REVIEW

Heavy Metal Pollution in Sub-Saharan Africa and Possible Implications in Cancer Epidemiology

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Abstract

The increasing scourge of cancer epidemiology is a global concern. With WHO emphasizing that 40% of all cancer cases are preventable, exposure to known and suspected carcinogens must be discouraged. The battle with communicable diseases and other third world challenges has greatly de-emphasized anti-cancer campaigns in sub-Saharan Africa. The abundant deposit of mineral resources in sub-Saharan Africa has attracted high mining activity with its negative environmental aftermath. Poor regulatory mechanisms have led to environmental contamination by products of mining including heavy metals. In addition to poor urban planning, the springing up of settlements in industrial areas has led to generation and exposure to more hazardous wastes consequent on poor disposal systems. Studies establishing close association between exposure to heavy metals and cancer epidemiology in sub-Saharan Africa are increasing. The current review assesses the level of environmental pollution by heavy metals in sub-Saharan Africa, and brings to the fore available evidence implicating such in the increasing cancer epidemiology in the sub-continent.

Keywords: Cancer - environmental pollution - heavy metals - sub-Saharan Africa - epidemiology

Asian Pacific J Cancer Prev, **14 (6)**, 3393-3402

Introduction

Cancer is the leading cause of death in developing countries (Jemal et al., 2011; Malvezzi et al., 2013). Among many factors, the increasing adoption of cancer-related food and activity are implicated in the increasing global burden of cancer. Due to the current scourge of communicable diseases including malaria and HIV/AIDS, and government attention focused on reducing the high maternal and child mortality, cancer is under-emphasized in the sub-Saharan Africa (SSA). According to the World Health Organization (Quaglia et al., 2013; WHO, 2013), 70% of the 7.6 million cancer deaths accounts (about 13% of all deaths) in 2008 occurred in low- and middle-income countries which include SSA. Country-based cancer statistics are available for most developed Nations, such information are however scarce in SSA. Data for cancer epidemiology and mortality are often provided as rough estimates which usually reflect the poor diagnostic capacity and under-reported cases in the death registry. The development of cancer registration is slow in Africa, such that current registries only cover about 11% of the population (Parkin, 2006; Ferlay et al., 2010). This reflects in the fact that, since 1965, only Mauritius and South Africa have contributed into the WHO mortality database from SSA.

Despite the paucity of reliable statistics, it has been estimated that 62% of adults over 45 years old, die of non-communicable diseases especially cancer, and about 1 in 5 of all deaths are due to cancer (Siegel et al., 2012). It has also been projected that a million Africans will die annually from cancer-related causes by the year 2030 (Sylla and Wild, 2011). Where available, country-specific cancer epidemiology in Africa is not less than those of developed countries. In South Africa for example, according to the National Cancer Registry as published by the Medical Research Council (2003) and Sassman (2011), cancer is the fourth leading cause of death and the eighth major contributor to disability adjusted life years (DALY) in the country. According to the Cancer Association of South Africa (CANSA), 17% men and 14% women will get cancer in their life time irrespective of their race or socioeconomic status (Sassman, 2011). In the order of prevalence, CANSA presents breast, cervical, colo-rectal, lung and oesophageal cancer as the commonest in South Africa. It has been estimated that new cancer cases in less developed and developing countries will increase from about 56% of the world total in 2008 to more than 60% in 2030 due to the increasing global cancer trends, the expected population growth and increases in life expectancy (Jemal et al., 2010).

The causes of cancer, generally classified into external (infectious organisms, chemicals, tobacco, radiation) and internal (inherited mutations, hormones, immunity and metabolism-induce mutations) factors, may act independently, in sequence or together to

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initiate carcinogenesis. Usually, a time-lag of ten years or more may exist between exposure to carcinogens and the presence of detectable cancer. Besides HIV-related cancers, the increasing urbanization, mining activities, and poor waste control are major routes of human exposures to known carcinogens in SSA. The WHO (2007; 2013) estimates that 40% of all cancers are preventable because exposure to their causes is avoidable.

Relevant and detailed cancer statistics in SSA including the burden, distribution, trend, causes and control measures is provided in some recent reviews (Parkin et al., 2008a; 2008b; Ferlay et al., 2010; Jemal et al., 2012).

Aim, Search Strategy and Selection Criteria

The paper provides a concise review on the available knowledge on the environmental pollution and human exposure to heavy metals in SSA with possible association with increasing cancer cases. The review was systematically conducted by searching the databases of MEDLINE, PUBMED, EMBASE and COCHRAINE libraries for original researches using such search terms as 'cancer', 'heavy metal pollution', 'mining and cancer', 'Africa' and specific searches for each country in SSA. Search resulted were collated and studied extracting results relevant to the review title. Searches were not limited to time or place of research but limited to publications available (originally or translated) in English language.

Heavy Metals as Human Carcinogens

Chemical carcinogens are substances which under certain circumstances can induce cancer in humans after excessive or prolonged period of exposure. They include natural and/or synthetic industrial, agricultural or commercial substances which exert carcinogenic effects via various mechanisms including the distortion of the conformation of DNA (and its functions) during replication and transcription; mutational activation of protooncogenes and/or inactivation of tumor suppression genes; non mutational processes such as the clonal expansion of pre-malignant cells.

Despite the bio-importance of some heavy metals as trace elements, their general biotoxicity is of greater health concern (Jarup, 2003). Heavy metals often refer to metallic elements with relatively high density and known to be toxic to humans even at low concentration. They include lead (Pb), cadmium (Cd), zinc (Zn), mercury (Hg), arsenic (As), silver (Ag), chromium (Cr), copper (Cu), iron (Fe), and the platinum group elements.

A number of studies have established some heavy metals as potential risk factors in the pathogenesis of cancer. There are a number of authoritative international and local organizations that collect information, scientific evidence and toxicological data which help to classify and regulate chemical carcinogens, complex mixture, occupational exposures, physical agents and life style factors. Perhaps the most important of these is the International Agency for Research on Cancer (IARC, Lyon, France), an international scientific organization of the World health Organization. It is a body of experts in toxicology, cancer biology, cancer epidemiology, oncology and related fields functioning as the WHO authoritative body since 1971. IARC has published a series of monographs on the evaluation of carcinogenic risks to humans. It currently classifies carcinogens including heavy metals after a body of evidence (Table 1).

Arsenic

Arsenic is a carcinogen that contaminates drinking water worldwide. Accumulating evidence suggests that both exposure and genetic factors may influence susceptibility to arsenic-induced malignancies (Karagas et al., 2012).

The concentration of arsenic in the air and water is generally low (Rasmussen and Andersen, 2003; Uthus, 2003). Several studies have revealed that as is toxic to human cells at very low concentrations. This includes the induction of mutagenesis, carcinogenesis and

Table 1. IARC Classification of Heavy Metal Human Carcinogens, Updated in 2012

Metal	Class*	IARC monograph reference
Aluminium preparation	1	34, supp 7, 100F
Antimony trioxide	2B	101
Antibody trisulfide	2B	47
Arsenic and inorganic arsenic compounds	1	23, supp 7, 100C
Beryllium and beryllium compounds	1	58, 100CC
Cadmium and cadmium compounds	1	58, 100C
Calcium carbide production	3	92
Chromium, metallic	3	49
Chromium III compounds	3	49
Chromium IV compounds	1	49, 100C
Cobalt and cobalt compounds	2B	52
Cobalt metal with tungsten carbide	2A	86
Cobalt metal without tungsten carbide	2B	86
Cobalt sulfate and other soluble cobalt (II) salts	2B	86
Ferric oxide	3	1 supp 7
Gallium arsenide		86, 100C
Indium phosphide	2A	86
Iron-dextran complex	2B	2, supp 7
Iron-dextrin complex	3	2, supp 7
Iron sorbitol-citric acid complex	3	2, supp 7
Lead	2B	23, supp 7
Lead compounds, inorganic	2A	87
Lead compounds, organic	3	23, supp 7
Mercury and inorganic mercury compounds	3	26 supp7
Nickel, metallic and alloys	2B	49
Nickel compounds	1	49, 100C
Plutonium	1	78, 100D
Potassium bromate	2B	73
Selenium and selenium compounds	3	9, supp 7
Titanium dioxide	2B	47, 93
Vanadium pentoxide	2B	86

*Group 1: carcinogenic to humans: implying the availability of sufficient evidence (epidemiological, occupational exposure and animal studies) of carcinogenicity to humans with a clear understanding of the relevant mechanism of human carcinogenicity. Group 2A: probably carcinogenic to humans: implying the availability of limited evidence of carcinogenicity to humans, sufficient evidence of carcinogenicity in experimental animals with strong evidence that the method of carcinogenesis is operable in humans. Group 2B: possibly carcinogenic to humans: implying the availability of limited evidence of carcinogenesis to humans, less than sufficient evidence in experimental animals; inadequate evidence in humans but sufficient or limited in experimental animals. Group 3: not yet classifiable as to its carcinogenicity to humans: availability of inadequate evidence in humans, inadequate or insufficient evidence in experimental animals, mechanism of carcinogenesis does not operate in humans. Group 4: probably not carcinogenic to humans: Negative evidence in humans despite suggestions from no-human studies

teratogenesis through alterations in cell differentiation and proliferation (Liu and Lu, 2010); the induction of chromosomal aberrations and sister chromatid exchange (Pant and Rao, 2010); and the mediation of increased cellular tyrosine phosphorylation, a mechanism associated with uncontrolled cell growth and cancer development, in exposed cells (Hossain et al., 2000; Flora, 2011). The processes of energy production from fossil fuel and the smelting of metals are the major anthropogenic activities that results in arsenic contamination of air, water and soil (Ravenscroft et al., 2011).

Cadmium

Cadmium is a soft, silver-white metal found naturally at low levels in rocks and soil. It is used in nickel-cadmium batteries, electroplating as a component in metallurgical and brazing – soldering alloys, in pigments, and as stabilizer for plastics. Further environmental sources are smelting of other metals like Zn, burning of fossil fuels and waste materials (often deposited as solid waste), use of high phosphate and sewage sludge fertilizers (Davis et al., 2001). Cd is a toxic metal with elimination half-life of 10-30 years, accumulating in the body especially in the kidneys. Its urinary excretion is a biomarker of its body burden (Su et al., 1011).

The kidney being its major target organ, the first manifestation of Cd toxicity is tubular dysfunction with increased urinary excretion of calcium and low-molecular weight proteins such as β2-microglobulin, -acetyl-β-D-glucoseaminidase (NAG), retinol-binding protein and α1-micro-globulin (Fujiwara et al., 2012). It is a major cause of end-stage renal disease. Long term exposure has been associated with bone diseases (such as osteomalacia and osteoporosis), alteration in lung function, lung cancer, prostate cancer and renal cancer in exposed workers (Nair et al., 2013; Verougstraetea et al., 2003). Much of Cd toxicity is attributable to its ability to substitute for Zn in biological reactions (Golovine et al., 2010). Zn is an essential metal required for the synthesis of DNA, RNA and protein as well as for the enzymatic activity of Zn-containing enzymes. Zn has been shown to reduce the carcinogenic effects of Cd in animal models (Saïd et al., 2010). This supports the antagonistic relationship between Cd and Zn. Cadmium is not known to offer any biological benefit to man. In contrast, following an array of experimental and epidemiological evidence, its carcinogenicity to man has been proven (Table 1). It inhibits apoptosis (Templeton and Liu 2010), induces single-strand DNA breaks (Nemmiche et al., 2011), exerts inhibitory effects on DNA repair system (Schwerdtle et al., 2010), disrupts cell adhesion and activates proto-oncogenesis (Waisberg et al., 2003).

Cobalt

Vitamin B12, also known as cyanocobalamin is a cobalt-containing compound in human and animal body necessary for tissue growth, and employed medically in the treatment of pernicious anaemia. The deficiency of Vitamin B12 results in defective DNA synthesis thus, cobalt is an essential trace element in man and animals. It is found in nature usually together with other metals like arsenic and nickel. Environmental contamination results from its recovery from the smelting of arsenical ores of lead, copper and nickel. Commercial use of cobalt include in magnets, alloys, cobalt steels and as binder for tungsten carbide cutting tools. Vitallium is a cobalt-chromium-molybdenum-nickel alloy employed as an implant orthopaedic surgery. The indication of possible carcinogenicity of cobalt arises from the production of fibrosarcoma, rhabdomyosarcoma and other sarcomas at injection sites following the injection of cobalt metal powders (Klatka et al., 2011). Following sufficient epidemiological data, IARC classified cobalt and its compounds as a possible carcinogen.

Iron

It is the most abundant metal in the earth's crust with soil level ranging from 7 g/kg to 550 g/kg. In fresh water, iron content depends on location and may vary from 0.01 to 1mg/L, with low values in rural areas, intermediate in urban and highest in areas close to iron foundries. The daily intake of iron in diet is about 9-35 mg, and this may also vary depending on the source of the diet as agricultural products mining areas contain higher iron contents. Occupational exposure to iron compounds mainly the oxides is common in mining, iron and steel foundry work and in arc welding. Iron has been shown to be mutagenic, and carcinogenic at high concentration (Prá et al., 2012). IARC found acceptable evidence for local sarcoma attributable to iron carbohydrate complexes, especially iron dextran, in several animal species.

Lead

It is extracted from several minerals, the most abundant being galena, containing the sulphide. Lead is also found as carbonate, sulphate, phosphate and chloride. The major consumer of lead is the automobile industry especially lead battery and alkyllead gasoline additive production. Other uses are; in alloys, paints printing, cables, pipes and glazes. In soil, lead is present at the range of 2-200 mg/kg, in drinking water, 0.01 mg/L and as high as 3 mg/L in lead pipes. In air, usually, less thn 0.1 $\mu g/m^3$ which may increase to 10 $\mu g/m^3$ or higher levels in urban areas with heavy traffic. Exposure occurs through contaminated food and water. Occupationally, exposure occurs in lead smelting and refining, in lead battery manufacture and in many other industrial processes. Lead poisoning is still common usually resulting from lead fume in refining operations, lead oxide in battery manufacture and tetraethyllead as gasoline additive (Nriagu et al., 2008). Lead is known to be toxic to the peripheral and central nervous system, reproductive system, the immune system, kidney and liver (Chandran and Cataldo, 2010).

Recent experimental and epidemiological studies have provided sufficient evidence that inorganic lead compounds are associated with increased risk of carcinogenesis. Plausible mechanism of lead carcinogenicity include direct DNA damage, inhibition of DNA synthesis or repair, clastogenicity, generation of reactive oxygen species leading to oxidative DNA damage (Chandran and Cataldo, 2010). Lead can also substitute for zinc in several enzymes that function as

transcriptional regulators reducing the binding of these proteins to recognition elements in genomic DNA which suggests an epigenetic involvement of lead in altered gene expression (Witkiewicz-Kucharczyk and Bal, 2005). Currently, lead and its inorganic compounds are classified by IARC in group 2B on the basis of sufficient evidence for carcinogenicity in experimental animals where it has been associated with renal and brain tumors.

Mercury

It is found in nature as sulphide in low concentrations in the earth's crust except for rich focal deposits where it may also be present in metallic form. It is fairly volatile element, released into the atmosphere and deposited again to form natural global cycle. Human exposure comes through activity in chloralkali industry, electrical and paint industries, measuring instrument, agriculture, dentistry and chemical industry. Soil level is about 50 μg/kg. There is usually low uptake by plants. Uncomplicated water has very low level while levels in the air in urban areas are around 50ng/m^3 (Boening, 2005). Mercury is currently grouped as a Class 3 carcinogen.

Nickel

Although evidence of beneficial roles of Ni in humans is not available, proven beneficial roles of nickel Ni-containing enzymes found in plants and microorganisms led to its classification as a trace element that is 'probably' essential by WHO in 1996. Ni compound have been shown to be cancer-inducing in human through a number of mechanisms including inhibition of intercellular communication (Vinken et al., 2010), immortalization of fibroblasts and epithelial cells (Yasaei et al., 2013), the induction of DNA deletions and aberrations (Carmona et al., 2011), production of DNA-protein cross-links, oxidative damage, inhibition of nucleotide excision repair (Goodman et al., 2011) and an increase in DNA methylation leading to inactivation of gene expression (Chen et al., 2010).

Implicating Heavy Metal Pollution in Cancer

Although, current evidence in literature may not be sufficient to establish causal-effect association between chronic environmental exposure to heavy metals and cancer, researchers globally continue to find a strong association between the two. Jaafar and co-workers reported cases of 3 patients who developed symptoms of skin cancer following chronic exposure to arsenic-contaminated well water in Malaysia (Jaafar et al., 1993). In an American study, Carrigan and co-workers found a strong association between pancreatic cancer and chromium, selenium and molybdenum (Carrigan et al., 2007). The authors quantify the heavy metal composition of pancreatic juice collected from patients with pancreatic cancer with no occupational exposures. In a similar study in an Egyptian population, serum cadmium levels were assessed in 31 newly diagnosed cancer patients. Compared to controls, the study found high cadmium levels in pancreatic patients who were exposed to the cadmium-polluted environment (Kriegel et al., 2006). A more recent study in Egypt concluded a causal association between high levels of cadmium and copper in the body and breast cancer (El-Harouny et al., 2011).

In a case-control study to investigate the role of environmental pollutants in the aetiology of lung cancer in Pennsylvania, residents who live close to a zinc smelter and a large steel manufacturing plant were studied. The risks for lung cancer were found to be double in these residents due to higher pollution of soil by arsenic and cadmium (Brown et al., 1984). In a Belgian study to assess possible association between environmental cadmium exposure and cancer, the authors recruited participants who resided close to zinc smelters and control groups with less exposure. Cadmium concentrations in the urine and the garden soils of participants were correlated. The incidence of cancer was followed for a period of over 17 years. The authors reported fourfold lung cancer cases among the high exposed population even though smoking habit was same in the two groups. The occurrence of other cancer types was also higher in the high-exposed group (Nawrot et al., 2006).

An ecological study that examined high nasal cancer incidence based on data from cancer registry for Rotorua, a New Zealand city where a number of mercury-generating geothermal plants are located was conducted. Compared to data from other parts of the country, the authors believe the study data was not sufficient to ascertain causal association but suggested possible roles of mercury exposure in the elevated rate of nasal cancer incidences (Bates et al., 1998). Chiang and co-workers (2010) correlated the incidence of oral cancer in Central and Eastern Taiwan with the levels of soil pollutions by heavy metals. In a similar study, following the discovery of the highest number of Taiwanese cases of oral cancer in Changhua whose exposure to the known risk factors (smoking and betel quid chewing) were not different from the rest of the country, Su and co-workers (2010a) conducted investigations for additional risk factors. The authors found high soil levels of heavy metal contaminants in the areas concerned. Epidemiological trends, using age-period-cohort models further isolated a decade old environmental exposure to heavy metals as a possible factor for the higher incidence of oral cancer in Changhua. Similarly, an observational study in Taiwan sought to investigate possible involvement of specific heavy metals as risk factors of oral cancer in addition to cigarette smoking and betel quid chewing. Using spatial analysis and Geographic Information System, the authors analysed 22,083 patients diagnosed with oral cancer over a period of 20 years alongside nation-wide levels of heavy metals in the soil. The study found geographical relationship between the incidence rate of oral cancer and the corresponding soil levels of arsenic and nickel in the patients' residential areas (Su et al., 2010b).

In Argentina, dose-response relationship between arsenic and bladder cancer was investigated with natural arsenic-contaminated drinking water following a Taiwanese study suggesting such. The authors reported consistently higher mortality from bladder cancer in areas exposed to arsenic in the drinking water (Hopenhayn-Rich et al., 1996). In a recent epidemiological study to

investigate risk factors in a northern Indian population where high prevalence of cancer was observed, Thakur and colleagues found among other factors, high concentration of heavy metals such as arsenic, cadmium, cadmium, chromium, selenium and mercury in the drinking water of the rural agricultural community. Compared to a control population, the study attributed high levels of pesticides and heavy metals in the food and water of the high-cancer incidence population (Thakur et al., 2008).

In a Chinese study to investigate probable association between long-term environmental exposure to lead and cadmium and cancer incidence, the authors found higher incidences of gastrointestinal and lung cancers among populations exposed to contaminated river over 30 years. In this ecologic study, the levels of heavy metals were determined in the drinking water and agricultural products consumed by the residents. Blood levels were then determined and compared to controls. Cancer mortality among the residents of the contaminated areas was found to correlate with cadmium and lead levels (Wang et al., 2011).

Environmental Pollution and Human Exposure to Heavy Metals in Sub-Saharan Africa

Most heavy metals are found naturally in the environment. Human exposure to them derives from drinking water, air and food. The interest in heavy metals are based on the fact that exposure to them is modifiable, being trace elements. Table 2 shows average exposure to some of the heavy metals.

Heavy metal pollution occurs when humans are exposed to heavy metals beyond certain limit. This has become an international medical issue since the Minamata metal poisoning in the 1960s which claimed the life of thousands of people in Japan (Wilcox et al., 2008). Heavy metals pollute the environment naturally and through anthropogenic activities. Concentrations of heavy metals in water are generally low. Marine products including fish, animals and plant species are sources of food for many people. Polluted sea water often leads to accumulation of heavy metals in sea products which portends potential health risk to man. The United Nations' Group of Experts on the Scientific Aspects of Marine Pollution (GESAM, 2000) defines marine pollution as the "introduction by man directly or indirectly of substances or energy into the marine environment (including estuaries) resulting in such deleterious effects as harm to living resources, hazards to human health, hindrances to marine activities including fishing, impairment of quality for use of sea water and reduction of amenities".

Table 2. United States Environmental Protection Agency (USEPA) Maximum Permissible Heavy Metal Contamination Levels in Air, Soil and Water (adapted from Duruibe and Co-workers (2007))

Heavy metal	Air ($\mu g/m^3$)	Soil (mg/kg)	Drinking water ($\mu g/L$)	Water supporting aquatic life ($\mu g/L$)
Arsenic	-	-	10	-
Cadmium	100-200	85	5	8
Calcium	5000	Tolerable	50000	>50000
Lead	-	420	0	5.8
Mercury	-	1	2	50
Silver	10	-	-	100
Zinc	-	-	10	-

Estimates of heavy metal exposure are often determined in the biological specimens including blood (whole blood, plasma, serum, and erythrocytes), urine, hair and nails. Interpreting the results presented must take into consideration the sample measured. Short term exposures are easily determined through plasma and serum measurements while measures in erythrocytes represent long-term exposures. Toenails are often preferable to other biological samples (eg urine, blood) for the measurement of heavy metal levels because they reflect longer-term exposure. Good correlations have been reported in studies using toenails between toenail measures and selenium intake (Lemos and de Carvalho, 2010; Kim and Kim, 2011).

Human exposure to heavy metals in the SSA has become a major health risk. This has received the attention of national and international environmentalists and concerned health bodies. Rapid population growth, increasing urbanization and the increasing appearance of slums and townships as a consequence of poor planning coupled with increasing industrial activities have resulted in overwhelming production of waste without adequate disposal systems. It has been reported by the United Nations Center for Human Settlements that only one-third of the solid waste generated in urban areas across the continent is collected and less than 2% recycled (Scheinberg et al., 2011). Increasing mining activities, use of lead gasoline, fugitive dusts, indiscriminate dumping and burning of toxic waste, concentration of production factories within residential areas and poor pollution legislations have contributed to unprecedented heavy metal pollution in the last decades. According to a WHO study on the environmental burden of disease, 25% of the total burden of disease and 2.97 million human deaths are yearly attributed to environmental risk factors (Oberg et al., 2010). Large deposits of mineral resources are found in Africa and mining activities have increased with poor environmental regulations and/or and compliance (Table 3). Thus heavy metals have constituted agents of toxic pollution of water, air, soil and food products.

In a United Nation Environment Programme (UNEP) report on environmental assessment in Ogoniland, southeastern Nigeria, released in 2011, drinking water, air and agricultural soil in 10 communities contain over 900 times permissible levels of hydrocarbon and heavy metals. The report acknowledges that recovery after extensive compliance with recommendations may take 30 years. A cursory look at published research work shows that heavy metal pollution is a continental trend in SSA (Table 4).

These levels of heavy metal pollution of the environment translate to high human exposure with attendant deleterious effects. For instance, a study in the Democratic Republic of Congo showed up to 43-fold

increase in the urinary concentration of cadmium, cobalt, lead and uranium in human subjects including children living in mining areas compared to controls (Banza et al., 2009). In a more recent study by Ibeto and Okoye (2010) in Eastern Nigeria, blood samples from 240 residents of an urban area including children and pregnant women were analysed for heavy metals. The results showed values significantly higher that the WHO recommended limits for nickel, manganese and chromium. The sampled population who are non-miners demonstrated the prevalence heavy metal pollutions in Nigeria among ordinary people. In a similar study in South Africa, the extent of heavy metal pollution was demonstrated by determining the concentrations of heavy metals in maternal and umbilical cord blood from residents of selected areas (Rollin et al., 2009). Although levels of some measured metals differ by site indicating different environmental pollution levels, unacceptably high levels of mercury, cadmium, lead and selenium were found. All analysed heavy metals were found in the umbilical cord whole blood samples. This further demonstrates the risks of heavy metal pollution, not only to adults, but unborn babies.

Heavy Metal Pollution and Cancer in Sub-Saharan Africa

As far back as 1967, Schulz (1967) had reported nine skin cancer cases in Pretoria, South Africa, traceable to arsenic exposure. Of the nine patients, five had taken arsenical preparations for the treatment of epilepsy, two had treatment history with Fowler's solution (an arsenic-containing medication), for chronic dermatoses, one

Table 3. Mineral Mining in Sub-Saharan Africa

Countries	Metals deposits and/or mined
Angola	Diamond, gold, iron ore, petroleum
Benin	Chromium, iron ore, petroleum
Botswana	Coal , copper, nickel
Burkina Faso	Bauxite, copper, gold, limestone, manganese, uranium
Burundi	Copper, gold, nickel, platinum
Central African Republic	Diamond
Chad	Uranium
Congo (Brazzaville)	Diamond, petroleum
Congo DRC	Bauxite, coal, cobalt, copper, gold, manganese, petroleum, silver, tin, zinc
Cote d'Ivoire	Diamond, manganese, petroleum
Equitorial guinea	Petroleum
Eritrea	Gold, potash, zinc
Ethiopia	Copper, gold, platinum
Garbon	Iron ore, manganese, petroleum, uranium
Ghana	Bauxite, gold, manganese, petroleum
Guinea	Bauxite, gold, iron ore, uranium
Madagascar	Bauxite, chromium, coal, graphite, manganese
Malawi	Limestone
Mali	Bauxite, gold, iron ore, manganese, phosphates
Mauritania	Copper, gypsum, iron ore
Mazambique	Coal, titanium
Namibia	Copper, diamond, gold, lead, uranium, zinc
Niger	Coal, iron ore, uranium
Nigeria	Coal, columbite, iron ore, petroleum, tin
Rwanda	Gold, tin ore
Senegal	Iron ore, phosphates
Sierra Leone	Bauxite, diamond, iron ore
Somalia	Uranium
South Africa	Chromium, coal, gold, iron ore, manganese, platinum, uranium
Sudan	Chromium, copper, iron ore, petroleum
Swaziland	Asbestos, coal
Tanzania	Diamonds, iron ore, phosphate, tin
Togo	Limestone, phosphate
Uganda	Cobalt, copper
Zambia	Coal, cobalt, copper, lead, zinc
Zimbabwe	Asbestos, chromium, coal, gold platinum

Table 4. Heavy Metal Pollution in Sub-Saharan Africa

Country/region	Pollution type/source	Pb	Cd	Hg	Cu	Co	Zn	Cr	Ni	As	References
Cameroon/ Yaounde											
	River sediment	20.3-249	2.8-15.6		42.8-142		26.8-341	94.7-199	2.68-32.7		Ekengele et al., 2008
Ethiopia	Vegetable		0.345				130	24.11			Prabu 2009
Ghana	Fish		0.028	0.006						2.321	Essumang 2009
Ghana/ Iture	Water	0.075	0.041				2.45				Fianko et al., 2007
Ghana/ Kumasi	Agricultural soil	54.6	2.87		1631.67		2606				Odai et al., 2008
Ghana/ Tarkwa	Water			2.3						1.3	Kwadwo et al., 2007
Kenya	Water	0.496	0.01					1.95			Mireji et al., 2008
Kenya/Nairobi	Soil	264	40	18.6	105		462	157			UNEP 2007
Namibia	Sediment				10500		205		1950		Taylor and Kesterton 2002
Nigeria	Herbal medicines*	27	4.75			97.5	25.5		78		Obi et al., 2006
Nigeria/Calabar	River sediment	20	0.2		64	15	184	65	67		Ntekim et al., 1993
Nigeria/Ibadan	River surface water	0.046	0.0044		0.0033	0.018	0.14		0.0031		Mombeshora et al et al., 1998
Nigeria/ Kano	Vegetable	13.19	0.735					12.89			Abdullahi et al., 2007
Nigeria/Lagos	Soil	67.5-426	1.61-5.31								Fakayode and Onianwa 2002
Nigeria/ Niger Delta	Water	0.025-0.064	0.01-0.11					0.03-0.081	0.03-0.09		Emoyan et al., 2006
Nigeria/Niger Delta	Fish	0.3	0.03					0.53	0.21		Oze et al., 2006
Nigeria/Ogun	Fish	3.4	2.1		5		20.35			2.3	Farombi et al., 2007
Nigeria/Osogbo	Soil	92.07	3.6		37.9		71.9		17.3		Fakayode and olu-owolabi 2003
South Africa	Water	16.3	72		42.6		27.6				Fatoki and Mathamatha 2001
Tanzania	Vegetable	4.9	0.3								Bahemuka and Mubofu 1999
Tanzania, along lake Victoria											
	Water sediment	54.6	7	2.8	26.1		83.7	12.9			Kishe and Machiwa 2003
Uganda	Vegetable	18.7	1.87								Nabulo et a. 2006
Uganda, along Lake Victoria											
	Water	1.44	0.02			0.16		0.02	0.13		Muwanga and Barifaijo 2006
Zambia	Sediment				12855±1445	1030±58					Pettersson and Ingri 2001
Zambia	Sediment	9-75	0.8				125	130	220		Von der Heyden and New 2004
Zimbabwe	Water	1.02	0.12					2.48	2.37		Meck et a. 2006
Zimbabwe Harare	Vegetable	6.77	3.68	0.05	111		221	16.1			Muchuweti et al., 2006

*Values are expressed in mg/5mL digested sample. All other values in the table are expressed as ppm (mg/L for water and mg/kg for others)

had consumed arsenic-polluted tap water and the last inhaled arsenic fumes while working in a copper mine in Zimbabwe. Similarly, a geographical pathology survey of a large area in Central Africa was conducted comparing the effects of the consumption of heavy metal-contaminated indigenous distilled spirits. The study observed spatial association between oesophageal cancer and the local gin consumption (McGlashan, 1969).

In a study conducted among miners in South Africa, an association between lung cancer and exposure to gold mining dust was confirmed (Hnizdo and Sluis-Cremern, 1991). The study assessed 2209 white South African gold miners whose exposure to mining dust started in 1936-1943, and were selected in 1968-1971 when they were aged 45-43. A further mortality follow up was done from 1968-1986 to conclude the findings. In a similar study involving black miners in southern Africa recruited from Lesotho, South Africa and Mozambique, spanning 1964-1996, mining activity and exposure to mining dust were closely associated with lung, liver, esophageal and lymphatic system cancer (McGlashan et al., 2003).

In a 1997 nested control study for lung cancer on a cohort study of 2260 South African gold miners it was concluded that subjects with high dust exposure are at greater risk of lung cancer (Hnidzo et al., 1997). Compared to controls, people exposed to nickel during mining in Zimbabwe had greater risk of lung cancer (Parkin et al., 1994). In a study carried out by Obiri and co-workers (2006), cancer risks were evaluated in a Ghanaian population who consume food crops cultivated in mining communities, where agricultural soil has shown high levels of heavy metals. This population showed 10,000-fold likelihood to suffer from cancer and/or cancer related diseases compared to controls. Similarly, Alatise and Schrauzer (2010) demonstrated a correlation between body levels of lead and volumes of tumor in breast cancer patients in a Nigerian population. In the said study, lead and other heavy metals including cadmium, mercury, chromium, tin and arsenic were detected in high concentrations in the blood and scalp hair of newly diagnosed breast cancer patients, all with infiltrating ductal carcinoma. By determining the blood, hair and tumour biopsy tissue levels of lead and selenium, the authors also demonstrated evidence for interactions between lead and selenium. While high levels of lead were directly correlated with tumour volumes in accordance with the known tumour-inducing effects of lead, selenium levels were inversely correlated with tumour volume which is consistent with the anti-proliferative effects of selenium.

Discussion

Although epidemiological evidence for outright implication of heavy metal exposure in cancer epidemiology in SSA seem to be scanty at the moment, studies in other continents and regions have established such association (Jemal et al., 2002; Boffetta, 2004; Khlifi and Hamza-Chaffai, 2010). The extent of heavy metal pollution in SSA is very high. This is due to the various anthropogenic activities in the region especially mining. SSA has vast mineral deposits which has attracted multinational companies in the mining industries. However, health regulations concerning these exploratory activities are weak and poorly enforced. Another major challenge is the increasing appearance of slums and concentrating urban areas. The consequence of this is high generation of wastes, high industrial activity and increased use of fume-generating equipment. All these contribute immensely to the heavy metal pollution as seen from the reviewed literature above. The planting of edible fruits and other agricultural products in urban areas is also very common in SSA. Most of these locations are in close proximity with roadside mechanic shops, petrochemical and other mining industries. The consumption of such food products contribute to health hazards in SSA. Government and health agencies need to pay more attention to the environment and the environmental pollution arising from these anthropogenic activities. Regulatory authorities in SSA also need to be more stringent in enforcing existing rules in other to protect the citizens from avoidable heavy metal contamination. While there are sufficient evidence globally linking heavy metal pollution to cancer epidemic, research in this area is sparse in SSA. Government attention on cancer in SSA is also inadequate. This is due primarily to the burden of communicable diseases including poliomyelitis and meningitis most of which are rare in other continents.

In conclusion, although epidemiological evidence is limited due to low study power, high exposure to heavy metals in SSA may impact negatively on cancer epidemiology. With research evidence establishing association between the exposure (occupational and environmental) to heavy metal with increasing cancer epidemiology, the likelihood that heavy metal pollution in SSA play significant role in cancer epidemiology is high. As a preventable phenomenon, concerned and interested parties must expend more resources and attention in this regard. Legislation and regulatory activity as they pertain to mining and environmental degradation should be enhanced and enforced.

Acknowledgements

Prof Orish Ebere Orisakwe was a recipient of short term fellowship sponsored by KIC Travel Grant of the South African National Research Foundation and Tygerberg Poison Information Centre, Division of Pharmacology, Dept. of Medicine, Faculty of Health Sciences, Stellenbosch University.

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