Health risk assessment
in the Lower Watut River

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Abstract

This assessment was undertaken to evaluate the health risks from consumption of fish and water from the lower Watut River among villagers in the Watut area of Wau/Bulolo, Papua New Guinea (PNG). Concerns surrounding the potential contamination of environmental media from mercury pollution as a result of small scale-alluvial mining activities taking place within the area have driven the need for this health risk assessment. The assessment was based on the Australian framework for environmental health risk assessment comprising the elements of hazard identification, dose- response assessment, exposure assessment and risk characterisation. Data examined in the calculations of risks were based on a report by Orathinkal, *et al.* (2011). Calculations of the risks using the water guideline approach showed no risk to the adults whereas the hazard quotient calculations revealed an elevated risk to both adults and children from mercury contamination when using the river as a drinking source as well as the consumption of fish harvested from the river. However, care must be taken when interpreting the results due to the limited number of samples involved in the study. On the other hand, it is prudent to adhere to precautionary principles particularly to ban pregnant mothers and children from consuming fish taken from the river as well as the need for proper regulation of small scale -alluvial mining within Papua New Guinea.

Keywords: Health Risk Assessment, Artisanal gold mining, Watut River, Methylmercury

Introduction

Artisanal and small scale mining refers to mining by individuals, groups, families or cooperatives with minimal or no mechanisation, often in the informal sector of the market (Hentschel, Hruschla and Priester, 2002). A common definition for artisanal and small scale mining is yet to be established however in some countries distinction is made between artisanal mining as that which is purely manual and on a very small scale, and small scale mining as that which is more mechanised and on a larger scale within the informal sector (Hentschel, Hruschla and Priester, 2002).

In PNG's context artisanal gold mining is preferred as the practice is purely manual and on a very small scale. Artisanal or small scale mining is common in many poorer, rural areas of developing countries in the world and Papua New Guinea is no exception. However for many of these countries the

government made small scale mining illegal by awarding mining rights only to large international mining companies (Heemskerk, 2005).

In contrast Papua New Guinea has a policy supporting small scale alluvial mining to support community based enterprises as a means of eradicating poverty. Artisanal mining in PNG uses amalgamation as the preferred extraction method whereby elemental or liquid mercury is used as the element to attract gold thus separating it from the alluvium. In the process alluvial gold is found mixed with sediments of which the lighter components can be removed easily by using gold–pan or sluice box. The miners then add drops of elemental mercury to the remaining sediments facilitating the adhesion of gold dust to the mercury thus resulting in globular amalgamations of mercury and gold (Aryee, Ntibery and Atorkui, 2003). Miners then boil the excess mercury, often in an uncovered container, releasing vaporised mercury into the atmosphere. Vaporised mercury when washed by rain ends up in the soil and river systems whereby it is converted into methylmercury thus contaminating fish predominantly.

It has been widely documented that amalgamation processes can result in high mercury exposure levels for miners and their families (Crispin, 2002; Hilson and Murck, 2000; Azapagic, 2002) and it can also lead to significant environmental contamination if proper control techniques and Best Practice Guidelines are nonexistent. Although artisanal gold mining has been in PNG for decades (Moretti, 2007) no assessments have been done to assess the risks to human health either from occupational exposure or environmental exposure to mercury.

There is now a growing concern from the communities within close proximity to the mining site as the slurry from the mine is disposed into streams that feed into rivers which serve the local people for drinking, cooking, bathing and fishing. Such is the case with the villagers of the lower Watut river in Wau/Bulolo. Accordingly, concerns regarding mercury poisoning are increasing among the villagers as they begin to understand the impacts mercury can have on the environment and ultimately on their health. It is due to this very reason that this health risk assessment is undertaken.

The aim of this assessment is therefore to determine if villagers near the Wau/Bulolo gold mine are at risk of significant total mercury, that is methylmercury and inorganic mercury poisoning from consumption of fish and water respectively from the lower Watut River. Environmental and biological samples were analyzed by the University of Technology, Lae with results presented in Table 1.

The Australian Health Risk Assessment framework (EnHealth Council, 2004; EnHealth Council, 2002) being consistent with the US Environmental Protection Agency health risk assessment model, is used in this assessment.

Table 1: Shows concentration of mercury in water and fish samples

Sample #	Sample Description	Results
W1	River sample from point 1	0.002 mg/L
W2	River sample from point 2	0.003 mg/L
W3	River sample from point 3	0.002mg/L
F1	Mallet	0.001µg/g
F2	Catfish	0.001µg/g
F3	Golden carp fish	0.24 µg/g

Source: Orathinkal, *et al.* 2011

Hazard identification

Mercury is naturally present in air, water and soil and is used in a variety of products and processes because of its unique properties. It exists in several forms namely metallic mercury or elemental mercury, inorganic mercury and organic mercury. The metallic or elemental mercury is used in artisanal and small-scale mining of gold and silver apart from chor-alkali production and also in products such as thermometers and dental amalgam fillings.

Metallic mercury can be released to the air, water and soil during production processes and uses or after disposal of mercury containing products and wastes (US Department of Health and Human Services, 1999). By far the most common organic mercury found in the environment is methyl mercury which is a major public health concern.

It is of significant health concern because it bio-concentrates as it moves up the food chain, accumulating in high concentrations in fish tissues (Oosthuizen and Ehrlich, 2001) particularly larger fish.

Physical and chemical properties

Mercury exists in a number of physical and chemical forms. In the elemental form which is the pure form of mercury or metallic form it is a liquid at room temperature, with its silvery mobile form, thus being called quicksilver by Aristotle (Clarkson, 1997). In a liquid form it is mobile, heavy, and preferably referred to as a liquid metal whilst in its solid form it is ductile, and has a malleable mass which is odourless. Metallic or elemental mercury possesses a high vapour pressure which can result in poisoning via inhalation among those who are involved in the heating of mercury in artisanal gold mining for example. The mercury vapour that arises from the heating process of mercury is colourless and odourless and can stay up in the atmosphere for a year where it can be transported to other places ultimately settling into rivers, streams and lakes where it transforms to methylmercury (WHO, 2007).

Methyl mercury ($C_2H_6Hg^o$) like phenyl mercury however exists as 'salts' and when in its pure form it is a white crystalline solid (WHO, 2008). It is soluble in sulphuric acid upon boiling; similarly it is readily soluble in lipids

and has also proven to be soluble in pentane. It has a low solubility in water and a low vapour pressure (US Department of Health and Human Services, 1999) thus bioaccumulation in fish can be high.

Toxicokinetics of methylmercury and metallic mercury

Methylmercury is rapidly and extensively absorbed, with about 95% of it being absorbed through the gastrointestinal tract following ingestion. Following its ingestion it is distributed throughout the body and easily penetrates the blood brain and placental barriers (Konig *et al*. 2005).

In the body the methylmercury combines with cysteine which is an amino acid found in most protein to form a methylmercury-cysteine conjugate, which is then transported into cells via a neutral amino acid carrier protein.

The methylmercury-cysteine conjugate can pass through not only the blood-brain barrier but also the placenta via an amino acid transporter. The passing of methylmercury cysteine conjugate through the placental barrier can have tremendous effects on an unborn child. Likewise as it passes through the blood brain barrier it can be oxidised and eventually accumulate in the brain resulting in chronic exposure that can lead to adverse health effects (Kanai, 2003; Kerper, 1992; Mottet, 1985; Sakamoto, 2004 cited in WHO, 2008).

Some methylmercury in the body is slowly converted to inorganic mecury. Methylmercury has a relatively long biological half-life in humans; which ranges from 44 to 80 days (WHO, 2008).

Absorption of inorganic mercury in the GI tract depends on solubility. Inorganic mercury which has a low solubility tends to have low absorption thus hindering systemic poisoning from occurring. On the other hand, soluble inorganic compounds are sufficiently well absorbed enhancing severe or even lethal systemic poisoning from occurring (Clark and Magos, 2006).

Absorption of metallic mercury vapour via inhalation is high, accounting for approximately 70-80% as opposed to oral exposure which is usually negligible (WHO, 2008). Due to its high lipophillicity, absorption of the inhaled vapour is followed by rapid diffusion across the alveolar membranes of the lungs into the blood (Clarkson and Magos, 2006).

It has been documented that exposure to 0.1–0.2 mg/m^3 elemental mercury vapour has resulted in approximately 74–80% of the inhaled elemental mercury vapour being retained in human tissues (Hursh *et al.* 1976; Teisinger and Fiserova-Bergerova 1965 cited in US Department of Health and Human Services, 1999).

Oral absorption of organic mercury is almost complete but respiratory absorption is not thoroughly documented (WHO, 2005). Similarly, there is limited information regarding dermal absorption of inorganic or organic mercury compounds in humans or animals.

The distribution for metallic, inorganic and organic mercury is somewhat consistent with the kidneys being the organ with the highest mercury bioaccumulation (Clarkson and Magos, 2006). Being highly lipophillic, metallic mercury can easily transfer through the placenta and the blood brain barrier (WHO, 2008). The oxidation of metallic mercury to inorganic divalent cation in the brain can lead to its retention in the brain for a long time (Clarkson and Magos, 2006).

> Metallic mercury can be oxidized to inorganic divalent mercury by the hydrogen peroxidise- catalase pathway in most tissues. This inorganic divalent cation is very unstable and therefore can be reduced further to metallic mercury (Clarkson and Magos, 2006 p. 384).

Excretion of methylmercury occurs primarily via the faeces, and less than one-third of the total excretion occurs through the urine (Clarkson, 1992; Department of Health and Human Services 2005). Methylmercury is also excreted through human milk however at much lower levels (WHO, 2004; US EPA, 1997d; ATSDR, 1999 cited in WHO, 2008).

Inorganic mercury is eliminated in urine and faeces while organic mercury is excreted predominantly via the faeces. Both inorganic mercury and methyl mercury are eliminated in breast milk (US Department of Health and Human Services, 1999).

Dose-response assessment

Toxicology

Acute exposure
Mercury causes severe disruption of any tissue in the human body with which it comes into contact when in sufficient concentration. However, the two main effects of mercury poisoning include neurological and renal disturbances (WHO 2005; Clarkson 1972).

Neurological disturbances are mainly due to poisoning by methyl and ethyl mercury (II) salts, in which liver and renal damage are of relatively little significance, while renal disturbances are characteristic of poisoning by inorganic mercury (WHO, 2005).

According to Clarkson (1997) organic mercurial, mercuric chloride and salts of mercuric mercury have an LD^{50} in the range 5 to 50 mg/kg and categorised as extremely toxic chemicals.

> In general, the ingestion of acute toxic doses of any form of mercury will result in the same terminal signs and symptoms, such as shock, cardiovascular collapse, acute renal failure and severe gastrointestinal damage (WHO, 2005 p.6).

Acute oral poisoning from organic mercury mainly lead to haemorrhagic gastritis and colitis; which ultimately causes damage to the kidney (WHO, 2005). Clinical symptoms of acute intoxication are clearly documented and include pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock. Symptoms that occur on a later stage include; swelling of the salivary glands, stomatitis, loosening of the teeth, nephritis, anuria and hepatitis (Stockinger, 1981 cited in WHO, 2005).

It is also documented that the ingestion of 500 mg of mercury (II) chloride can cause severe poisoning and sometimes death in humans (Bidstrup, 1964 cited in WHO, 2005). Likewise, inhalation of air containing mercury vapours at concentrations in the range of 0.05–0.35 mg/m3(Teisinger & Fiserova-Bergerova, 1965; Neilsen-Kudsk, 1972 cited in WHO, 2005) can cause acute effects.

> Exposure for a few hours to 1–3 mg/m3 may give rise to pulmonary irritation and destruction of lung from the tissue and occasionally to central nervous system disorders (Skerfving & Vostal, 1972 cited in WHO, 2005). Dermal exposure to alkylmercurials may give rise to acute toxic dermatitis and eczematous changes (WHO, 2005 p.6).

Long-term exposure
Numerous studies have indicated that the classical signs and symptoms of elemental mercury vapour poisoning such as tremors, mental disturbances and gingivitis may be expected to appear after chronic exposure to air mercury concentrations above 0.1 mg/m3 (IPCS, 1991 cited in WHO, 2005). Similarly, non-specific neurological and physiological symptoms can also be associated with lower exposure levels. .

Children and pregnant women (Konig *et al.* 2005; Hudson *et al.* 1987 cited in WHO, 2005) are especially vulnerable to chronic poisoning from methylmercury from direct exposure through eating contaminated fish.

> Pregnant women who consume mercury contaminated fish may encounter neuro-developmental problems in the developing foetus. Neurological symptoms include mental retardation, seizures, vision and hearing loss, delayed development, language disorders and memory loss. In children, a syndrome characterized by red and painful extremities called acrodynia has been reported to result from chronic mercury exposure (WHO, 2007 p. 7).

No evidence of carcinogenicity, reproductive toxicity, genotoxicity or tetarogenicity has been reported in epidemiological studies (Department of Health and Human Services, 2005). On the other hand, toxicological studies on rats has indicated teratogenic effects with foetal malformations and particularly brain defects in 23% of all live foetuses studied (WHO, 2005). This study found that "at mid-gestation, the minimum effective teratogenic dose of

mercury (0.79 mg/kg of body weight) was high in relation to the maternal LD50, and the incidence of fetal malformations, mainly brain defects, was 23% in all live fetuses" (WHO, 2005).

Moreover, the same study found that mercury chloride has the potential to increase incidence of some benign tumours as well as cause point mutations but not genotoxic effects (WHO, 2005).

Tolerable intake values - acceptable daily intake

Various ADI values for methylmercury have been set by different agencies; United States Environment and Protection Agency (US EPA) has set an ADI of 0.1 µg/kg body weight/day; Joint FAO/WHO Expert Committee on Food Additives (JECFA) has set 0.23 µg/kg body weight/day; and World Health Organisation (WHO) has set 0.4 µg/kg body weight/day (Clarkson and Magos, 2006).

The US EPA establishment of an ADI of 0.1 µg/kg body weight/day for methylmercury involved the benchmark dose used to calculate the NOAEL and the uncertainty factor of 10 applied to the NOAEL (Clarkson and Magos, 2006). The RfD was based on a number of studies including the Faroe islands, New Zealand and Seychelles studies which take into consideration the most sensitive stage of life span particularly the prenatal period of the brain development (WHO, 2008).

The study on 900 Faroese children showed that prenatal exposure to methylmercury resulted in neuropsychological deficits at 7 years of age (Grandjean *et al*. 1997 cited in WHO 2008).

The joint FAO/WHO expert committee on food additives (JECFA) has set an ADI of 0.23 µg/kg body weight/day which is equivalent to the reference level of 1.6 µg MeHg/kg body weight/week (PTWI) for methylmercury (WHO, 2004 cited in WHO, 2008; Clarkson and Magos, 2006). The reference value was applied because it represents permissible human weekly exposure taking into account the protection of the most vulnerable life stages such as the embryo and foetus (WHO 2007 cited in WHO 2008).

The TDI for inorganic mercury as set by the International Programme on Chemical Safety (IPCS) with the World Health organisation is 2 µg/kg body weight/day.

This was derived based on the NOAEL of 0.23 mg/kg of body weight per day for kidney effects in a National Toxicology Program of 26-week study in rats that applied an uncertainty factor of 100 to account for inter and intraspecies variation (WHO, 2008). This was also based on the assumption that a 60-kg adult drinks 2 litres of water per day and then allocating 10% of the TDI to drinking-water, since the major sources of exposure are through food (WHO, 2008).

Health Canada has set a tolerable daily intake of 0.47µg/kg/day for methylmercury and 0.71µg/kg/day for total mercury respectively (Health Canada, 2000).

Guideline values for water and food (fish)

The Drinking Water Quality Standards in PNG are adopted from the World Health Organization's Drinking Water Quality Guidelines (PNG Water Board, 2002). The Standards are regulated under the PNG Public Health Act and were reviewed in 1998 but guideline values are not easily accessible for reference. However the guideline value for total mercury, that is inorganic and methylmercury, in water as per the World Health Organisation's guideline value is 1µg/L (WHO, 2007) or 0.001 mg/L.

The Codex Alimentarius Commission guideline levels are 0.5 mg/kg for non-predatory fish and 1mg/kg for predatory fish for methylmercury (WHO, 2007). Since the PNG guidelines are inaccessible this assessment will derive the guideline values for inorganic mercury based on the World Health Organisation and the International Program on Chemical Safety TDI for inorganic mercury. The guideline value fothe r methylmercury in fish will be based on the Codex Alimentarius Commission guideline levels for non-predatory fish for both adults and children.

The guideline value is generally calculated based on the highest dose of a chemical or contaminant that causes no adverse effects in long-term experiments on laboratory animals (NOEL) using the following formula:

$$\text{Guideline (mg/L)} = \frac{\text{animal dose x human weight x proportion of intake from water}}{\text{Volume of water consumed x safety factor}}$$

Where, animal dose = NOEL (g/kg/day)
Human weight = average weight of a Papua New Guinean adult = 60kg, child = 10kg
Proportion of intake from water = 10%
Volume of water consumed = 2L for an adult, 1L for a child each day
Safety factor = between 100 and1000.

Derivation of a guideline value for this scenario

In this scenario, the ADI of 2 µg/kg/day as set by the International Program on Chemical Safety and the World Health Organisation, is used. This chosen ADI was derived based on the NOEL of 0.23 mg/kg/day for kidney effects in a 26 week study on rats. This study has applied an uncertainty factor of 100 to account for inter and intra species variation. This ADI is also chosen as it was based on assumptions that a 60kg adult drinks 2 litres of water a day thus allocating 10% of the TDI to drinking water.

Therefore since we have an ADI of 0.002 mg/kg/day for inorganic mercury, the PNG guideline value will be calculated using the following formula:

Guideline (adult) = $\frac{\text{ADI x human weight x proportion of intake from water}}{\text{Volume of water consumed}}$

$\qquad\qquad$ **=** 0.006 mg/L
Guideline (child) = 0.002 mg/L

Proportion of water intake from water, volume of water and weights for the adults and children are taken from the Australian Drinking Water Guidelines (2004).

Exposure assessment

Environmental behaviour, occurrence and distribution

Air
Metallic mercury or elemental mercury is very volatile due to a very high vapour pressure and therefore can be oxidized by ozone to other forms such as Hg^{t2} which can be removed from the atmosphere by precipitation (Brosset and Lord 1991 cited in WHO, 2007). The residence time of elemental mercury in the atmosphere is estimated to be 6 days to 2 years depending on atmospheric conditions (Lindqvist 1991b cited in WHO, 2008). For instance, in clouds, a fast oxidation reaction occurs within hours between elemental mercury and ozone. The main transformation process for elemental mercury or other mercury compounds is photolysis (EPA 1984b; Johnson and Bramen 1974; Williston 1968 cited in WHO, 2005).

Water
In water, mercury can exist in various forms such as mercuric Hg^{t2} and mercurous Hg^{t1} and mercuric ion compounds with varying water solubilities. The transport and partitioning of mercury in surface waters is influenced by the particular form of the compound. More than 97% of the dissolved gaseous mercury found in water consists of elemental mercury (Vandal *et al.* 1991 cited in US EPA 1999). Metallic mercury apart from dimethymercury being volatile tend to evaporate to the atmosphere, whereas solid forms partition to particulates in the water column and are transported downward in the water column to the sediments (Hurley *et al.* 1991 cited in US EPA 1999) where adsorption occur. Elemental mercury is highly volatile and so through photolysis it volatises from its vapour form from surface waters when the water temperature is high and the conditions are acidic.

Any form of mercury in the water can be transformed to methylmercury by microbes; particularly sulphur reducing bacteria play a role in the mercury methylation process in anaerobic conditions (Regnell and Tunlid 1991 cited in US EPA, 1999). In addition, the acidity of the water plays an important role in the transformation of mercuric ions to methylmercury. This happens when "under acidic conditions, the activity of the sulfide ion decreases, thus

inhibiting the formation of mercuric sulfide and favouring the formation of methylmercury" (Bjornberg *et al.* 1988 cited in US EPA p.408).

Soil

As in water mercury also exists in various forms such as mercuric Hg^{t2} and mercurous Hg^{t1} in soil. The transport and partitioning of mercury in soils is influenced by the particular form of the compound. For instance, the volatile forms such as metallic mercury and dimethylmercury evaporate to the atmosphere, whereas solid forms partition to particulates in the soil (Hurley *et al.* 1991 cited in US EPA, 1999).

Vaporization of mercury from soils may be controlled by temperature, with emissions from contaminated soils being greater in warmer weather (Lindberg *et al*. 1991 cited in US EPA, 1999). It has been reported that "the dominant process controlling the distribution of mercury compounds in the environment appears to be the sorption of non-volatile forms to soil and sediment particulates sediments back into the water column" (Bryan and Langston 1992 cited in US EPA, 1999 p. 409). The adsorption of mercury to soil disallows it to leach down into underground water (Krabbenhoft and Babiarz 1992 cited in US EPA, 1999).

It is also documented that with increasing pH or chloride ion concentrations the adsorption of mercury in soil tends to decrease (Schuster 1991 cited in US EPA, 1999). This can explain the reasons of high concentrations of mercury in sediments as opposed to the water column as is the case also in fish where bioaccumulation of mercury occurs. Inorganic mercury sorbed to particulate material is not readily desorbed and therefore, freshwater and marine sediments are important repositories for inorganic forms of the element, but leaching can be a relatively insignificant process in soil processes (Meili 1991 cited in US EPA, 1999). With surface runoff however the movement of mercury from soil to water can be facilitated by soils with high humic contents.

Table 2: Exposure levels for this assessment

Sample #	Sample Description	Results
W1	River sample from point 1	0.002 mg/L
W2	River sample from point 2	0.003 mg/L
W3	River sample from point 3	0.002mg/L
F1	Mallet	1×10^{-7} mg/g
F2	Catfish	1×10^{-6} mg/g
F3	Golden carp fish	2.4×10^{-4} mg/g

While the above values have been used exclusively in the assessment of health risk for the mentioned population it is important to note the limitations associated with the study. A very limited number (3) of fresh water fish and fresh water samples (3) were used in the study by Orathinkal (2011) and as such the possibility of random error cannot be ruled out.

The standard protocols of how the environmental and biological samples were analyzed are not available for input in this assessment hence the possibility of ruling out any error due to sampling measurement and variation are acknowledged.

Likewise, the measurement equipments and how the samples were collected are not made available and therefore there may be uncertainties surrounding the validity of the above values. That is whether the actual values are higher or lower than the measured values. Nevertheless, it is undeniable that the Wau/Bulolo artisanal gold mine has been in operations in the area for decades and with no proper control and best practice guidelines to guide the daily operations of this enterprise one can be confident that there is no better way of containing mercurial waste apart from the easiest and most destructive which is to pollute the surrounding environment and ultimately the people nearby.

Calculation of an Estimated Daily Intake (EDI)

The EDI for this study consists of the estimated dose from drinking water plus the estimated dose from the ingestion of fish. Assumptions are that: there is no absorption via skin contact; the river is the only source of water for drinking and fishing.

Therefore, Estimated Daily Intake = $ED_w + ED_f$
Where, ED_w is the estimated water ingestion dose and ED_f is the estimated fish ingestion dose.

The amount of inorganic mercury absorbed through drinking water is estimated using the equation in Figure 1 while the estimated dose of methylmercury absorbed through ingestion of fish is worked out using the equation in Figure 2.

Figure 1: Equation use in calculating EDw, (adopted from Health Canada)

The amount of total mercury absorbed into the body through drinking water EDw can be estimated using the following equation:

$$EDw = \frac{C \times IR \times EF}{BW}$$

Where,
EDw = Estimated dose from drinking water: the water ingestion dose is expressed as milligrams of total mercury ingested, per kilogram of body weight per day (mg/kg/day)
C = Concentration of total mercury in milligrams per litre of water (mg/L)
IR = Ingestion rate: the amount of water a person drinks in a day, in litres per day (L/day)
EF = Exposure factor: indicates how often the individual has been exposed to the contaminant over a lifetime
BW = Body weight: the average body weight in kilograms (kg) based on individuals age group

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Figure 2: Equation used in calculating ED$_f$ (adopted from Health Canada)

The amount of methylmercury absorbed into the body through ingestion of fish (ED$_f$) can be estimated with the equation:

$$ED_f = \frac{CF \times CR \times EF}{BW}$$

Where:

ED$_f$ = Estimated dose from food (fish): The food ingestion dose is expressed as milligrams of methylmercury eaten per kilogram of body weight per day (mg/kg/day)

CF = Concentration of methyl mercury in fish which is expressed in milligrams of methylmercury per gram of food (mg/g)

CR = Consumption Rate: The amount of fish eaten, expressed in milligrams per day (mg/day) using Health Canada's standard values

EF = Exposure factor: indicates how often the individual has eaten contaminated fish in a lifetime (unit -less).

BW = Body weight: Average body weight in kilograms (kg) based on individuals age group, obtained from standard values from Table 2 – Health Canada

Therefore to calculate the EDI using the highest concentration, as in the worst case scenario our calculations would be as following:

For adults: ED$_W$ = [C x IR x EF] / BW

Where,   EF = 1 because we are using the worst case scenario

ED$_W$ = 0.0001 mg/kg/day

$$ED_f = \frac{CF \times CR \times EF}{BW}$$
= 0.00732 mg/kg/day.

For children: ED$_W$ = [0.003 mg/L x 1L/day x 1] / 10 kg
= 0.0003 mg/kg/day
ED$_f$ = 0.0216 mg/kg/day

Therefore, EDI = ED$_W$ + ED$_f$

EDI adult for total mercury= 0.00832 mg/kg/day
EDI children for total mercury = 0.0027 mg/kg/day

Risk characterisation

From the derived guideline values for inorganic mercury and guideline value for methylmercury in fish based on the Codex Alimentarius Commission's values the following statements can be made:

Inorganic mercury for adults indicated that the lowest measured value is less than the derived guideline value (0.006 mg/L), and the highest measured value is also less than the derived value. For children, the lowest measured value is equal to the derived value (0.002 mg/L) while the highest measured value is higher than the derived value. Nevertheless if we use the World Health Organisation's guideline value for mercury in drinking water which is 0.006 mg/L both the lowest and the highest measured values would be low.

Interestingly, if we use the PNG's prescribed water quality guideline for mercury in fresh water which is 0.002 mg/L, the lowest measured value would be equal to it while the highest measured value would be higher.

Based on the Codex Alimentarius Commission's guideline value for methylmercury in fish, 0.5 mg/kg it is evident that both the lowest and the highest measured values are lower than this guideline value.

Applying the Hazard Quotient (HQ) approach:
        HQ =Exposure estimate / Reference dose
            = EDI / ADI

For adults: HQ = 4.16
For children: HQ = 1.35

Risk characterisation summary and decision

Using the worst case scenario it is obvious that the concentrations of total mercury are higher than the PNG prescribed water quality guidelines for fresh water. Based on these results the continuous consumption of water from the lower Watut River may pose risks to human health and therefore appropriate control or treatment measures may be required to be implemented. Moreover, the results obtained using the Hazard Quotient approach indicated an elevated risk indicating that those already exposed are likely to suffer adverse effects from consumption of fish and water from the Watut River.

Considering that the worst case scenario is applied here it may be argued that the actual risk may be lower. However in reality the local people entirely depend on this water source for their living as there are no alternative water supply systems in place. In addition, children who grow up and live in the area will be exposed to much higher levels of mercury as mining activities continue their operations and therefore in light of these facts it is fundamentally important to ere on the side of safety. And more importantly, due to the subtle effects from prenatal exposure such as delayed development and cognitive changes in children we should always ere on the side of caution.

With due considerations to the limitations of this study, particularly regarding the sampling methods and measurements the values presented here may not truly reflect the concentration of mercury in the water and the fish. It is therefore recommended that a more thorough study be carried out on both

environmental samples and also on small scale miners taking into account the limitations of this study.

Risk management

In light of the above risk assessment, the evaluation of the risk requires that the risk should be thoroughly treated. The immediate action to take would be risk communication to the concerned villagers. This is absolutely necessary because most of the villagers are illiterate and have no knowledge of the effects of mercury on their health. Thus the communities need to be well informed of mercury and the effects it has on their health based on the findings of this study. The local member or parliamentarian representing the local people can be collaborated with to address this issue. For instance, the Parliamentary leader may provide water tanks for the affected communities to cater for their cooking and drinking needs while the river can be used for bathing only since mercury cannot be absorbed through water contact.

The size and portion of fish to be consumed need to be restricted. For instance smaller fish are much preferred over larger ones due to a less effect of bioconcentration of methylmercury in their tissues, however this may not be feasible in the long run. More so the most vulnerable populations such as pregnant mothers and young children needs restrictions on their fish consumption rate or may be banned from consuming fish taken from the lower Watut River.

All the above measures are temporary as they are the end result of mercury pollution from the Wau/Bulolo artisanal gold mine. Therefore, it is prudent to deal with the source of the problem to prevent all other problems associated with it. The most fundamental step to start with is the recognition that there are no pollution control mechanisms in place. By pollution control mechanisms. it is implied that the mining industry should have techniques, machines, or systems that can be employed by the rural people to extract gold using mercury and yet with the minimum damage to the environment and human health. The PNG Mineral and Resources Department can ensure that the mining industry either formal or informal is kept abreast with these requirements by first making sure that they are available for them to use and comply with. Since artisanal mining is legal in PNG the government is ultimately responsible to ensure the best techniques are employed for controlling mining related pollution.

Other responsible authorities such as the Environment and Conservation Department, the Health Department, and the Labour Department, need to collaborate with the Mineral and Resources department and the Mining industry on the development and implementation of a Best Practice Guideline for use in artisanal gold mining areas of PNG.

Conclusion

Small scale alluvial mining is an income generating 'machine' in mainly developing countries of the world and PNG is no exception. With it comes the price of environmental contamination from mercury through the employment of amalgamation techniques. Since there are also positive benefits from this informal business it would be beneficial for all that prudent and strict measures on environmental pollution are developed and complied with. Best Practice Guidelines are fundamentally necessary to guide in the day to day operation of such activities. This is of vital importance because from this health risk assessment it has been revealed that there is likely risk from mercury pollution as calculated using the hazard quotient approach relating to the particular community in PNG.

It is highly recommended that a health risk assessment be carried out to assess the risk from occupational exposures as this assessment was only based on the indirect effect of mercury pollution through environmental pollution. Even though this may have been an indirect method for quantifying health related problems from mercury pollution it can be indicative of the problem in the communities and thus it is only ideal to do a health risk assessment of the workers directly involved in the handling of mercury in small scale mine settings.

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