Lead and Arsenic Poisining through Fish and Chicken

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Abstract: General population exposure to arsenic and lead is mainly *via* intake of food and drinking water. Food is the most important source as fish meat, chicken meat and hens eggs but in some areas, arsenic in drinking water is a significant source of exposure to inorganic. Lead poisoning and arsenic exposure have become serious health risks in the past few decades. Lead poisoning or plubism is called toxicological condition, in which its exposure can cause either acute or chronic poisoning. Interferes with a number of body functions primarily affecting the central nervous, brain damage hematopoietic, hepatic and renal system producing serious disorders, high blood pressure, miscarriages, and behavioral problems in children. Contaminated soils such as mine-tailings are also a potential source of arsenic exposure. Absorption of arsenic in inhaled airborne particles is highly dependent on the solubility and the size of particles. Soluble arsenic compounds are easily absorbed from the gastrointestinal tract. However, inorganic arsenic is extensively methylated in humans and the metabolites are excreted in the urine. Arsenic (or metabolites) concentrations in blood, hair, nails and urine have been used as biomarkers of exposure. Speciated metabolites in urine expressed as either inorganic arsenic or the sum of metabolites (inorganic arsenic + MMA + DMA) is generally the best estimate of recent arsenic dose. Fish may accumulate toxinswhich can pass to human through its meat consumption. Accumulation of metals in fishdepends on concentrations of metals in water and food organism, on physiochemicalfactors and exposure duration. Heavy metals pollution in chickens is a problemdue to the health risk to human as a result toconsumptionits meat and eggs where inorganic arsenic is accumulated in the chicken breast meat and lead is deposited into the hen's eggs. It causes economic loses in poultry industry. It was found that arsenic toxicity results in many adverse conditions specially in broilers and broiler breeders chickens in form of increase mortalities and decrease in egg production.
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Introduction:

Lead:

The symptoms of acute lead poisoning are headache, irritability, abdominal pain and various symptoms related to the nervous system. Lead encephalopathyis characterized by sleeplessness and restlessness. Children may be affected by behavioural disturbances, learning and concentration difficulties. In severe cases of lead encephalopathy, the affected person may suffer from acute psychosis, confusion and reduced consciousness **(1-2)**.

People who have been exposed to lead for a long time may suffer from memory deterioration, prolonged reaction time and reduced ability to understand. Individuals with average blood lead levels under 3 mol/l may show signs of peripheral nerve symptoms with reduced nerve conduction velocity and reduced dermal sensibility. If the neuropathy is severe the lesion may be permanent. The classical picture includes a dark blue lead sulphide line at the gingival margin. In less serious cases, the most obvious sign of lead poisoning is disturbance of haemoglobin synthesis, and long-term lead exposure may lead to anaemia **(3-6).**

Recent research has shown that long-term low-level lead exposure in children may also lead to diminished intellectual capacity **(7-9)**.

Arsenic:

It is a widely distributed metalloid, occurring in rock, soil, water and air. Inorganic arsenic is present in groundwater used for drinking in several countries all over the world (*e.g.* Bangladesh, Chile and China), whereas organic arsenic compounds (such as arsenobetaine) are primarily found in fish, which thus may give rise to human exposure **(10-12)**.

Smelting of non-ferrous metals and the production of energy from fossil fuel are the two major industrial processes that lead to arsenic contamination of air, water and soil, smelting activities being the largest single anthropogenic source of atmospheric pollution. Other sources of contamination

are the manufacture and use of arsenical pesticides and wood preservatives **(13-16).**

The working group of the EU DG Environment concluded that there were large reductions in the emissions of arsenic to air in several member countries of the European Union in the 1980s. In 1990, the total emissions of arsenic to the air in the member states were estimated to be 575 tonnes **(17).**

In 1996, the estimated total releases of arsenic to the air in the UK were50 tonnes**. (18).**

Concentrations in air in rural areas range from 1 to 4 ng/m3, where as concentrations in cities may be as high as 200 ng/m3. Much higher concentrations (1000 ng/m3) have been measured near industrial sources **(19).**

Water concentrations are usually 10 g/l, although higher concentrations may occur near anthropogenic sources. Levels in soils usually range from1 to 40 mg/kg, but pesticide application and waste disposal can result in much higher concentrations **(22-21)**.

General population exposure to arsenic is mainly *via* intake of food and drinking water. Food is the most important source, but in some areas, arsenic in drinking water is a significant source of exposure to inorganic arsenic. Contaminated soils such as mine-tailings are also a potential source of arsenic exposure.

Absorption of arsenic in inhaled airborne particles is highly dependent on the solubility and the size of particles **(23-24)**.

Soluble arsenic compounds are easily absorbed from the gastrointestinal tract. However, inorganic arsenic is extensively methylated in humans and the metabolites are excreted in the urine.

Arsenic (or metabolites) concentrations in blood, hair, nails and urine have been used as biomarkers of exposure.

Arsenic in hair and nails can be useful indicators of past arsenic exposure, if care is taken to avoid external arsenic contamination of the samples. Speciated metabolites in urine expressed as either inorganic arsenic or the sum of metabolites (inorganic arsenic + MMA + DMA) is generally the best estimate of recent arsenic dose. However, consumption of certain seafood may confound estimation of inorganic arsenic exposure, and should thus be avoided before urine sampling **(25-27).**

In Fish:

The distribution of metals varies between fish species, depending on age, season, development status and other physiological factors **(28)**. Metals accumulate mainly in kidney, liver and intestine epithelium. Fish muscles contain low levels of metals, but are often examined for metal content due to their use for human consumption **(29)**. Analytical results were recorded for concentrations of arsenic, cadmium, copper, lead, mercury, selenium and zinc in freshwater fish collected in 1984 and temporal and geographic trends were evaluated by comparison with earlier finding **(30).**

Lead can cause deficits or decreases in survival, growth rates, development and metabolism **(31)**.

In Chickens:

The toxicity of these metals has two main aspects, the fact they have no known metabolic function but when present in the body disrupt normal cellular processes. Arsenic leading to toxicity and the potential of these heavy metals to accumulate in biological tissues, a process known as bio accumulation **(32)**. This occurs because the metals are stored in particular organs, for example the liver and the kidney **(33)**. Arsenic-based drugs were approved to prevent parasites in the chicken's gut. Poultry producers would routinely add roxarsone (inorganic arsenic, a known carcinogen), to their chickens feed, because arsenic is believed to speed the growth of chickens, and to give chicken meat a pink color that's pleasing to the shopper's eye. Inorganic arsenic accumulated in the chicken breast meat **(34 -35)**.

It was found that experimental exposure to heavy metal such as arsenic and lead in concentration of 0-30 ppm results in decrease broiler chickens feed intake and body weight after 56 days period of exposures **(36)**.

Chickens that develop acute lead toxicity or poisoning will often present with sudden onset of muscle weakness, loss of appetite, marked weight loss, ataxia, drop in egg production and severe anemia. Chronic toxicity: Chronic lead toxicity may eventually result in degeneration of motor nerves in the chicken's spinal cord and axonal loss in the peripheral nerves. Muscle atrophy and myodegeneration are often also present.

Moreover experimental exposure of broiler breeder to arsenic (0.8 ppm) and lead (6.7 ppm) results in decrease egg production, egg weight and increase percentage of embryonic mortalities **(37)**.

References

1. *Department of Epidemiology*, Lars Järup, Hazards of heavy metal contamination 2017.
2. Staessen JA, Roels HA, Emelianov D, Kuznetsova T, Thijs L, Vangronsveld J *et al.* Environmental exposure to cadmium, forearm bone density, and risk of fractures: prospective population study. Public Health and Environmental Exposure to Cadmium (PheeCad) Study Group. *Lancet*1999; 353: 1140–4.
3. Alfven T, Elinder CG, Carlsson MD, Grubb A, Hellstrom L, Persson B *et al.* Low-level cadmium exposure and osteoporosis. *J Bone Miner Res* 2000; 15: 1579–86.

4. Nordberg G, Jin T, Bernard A, Fierens S, Buchet JP, Ye T, Kong Q, Wang H. Low bone density and renal dysfunction following environmental cadmium exposure in China. *Ambio*2002; 6:478–81.
5. Nishijo M, Nakagawa H, Morikawa Y, Tabata M, Senma M, Miura K *et al.* Mortality of inhabitants in an area polluted by cadmium: 15 year follow up. *Occup Environ Med* 1995; 52: 181–4.
6. IARC. Cadmium and cadmium compounds. In: *Beryllium, Cadmium, Mercury and Exposure in the Glass Manufacturing Industry*. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. 58. Lyon: International Agency for Research on Cancer, 1993; 119–237.
7. Kolonel LN. Association of cadmium with renal cancer. *Cancer* 1976; 37: 1782–7.
8. Mandel JS, McLaughlin JK, Schlehofer B, Mellemgaard A, Helmert U, Lindblad P, McCredie M, Adami HO. International renal-cell cancer study. IV. Occupation. *Int J Cancer* 1995; 61: 601–5.
9. Pesch B, Haerting J, Ranft U, Klimpel A, Oelschlagel B, Schill W. Occupational risk factors for renal cell carcinoma: agent-specific results from a case-control study in Germany. MURC Study Group. Multicentre urothelial and renal cancer study. *Int J Epidemiol*2000; 29: 1014–24.
10. WHO. *Inorganic Mercury.* Environmental Health Criteria, vol. 118. Geneva: World Health Organization, 1991.
11. WHO. *Methyl Mercury.* Environmental Health Criteria, vol. 101. Geneva: World Health Organization, 1990.
12. Sallsten G, Thoren J, Barregard L, Schutz A, Skarping G. Long-term use of nicotine chewinggum and mercury exposure from dental amalgam fillings. *J Dent Res* 1996; 75: 594–8.
13. Lindh U, Hudecek R, Danersund A, Eriksson S, Lindvall A. Removal of dental amalgam and other metal alloys supported by antioxidant therapy alleviates symptoms and improves quality of life in patients with amalgam-associated ill health. *Neuroendocrinol Lett*2002; 23:459–82.
14. Langworth S, Bjorkman L, Elinder CG, Jarup L, Savlin P. Multidisciplinary examination of patients with illness attributed to dental fillings. *J Oral Rehabil*2002; 29: 705–13.
15. Weiss B, Clarkson TW, Simon W. Silent latency periods in methylmercury poisoning and in neurodegenerative disease. *Environ Health Perspect*2002; 110 (Suppl 5): 851–4.
16. Salonen JT, Seppanen K, Nyyssonen K, Korpela H, Kauhanen J, Kantola M, Tuomilehto J, Esterbauer H, Tatzber F, Salonen R. Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnishmen. *Circulation* 1995; 91: 645–55.
17. Guallar E, Sanz-Gallardo MI, van't Veer P, Bode P, Aro A, Gomez-Aracena J, Kark JD, Riemersma RA, Martin-Moreno JM, Kok FJ; Heavy Metals and Myocardial Infarction Study Group. Mercury, fish oils, and the risk of myocardial infarction. *N Engl J Med* 2002; 347:1747–54.
18. Yoshizawa K, Rimm EB, Morris JS, Spate VL, Hsieh CC, Spiegelman D, Stampfer MJ, Willett WC. Mercury and the risk of coronary heart disease in men. *N Engl J Med* 2002; 347:1755–60.
19. Mortada WI, Sobh MA, El-Defrawy MM, Farahat SE. Study of lead exposure from automobile exhaust as a risk for nephrotoxicity among traffic policemen. *Am J Nephrol*2001; 21: 274–9.
20. Elliott P, Arnold R, Barltrop D, Thornton I, House IM, Henry JA. Clinical lead poisoning in England: an analysis of routine sources of data. *Occup Environ Med* 1999; 56: 820–4.
21. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. *Brain* 2003; 126: 5–19.
22. Steenland K, Boffetta P. Lead and cancer in humans: where are we now? *Am J Ind Med* 2000;38: 295–9.
23. WHO. *Arsenic and Arsenic Compounds.* Environmental Health Criteria, vol. 224. Geneva: World Health Organization, 2001.
24. Chilvers DC, Peterson PJ. Global cycling of arsenic. In: Hutchinson TC, Meema KM (eds) *Lead, Mercury, Cadmium and Arsenic in the Environment*. Chichester: John Wiley & Sons 1987; 279–303.
25. DG Environment. Ambient air pollution by As, Cd and Ni compounds. Position paper, Final version, October 2000. Brussels: European Commission DG Environment.
26. Nriagu JO. History of global metal pollution. *Science* 1996; 272: 223–4.
27. Annest JL. Trends in the blood level leads of the US population: The Second National Healthand Nutrition Examination Survey (NHANES II) 1976–1980. In: R.
28. Mendil, D., Demirci, Z., Tuzen, M., Soylak, M., (2010) Seasonal investigation of trace element contents in commercially valuable fish species

from the Black Sea, Turkey. Food and Chemical Toxicology, 48, 865-870.
29. Lidwin-Kaźmierkiwicz, M., Pokorska, K., Protasowicki, M., Rajkowska, M., Wechterowicz, Z., (2009) Content of selected essentials and toxic metals in meat of freshwater fish from West Pomeranian, Poland. Polish Journal of Food Nutrition and Science, 59(3), 219-224.
30. Schmitt C.J. and W. G. Brumbaugh (1990): National Contaminant Biomonitoring Program: Concentrations of Arsenic, Cadmium, Copper, Lead, Mercury, Selenium, and Zinc in U.S. Freshwater Fish, 197 & -1984. Arch. Environ. CO (lt~. Toxico!. 19,731-747 (1990).
31. Yılmaz, A.B., Sangün, M.K., Yağlıoğlu, D., Turan C., (2010) Metals (major, essential to non-essential) composition of the different tissues of three demersal fish from İskenderun Bay, Turkey. Food Chemistry, 123, 410-415.
32. Mariam, I.S. and Nagre, S., (2004). Distribution of some trace and macro minerals in beef, mutton and poultry. International Journal of Agric and Biology, 6:816-82.
33. Demirezen, O. and Uruc, K., (2006). Comparative study of trace elements in certain fish, meat and meat products. Food chemistry, 32: 215-222
34. Schmidt, C.W. (2013): Arsenical Association: Inorgang Arsenic may accumulate in the meat of the treated chicken *Environmental Health Perspectives*.
35. Nachman Keeve E., Patrick A. Baron, Georg Raber, Kevin A. Francesconi, Ana Navas-Acien and David C. Love (2013) *Arsenic in Chicken Meat, Environmental Health Perspectives May 2013*.
36. Okeke O. R., I. I. Ujah, P. A. C.Okoye., V. I. E Ajiwe and C. P. Eze (2015): Effect of Different Levels of Cadmium, Lead and Arsenic on the Growth Performance of Broiler and Layer Chickens. *IOSR Journal of Applied Chemistry (IOSR-JAC 8, 1 Ver. II. (Jan. 2015), PP 57-59*.
37. Vodela, J. K., J. A. Renden, S. D. Lenz, W. H. McElhenney, and B. W. Kemppainen, 1997. Drinking water contaminants (arsenic, cadmium, lead, benzene, and trichloroethylene). 1. Interaction of contaminants with nutritional status on general performance and immune function in broiler chickens. Poultry Sci. 76:1474–1492.

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