soils not affected by human activities, i.e. they are the reference values for 109 most countries. Soil guide values have been introduced in the late 1950s in Japan, in 110 1980 in The Netherlands, in 1986 in Switzerland, in 1987 in Great Britain, in 1994 111 in Germany. Since that time, many countries, notably the U.S.A., Canada, Great 112 Britain and the Netherlands, have progressed further in setting standards for 113



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hazardous constituents in soil, health-risk based soil screening levels and soil remediation. However, legislation on maximum admissible levels of heavy metals in the environment in the EU is rather confusing. Indeed, a general regulatory guideline on the maximum trace element concentration in soils has not yet been established, the current references being related to the total metal content in waste and sewage sludge to be spread on soil (Adriano et al. 1995).

120 **Bioavailability and Bioaccessibility**

Although total metal concentrations do not give an actual indication of the potentially plant-available or 'bio-accessible' fractions of a metal in a soil, they provide a useful indication of whether a soil has anomalously high or low concentrations. This will show whether this soil is contaminated and/or geochemically enriched and 124 thus poses a possible risk of toxicity to some species of plants, soil fauna or 125 microorganisms (Alloway 2013). For example, serpentine soils present Ni concentration up to 800 mgkg⁻¹, but only a small part of it is available/accessible to plants 127 growing on this kind of substrate, with the exception of hyperaccumulator plants 128 (e.g. Alyssum bertoloni, a well known endemic species growing on serpentine soils) 129 that are able to absorb high quantities of metals (Brooks 1998). 130

Knowledge of the total metal concentration in soils, in most cases, is not sufficient to assess element mobility and bioavailability (Abollino et al. 2009; Rao et al. 2008; Menzies 2007). Availability is driven by several factors and processes that enhance metal mobilization, namely: pH, redox status, complexing ligants, soil solution activity, plant roots exudates, available water, etc. (Alloway 1995).

137 As a matter of facts, the potential toxicity of contaminants is strongly determined by the speciation of the elements involved. Total element concentration includes 138 all forms of that element in soil: readily soluble in the soil solution, bound to 139 organic matter, adsorbed on surfaces of clay minerals, oxides, carbonates, bound in 140 the crystal lattice. In many cases, much of the total content of an element is not available for immediate uptake by plants. Conversely, the available metal fraction refers to the portion of the total content which is potentially available to living organisms (Alloway 2013). Sequential extractions, therefore, are currently performed to determine element fractionation. Several reports on fractionation 145 methods have been published in recent years (see f.i. Abollino et al. 2009; Rao et al. 2008; Menzies et al. 2007; Peijnenburg et al. 2007). The suggested procedures allow evaluating element mobility and bioavailability (Obrador et al. 2007; Kabata-148 Pendias and Mukherjee 2007; Quevauviller 1998). Water-soluble and EDTAexchangeable elements are considered bioavailable to plants, while those linked to Fe-Mn oxides, to carbonate and to organic matter are considered potentially bioavailable (He et al. 2005). In recent years, single extractants are widely utilized (Rao et al. 2008) to evaluate the plant uptake and possible transfer to the aerial parts and to the food chain. Single extractants most utilized are chelating agents as EDTA

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or DTPA, and neutral salts (e.g. CaCl₂), which proved the best agents to estimate 155 available metal pool, plant uptake and transfer to humans through the food chain 156 (Menzies et al. 2007).

There are various exposure pathways of toxic substances to humans: direct 158 pathways are soil ingestion, dust inhalation, dermal contact; but indirect ingestion 159 through the food chain is one of the most important pathways for the entry of these toxic pollutants into the human body (Khan and Cao 2012). Food chain contami- 161 nation is of increasing concern because of the adverse impact on the quality of food and health. Bioaccumulation in the edible parts of vegetables depends on numerous factors including soil characteristics (e.g. pH, SOC), metal concentration in soil, 164 physiological characteristics of vegetables and transfer routes. SOC acts as a huge sink of heavy metals and organic pollutants, and modulate metal bioavailability.

Vegetables grown on wastewater contaminated soils accumulate metals at a 167 concentration enough to cause human health risk. Several mechanisms including 168 sorption (from soil particles), uptake rate through transpiration, volatilization, and 169 re-deposition on plant leaf surfaces are responsible for the metal transfer from soil 170 ecosystem to plant tissues and to the food chain. Yet, food is considered the major 171 source of PHEs and POPs to humans, accounting for 70 % of the total exposure. 172 Therefore, it is necessary to investigate the source, origin, pathways, distribution in 173 agricultural soils, and bioaccumulation of metals to assess the possible human 174 health risk caused by consumption of metal-contaminated vegetables (see 175 Chap. 3, this volume). Agricultural soils contaminated with metals result in elevated uptake and transfer of metals to vegetables; consequently, severe human 177 health risk can be caused by the consumption of metal-contaminated vegetables. 178 Bioaccumulation of heavy metals in edible parts of vegetables is responsible for 179 major health concern. The benchmark contamination levels for HM vary from 180 country to country, but so far many countries have not established the tolerable 181 limits for HM in both soil and vegetables (Kabata-Pendias and Mukherjee 2007).

3 **Bioaccessibility and Human Health**

Many chemicals are recalcitrant, mutagenic and carcinogenic pollutants, present in 184 the environment as a result of different anthropogenic activities, and are implicated in different types of diseases, including breast, lung and colon cancer in humans 186 (Khan and Cao 2012). 187

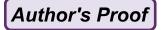
In human health risk assessment, oral exposure is typically stated in terms of the 188 external dose or intake, instead of in terms of absorbed dose or uptake (Lim 189 et al. 2008). Intake is typically defined as the process by which an agent crosses the outer exposure surface of a human without passing an absorption barrier, while uptake is the process by which an agent crosses an absorption barrier into living organisms (plants, animals or humans). For example, it has been demonstrated by several studies (Zupan et al. 1995; Basta et al. 2005; Bini et al. 2008, 2013; Maleci 194 et al. 2013) that Cr is unable to cross the root barrier opposed by several vascular 195

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C. Bini



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196 plants, including food crops (e.g. plantain, marigold, dandelion, wheat and maize) 197 to metal fluxes.

Bioavailability of a compound to humans can be defined as the fraction of an 198 administered dose that reaches the central blood compartment (i.e. the vascular 199 system), whether through the gastrointestinal track, skin or lung, Bioaccessibility is 200 the fraction of a chemical in an environmental medium that is available for 201 absorption based on "in vitro" extraction, but not necessarily absorbed (Lim 202 et al. 2008). The term "bioaccessible" is used to indicate the "in vitro" fraction of 203 the chemical intake that is directly available for absorption. Therefore, bioaccessible metal concentrations are more important for risk assessment than 205 total metal contents in the environment.

207 4 Bioaccumulation Factors

Element distribution in soil is determined by various processes occurring in soil (Kabata-Pendias 2011): weathering, decomposition, precipitation-dissolution, absorption-desorption, oxidation-reduction, chelation (He et al. 2005; Adriano 2001). Such processes in soil are regulated by soil parameters: pH, redox potential, cation exchange capacity, clay content, organic matter. The aqueous phase (soil solution) is the medium where most chemical equilibria, including metal absorption by roots, occur. The root sorption process, that is the prominent mechanism of element flux from soil to plants, is influenced by soil properties, element speciation, plant physiology, environmental conditions (Adriano 2001).

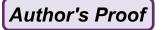
The metal transfer from soil to plants, and from plants to animals (including humans), is achieved through the food chain. The bioaccumulation factor is one of the key pathways of human exposure to PHEs through consumption of vegetable and/or the food chain, and is commonly assessed with specific coefficients. The value of the bioaccumulation factor depends on the nature of PHEs, physiology of plant, and content of elements in growing media. Metal concentrations in soils, roots, shoots and fruits of edible vegetables are currently utilized to calculate the PHEs bioaccumulation factors, since soil-to plant transfer is one of the major pathways for pollutants to enter the food chain (Bini et al. 2013).

In order to investigate the health risk associated with PHEs, it is essential to assess the bioaccumulation factors.

Several bioaccumulation factors have been proposed so far. The Biological Adsorption Coefficient (BAC) (or Transfer Coefficient – TC – according to Hao et al. 2009), is defined as the ratio between metal concentration in plant roots and metal concentration in soil (Chojnacka et al. 2005):

$$BAC = Cplant/Csoil$$

The Translocation Factor, i.e. the metal flux from plant roots to the aerial parts, is



calculated by the ratio of metal concentration in shoots and roots (Singh et al. 2010; 233 Mendez and Maier 2008; Yoon et al. 2006): 234

$$TF = C \text{ shoot/Croot}$$

Evaluation of human exposure as a consequence of pollutants translocation from 235 contaminated soils to edible vegetables has been proposed quite recently by Khan 236 and Cao (2012), who suggest to consider separately all the vegetable components. 237 Root concentration factor (RCF), shoot concentration factor (SCF) and fruit concentration factor (FCF) are calculated as follows: 239

where Croot, Cshoot, Cfruit and Csoil represent the contaminant concentration in 240 root, shoot, fruit and soil on dry basis, respectively. 241

The PHEs daily intake due to consumption of contaminated vegetables results 242 from the following equation: 243

$$DI_{PHEs} = C_{PHEs} \times C_{factor} \times V_{intake}/BW$$
,

where DI is the total PHEs daily intake, C PHEs is the total PHEs concentration in 244 vegetables mgkg⁻¹), C_{factor} is a conversion factor from fresh weight of vegetables 245 to dry weight (0.085 following Rattan et al. 2005), V_{intake} is the daily intake of 246 vegetables (0.350 kg person⁻¹day⁻¹), and BW is the average body weight (64 kg). 247

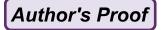
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Risk Assessment

While risk may be defined as the combination of the probability, or frequency, an 249 hazardous event to occur, and the magnitude of consequences of its occurrence, 250 hazard is commonly defined as "a property or situation that in particular circum- 251 stances could lead to harm" (Critto and Suter 2009). Consistently, risk assessment is 252 the systematic procedure with which the risks associated with hazardous sub- 253 stances, processes, activities or events are identified, described, analyzed and 254 estimated either qualitatively and quantitatively (Korre et al. 2002).

Risk assessment procedures include two components, the Environmental Risk 256 Assessment and the Human Health Risk Assessment. The former has been used 257 mainly for comparative and priority setting purposes with reference to contami- 258 nated sites. The latter refers to the possible consequences of human exposure to 259 contaminant sources on human health. In the contest of comparative risk analysis, 260 risk is used as an indicator, not as an absolute quantitative measurement of the 261



262 adverse effects of contamination on the environment and/or on human health 263 (Korre et al. 2002).

264 5.1 Quantitative Risk Assessment

Many countries utilize a multi-level risk-based methodology in regulating and 265 managing contaminated sites (ASTM 2000; Critto and Suter 2009; UK-EA 2009). 266 The risk-based methodology provides quantitative methods for the estimation of 267 human and ecological risks using analytical models of contaminant fate and 268 transport, and assessment of human and ecological exposure (Geng et al. 2013). 269 Common to this approach is the adaptation of a multi-tiered framework, along with 270 the requirement for developing risk-based screening levels in an earlier tier, and site-specific target levels in a later tier, thus allowing risk assessment to be undertaken in a progressive and cost-effective manner. 273

Conversely, development of a robust conceptual model of a contaminated site is an integral part of a successful risk assessment. It provides a qualitative evaluation 275 of potentially contaminant sources, pathways and receptors at the site, based on 276 plausible contaminant - pathway - receptor linkages under current and future land 277 use of the site. For example, Geng et al. (2013) investigated a Mo-contaminated industrial site in China, whose future land use would be residential. After examination of the historic records of the site and field-scale site investigation, they 280 developed a conceptual model on the site risk assessment. The principal linkages in 281 the assessment procedure (source – pathways – receptors) are identified as follows: 282 groundwater – drinking water and dermal contact – residents and workers. These 283 pollution linkages have been subjected to a quantitative risk assessment and have 284 been used as the basis for calculating the site specific target level. The results of 285 model application proved Mo contamination in the local streams due to inflow of 286 polluted groundwater will not pose unacceptable risks to aquatic biota (Geng 287 et al. 2013). Conversely, Mo was highly concentrated in the groundwater, and the 288 risk associated with this should not be neglected. Based on the toxicity data from 289 290 the database of the International Agency for Research on Cancer (WHO 2013) and the Integrated Risk Information System (USEPA 2013), Mo has no carcinogenic 291 effect on living organisms. Therefore, only non-carcinogenic risk was assessed: Mo will pose unacceptable non-cancer risks for on-site children when they directly 293 drink the groundwater.

295 5.2 Environmental Risk Assessment

The environmental risk assessment is the procedure of evaluating risks resulting from hazards in the environment that threaten soil, ecosystems, plants, animals and ultimately human population.

10 Risk Assessment of PHEs

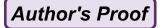
Within the environmental risk assessment procedure, in the 1990s the US 299 Environmental Protection Agency (US-EPA 1992, 1998) set up a framework and 300 the related guidelines for the Ecological Risk Assessment as the process of orga-301 nizing and analyzing data, assumptions and uncertainties to evaluate the adverse 302 ecological effects on ecosystems (Critto and Suter 2009). Ecological Risk Assess- 303 ment (ERA) is the appropriate process for identifying environmental quality objec- 304 tives and the ecological aspects of major concern (Semenzin et al. 2009). The 305 principles and procedures that have been established at international level (US-EPA 306 1998, UK-EA 2003 and several applications) point to a ERA framework based on a 307 hierarchical approach, including: (1) a screening phase allowing the definition of 308 land use-based soil screening values, and (2) a site-specific phase in order to 309 achieve a more comprehensive risk characterization. Within risk characterization, 310 US-EPA (1998) suggests to apply Weight of Evidence methods to determine 311 possible ecological impacts determined by chemical contamination, following 312 three lines of evidence: chemicals characterization, laboratory-based toxicity 313 tests, and characterization of living organisms communities, as indicated by Long 314 and Chapman (1985). The first level includes preliminary investigations that can be 315 stopped when the estimated risk is considered acceptable by experts. In the case it is 316 unacceptable, the second level is run to reduce uncertainty in the risk estimate. Sitespecific aspects of particular interest (e.g. specific plant communities or typical 318 endemics) can be analyzed in the third level, thus reducing costs and duration of 319 analysis. A specific software, called ERAMANIA, has been developed with the aim 320 at comparing the different tests included in the three levels of analysis (see 321 Semenzin et al. 2009 for details). To overcome the uncertainties included in the 322 assessment procedure, increased attention has been paid to developing a probabi- 323 listic risk assessment methodology, as opposed to the deterministic risk assessment 324 approach (US-EPA 1999).

The ecological risk is generally considered a second priority in comparison to 326 human health risk; however, it is very difficult to propose remediation techniques 327 for restoration of contaminated sites if the potential risks to biological communities are not considered (Moreno-Jimenez et al. 2011). For example, the risk posed by mining sites, which comes mainly from tailings with high concentration of potentially harmful elements (PHEs) (e.g. As, Cd, Cu, Pb, Zn), represents a serious 331 environmental concern. Since abandoned mine sites are widely diffused, particularly in developed countries (Bini 2012; see also Chap. 5, this volume), there is the need to develop new (or to implement existing) methods of risk assessment to be 334 applied to these sensible areas, in order to better quantify the potential environmental risk, also in the perspective of secondary poisoning to animals and even 336 humans.

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A new screening methodology for quantitative impact assessment based on 338 Environmental Risk Assessment (ERA) has been proposed recently (Moreno- 339 Jimenez et al. 2011); it applies general principles for chemical risk assessment, as 340 described in EU Technical Guidance Document (EC 2003), and develops in two 341 steps. The first establishes eight risk levels according to the values of hazard 342 quotients (HQs). HQs are calculated as the ratio between environmental 343



t.1	Table 10.1	Ranking of risk indexes based on hazard quotients and level of disturbance (Adapted
	from Morer	o-Jimenez et al. 2011)

t.2	Hazard quotients	Risk index	Level of disturbance
t.3	<1 (predicted non-effect)	0	Negligible effects
t.4	1-10 (predicted non-effect)	1	Sensitive species
t.5	1-10 (non-observed chronic effects)	2	Standard species
t.6	10-100 (non-observed chronic effects)	3	Community
t.7	>100 (non-observed chronic effects)	4	Ecological structure
t.8	1-10 (50 % lethal acute effects)	5	Standard species
t.9	10-100 (50 % lethal acute effects)	6	Community
t.10	>100 (50 % lethal acute effects)	7	Ecological structure

concentrations (i.e. exposure or daily intake) and toxicity values (i.e. reference dose or acute toxic dose). Consistently, a scoring system of 8 risk indexes (RIs) is defined according to the level of disturbance. RIs from 0 to 4 are based on chronic exposure and RIs from 5 to 7 are based on acute exposure (Table 10.1) (see Moreno-Jimenez et al. 2011, for details).

The second step evaluates the overall potential impacts of a contaminated site based on the Impact Index (ImI). This is obtained as the sum of the chronic and acute RIs. Finally, the ImI obtained is assigned to five different categories, from negligible to very high impact according to the following criteria:

$$\begin{split} RI(HQchronic) + RI(HQacute) = & \quad ImI \leq 1 \quad (negligible \ impact) \\ ImI \leq 2 \quad (low \ impact) \\ ImI \leq 7 \quad (moderate \ impact) \\ ImI \leq 9 \quad (high \ impact) \\ ImI > 9 \quad (very \ high \ impact). \end{split}$$

In order to select the ecological receptors and exposure routes, three protection goals are considered: soil organisms (plants, earthworms and microorganisms), aquatic organisms (algae, invertebrates and fish) and terrestrial vertebrates (birds and mammals).

The exposure concentration of soil organisms (Csoil) is represented by the metal concentrations measured in soil. The same Csoil values are also taken into account when assessing the exposure of terrestrial vertebrates.

For the aquatic compartment, the exposure for aquatic organisms and terrestrial vertebrates (through drinking water) is represented by the metal concentration in the water samples (Cwater).

Three main exposure routes are considered for terrestrial vertebrates: oral food ingestion, soil accidental ingestion and drinking water. Unlike to soil and aquatic organisms, exposure levels for terrestrial vertebrates are estimated using exposure models, admitted by different regulations (EC 2002a, b, 2003). The daily dose of metals through oral food ingestion (DDfood) is calculated by the following equation:



 $DDfood = FIR/W \times Cfood \times (100 - MC)/100 \text{ mg/kg b.w./day}$

where FIR is the food intake rate of indicator species (kg food fresh material per 369 day), W is the body weight (b.w.) of indicator species (kg), Cfood is the concentration of metal in food related to fresh material (mg/kg food), and MC is the 371 moisture content of food source (%).

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The total DDfood for terrestrial vertebrates is the sum of values obtained from 373 each exposure pathway (food, soil and drinking water). The risk quantification 374 indicates potential risk to sensitive species (HQ >1) in all the investigated compartments. Soil organisms are expected to suffer acute effects both to species and 376 community, while chronic effects to standard species are likely to occur, and this 377 may lead to possible overestimation of risk. A more realistic risk estimate is 378 quantification of the bioavailable metal fraction in soils, although it is not generally 379 admitted (Berthelot et al. 2008). In any case, the whole ecosystem seems to be 380 highly impacted by (heavy) metals, and the site recovery seriously compromised.

Taking into consideration time and costs of risk assessment procedures, an 382 alternative way to reduce uncertainty, time and costs is applying regression models, 383 based on soil properties, for estimating metal concentration in vegetables (Karo Bester et al. 2013). Yet, regression models identify statistically significant soil 385 properties which have an influence on the accumulation of metals by plants, Identifying effective soil properties enables to drive metal transfer to the food 387 chain, reducing the risk to human health. As application of the method, Karo Bester 388 et al. (2013) developed a regression model to predict Cd concentration in selected vegetables grown on garden soils as a function of significant soil parameters (pH, 390 SOC, clay content). Based on regression analysis, the most predictive soil proper-391 ties for metal uptake by most of the vegetables examined were soil Cd concentration, pH and SOC, as expected. The Cd level exceeded the EU legislative maximum 393 level (EC 1881, 2006) in carrots, followed by red beet, onion, chicory and endive.

The main advantage of regression models is that they are time and cost effective, reducing the size of data sets needed to identify the statistically significant soil 396 properties. However, the results of regression models are reliable when the sampled 397 soils and vegetables are a statistically representative sample. Moreover, there may be also interactions between soils and other environmental properties, as geology and meteorological conditions. Therefore, the model results must be interpreted 400 correctly.

5.3 Exposure Assessment

The exposure assessment identifies the pathways by which living organisms are 403 potentially exposed to toxicants and estimates the magnitude, frequency and dura-404 tion of these actual and/or potential exposures (Lee et al. 2008). 405

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Estimate of exposure levels is a central step in Ecological Risk Assessment to evaluate ecotoxicity risks posed by chemicals (Bertazzon et al. 2006). Although in general regulatory guidelines emphasize that exposure conditions are a function of spatial factors, exposure estimate methods ignore frequently site-specific conditions that can be accounted for in risk assessment. Therefore, estimates derived from non-spatial models are unreliable and potentially misleading.

A method for the characterization of contaminant exposure, based on data spatial 412 dependence which produces a spatial interpolation of the sampling points in a GIS 413 framework, has been proposed by Bertazzon et al. (2006). According to the scale of the problem to be assessed, the method provides two different approaches, the site-415 specific spatial risk assessment, and the regional one (Critto and Suter 2009). The former is performed at local scale using site-specific data to define the spatial 417 distribution of risk, and provides reliable risk maps; its use has increasing interest, 418 combining quantitative information with spatial data. The latter is of more general interest, dealing with problems that affect large geographical areas with multiple 420 habitats; it is used especially by policy makers facing problems caused by multiple 421 sources of hazards (e.g. subsidence, sediment contamination, benthic communities population, fishing, etc.). For example, Micheletti (2006) produced maps showing 423 the spatial distribution of single and cumulative ecological risk for As, Cd, Ni, Zn 424 and PCBs in the lagoon of Venice, while Ungaro et al. (2008) expanded a previous 425 soil survey mapping the As probability of exceeding regularity thresholds in the 426 427 Venice lagoon watershed.

Interest in site-specific spatial risk assessment is more and more increasing. This novel approach combines quantitative risk assessment procedures and spatial distribution of stressors (e.g. contaminated sites or groundwater) and receptors (e.g. plant community, human population) to facilitate understanding and communication (Gay and Korre 2006).

433 Bertazzon et al. (2006) applied the spatial analysis to a complex case study, the lagoon of Venice (Italy), characterized by noteworthy spatial variability both in 434 morphology and in contaminated sediment distribution and properties. The pro-435 posed model allowed estimating chemicals exposure levels of receptor organism 436 (clam: Tapes philipinarum) at any location in the lagoon, as well as hot spots, thus 437 438 preventing any concern with respect to human health by consuming contaminated clams. Different site-specific risk assessment applications are available, concerning 439 440 in particular contaminated sites. Carlon et al. (2008) developed a technical software (DESYRE), aimed at performing a spatial risk assessment, accessed directly in the 441 popular GIS platform ArcGIS 9.2. Pizzol et al. (2009) illustrated in detail the 442 443 structure of DESYRE. It is a Decision Support System (DSS) structured into six modules (Socio-economy, Characterization, Risk Assessment, Technological 444 Assessment, Residual Risk Assessment, Decision) which represent the main phases of contaminated sites management. The DSS objective is the creation of different 446 scenarios and their comparison in terms of residual risk following site remediation. 447 448 It integrates large volumes of georeferenced heterogeneous information (e.g. land uses, industrial activities, population density, traffic, geology, hydrology, contam-449 inant concentration), performing a spatially resolved environmental risk assessment

(see Pizzol et al. 2009, for details). The risk assessment module, in particular, 451 provides tools for human health risk analysis of soil contaminants, and allows a 452 risk-based zoning of the site. Considered exposure pathways in the module are the 453 ingestion and dermal contact with soil and groundwater, and inhalation of vapours 454 and particulate emissions.

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Currently, risk assessment approaches are becoming widely used in Europe to 456 support the EU recent policies and EC Directives on environmental concerns 457 (e.g. Commission's White Paper 2001, European Thematic Strategy for Soil Pro- 458 tection 2006, REACH regulatory 2013), also with specific networks for contaminated sites, such as CARACAS, CLARINET and NICOLE (Critto and Suter 2009). 460 Although there exist several differences among the Member States in terms of 461 approach, there is a general consensus for developing a common data base and a set 462 of models devoted to risk assessment of natural hazards, including contaminated 463 sites. With this perspective, the EU Council has developed a set of guidelines for 464 national risk assessment and mapping (EC 2010), in order to implement the 465 methodology and to provide risk management instruments for policy-makers. 466 Three basic steps are defined:

- 1. Risk identification, which is the process of recognizing and describing risks;
- 2. Risk analysis, which is the process of understanding the nature of risk and 469 quantifying the risk level in function of probability, exposure and vulnerability; 470
- 3. Risk evaluation, which is the process of comparing risk analysis with risk criteria 471 to determine the acceptance level. 472

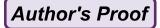
To support the risk assessment process, risk maps should be developed to inform 473 about the hazards and the vulnerability of land and residents, and to show the spatial 474 distribution of major hazards, in order to develop a risk attenuation strategy. Risk 475 assessment and management techniques, therefore, are a useful tool for: (i) the 476 development of environmental regulations, (ii) providing a basis for site-specific 477 decisions, (iii) ranking environmental risks, and (iv) comparing risks (Critto and 478 Suter 2009). 479

5.4 Human Health Risk Assessment

The second aspect of risk assessment procedure refers to the probability of occur-481 rence of an event, and the probable magnitude of adverse health effects on human 482 exposure to environmental hazards (NRC 1983; Paustenbach 2002).

Human health risk assessment has been used to determine if exposure to a 484 chemical, at any dose, could cause an increase in the incidence or adverse effects 485 on human health (Lim et al. 2008). 486

According to the procedure for human health risk assessment proposed by the 487 US National Academy of Sciences (1993), as reported by Chon et al. (2011), four interactive and iterative steps compose the basic framework for risk assessment: 489



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- 490 1. hazard identification;
- 491 2. exposure assessment;
- 492 3. toxicity (dose-response) assessment;
- 493 4. risk characterization.
- 1. *Hazard identification*. The purpose of hazard identification is to identify chemical substances which can affect a harmful effect in human body. A hazard is a source of risk but not a risk itself. The concern of chemicals (COCs) is selected by a risk assessor in this stage (Lim et al. 2008). The hazard identification process is accomplished through the sampling of different environmental media (soils, waters and plants), and the subsequent determination of the contaminant level of PHEs in these samples.
- 2. Exposure assessment.- Exposure assessment, as in the ecological risk 501 described above, is an important analytical tool for evaluating the extent of actual 502 or potential exposure of receptors to the source of a chemical hazard, and is an 503 important component of any health risk assessment and epidemiological study 504 (Nieuwenhuijsen et al. 2006). The aims of exposure assessment are identification 505 of potential receptor(s), evaluation of exposure routes and pathways, and quantifi-506 cation of exposure. The exposure assessment identifies the pathways by which 507 508 humans are potentially exposed to toxic substances and estimates the magnitude, frequency and duration of these actual and/or potential exposures (Chon 509 et al. 2011). As direct associations need to be established between actual human 510 exposure and health effects, exposure assessment is a crucial element of epidemiological research (Nieuwenhuijsen et al. 2006). Some environmental epidemiological studies use simple proxies such as distance from a point source (e.g. a factory), while others are categorised as industrial sources, agriculture land use, mining or urban zones. Many of the former studies have reported positive associations with health outcomes; however, it is difficult to attribute the incidence or prevalence of a 516 disease to a particular industry or chemical.

Conducting an exposure assessment involves analyzing contaminant releases, identifying exposed populations, identifying all potential pathways of exposure, estimating exposure point concentrations for specific pathways, and estimating contaminants intakes for specific pathways (Lee et al. 2008; Chon et al. 2011). As already stated, the most common pathways for toxicant intake are via direct (oral) soil ingestion, food and drinking water assumption, dust inhalation, skin absorption. The (receptor) exposure estimate is achieved through calculation of the average daily dose intake of a given contaminant, or of the sum of several contaminants, whose effect could be biomagnified.

The average daily dose (ADD) of the contaminant via the identified pathways (i.e. soil ingestion, dust inhalation, food ingestion and drinking water pathways) indicates the quantity of chemicals ingested per kilogram of body weight per day (Kolluru et al. 1996; Paustenbach 2002):

$ADD = C \times IR \times ED \times EF/BW \times AT \times 365$,

where: 531 C = concentration of the contaminant in the environmental media (mgkg⁻¹),532 IR = ingestion rate (mg/day)533 ED = exposure duration (years)534 EF = exposure frequency (days/year)535 BW = body weight of the receptor (kg) 536 AT = averaging time (life expectancy)537

365 = conversion factor from year to days.538 Based on US-EPA database IRIS (US-EPA 1997), Chon et al. (2011) applied 539 this model to As-contaminated agricultural soils in Korea, and found that exposure 540

factors to chemicals of an adult farmer (IRsoil = 50×10^{-6} kg/day; ED = 30 541 years; $EF = 350 \, days$; $AT = 76 \, years$, $BW = 60 \, kg$), accounted for an As average daily dose of 7.8×10^{-5} mg/kg-day by soil ingestion, 2.56×10^{-4} mg/kg-day for 543 drinking water consumption, and 2.3×10^{-3} mg/kg-day for rice consumption 544 (i.e. two orders of magnitude with rice in comparison to direct soil ingestion), by far highest than rice consumed at non-contaminated sites, as reported by Lee 546 et al. (2008) for similar conditions.

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3. Toxicity assessment – The purpose of toxicity assessment (i.e. dose–response 548 assessment) is to estimate the potential for selected chemical substances to cause 549 harmful effects in exposed people and to provide an estimate of the relationship 550 between the extent of exposure and the increased likehood of harmful effects (Lim 551 et al. 2008). The two principal toxicity indexes, as proposed by US-EPA (1992) are 552 known as slope factor (SF) and reference dose (RfD). The SF is a conservative 553 estimate of the increasing probability of an individual developing cancer as a result 554 of exposure over a lifetime, and RfD is the estimated amount of the daily exposure 555 level for the population that is likely to be without an appreciable risk of deleterious 556 effects during a lifetime. The toxicity indexes of selected elements are presented in 557 Table 10.2

Concerning PHEs, accurate prediction and quantification of the toxicological 559 risk for population resident in a contaminated region should be performed. Hazard 560 indexes for non-cancer risk (HInc) induced by selected elements from mine sites 561 have been calculated by Chon et al. (2011). Arsenic and Cd presented HInc > 1 in 562 most samples, thereby indicating possible individual threats for human health, 563 while Zn HInc was <1, suggesting threats to be unlikely. Conversely, cancer risk 564 for As via soil ingestion pathway was acceptable in all the examined sites (range 565 2.0×10^{-5} – 6.8×10^{-7}), and was unacceptable with water consumption and especially with food (up to 8.5×10^{-4}).

Currently, the risk assessment models incorporate PHEs data for a range of 568 important exposure pathways (drinkable water, beverages, food, dust inhalation, 569 soil ingestion) from which a total human intake is derived (Kolluru et al. 1996; 570 Heikens 2006; Yi et al. 2011; Chon et al. 2011; Alvarenga et al. 2013). 571



t.1 t.2 t.3 t.4 t.5 t.6 t.7

Table 10.2	Toxicity indexes
of selected e	elements (Adapted
from Zhou e	et al. 2010)

Element	Slope factor dose	Reference
As	1.5	0.003
Cd	6.1	0.001
Cr	_	0.005
Cu	_	0.038
Hg	_	0.0001
Pb	_	0.004
Zn	_	0.30

4. *Risk characterization* – Toxic risks refer to the non-carcinogenic harm occurring due to the exposure level (Chon et al. 2011), and the extent of the harm is indicated by US-EPA (1992, 1998) in terms of hazard quotient (HQnc):

$$HQnc = E/RfD$$
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The exposure level (E), is the average daily dose (ADD), and the reference dose (RfD) is the daily dosage that enables the exposed individual to sustain this level of exposure over a prolonged time period without experiencing any harmful effect.

The hazard index (HI) is the overall toxic risk resulting from the sum of individual toxic risk due to a single PHE. If HI < 1.0, the non-carcinogenic adverse effect is considered negligible.

The cancer risks refer to the probability one may develop cancer at a given lifetime exposure level. High carcinogenic risk levels are expressed by the following equation (US-EPA 1998):

$$Risk = 1 - \exp(-CDI \times SF),$$

where CDI is the chronic daily intake over 70 years and SF is the slope factor (see above).

Chon et al. (2011) proposed a similar calculation of cancer risk, which is determined as the product of the lifetime exposure level (ADDlife) by the slope factor (SF):

Cancer risk = ADDlife
$$\times$$
 SF.

The above model has been widely accepted and used, but has been also improved and adapted to specific purposes, combining human health with ecological risk assessment (Korre et al. 2002).

For example, an integrated procedure has been proposed so far by Covello and Merkhofer (1993), which consists of seven steps, and include:

- 594 (a) problem formulation;
- 595 (b) hazard identification;
- 596 (c) release assessment;
- 597 (d) exposure assessment;

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10 Risk Assessment of PHEs

(e) consequence assessment;	598
(f) risk estimation;	599
(g) risk evaluation.	600

The problem formulation is the planning process for performing the risk assess- 601 ment. Its main goals are: (i) the selection of assessment endpoints (i.e. one or more 602 ecosystem components or attributes); (ii) identification of the pathways by which 603 human activities induce effects on the assessment endpoints, and (iii) identification 604 of data needs and methods of data generation to continue the risk assessment (Critto 605 and Suter 2009).

The hazard identification is aimed at identifying the potential threat for the 607 environment (e.g. a landslide or flooding, or waste disposal on soil and groundwa- 608 ter) or which can determine a harmful effect in human body (e.g. PHEs). As previously stated, the hazard identification process is accomplished through the 610 collection of environmental data (e.g. sampling of soils, waters and plants, and the 611 subsequent determination of the contaminant level of PHEs in these samples).

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The release assessment step involves the identification and monitoring of the 613 source, and the use of statistical analysis, spatial analysis (Bertazzon et al. 2006) 614 and modelling techniques to quantify the source of risk.

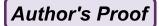
The exposure assessment process entails the characterization of exposure, identification of the exposure routes, and description of the exposed population, and the 617 analysis of all the critical variables of the exposure scenario (Korre et al. 2002). The 618 analytical phase points to the consequence assessment, and allows quantification of 619 the land constraints (e.g. rock detachment, soil liquefaction, release and fate of 620 contaminants), and characterization of ecological effects, and defines the exposureresponse relationships.

The last two steps, risk estimation and risk evaluation provide risk estimates 623 through the integration of results of exposure and effects. 624

Concerning human health, in general, the PHEs exposure routes related to soil 625 are direct soil ingestion and dermal absorption. It is known from literature 626 (Veerkamp 1994; Korre et al. 2002) that dermal absorption is significant in the 627 case of organic substances (dioxins, PAHs) and organometallic compounds 628 (e.g. Hg-methyl, Pb-tetraethyl, Sn-trialchylchloride), but is negligible in the case 629 of heavy metals. Therefore, only the ingestion pathway is generally investigated. 630 For example, with lead exposure the Chronic Daily Intake (CDI) of Pb deriving 631 from the pathway of direct ingestion of contaminated soil is calculated as follows 632 (Korre et al. 2002): 633

$$CDI = (Cs \times IR \times CF \times FI \times EF \times ED)/(BW \times AT)$$

where Cs is the Pb concentration in soil (mgkg⁻¹), IR is the ingestion rate of soil 634 from all sources (mgday⁻¹), CF is a conversion factor (10⁻⁶ mgkg⁻¹), FI is the 635 fraction ingested from the site as a fraction of the total from all sources (in range 636 0.0–1.0), EF in the exposure frequency (days year⁻¹), ED is the exposure duration 637



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638 (years), BW is the body weight (kg) and AT is averaging time (days). For 639 non-cancer risk $AT = ED \times 365$.

Advanced geostatistics coupled with exposure assessment of residents at a Pb-contaminated site (Korre et al. 2002) allowed to estimate the fraction of Pb absorbed into the vascular system after ingestion; it was considered maximum and was set to one. This represent the worst-case scenario, which enlarges the influence of the ingestion pathway.

The US-EPA generic reference dose (RfD) is a commonly used estimate of exposure for the human population (US-EPA 1992, 1998). Several RfD values for Pb exist in the literature; among the most commonly reported, US-EPA proposes 0.1 mgkg⁻¹day⁻¹ (Petts et al. 1997), and AERIS (Aid for Evaluating the redevelopment of Industrial Sites) proposes 0.0035 mgkg⁻¹day⁻¹ (AERIS 1991). The former is a generic reference level (maximum value in the literature), the latter one is the smallest oral exposure RfD.

After the completion of the risk assessment steps, Korre et al. (2002) calculated a sensitivity index (SI) based on the model parameters, applying the following equation:

$$SI = 1 - CDImin/CDImax$$

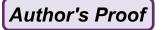
where CDI is the chronic daily intake. The closer to zero is SI, the smaller is the correlation between the input parameter and the resultant chronic daily intake. If SI is close to one, the investigated parameter was labelled as sensitive, and has a significant effect on the resultant CDI.

The CDI of Pb at contaminated sites, deriving from the pathway of direct ingestion of contaminated soil was estimated for two population groups (see the CDI equation above): male adults with maximum exposure to soil (gardeners, farmers) and children 1–6 years old. The spatial representation of the results by a GIS model yielded a comprehensive picture of the risk to human health from direct ingestion of soil.

The results showed that the highest risk probability is consistent with high heavy metal concentrations. Yet, the effect of the selection of the reference dose was significant: when a low RfD was chosen, the probability of high exposure was significant for the target populations. On the contrary, when a high RfD was chosen, the probability of high exposure was smaller.

670 6 Urban Soils Contamination and Risk Assessment

Most frequently, high contamination levels affect urban soils, as a consequence of various anthropic activities (industry, traffic, waste disposal etc.), which determine PHEs concentration levels that can pose significant human health risks due to direct soil ingestion, inhalation of volatiles and dermal contact (Siciliano et al. 2009), especially in public parks and playground areas (see Chap. 6, this volume).



Particularly children and senior citizens are most vulnerable, because of their less 676 immunological defence and the possible exposure to PHEs (Luo et al. 2012). For 677 instance, lead contamination is ubiquitous in urban soils, and can be re-suspended 678 in the air posing ongoing threats particularly to children because of its effects as a 679 neurotoxin that inhibits development (Schmidt 2010).

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In comparison with agricultural soils which mainly influence human health 681 indirectly via food chain (see Chap. 3, this volume), soils in residential areas and 682 urban parks have special recreational functions. Due to open space activities such as 683 jogging, sporting, playground etc., re-suspended dust and hand-to-mouth oral 684 ingestion can be a critical pathway of exposure for both adults and children, and 685 it is important to quantify the various PHEs exposure risk levels for citizens.

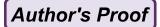
Urban soil contamination has been generally evaluated by analysing total metal 687 concentrations compared with corresponding soil guidelines values, that may overestimate the actual health risks, as is the case of Ni in serpentine soils (Angelone et al. 1993).

The risk posed by PHEs with ingested soil depends on the element fraction that 691 is soluble in the gastrointestinal tract available for subsequent absorption (i.e. only a 692 fraction of the total soil metal content is bioaccessible). Luo et al. (2012) have 693 developed an in vitro digestion model to assess the human bioaccessibility, simulating the successive solubility of metal under stomach (gastric acid) and intestinal 695 tract conditions. The proposed in vitro bioaccessibility extraction test is a static 696 gastric model by which bioaccessible metals are extracted under acid conditions 697 simulating those in human stomach. The percentage of ingested bioaccessible 698 fraction (BAF%) of each metal is calculated as the percentage of the fraction 699 soluble in simulated stomach acid (Cbioacc mgkg⁻¹) relative to the pseudo-total concentration (Ctotal, $mgkg^{-1}$) of the sample using the following equation: 701

$$BAF\% = Cbioacc/Ctotal \times 100.$$

Exposure of humans to PHEs in urban soils can occur via three main pathways: 702 direct oral ingestion of substrate particles (also called Chronic Daily Intake by 703 ingestion: CDI ingestion), inhalation of dust re-suspended from soil through mouth 704 and nose (Chronic Daily Intake by inhalation: CDI inhalation), and absorption of 705 heavy metals-bearing soil particles by exposed skin (Chronic Daily Intake by 706 dermal contact: CDI dermal). Both non-carcinogenic and carcinogenic risks of 707 these exposure routes are considered in literature, taking particularly care of the 708 non-carcinogenic hazard exposure for children (Luo et al. 2012). The dose received 709 (chronic daily intake, CDI, i.e. average daily dose ADD) through the main exposure 710 routes is calculated by the adapted US-EPA (1989, 1997) and US-DOE (2011) 711 models (acronyms are the same as above).

Non-carcinogenic hazard for children is determined as the summation of:



CDI ingestion =
$$(C \times IR \times EF \times ED/BW \times AT) \times 10^{-6}$$

CDI inhalation = $C \times EF \times ET \times ED/PEF \times 24 \times AT$
CDI dermal = $(C \times SA \times AF \times ABS \times EF \times ED/BW \times AT) \times 10^{-6}$
(see Luo et al.2012, for details)

The carcinogenic risk for adults is calculated for the lifetime exposure, estimated as the incremental probability of an individual developing cancer over a lifetime as a result of total exposure to the potential carcinogen. The dose received (chronic daily intake, CDI, i.e. average daily dose ADD) through the main exposure routes is calculated by the adapted US-EPA (1989, 1997) and US-DOE (2011) models (acronyms are the same as above).

Carcinogenic risk for adults (see Luo et al. 2012, for details):

CDIingestion =
$$(C \times IR \times EF/AT) \times 10^{-6}$$

CDI inhalation = $(C \times EF \times ET \times ED/PEF \times 24 \times AT) \times 10^{3}$
CDI dermal = $(C \times ABS \times EF \times DFS/AT) \times 10^{-6}$
DFS = soil dermal contact factor-age-adjusted.

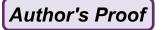
Though interactions between some metals might result in a synergistic response (Xu et al. 2011), it is assumed that all the metal risks are additive, hence it is possible to calculate the cumulative non-carcinogenic hazard expressed as the Hazard Index as the sum of the three Hazard Quotients (HI = \sum HQ ing + HQ inhal + HQ der), and carcinogenic risk expressed as the total cancer RISK:

$$(total\ RISK = \sum Risk\ ing + Risk\ inhal + Risk\ der).$$

Considering the site-specific oral bioaccessibility, the human exposure estimate to the main exposure pathway (soil ingestion) is adjusted when calculating the HQ ingestion and Risk ingestion:

CDI ingestion – adjusted = CDI ingestion
$$\times$$
 BAF%.

Concerning hazard quotient, as a general rule, the greater is the (positive) value 729 of HQ, the greater is the likehood to have adverse health effects. Hence, HQ ≤ 1 730 suggests unlikely adverse health effects, whereas HQ > 1 suggests adverse health effects to be likely. Concerning (total) cancer risk, the value 10^{-6} is considered the carcinogenic target risk by USEPA (2011): the cancer risk lower than 10^{-6} 733 (a probability of an individual in one million to develop cancer) is considered to be negligible, while cancer risks above 10^{-4} are considered unacceptable by most international regulatory agencies (Luo et al. 2012). Though the human health risk 736 levels of PHEs at urban sites are not negligible, the metal levels are often lower than various current guideline values. Hence, soil quality criteria still based on total metal concentrations might not accurately estimate the real risks (Baize and Van 740 Oort 2013) and are just appropriate for worst-case scenarios. Overall, the actually 741 occurring adverse effects of heavy metals in urban soils are site-specific. Moreover,



the importance of exposure pathways, metal bioaccessibility and soil properties in 742 assessing the realistic risks of soil metals should be highlighted. 743

7 Land Uses and Risk Assessment

Besides urban soils, different land use types significantly control the soil metal 745 exposure and corresponding human health risks. Six types of land use are generally 746 considered: residential, agricultural, forest, water, mine and bare land). Residential 747 soils, as urban soils, may be affected by PHEs released by human activities 748 (e.g. traffic, road dust); agricultural soils suffer for large use of fertilizers and 749 herbicides (Simon 2013); mine soils are naturally enriched in PHEs that might 750 pose serious problems to human health (Chap. 5, this volume), and also forest soils 751 may be enriched in heavy metals released by the geological composition of parent 752 rocks (Chap. 4, this volume), especially with acidic conditions that enhance mineral 753 weathering. Therefore, in some developed countries such as Canada (CCME 2007), 754 UK (UKEA 2009) and USA (USEPA 2011), the soil guideline values of contam- 755 inants have been proposed for different land use (Table 10.3). 756

A methodology to calculate the human health risk from heavy metals has been 757 proposed recently by Zhao et al. (2012), based on a dose-response model, including 758 different land uses. Three factors are considered in a dose-response health risk 759 assessment: the sources, pathways and receptors. The first two are different for each 760 land use (residential, agricultural, forest, water, mine and bare land). Sources can be 761 dust, water, crops/food; exposure pathway is generally ingestion (soil and food) or 762 inhalation (dust). Receptors are living organisms and especially local population 763 that lives near the source.

The models used to predict the heavy metal contents of food (rice and vegeta-765 bles) are developed using a multiple regression analysis based on metal concentra-766 tion in food and in soil, in relation to soil pH:

$$logMetal(crop) = a + b \times logMetal(soil) + c \times pH soil,$$

where a, b, c are coefficients that vary depending on the soil, heavy metal, climate 768

By applying this model to rice and large leaf vegetables, Zhao et al. (2012) found 770 a significant positive correlation for metal concentration (Cd, Cu and Zn) in 771 vegetables and in soil. This is typical of metal tolerant (i.e. indicator) plants, as 772 stated by Baker (1981). The relationship of metal intake to its relative reference 773 dose (HQ = CDI/RfD) was used to assess the human health risk. Food daily intake 774 was estimated to be 370 g/day for adults (65 BW); the reference dose was in the 775 range 0.001–0.3 mg/kg/day (the lowest for Cd and the highest for Zn), consistent 776 with US-EPA (2006, 2009). The maximum HQs for Cd and Cu exceeded 1.0, 777 indicating a potential human health risk associated especially with Cd concentra-778 tion in soil and its transfer to food chain and local population. Yet, Cd level in blood 779

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Location		Land use	Cd	Cr	Cu	Ni	Pb	Zn
China (21 cities)		Residential	0.39	69	40	25	55	109
Europe		Agricultural	0.79	53	195	27	39	68
Europe (34 cities)		Residential	0.95	59	46	22	102	130
Canadian guidelines		Agricultural	14	64	63	50	70	200
Canadian guidelines		Residential	10	64	63	50	140	200
Canadian guidelines		Industrial	22	87	91	50	600	360
UK guidelines		Residential	10	100	36	130	85	140
UK guidelines		Industrial	230	_	_	1,800	_	_
US-EPA soil screen	ing level	Residential	70	120	3,100	1,500	400	23,00
US-EPA soil screen	ing level	Industrial	800	1,500	41,000	20,000	800	31,00
Italy		Mine soils	33	95	412	54	306	657

1 Table 10.3 PHEs concentrations at various sites and guidelines values for different land uses

780 of local residents in the investigated area was in the range 24.10 (highest exposure) 781 -1.87 (lowest exposure) μ g/L, as reported by Wang et al. (2011).

The modelling proposed (Zhao et al. 2012) provides a reasonable assessment of human health risk by integrating a spatial analysis of contaminant concentrations and land use.

Other approaches have become common in the last decade in the Environmental 785 Risk Assessment. One of these is based on the Weight of Evidence (WoE), a system 786 which allows determination of environmental risks by weighting multiple Lines of 787 Evidence (LoEs) that report the quality, extent, and congruence of data (e.g. chemical analyses) that pertain to important aspects of the environment 789 (Smith et al. 2002). Another approach is the Sediment Quality Triad (SOT), which is based on a standard combination of three LoEs, namely Sediment chemistry, Sediment toxicity and Benthic community structure. Each LoE provides 792 distinct and complementary information about the investigated environment. Moreover, the latter is the assessment endpoint that changes in response to exposure to a certain stressor: the model is widely used, and is continuously improved (Chapman 795 796 et al. 2002).

97 8 Biomonitoring

As previously stated, it is difficult to attribute the incidence of a disturbance at any 798 799 environmental compartment, or some adverse effects to humans, to a particular industrial activity or a chemical substance (Nieuwenhuijsen et al. 2006), as is the 800 case of arbitrary waste disposal on the land (e.g. chromium-rich effluents from leather industry, see f.i. Bini et al. 2008), or emissions in the atmosphere (see 802 Chap. 1, this volume). This is further complicated by other factors such as the fate 803 of chemicals (Alloway 1995), their pattern of dispersion, or the influence of local 804 geological, hydrological and meteorological conditions (Dall'Aglio et al. 1966). 805 Biological monitoring, also called biomonitoring, is a promising method of

assessing environmental and human health risk by analysing PHEs concentration in 807 environmental matrixes (e.g. plants, animals), or in human tissues (hairs, nails), or 808 in a biological matrix (blood, urine). 809

Concerning human health, biological monitoring is usually described as the 810 measurement of a particular chemical substance, or a metabolite of that substance, 811 in a suitable biological matrix (e.g. blood, urine, serum, and tissues such as hairs, 812 nails, sweats), that act as an effective biomarker. For example, Cd levels in toenail 813 were analysed in relation to prostate cancer (Vinceti et al. 2007); Chuang 814 et al. (2007) found significant relationships between blood PHEs and hearing 815 function; Se in human blood was related by Schalin (1980) to the aetiology of 816 multiple sclerosis; depleted U was determined in urine of military personnel 817 involved in the Bosnia war (Roth et al. 2001), and Cd in urine of exposed workers 818 was found as a result of various health problems due to prolonged exposure (Han 819 et al. 2009); quite recently, Giaccio et al. (2012) considered heavy metals in serum 820 as the main responsible for male infertility.

The application of biomonitoring as a direct method of measuring possible 822 contaminant exposure is frequently limited by the availability of adequate samples 823 (Nieuwenhuijsen et al. 2006). For example, in the environmental compartments, 824 mosses proved so far useful traps for airborne PHEs supply (Steinnes 1980), and 825 unusually low Se concentration in pastures was considered responsible for disorders 826 in grazing animals in several parts of the world (Schalin 1980).

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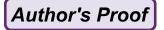
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In human population, blood and urine are often the preferred media, since many 828 toxic substances are easily measured in these media. For example, urine Cd proved 829 consistently associated with various renal and bone diseases (Bernard 2008), and 830 effective biomonitoring with urine and nails showed that human As exposure 831 decreased with distance from a power station (Wilhem et al. 2005). Biomonitoring 832 techniques can also be used to assess early biological or physiological changes that 833 are correlated with the uptake of toxic substances. These may induce molecular 834 and/or cellular alterations that occur along the temporal pathway connecting ambi-835 ent exposure to a chemical toxicant, as reported by Maleci et al. (2013) in 836 Taraxacum officinale. Similarly, Sarkar et al. (2013) found that the outcome of 837 Cd exposure could be apoptosis, growth inhibition, proliferation or carcinogenicity 838 in animal cells.

Indirect methods of estimating exposure such as simulation studies, GIS map- 840 ping, mathematical models as well as other statistical techniques are also currently 841 explored since the last years of last century (Goovaerts and Journel 1995; Bailer 842 et al. 1997; Ungaro et al. 2008). One of the most known environmental and human 843 concern is exposure to asbestos, a sneaky carcinogenic substance which may have 844 adverse effect on human health (lung cancer) with long incubation. A number of 845 studies were conducted during the period 1950–1990 to determine exposure of car 846 mechanics to asbestos released from brakes, but unfortunately certain characteristics of exposure were not studied at that time when the risk posed by asbestos was 848 not considered (Nieuwenhuijsen et al. 2006).

Simulation studies have been conducted recently to fill possible data gaps (see 850 f.i. Paustenbach et al. 2003), particularly with the finest aerosol particulate (PM₁, 851



PM_{2.5}; see Rampazzo et al. 2013). Also useful to this aim are retrospective simulation studies with historical records on metal distribution in water, soils and sediments carried out since the 1960s (e.g. Dall'Aglio et al. 1966), and in human bones (Martinez-Garcia et al. 2005). More recently, Khan and Cao (2012) paid particular attention to organic environmental contaminants such as dioxin, PCBs and PAHs. This information, coupled with the recently updated mortality data, could be used to estimate accurately the daily intake and the carcinogenic potency of such toxicants.

In recent years, mathematical modelling has become an important tool in 860 environmental research (Goovaerts 1997), and has been greatly enforced by the 861 use of geographical information systems (GIS) and geostatistical techniques. A 862 model may help to explain a complex system and to study the effects of different 863 components, and to predict the behavior of components (e.g. PHEs in environmen-864 tal media). Models are typically applied to study impacts of individual sources 865 (hot-spots), multiple-source industrial facilities, metropolitan areas, or larger 866 regional areas (Rampazzo et al. 2013). For example, regional models allow solving 867 important pollution phenomena and concentration gradients in areas where point 868 sources are present (e.g. mine areas). The spatial scales range from up to few 869 kilometers (for large industrial point sources), to 100 kilometers (for individual 870 urban areas), to few 1,000 kilometers (for larger regional areas). The development 871 of GISs (Geographical Information Systems) has further enhanced the facility of analysis, combining all the territorial data sets (e.g. population statistics, social and environmental data, land use, etc.) in information layers, which allow understand-874 ing relationships not always evident from single data sets. In the last decades, modelling techniques have greatly improved the assessment of local pollution patterns on the basis of monitored data, and are likely to be important in further 877 878 studies.

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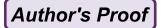
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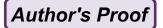
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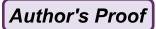
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Author Queries

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Query Refs.	Details Required	Author's response	
AU1	AU: Please check if inserted closing double quotes is okay.	ok	
AU2	AU: References "Menzies (2007), Kabata-Pendias (2011), UK-EA (2003), EC (1881), Commission's White Paper (2001), European The- matic Strategy for Soil Protection (2006), REACH regulatory (2013), US-EPA (2006, 2009)" are cited in text but not given in the reference list. Please provide.	done when necessary some achronims refer internal reports	
AU3	AU: The citations "Basta et al., Chojnacka (2005), Dall'Aglio (1966), Steinnes (1984), EA (2009)" have been changed to "Basta et al. (2005), Chojnacka et al. (2005), Dall'Aglio et al. (1966), Steinnes (1980), UKEA (2009)". Please check if appropriate.	ok	
AU4	AU: Please provide opening parenthesis in the sentence "where DI is the total PHEs daily intake".	done	
AU5	AU: Please confirm the paragraph starting in "4. Risk characterization".	ok	
AU6	AU: Please provide opening parenthesis in the sentence "Six types of land use are generally".	done	
AU7	AU: Please provide the in-text citation for the references "Angelone and Udovic (2013) and Wahsha (2013)".	done	
AU8	AU: Please update volume number and page range for Alvarenga et al. (2013), Geng et al. (2013), Maleci et al. (2013).	done	
AU9	AU: Please confirm the updated details for Angelone and Udovic (2013), Baize and Van Oort (2013), Rampazzo (2013), Simon (2013), Wahsha (2013).	done	
AU10	AU: Please confirm the inserted volume number for the references "Chapman et al. (2002), Singh et al. (2010)".	ok	

AU11	AU: Please confirm the inserted journal title for the reference "Chuang et al. (2007)".	ok
AU12	AU: Please confirm the inserted publisher location for US Department of Energy (USDOE) (2011), US Environmental Protection Agency (USEPA) (1989).	ok
AU13	AU: Please provide publisher name for US Environmental Protection Agency (USEPA) (1997).	not needed, it is a forum

