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URBAN SOILS AND HUMAN HEALTH

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Abstract

Since the dawn of civilization, the anthropic activity has lead to a legacy of increased land degradation/contamination. Potentially harmful elements (PHEs) are among the most effective environmental contaminants, and their release into the environment is increasing since the last decades. Interest in trace elements has risen as major scientific topic over the last 50 years, when it was realized that some elements were essential to human health (e.g. Fe,Cu,Zn), whereas some others were toxic (e.g. As,Hg,Pb), and likely responsible for serious human diseases and lethal consequences. Since that time, great progresses in knowledge of links between environmental geochemistry and human health have been achieved. The urban environment (nowadays the main habitat for human population) is a potential PHEs source, with high risk for residents'health. Indeed, PHEs concentration and distribution are related to traffic intensity, distance from roads, local topography and heating. Industrial emissions also contribute to the release of toxic elements. Understanding the extent, distribution and fate of PHEs in urban environment is therefore imperative in order to address the sustainable management of urban soils and gardens in relation to human health.
Despite the copious research addressed to this topic, the effects of most trace metals on human health are not yet fully understood. Uncertainty is still prevailing, particularly with non-essential elements that are "suspected" to be harmful to humans, causing serious health problems as intoxication, neurological disturbances and also cancer. Some of them (e.g. As,Cd,Hg,Pb) have attracted most attention worldwide, due to their toxicity towards living organisms. Other elements (Al,B,Be,Bi,Co, Cr,Mn,Mo,Ni,Sb,Sn,Tl,V,W) are likely harmful, but may play some beneficial functions not yet well known, and should be more investigated.

Introduction

It is generally recognized that environmental contamination with potentially harmful elements (PHEs) has increased dramatically since the dawn of the Industrial Revolution (Nriagu 1979, 1988), and the main receptor of contamination is soil (Brevik, 2013). Yet, it is the primary source of several elements and substances that humans intake with daily diet, and 98 % of food derives directly or indirectly from the soil (Coccioni 2011).

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Urban soils are particular ecosystems strongly influenced by human activity. These soils have thus become sink and storage areas of anthropogenic materials (e.g. chemicals, glass, plastic, oil) that release PHEs into the environment in such quantities as to change the natural background, sometimes up to 10–100 times and even more (Angelone and Udovic, 2014).
Urban soils include: public green and parks (e.g. Venice, S. Giuliano round point, where soils containing over 50 mg/kg As have been restored with fern), private gardens and horts, urban garbage and solid waste areas, industrial areas (e.g. Venice-Marghera, where severe polimetallic contamination has been ascertained, with elevated concentrations of As (137 mg/kg), Cd (57 mg/kg), Hg (30 mg/kg), Ni (23 mg/kg), Pb (700 mg/kg), Zn (5818 mg/kg) (Bini, 2008).

History

Besides local pollution episodes, the most significant world soil pollution events in the recent history date back to the last century (Bini and Wahsha, 2014).
A relevant amount of methyl mercury was released by a chemical factory in Japan over a long period (1932–1968), and entered the food chain determining acute poisoning to the local population, provoking severe neurological disturbance, known as Minamata disease.
In the same years (1950), Cd-enriched wastewater from a mining plant was discharged on rice fields in Japan, determining chronic poisoning (itai-itai disease) with severe consequences in kidney functioning and bone deformation (Bernard, 2008).
A significant long-term contamination, lasting 15 years (1952–1966), occurred in the town of Hinkley (California, USA), where groundwater contaminated with Cr(VI)-rich wastewater from a chemical plant determined several cases of cancer to resident population.
More recently (1976), an accident to the ICMESA chemical plant in Italy determined the release of dioxin in the atmosphere, and successive fallout to soils of a large area in Lombardy, with ca 250 persons affected by chlorine dermatitis. Moreover, dioxin is a known highly teratogenic substance responsible for severe foetal malformation, with still relevant effects after more than 30 years (Bini and Wahsha, 2014).
At Bhopal (India), in 1984, 40 tons of methyl isocyanate were discharged from a chemical plant producing pesticides, provoking 10,000 victims, and the mortality is still higher in that area than in other parts of India.
Chromium-sludge discharged (1970-1990) by leather tannery plants in the industrial district of Vicenza (Italy) has been shown to have contaminated hundreds ha of agricultural land (and water) up to 10,000 mg kg^{-1} Cr (Bini et al., 2008).
Petrol-chemical plants active at Porto Marghera (Venice, Italy) since the 1950s have been considered to be responsible for the contamination of the lagoon of Venice with several PHEs (As, Cd, Cr, Cu, Hg, Mn, Pb, Sb, Se, Zn) and organic chemicals (PAH, PCB, Dioxin). The whole area has been classified as contaminated site of national interest, and a restoration project is ongoing (Bini, 2008).

Perhaps the most known and impressive environmental disasters are those provoked by nuclear accidents like Chernobyl (1986) and Fukushima (2011). The consequences of the accident on human health, particularly in Ukraine and Russia, were (and still are) very impressive: although the official report indicates only 65 persons dead, and possibly 4,000 affected by cancer and leukaemia, an estimate of presumable deaths related to the accident indicates tens thousands to millions of victims.

PHEs input in soils

The major inputs of PHEs into soil are due to different sources:
- Atmospheric deposition;
- Application of manure, fertilizers, sewage sludge;
- Industrial, mining and smelting activities.

In the urban environment, the main sources of trace elements are emissions from vehicular traffic, followed by atmospheric emissions (heating and industrial) and wastewater. All these sources contribute to the release of elements such as Cd, Cu, Cr, Fe, Hg, Pb, Zn, and organic compounds such as PCB, PAH.

Urban soils may host potentially harmful substances such as
- Chemicals

Inorganic: major and trace elements (Fe, Al, Mn, Cr, Ni, Cd, Pb, Cu, Zn, As…), oxides (e.g.TiO_2, Fe_2O_3…) and anions (NO_3^-,CrO_4^{2-},Cl^-,S^{2-}…);
Organic: PCB, PAH, Dioxin.
- Plastic, glass, ceramic, bricks, wood, paints.

PHEs' Impact and Human Health

Presently, there is a growing tendency to promote urban agriculture in private gardens and horts. Food production in urban areas, therefore, implies a thorough overview and monitoring of the PHEs presence in urban soils.
Which are the possible consequences of human exposure to these substances? Resident population is continuously exposed to high levels of PHEs; their adverse health effects have been frequently recorded, in terms of:
- Sanitary risk for human health (by inhalation, ingestion, contact);
- Casual/professional diseases: toxication, plumbism, mercurialism.

Soil ingestion is generally considered to be the most important exposure pathway, and it is usually associated with eating of dirt in children, and with occupational exposure in adults.
Ingestion. The suggested values of daily-ingested soil are up to 137 mg d^{-1}, or even 1,432 mg d^{-1}, when pica behavior is present (Moya et al. 2004). The ingestion limits proposed by the US EPA differ in relation to the intake pathway, and are 50 mg d^{-1} for soil solely, 100 mg d^{-1} for soil and dust, and 1g d^{-1} for soil pica behavior (US EPA 2008).

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Many studies have focused on children, since they represent the most vulnerable group of people. Children are exposed to soil PHEs by dust and/or soil tracked into homes on shoes or family pets (Hunt et al. 2006), by dust deposition in closed spaces (Laidlaw and Filipelli 2008), by their mouthing behavior and during their recreational outdoor activities (Ko et al. 2007; Abrahams 2012). In addition, children stature usually coincides with the lower airborne layer produced by vehicular traffic emissions.

Particle size inhalation. Small particles (PM 2.5) can enter the human body also via inhalation, leading to a significant PHE absorption in the respiratory tract, in addition to the gastrointestinal absorption due to the ingestion of soil particles (PM10+) and to the consumption of vegetables and water contaminated with airborne particles (Rampazzo et al, 2014).

PHEs impact on Human Health. PHEs are known to have significant negative effects on human health at different levels.

Toxic effects may occur with acute, subacute, subchronic and chronic symptoms.

Acute reactions are generally due to the exposure to increased levels of PHEs, while long-term exposure may cause chronic illness, including cancer.

Long-term exposure to PHEs may lead to several diseases, among them a high potential to develop cancer (Nriagu 1988; Kurt-Karakus 2012). Hubbard et al. (1996), for example, reported about significant exposure-response effects regarding the cryptogenic fibrose alveolitis among subjects occupationally exposed to metal or wood dust.

Exposure to mercury from industrial activities has been found to be correlated to the increased kidney disease mortality among the population in the nearby residential zone. Similarly, epidemiological studies have reported an elevated incidence of beryllium sensitization (BeS) among workers occupationally exposed to Be-bearing dust particles, that may develop into the potentially fatal lung disease, the chronic Be disease (CBD) (Virji et al. 2011).

Chronic exposure to Cd may have effects such as lung cancer, prostatic proliferative lesions, bone fractures, kidney dysfunction and hypertension (Zhao et al. 2012). Prolonged oral and inhalation exposure to As can lead to skin lesions and lung cancer, respectively. Exposure to Pb may cause plumbism, anaemia, gastrointestinal colic and central nervous system disturbance (Zukowska and Biziuk 2008). Nickel can cause lung cancer, chronic bronchitis, emphysema and asthma.

Willis et al. (2010) have studied the connection between the incidence of Parkinson disease and PHEs (Cu, Pb and Mn) emission in urban areas.

Some examples of Widely Recognized Harmful Elements

Arsenic. Arsenic is a lethal poison, as represented also by the register Frank Capra in the famous fiction "Arsenic and old lace" (1944). It may provoke skin lesions and tumours, although at clinically achievable concentrations it is used to induce apoptosis in malignant cells. A type of baked clay (sikor), commonly consumed by

Bangladeshi women in Bangladesh and in the United Kingdom, may be an important source of As, Cd and Pb intake (Al-Ramalli et al. 2010), with severe consequences, (cancer, kidney damage and brain damage), especially during pregnancy. The average daily consumption of sikor (50g/d) would contribute up to 370 and 1,236 mg kg^{-1} of As and Pb to the diet, respectively.

Cadmium. The International Agency for Research on Cancer (IARC 2006) classifies Cd in Class 1: "the agent is carcinogenic to humans", and is a "priority hazardous substance" under the EC Water Framework Directive (2006).
Cadmium has been ranked at the sixth in the Top 20 list of toxic substances for significant human health hazard by USA, and has been a focus of study on environmental pollution in the UN Environmental Programs (UNEP) and the International Commission on Occupational Health Organization (Han et al. 2009).
The acute toxicity, as first described by Friedrich Stromeyer (1817), can lead to kidney, bone, and pulmonary damages. Chronic exposure to Cd can have severe effects such as lung cancer, prostatic proliferative lesions, bone fractures, kidney dysfunction and hypertension (Bernard, 2008).

Lead. So far, much attention has been given especially to Pb, which remains one of the major public health problems in the United States (Todd et al. 1996) and elsewhere. About 5.4 million tons of Pb additives were used for vehicular traffic in urban areas in the USA in the period between 1927 and 1994, and this proved the main source of Pb exposure.
Another important source of soil pollution in urban areas is the Pb-based painting. On February, 21, 1988, local newspapers in U.S.A. reported that it was a miracle that Mr and Mrs Wallace survived to lead intoxication induced by kitchen artistic pottery. Long-time exposure to PHEs may also impair fertility. Louis et al. (2012) found the Pb and Cd blood levels to be significantly associated with reduced couple fecundity.
It should be also highlighted that maternal exposure to PHEs, in particular to Pb, resulting in maternal blood levels >1 $\mu L\ L^{-1}$, can have severe negative effects on the developing fetus, impairing the cognitive and motor abilities of the child, or even inducing spontaneous abortion (Bellinger, 2005; Schell et al., 2006).
Lead and Children. Lead has a high uptake percentage into the children's organism; 50 % of the ingested Pb is retained into the organism, compared to 5 % in adults (Laidlaw and Filipelli, 2008). As Pb accumulates in the developing neural system and in bones, it may lead to permanent neural deficiencies, impaired intellectual performance, learning disorders, attention deficit/hyperactivity disorder (Hassanien and El Shahawy, 2011).

Other Heavy Metals. Not only Pb and Cd, but also Zn, Cr, Cu, and Ni are largely present in urban soils and dusts (Mielke et al. 2000; Wei and Yang 2010). Some PHEs, such as Cu, are harmless in small quantities, whereas some others, including Pb and Cd, may have neurotoxic effects.

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Exposure to high levels of PHEs could have serious negative effects on humans, e.g. accumulation in fatty tissues, negative effects on central nervous system and damage of lungs, kidneys, liver and other vital organs (Dockery and Pope 1996).

Some examples of Emerging Harmful Elements

Aluminium - directly related to neurotoxic disturbances (haedache, epilexy) at high exposure levels (Polizzi et al., 2002). Implicated in the aetiology of neurological disorders (Alzheimer, arteriosclerosis).

Beryllium - highly toxic to living organisms, and affects exposed workers; the target organs are bones, liver, kidney, and lung. Pulmonary diseases that affect Be-workers are particularly serious.

More attention should be paid to this emerging element.

Fluorine - Elevated F levels in drinking water can produce both mutagenic and carcinogenic changes in the kidneys.

- Acute fluorine intoxication is characterized initially by gastro-enteric disturbances, vomit, abdominal pains, diarrhoea. Afterwards, muscle tremors, urinary incontinence, cardiocirculatory collapse may occur. Respiratory paralysis and heart failure determine lethal consequences.
- Chronic intoxication is characterized mostly by evident disturbances of skeletal apparatus, with scarcely relevant bone lesions, thickening of the long bones of the limbs, calcification, spontaneous fractures, dental lesions.

Nickel - The toxicity of Ni is relatively low, but Ni allergy is a significant problem in humans, even at low exposure doses. Instead, the toxicity and carcinogenicity of high doses of Ni are well documented and depend mainly on its potential to damage proteins and nucleic acids. Yet, Ni is known as producer of ROS (e.g. OH), lipid peroxidation (LPO) and oxidative DNA damage (Franco et al. 2009). Investigations on the toxicity of Ni have indicated various effects of its excess, among which the most important are developmental, genotoxic, neurological, reproductive, and carcinogenic.

Thallium - The toxicity of Tl has not been greatly studied, but its harmful impact has been observed in both humans and animals; Tl is more acutely toxic than Hg, Cd, Pb, Zn and Cu in mammals (Peter and Viraraghavan 2005). Initial thallium poisoning symptoms in humans are palmar erythema, acne, loss of hair and hallucinations. The principal features of acute thallium poisoning are gastroenteritis, polyneuropathy and alopecia (Kazantzis 1986, 2000). With acute intoxication, there is usually an initial hypotension and bradycardia, followed by hypertension and tachycardia. The central and peripheral nervous system is the main critical organ in thallium intoxication. Major symptoms of Tl poisoning include anorexia, headache, pains in abdomen, upper arms and thighs and even in the whole body. In extreme cases, alopecia, blindness and even death may be caused.

Tin - Increased Sn concentration in food may cause acute gastric irritation, impaired reproductivity, and bone strength failure. It is also considered to be genotoxic. Some organotins are highly dermal irritants (Kabata-Pendias and

Mukherjee, 2007). As in the case of other elements (e.g. Mo, Se), a dietary deficiency of Sn is likely to induce some disturbances in humans, as hair loss, depressed growth, response to sound, feed efficiency, synergic decrease of other elements in various organs (e.g. Fe in kidney, muscle and spleen).

PHEs and cancer

Some elements, such as mercury (Hg), cadmium (Cd), arsenic (As) and chromium (Cr), are toxic or carcinogenic even at low concentrations. Allergies may also occur and repeated long-term contact with some metals, or their compounds, may become carcinogenic.
Carcinogenic elements present common exposure pathways; the main way is inhalation, and the main target organs are those of the respiratory system (bronchus and lung), although other organs such as skin, stomach, prostate, kidney, urinary bladder, are reported as possible target organs (Apostoli and Catalani 2008).
Carcinogenic metals are only little mutagenic. As, Cd, and Ni inhibit DNA repair mechanisms, contributing to augment tumour inizialization induced by other agents, playing an important role in cellular proliferation, and favouring neoplasm development. Some metals, moreover, may induce codifying genes for cell-protective proteins such as metallothionein, and stress proteins (Apostoli and Catalani 2008).

ROS

Among the mechanisms which make plausible the carcinogenic action of metals, is reported their ability to generate reactive oxygen species (ROS) and other intermedia able to induce direct damage to DNA by interacting with several enzymes and with cellular proliferation regulators.
ROS act on cells with a direct effect on proteins, altering the activity and the conformation, or acting on redoxisensible proteins. The formation of metal-protein complexes, moreover, may interfere on cellular homeostasis, and determine conditions for an increase of cellular clones with mutagenic phenotypes.
It is generally accepted that the metal species influences in a determinant way the biological and toxicological activity of that metal. The effect induced by that element depends on its ability to enter the cell and to interact with target sites such as DNA (Sarkar et al., 2013).
Crucial, therefore, are the chemical species, the oxidative state, charge, solubility, binding properties, stereochemistry, possibility to interact with other xenobiotics (Apostoli et al. 2006).

Supplementary Material (adapted from Angelone and Udovic, 2014)

1 - Technological substrates and related waste commonly present in urban areas:
- Source Waste material
- Construction and housing brick, concrete, mortar, plaster

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- Road work Bitumen asphalt, tar asphalt
- Ironwork, Steelworks, Foundries,
- Heavy metal works
- Steel and furnace slag, sand of foundry, pumice
- Incinerator fly and bottom ash
- Household Glass, metal, paper, plastic, ceramic, organic garbage, wood, bulky refuse

2 - Sources of potentially harmful pollutants in the urban environment:
- Cars (exhaust, fuel, oil,) Ba, Cd, Pb, Cr, Cu, Pd, Pt, Rh, Zn, V, NOx, PAH, CO, SO_2, Phenols, Hydrocarbons);
- Tires (Cr, Cd, Cu, Ni, Zn)
- Brakes wear (Cu, Zn)
- Road and urban surface weathering: PAH (asphalt), metals;
- Road and urban surface winter management:Detergents, salts, Cd, Cu, Fe, Ni, Zn;
- Corrosion of construction materials: Cd, Zn;
- Domestic heating systems: Co, Mn, Ni, V;
- Garbage incineration, fire releasing from various materials: Cd, Cu, Zn, Ba.

Conclusions

More accurate epidemiological investigations, combined with environmental and biological data, which actually may qualify population exposure, and in collaboration with other disciplines as metallurgy, industrial hygiene, analytical chemistry etc. could enhance comprehension of the actual risk posed by PHEs to human health, with particular reference to urban environment.

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