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Selenium - Mercury Antagonism in Human Blood Samples from Residents of Wassa West District, a Mining Area in Ghana

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Abstract:
This paper sought to evaluate Se to Hg (Se:Hg) molar ratio in human blood samples from residents of Wassa West district of Ghana, a region which has a long history of gold mining. Determining Hg levels alone in either environmental samples or human body fluids or tissues is necessary but not essential enough to assess the health risk of a given population. The levels of Se in such matrices are the fundamental factor in health risk assessment of Hg exposure and toxicity. Fifty blood samples were collected from volunteers from Wassa West District and were analysed for Hg and Se concentrations. The molar concentrations of each element was calculated and the corresponding Se:Hg molar ratios were determined. The results indicated that, the molar concentrations of Se ranged from 0,35 nM to 62.38 nM with a mean value of 25.26 nM, while Hg molar concentrations ranged from 0.05 nM to 4.65 nM with a mean value of 0.38 nM. The ratios ranged from 0.97 to 958 with a mean value of 155.40. Apart from, one subject, all the Se:Hg molar ratios were above one, which may imply that the residents have enough Se (well nourished) to prevent Hg toxicity. The precision and accuracy of the analytical methods were determined by the standard reference materials, DORM-2 and GBW 09101. The measurement precision which was determined using relative standard deviation fell within 4%.The results of the analysed samples were within ±5% of the certified values of the standard reference materials.

Key words: *Blood, Selenium, mercury, antagonism, molar ratio, mining, health, risk.*

1. Introduction

The investment boom in the mining industry, coupled with increasing artisanal activities, due to the implementation of the Structural Adjustment Programme in 1983, resulted in accidental and occasional pollutant spillages into the environment in the study area(Akabzaa, 2001). Although the pollutant spillages in Wassa West District were under-reported, the major reported spillages of pollutants besides the pollutants (chemicals) used by artisanal miners occurred in 1989, 1991, 1994, 1996, 1999 and 2001 (Berit, 2007), One of such spillages which received condemnation from all segments of the society was on 18th June 1996, a situation which Thomas Akabzaa, termed as "Teberebie cyanide spillage saga". It appears on all such occasions, individual researchers and the Environmental Protection Agency normally analysed environmental samples (water, soil and sediment) in order to assess the extent of damage caused to the environment without analysing human tissues and body fluids to assess the impact of the spillage on the inhabitants, hence this study intends to focus on this gap.

1.1. Selenium-Mercury Antagonism (Se-Hg)

Normally, two harmful chemicals do not interact at all with each other. However, in those unusual instances, if they do interact, two opposite things may happen, the two contaminants can nullify each other's toxicity, a phenomenon called antagonism ,or the two can magnify each other's toxicity which is called synergism**.** (USGS, Science for Changing World, 1999)**.**

The study of selenium physiology has become one of the fastest growing areas in biomedical research, as well as its role in protection against mercury toxicity(Laura, 2004).

The interactions between the toxicities of mercury and selenium are perhaps the best known of all interactions among environmental contaminants. Moreover, detoxification of toxic heavy metals is one of the expected roles of Se. The element,Se has been shown to react with Hg in the bloodstream by forming complexes containing the two elements at an equimolar ratio when selenite and mercuric chloride are co-administered. According to El-Demerdash (2001), this reaction may also explain the consistent equimolar ratio of Se and Hg in tissues of seals and other marine mammals and mercury mine workers. Burger (2012) suggested that Se: Hg molar ratios of over 1 are protective against mercury toxicity for humans. However, it is also known that exposure to both Se and Hg have been shown to result in toxic effects (synergism).

El-Demerdash (2001), investigated the effects of Se and Hg on some biomarker enzymes, protein and oxidative stress in adult rats receiving Se as sodium selenite, and Hg as mercuric chloride, or a combination of both showed that selenium counteracts the toxicity of mercury in rat serum, brain and liver.

According to Laura (2004), measuring the amount of mercury present in the environment or food sources may provide an inadequate reflection of the potential for health risks if the protective effects of selenium are not also considered. Selenium's involvement is apparent throughout the mercury cycle, influencing its transport, biogeochemical exposure, bioavailability, toxicological consequences, and remediation(Laura, 2004).Laura(2004), indicated that selenium, present in many foods (including fish), protects against mercury exposure. A Study by Laura (2004), have shown that mercury exposure reduces the activity of selenium dependent enzymes, through diversion of selenium into formation of insoluble mercury-selenides. Thus, mercury may inhibit the formation of selenium dependent enzymes, supplemental selenium supports their continued synthesis.

It was also shown that, owing to the extremely high affinity between mercury and selenium, selenium sequesters mercury and reduces its biological availability. It is obvious that the converse is also true as a result of the high affinity complex formed, mercury sequesters selenium. This is important because selenium is required for normal activity of numerous selenium dependent enzymes. (Laura, 2004). Further research into mercury-selenium interactions will help us understand the consequences of mercury exposure and identify populations which may be protected or at greater risk to mercury's toxic effects (Laura, 2004).

It is well recognized that mercury and sulphur bind together to form complexes. This binding property is the basis of chelating therapy used as a treatment in cases of acute mercury poisoning. The complexes between mercury and selenium are less generally known but of much higher affinity(Laura, 2004). Physiologically, sulphur is far more abundant than selenium, yet because of selenium's higher affinity for mercury, mercury selectively binds with selenium to form insoluble mercury-selenides (Hg-Se). This interaction has been assumed to be a 'protective' effect whereby supplemental selenium complexes the mercury and prevents negative effects in animals fed with toxic amounts of mercury(Laura, 2004).The ability of selenium compounds to decrease the toxic action of mercury has been established in all investigated species of mammals, birds, and fish(Laura, 2004).

Alarmingly, while the placental barrier can stop many toxic elements, methylmercury is an exception, in that it does not only crosses the placenta, it also, accumulates at higher concentrations on the fetalside than on the maternal side,worsening the situation for the developing fetus.Mercury also crosses the blood-brain barrier and exhibits long-term retention once it gets across(Laura, 2004). According to the author, because of the binding interaction between these two elements, selenium appears to have an effect on the bioavailability of mercury, both biologically and environmentally. Laura (2004) suggested an important role of selenium in the bioaccumulation of mercury in fish.Belzile (2000), determined the concentrations of Se and Hg in muscles of two freshwater fish species,namely perch (*Percaflavescens*) and walleye (*Stizosedionvitreum*). According to Belzile (2000), samples of various sizes were collected from ten lakes of Northern Ontario. The correlation analysis revealed a clear and strong antagonistic effect between Se and Hg in muscles for both perch (r= - 0.790) and walleye (r = - 0.973), with the concentrations of Hg decreasing exponentially as Se increase.

A study by Zhang (2014), in mammals, however, have shown that, adding Se in amounts beyond the reasonable range for safety caused an "additive effect" or "synergistic effect" of Hg-Se toxicity instead of the desired antagonistic effect. Zhang (2014), suggests that the total dissolved Se concentration in water should not exceed the safe range of 5.0μg/L when Se is added to reduce the methylmercury (MeHg) enrichment in fish.

The antagonistic effect of Se on inorganic and MeHg was confirmed by Zhang (2014),in addition, Hg: Se ratio was found to be 1:1 in experiment on marine mammals and mercury-mine workers.

A large number of animal studies have shown that increasing dietary Se intake within a physiologically appropriate range can significantly increase the detoxification signal for symptoms of MeHg poisoning. A case study that focus on residents in Hg mine areas with inorganic Hg poisoning, indicated that moderate supplementation with organic Se can increase the Hg excretion in local residents(Zhang, 2014).

1.2. Possible Mechanisms of Protective Effect of Selenium against Mercury Toxicity

Although, selenium inhibits the toxicity of Hg, the actual mechanism is not clear. However, it is suggested that, the toxicity of Hg can be reduced if Hg can bind selenide to form Hg-Se or a highly stable Hg-Se protein complex, which is likely to be the mechanism for mitigating MeHg poisoning in marine mammals that are high in food chain and whose MeHg removal rate cannot keep up with their MeHg intake rate(Zhang, 2014). This mechanism may explain the 1:1 Se-to-Hg (Se:Hg) molar ratio in these animals and why higher levels of Hg do not appear to induce toxic effects in these mammals.(Zhang, 2014).

Another mechanism proposed is the redistribution of inorganic Hg among various organs in biological systems under the influence of Se. Also, Se-Hg complexation induced by Se deficiency may be another mechanism. This is because the affinity of Hg^{2+} and MeHgfor –SeR is higher than for –SR. It is therefore reasonable to assume that Hg and MeHg will bind –SeR with higher priority resulting in bioavalability of Se in organisms.(Zhang, 2014).

1.3. New Model for the Assessment of Health Risk of Hg

At present, the Hg concentration is the only standard used in the health and safety risk assessment. It was proposed as early as 1972 that the Hg-to Se molar ratio should be used as a reference standard for Hg pollution. However, this proposal attracted little attention because the specific mechanism has not been unraveled (Zhang, 2014).

To simplify the evaluation of the risks of MeHg exposure and the nutritional benefits of Se, Zhang (2014) proposed the Se Health Benefit Value (Se-HBV) and used this index to successfully explain the ''contradictory'' research results that the conventional Hg-

exposure evaluation model fails to explain clearly. According to Zhang (2014) ,the Se-HBV combines the absolute values and the relative ratios of Se and Hg, and the relative ratios of the two are used to correct the absolute amounts of Hg and Se. The Se-HBV can measure the risks and benefits of the dietary intake of Hg and Se using only one index, and it is more easily explained than the conventional model in which only Se or Hg is considered(Zhang, 2014). According to Zhang (2014), a positive index indicates a health benefit, and a negative index indicates a health risk. The Se-HBV value matches the expected benefits or risks. The two formulae are expressed in molar units to reflect the stoichiometric relation between the two substances. The recommended formula, namely, Se-HBV = Se(Se/Hg) - Hg(Hg/Se), has begun to be used in many recent studies to evaluate and discuss the risks of the combined effects of Se and Hg (Zhang, 2014). Unfortunately, both this formula and the conventional Hg-to-Se molar ratio method have an obvious shortcoming. This shortcoming is as follows: in some extreme cases, in the presence of a lower-than-standard Se intake (i.e., the danger of selenium deficiency) or when the Se intake far exceeds the standard (i.e., the potential for Se poisoning), it is still possible to satisfy the condition of an Se-to-Hg molar ratio of 1, or a positive Se-HBV index(Zhang, 2014). These two extreme cases are hidden within this single Se-HBV index and superficially manifest as highly beneficial situations, suggesting that either risk-evaluation method may be somewhat misleading.(Zhang, 2014)

In addition, the specific Hg-to-Se molar-ratio threshold for the production of protective effects is presently unknown. Logically speaking, it is also very difficult to determine a universal ratio because of the differences in such aspects as physiology and dietary habits among various regions and populations (Zhang, 2014). More importantly, recent studies have found that there is no apparent threshold for the toxic effects of MeHg exposure. Therefore, based on the considerations of controllability and ‘‘conservative principles’’ in risk management (i.e.,one would rather underestimate the health benefits and overestimate the risks in uncertain conditions), the author suggests that it is best to combine the conventional intake-dose-only index and the newly proposed index of the Se-to-Hg molar ratio, which permits the comparison of various combined relations(Zhang, 2014). The purpose is not to define an ‘‘accurate’’ measure of the relative risks and benefits but instead to use this comparative assessment model as a guide for the general population(Zhang, 2014).A study by Sampaio (2013) suggested that Se ingestion within a safe dose interval may constitute an antidote to Hg toxicity.

Burger (2012), suggested that an excess of Se over Hg protects against methylmercury toxicity, as measured by Se:Hg molar ratio, and that higher ratios are more protective than lower ratios. The actual ratio which is protective is unknown, both for vertebrate classes, species, and individuals and for sensitive tissues.

This paper seeks to evaluate Se to Hg molar ratios in blood,and using it to establish whether or not the residents are at risk to Hg exposure in the mining impacted region of Ghana.

2. Materials and Methods

2.1. Sample Collection

The study area has already been described in (Samlafo, 2014). All the fifty blood samples were collected from Tarkwa Government Hospital, ABA Goldfield Hospital (Tarkwa), Ami Maternity Home(Tarkwa), GAG Hospital-Iduapriem, Ghana Manganese Company Hospital (GMC)-Nsuta and Pentecost Clinic.

The blood samples were collected randomly from the subjects on three occasions with ethical approval from the Ghana Health Service.The blood samples were collected in already washed tubes containing ethylene diamine tetra acetic acid (EDTA, BHD chemicals, England) as an anticoagulant and labelled Bx, where x=1-50.The blood samples were packed into a fridge(at 2-4ºC).

The subjects were made to respond to a detailed questionnaire regarding dietary habits, gender, age, occupation etc. A total of 50 subjects responded to the questionnaire. This consists of 40 females and10 males.

2.2. Preparation of the blood samples

One millilitre of the whole blood sample was taken by calibrated 1000µL eppendorf pipette and diluted to 100.0 mL using double distilled water. The mixture was acidified with 12.0M H_2SO_4 drop by drop until the pH of the mixture dropped to 1.5-2.0 for Hg extraction. About 20.0 mL of 0.02% dithizone/Chloroformmixture was added to the mixture in a conical flask and shaken and allowed to stand for 10 munites. The mixture was then extracted three times with 20.0 mL aliquot of 1.5% sodium thiosulphate ($Na_2S_2O_3$). The organic phase was then separated from the aqueous phase into a labelled beaker.

The aqueous phase in the separatory funnel was again extracted with 20.0mL aliquots of dithizone/chloroform solution three times. The organic phase was added to the beaker containing the organic layer while the aqueous phase was discarded. The total volume of organic phases obtained from the three extractions was left in a fume hood for two days to evaporate to dryness. The evaporation stage reduces the sample volume and concentrates the analyte of interest. Five sub-samples of 100mg of each organic phase was weighed and put into 1.5mL vial and heat sealed. The sealed vials were then packed into medium vials for irradiation. The packed samples were irradiated together with single element Hg comparator standards and DORM-2, the certified standard reference material.

2.3. Sample irradiation and counting

Irradiation and counting of samples using the Ghana Research Reactor-1 facility has been described earlier (Nyarko, 2003). The packed extracted blood samples, comparator standard samples and the certified reference material DORM-2 were irradiated at the inner irradiation sites of the Reactor-1 facility, using a pneumatic transfer system at a thermal neutron flux of 5.10^{11}n.cm^{2}s^{-1} and a pressure of 1. 723 bars. The samples were irradiated for 1 hr (t_i=1hr), and allowed to decay for 24 hrs (t_d=24hrs) and counted for 10 minutes (t_c=10min.).

The counting of samples was done using a gamma-ray spectroscopy system. The system was made up of N-type HPGe detector model GR2518, and HV Power Supply Model 3103, a spectroscopy Amplifier Model 2020, an ACCUSPEC Multi-channel Analyzer (MCA) emulation software card, 486 micro-computer, all manufactured by Canberra Industries, Inc. The efficiency of the detector was 25%. Each sample was placed at a distance of 2.6 cm from the detector surface and counted (Nyarko, 2003).
The accumulated spectra intensity was analyzed qualitatively and quantitatively. The qualitative analysis involved the identification of Hg using 77.4 keV of ^{197}Hg. The quantitative analysis was done by converting the counts (area) under the photopeak of the radionuclides by comparator NAA (Nyarko,2003)method using Hg single standards for total Hg (THg). Cobalt-60, Caesium-137 and Barium- 133 sources were used to calibrate the detector periodically whenever counting was going to be done and between counting of the samples.

2.4. Digestion of whole blood for total Se determination

One hundred milligrams (100mg) of whole blood samples were weighed, diluted with 25.0 mL of double distilled water. Ten millilitres (10.0mL) of concentrated nitric acid was added to the mixture and allowed to stand for 10 minutes. Five millilitres (5.0mL) of 30% H_2O_2 was also added to the mixture using calibrated eppendorfpippete to dissolve any organic matter that might be left in the mixture. The resulting mixture was put in a microwave, digested for 25min in a Milestone microwave oven (Ethos 900) using the following operation parameters; 250W for 2min, 0W for 2min, 250W for 6min, 400W for 5min, 650W for 5min and 5min allowed for venting(Milestone Technical papers , 2001). A 2.0g of certified standard human hair reference material GBW 09101(certified by Shanghai institute of Nuclear Research) was similarly digested together with a blank as a quality control measure.
Five millilitre of the digested blood, samples were taken into each test tube and 5.0 mL of 5.0M HCl was added to the test tube containing the samples. The mixture was heated for 30.0 minutes at a temperature range of 90-100^{o}C in a water bath to reduce Se (VI) to Se (IV). The digest was assayed for the presence of total selenium (TSe) at a wavelength of 196.0 nm,using Varian Fast Sequential Atomic Absorption Spectrometer, model AA240FS. Acetylene gas was used as the carrier gas, while inert argon was pass through the system to remove interfering gases between each reaction time. The instrument detection limit was 0.002µg/mL.

2.5. Calculation of Se -to –Hg (Se:Hg) molar ratio

Molar- based concentrations (µM) of both total Hg (THg) and total selenium (TSe) were calculated from mass-based concentrations(µg/g) in each blood sample using a density of 1.037g/L for whole blood and molecular mass (µg/µmol) of each element(Lucero, 2013).

$$[THg]\mu M = ([THg]\mu g/g \times \text{Density of blood g/L})/200.59\mu g/\mu mol \quad (1)$$

$$[TSe]\ \mu M = ([TSe]\mu g/g \times \text{Density of blood g/L})/78.96\mu g/\mu mol \quad (2)$$

The Se:Hg molar ratio was calculated from the relation

$$Se:Hg = \frac{\text{Molar concentration of Se}}{\text{Molar concentration of Hg}} \quad (3)$$

The molar concentration ratio of [TSe] : [THg] designated as Se:Hg was determined for each blood sample(Lucero, 2013).

3. Results and Discussions

3.1. Validation of the Analytical Results

The precision and accuracy of the Instrumental Neutron Activation Analysis (INAA) method was determined by standard reference material DORM-2 as shown in Table 1. The levels of the elements in DORM-2 were strongly correlated with the certified values as Pearson's correlation coefficient was 0.99. The results of the samples were within ±5% of the certified values.
Similarly, GBW 09101 reference material was used to validate the Hydride Generation Atomic Absorption spectrophotometric (HGAAS) method. The recoveries made lies between 96-110%. A paired student t-test was used to compare the molar concentrations of Se and Hg in order to established significant statistical differences between the molar concentrations. The level of probability at which significant differences existed in molar concentrations was set at $p< 0.05$ at 95% confidence level. The null hypothesis is that all Se to Hg (Se:Hg) molar ratios in blood samples are not greater than 1 was tested.

Element	HGAAS- GBW 09101		INAA -DORM-2	
	Measured value	Certified value	Measured value	Certified value
Se	0.56± 0.02	0.58±0.05	1.51±0.06	1.40±0.09
Hg	2.20± 0.15	2.16±0.21	4.68±0.51	4.64±0.26
Mn	2.98±0.25	2.94±0.20	3.20±0.62	3.66±0.34
Fe	71.85±4.5	71.20±6.6	143.5±12	142.0±10
Cu	23.12±2.5	23.0±1.4	2.41±0.18	2.34±0.16

Table 1:Mean measured values (µg/g) of some elements in GBW 09101 and DORM-2 compared with the certified values with standard deviations (n=5),

Sample code	Se µg/mL	Hg µg/mL	Se nM	Hg nM	Se:Hg Ratio
B1	3.15±0.80	0.04±0.01	41.36	0.21	196.95
B2	2.66±0.60	0.03±0.01	34.93	0.16	218.31
B3	2.97±0.40	0.09±0.01	39	0.46	84.78
B4	1.26±0.20	0.19±0.03	16.54	0.98	16.89
B5	3.19±0.24	0.13±0.02	41.89	0.67	62.52
B6	2.16±0.12	0.01±0	28.36	0.05	567.2
B7	1.0±0.20	0.02±0.01	13.13	0.1	131.3
B8	3.21±0.80	0.05±0.01	42.15	0.25	168.6
B9	0.72±0.10	0.02±0.01	9.45	0.1	94.5
B10	3.73±0.90	0.04±0.01	48.98	0.21	233.2
B11	2.70±0.20	0.03±0.01	35.46	0.16	221.62
B12	0.39±0.10	0.05±0.02	5.12	0.25	20.48
B13	0.38±0.20	0.20±0.01	4.99	0.1	49.9
B14	3.25±0.12	0.04±0.01	42.68	0.21	203.2
B15	1.20±0.24	0.02±0.01	15.75	0.1	157.5
B16	1.11±0.20	0.03±0.01	14.57	0.16	91.06
B17	0.80±0.22	0.03±0.01	10.51	0.16	65.68
B18	0.58±0.12	0.05±0.01	7.62	0.25	30.48
B19	0.30±0.01	0.03±0.01	5.21	0.16	32
B20	1.02±0.24	0.05±0.01	13.39	0.25	53.56
B21	0.54±0.10	0.05±0.01	7.1	0.25	28.4
B22	3.46±1.20	0.07±0.02	45.44	0.36	126.22
B23	2.45±0.68	0.05±0.01	32.18	0.25	128.72
B24	3.67±0.90	0.05±0.01	48.2	0.25	192.8
B25	0.66±0.12	0.03±0.01	8.66	0.16	54.13
B26	0.49±0.22	0.04±0.01	6.44	0.21	30.67
B27	0.66±0.15	0.04±0.01	8.66	0.21	41.23
B28	3.45±0.86	0.11±0.02	45.3	0.56	80.89
B29	3.16±0.64	0.04±0.01	41.5	0.21	197.62
B30	0.28±0.01	0.10±0.01	3.68	0.52	7.07
B31	3.83±0.20	0.03±0.01	50.3	0.16	314.37
B32	0.77±0.12	0.04±0.01	10.11	0.21	48.14
B33	1.13±0.14	0.05±0.01	14.84	0.25	59.36
B34	1.0±0.20	0.03±0.01	13.13	0.16	82.06
B35	3.35±0.60	0.01±0	43.99	0.05	879.8
B36	4.75±0.80	0.05±0.01	62.38	0.25	249.52
B37	4.19±0.64	0.06±0.02	55.02	0.31	177.48
B38	0.05±0.01	0.07±0.01	0.65	0.36	1.81
B39	4.41±0.96	0.03±0.01	57.91	0.16	361.93
B40	3.30±0.78	0.01±0	43.33	0.05	866.6
B41	0.45±0.01	0.09±0.01	5.91	0.46	12.84
B42	0.70±0.20	0.11±0.02	9.19	0.56	16.41
B43	0.75±0.15	0.07±0.01	0.35	0.36	0.97
B44	3.91±0.50	0.12±0.01	51.35	0.62	82.82
B45	3.65±0.60	0.01±0	47.94	0.05	958.8
B46	0.21±0.01	0.09±0.01	2.76	0.46	6
B47	0.52±0.02	0.07±0.01	6.83	0.36	18.97
B48	0.40±0.10	0.11±0.02	5.25	0.56	9.38
B49	2.38±0.50	0.90±0.10	31.25	4.65	6.72
B50	2.45±0.20	0.22±0.03	32.18	1.13	28.48

Table 2: Mean concentrations of Se and Hg (µg/mL) in whole blood samples and corresponding molar concentrations and molar ratios, n=5

Mean Selenium levels in whole blood ranged from 0.05µg/mL to 4.75µg/mL with a mean of 1.94µg/mL while mercury levels ranged from 0.01µg/mL to 0.90µg/mL with a mean of 0.078µg/mL as shown in Table 2 randomly. Se and Hg were found in all the whole blood samples.

The mean molar selenium concentrations ranged from 0.35 nM to 62.38 nM with a mean of 25.26 nM, while Hg molar concentrations ranged from 0.05 nM to 4.65 nM with a mean of 0.38 nM. There was no correlation in molar concentrations of Se and Hg as Pearson correlation coefficient was 0.03. Hence molar concentrations of Se were far greater than molar concentrations of Hg. Significant statistical differences exist between molar concentrations of Se and Hg as $P= 1.02\times10^{-12}< 0.05$. This difference might be due to the fact that the residents are well nourished, and has the required amount of Se to meet the physiological requirement of the body. Moreover, Se is an essential element and is required for the proper functioning of the body. On the other hand, mercury is a hazardous element and may not be needed by the human body, but is found in the blood as a result of exposure to mercury through mining activities or diet (eating of sea fishes) the main source of mercury exposure.

From the Se:Hg molar ratios calculated, it was observed that the Se:Hg molar ratio ranged from 0.97 to 958.80 with a mean of 155.40. These ratios are far greater than 1, except subject B43 which recorded Se:Hg ratio of 0.97(less than 1). The ratio of subject B43 might be due to the fact that the subject is chronically deficient in Se and might be suffering from physiological stress of Se (malnutrition). Since 98% of the subject had Se:Hg ratio greater than 1, the null hypothesis can be rejected.

The high Se:Hg ratios observed in the district explains why cases of mercury poisoning was not reported among the residents of Wassa West district even though, several studies recorded high levels of mercury in environmental samples and staple food crops. A Se: Hg ratio of 1 is a level below which mercury toxicity is likely to occur and this ratio should be an important consideration for risk assessment (Burger J. G., 2013). However, one study argued that the practical implications of the modifying effect of Se on Hg toxicity are unclear because of the variability in toxicodynamics.

Selenium does bind to mercury and can confer some protection against mercury toxicity, while at the same time excess selenium compensates selenoenzymes synthesis for any selenium sequestered by mercury. However, selenium toxicity from supplementation is a concern. Also, the amount of selenium required to protect against any particular concentration of mercury is unclear, variations within individuals makes it difficult to use the mean Se:Hg molar ratio as a measure of reduced mercury toxicity(Burger J. G., 2013). Figure 1, however did not show any relationship between Se:Hg ratio and Hg molar concentrations.

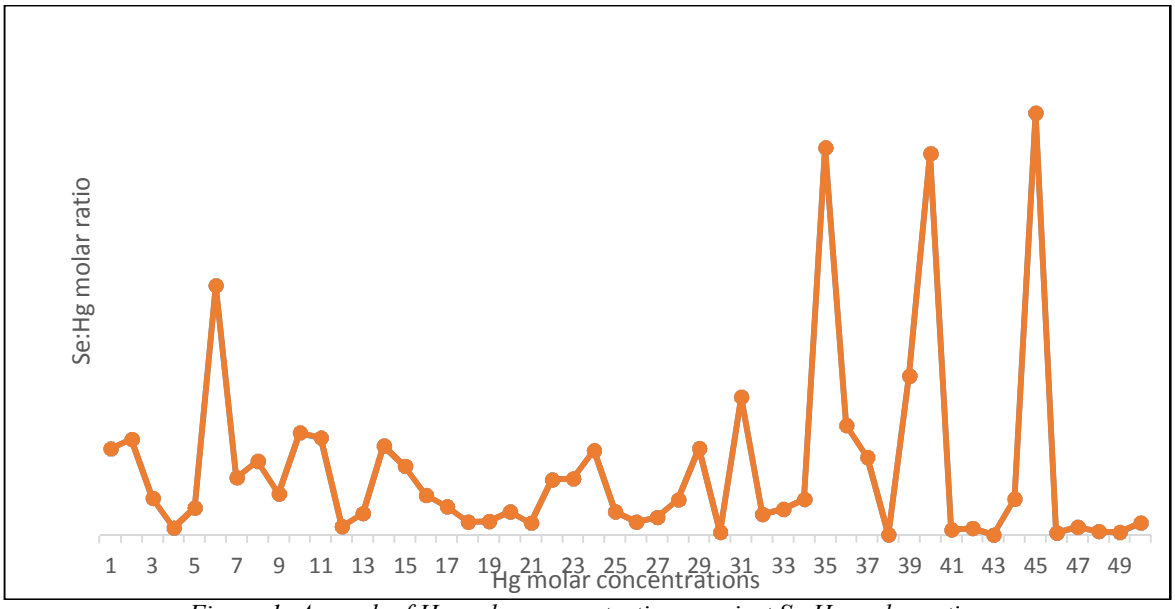

Figure 1: A graph of Hg molar concentrations against Se:Hg molar ratios

4. Conclusion

Generally, with the exception of one subject, which constitute 2%, the observed subjects all had Se:Hg molar ratio above one. This implied that although the residents are exposed to Hg, the higher levels of Se in their diet (well nourished) protects the people from Hg toxicity, hence no reported cases of Hg poisoning in the district

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