

Health Risk Assessment of Inorganic Arsenic Intake of Ronphibun Residents via Duplicate Diet Study

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Objective: To assess health risk from exposure to inorganic arsenic via duplicate portion sampling method in Ronphibun residents.

Material and Method: A hundred and forty samples (140 subject-days) were collected from participants in Ronphibun sub-district. Inorganic arsenic in duplicate diet sample was determined by acid digestion and hydride generation-atomic absorption spectrometry. Deterministic risk assessment is referenced throughout the present paper using United States Environmental Protection Agency (U.S. EPA) guidelines.

Results: The average daily dose and lifetime average daily dose of inorganic arsenic via duplicate diet were 0.0021 mg/kg/d and 0.00084 mg/kg/d, respectively. The risk estimates in terms of hazard quotient was 6.98 and cancer risk was 1.26×10^{-3} .

Conclusion: The results of deterministic risk characterization both hazard quotient and cancer risk from exposure inorganic arsenic in duplicate diets were greater than safety risk levels of hazard quotient (1) and cancer risk (1×10^{-4}).

Keywords: Inorganic Arsenic, Duplicate Diet, Risk Assessment, Ronphibun

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Human health problems in Ronphibun district were reported to the public when the first serious cases of keratosis and hyperpigmentation were diagnosed on a resident who suffered from arsenical skin cancer in 1987. Exposure to inorganic arsenic is a significant causal factor in human carcinogenesis and the development of a range of non-cancer effects in several countries. Assessing the risk on human health associated with inorganic arsenic intake from food and water is more important than total arsenic intake. Almost no information is available on the effects of organic arsenic compounds in humans. At present, risk assessment is based on exposure to inorganic arsenic only⁽¹⁻³⁾. Most of the data are on concentrations of arsenic in food; in several surveys on Ronphibun were usually presented using total organic arsenic rather than inorganic

arsenic compounds^(4,5). Uses of concentrations of total arsenic in food to assess risk may lead to over-estimation of the arsenic intake.

Food and water have been shown to be the major sources of arsenic exposure. Arsenic concentrations may differ between uncooked and cooked food. After cooking, most of the water evaporated but arsenic is still present and concentrated in the food and consequently an increase in the toxicological risk for the exposed population. Therefore, tests to assess risk by food consumption should take into account ready-to-eat foods. Other sampling methods for estimating the daily arsenic intake can not take into account the effects of the cooking process or the cooking water. To determine the actual intake, duplicate meal method is required⁽⁶⁻⁸⁾. In addition, there is little or no information regarding inorganic arsenic concentration in duplicate diet in this area.

The purposes of the present study were to estimate the inorganic arsenic intake and to conduct a

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risk assessment from consuming arsenic contaminated food by duplicate portion sampling method in adults who are living in an arsenic affected district of Ronphibun by deterministic approach.

Material and Method

Sample collection and preparation

The present study focused on four villages of Ronphibun sub-district including villages number 1, 2, 12, and 13 because almost all the patients that suffered from chronic arsenic-related diseases lived in these areas⁽⁹⁾. The present study used purposive sampling method for collecting samples from 20 people (10 males and 10 females) for 7 consecutive days. A hundred and forty samples (140 subject-days) of duplicate diet were collected from all participants. Participants received compensation before each sampling period. Participants collected equal amounts of food and beverages they consumed each day. Each diet sample was collected in separate plastic bags. Drinking water and beverages samples were collected from the present drinking water sources of each participant in clean bottles. After the collection phase, the samples were stored in a cold box and transported to the laboratory by train.

An interview was conducted to collect all exposure factors. A structured questionnaire included detailed questions on variables used to estimate intake such as: body weight, duration frequency, and exposure duration. The questionnaire had a reliability alpha coefficient value of 0.87. Administration of questionnaires was produced by staff for this research. Two hundred randomly selected people were successfully interviewed (128 females and 72 males; aged: 20-71 years; occupations: 90% farmer and 10% other occupations). As the migration population is significant, samples were collected from residents only if their length of residence in the area was more than 2 years. The features of arsenical toxicity appear gradually and slowly with time. Six months to 10 years (average 2 years) may be required for the development of clinical features⁽¹⁾. The present study was carried out between November 2006 and December 2007.

In the laboratory, inedible parts of the foods (*e.g.* bone, seeds of fruits) were discarded. Edible parts were minced and mixed in all duplicate diets (foods, beverages, and other materials intake) each day and was blended to give a homogeneous sample. The sample was weighed, frozen, freeze-dried, and stored in polyethylene bags until analysis. In the present study, beverages intake rate was not reported because

it was embedded in the expression used for amount of arsenic concentration in duplicate meal sample (mg/g, dry weight).

Determination of inorganic arsenic

The methods employed for determination of inorganic arsenic have been described by Munoz et al⁽¹⁰⁾ and Huang et al⁽¹¹⁾. Briefly, an accurate weight (0.5 g) of the lyophilized sample was placed into a 50 ml screw-top centrifuge tube. Five milliliters of deionized water was added and agitated until the sample was completely moistened. Then, 20 ml of concentrated hydrochloric acid was added and agitated again for 1 hour and left to stand overnight. The reducing agent (1 ml of 1.5% w/v hydrazine sulfate solution and 2 ml of hydrobromic acid) was added and agitated for 30 seconds. An amount of 10 ml of chloroform was added to the sample for a further agitation of 3 minutes. The phases were separated by centrifuging at 2,000 rpm for 5 minutes. It was separated the chloroform phase by aspiration and poured into another tube. The extraction process was repeated two more times. The chloroform phases were combined and centrifuged again. The remnants of the acid phase were eliminated by aspiration. Eliminate possible remnants of organic material in the chloroform phase were eliminated by passing it through a nylon filter. The inorganic arsenic in the chloroform phase was back-extracted by agitating for 3 minutes with 10 ml of 1 mol/l hydrochloric acid. The phases were separated by centrifuging at 2,000 rpm, and the aqueous phase was then aspirated and poured into a beaker, this stage was repeated once again and the back-extraction phases obtained were combined. When the back-extraction phase generated emulsions that could not be broken by centrifuging at over 2,000 rpm, the emulsion was transferred to the beaker. Nitric acid was added in the beaker and it was heated on a hot plate. The emulsion was then broken and the chloroform phase formed was removed by aspiration.

Ashing aid suspension (2.5 ml) and concentrated nitric acid (10 ml) were added to the combined back-extraction phases. Then, it was evaporated on a hot plate until total dryness and after cooling the residue was diluted in distilled water and filtered through filter paper (Whatman[®] no. 42). Next, the solution was transferred into a 25-ml volumetric flask and adjusted to volume with 5% nitric acid. Determination of inorganic arsenic was performed with a hydride generation-atomic absorption spectrometry (HG-AAS; Perkin Elmer[®] AAnalyst 300). The detection

limits for inorganic arsenic was 0.02 µg/g. Each sample was analyzed in triplicate and arsenic was calculated in milligram per gram of dry weight.

The validity of the analysis was confirmed by analyzing the standard reference materials (SRM) tomato leaves (SRM1573a). The SRM was obtained from the National Institute of Standards and Technology (NIST), USA. All chemicals were purchased from Merck® and the reagents were of analytical grade. Deionized water was used throughout the whole experiment for preparation of reagents and standards. The average recovery in spiked sample was 93.41% (n = 10).

Health risk assessment process

Several sources of information have described risk assessment. The present study is referenced throughout using the United States Environmental Protection Agency (U.S.EPA) guidelines^(3,12). The basic definition of risk assessment is a process in which information is analyzed to determine if a hazard might cause adverse effects to humans following exposure under defined conditions to a risk source⁽¹²⁾. U.S.EPA uses it as a tool integrate exposure and health effects information into characterization of the potential for health hazards in humans and uses risk assessment as a source of scientific information for making decisions about managing risks to human health and the environment. Risk assessment consists of four steps, namely hazard identification, dose-response assessment, exposure assessment, and risk characterization. U.S.EPA addresses the first two components (hazard identification and dose-response assessment) through its Integrated Risk Information System (IRIS) databases. Hazard identification is determined whether exposure to chemicals can cause an adverse effect and whether it effect is likely to occur in humans. In cases of inorganic arsenic, sufficient information shows that inorganic arsenic is producing widely adverse effects in humans and animals both non-carcinogenic and carcinogenic effects. In the dose-response assessment, it was performed based on data concerning relationship between exposure and adverse health effects. The results of dose-response assessment for ingested toxicants are expressed in terms of reference dose (RfD) for non-cancer effects and cancer slope factor (CSF) for cancer effects. The current RfD and CSF for ingested inorganic arsenic are based on Taiwan epidemiological studies. Taiwan studies have several strengths for quantitative dose-response assessment including the sample size of the exposed population,

which is large (40,412), the number of skin cancer and skin disorder cases are also relatively large⁽¹³⁻¹⁵⁾. Currently, RfD and CSF of ingested inorganic arsenic are 0.0003 mg/kg/day and 1.5 (mg/kg/day)⁻¹, respectively⁽²⁾. These values are used in risk characterization combined with exposure assessment information.

Exposure assessment

A description of exposure to the chemical is a very important component of risk assessment process. The objective of this step is to estimate the type and magnitude of exposures to the chemical for potential concerns that are present at a site. Exposure is dependent on the concentration of the contaminant, frequency and duration of contact. These are typically expressed in terms of concentration per dose or units in the media to which humans are exposed. The most common measures are the average daily dose (ADD), which is used to assess the non-cancer effects and the lifetime average daily dose (LADD) for cancer effects. Inorganic arsenic intake from duplicate diet in the present study was estimated by the following equations⁽¹²⁾:

$$ADD = \frac{C \times IR \times ED \times EF}{BW \times AT_{nc}} \quad (1)$$

$$LADD = \frac{C \times IR \times ED \times EF}{BW \times AT_c} \quad (2)$$

where: C is the concentration of inorganic arsenic in a duplicate diet (mg/g); IR is the ingestion rate (g/day); ED is the exposure duration (years); EF is the exposure frequency (days/year); BW is the body weight (kg); AT_{nc} is the averaging time, non-carcinogen (ED x 365 days/year); AT_c is the averaging time for cancer effects, equal to the life expectancy time (70 x 365 = 25,550 days). Arsenic concentration in a duplicate diet was assumed to be 100% of absorption because data on arsenic absorption via food has not been reported⁽¹⁾. Exposure parameters were evaluated from interviewed data. The principal exposure factors that have been taken into account to carry out the risk assessment calculations are presented in Table 1.

Risk characterization

Risk can be characterized according to several types of risk description. The present study has an estimated risk by using deterministic risk assessment only. A risk assessment in which deterministic or point estimate of risk is calculated from a set of single values for exposure and toxicity to represent variables in a

Table 1. Distribution of exposure parameters

| Statistical value | Exposure factor | | | | | |
|-----------------------------|-----------------|------------|------------|----------------|---------|-------------|
| | C (mg/g) | IR (g/day) | ED (years) | EF (days/year) | BW (kg) | ATnc (days) |
| Min | 0.00014 | 273.2 | 2 | 200 | 30 | 730 |
| Mean | 0.00028 | 335.91 | 28 | 350 | 58.26 | 10,220 |
| SD | 0.00006 | 39.19 | 17.13 | 30.62 | 10 | 6,252 |
| 5 th percentile | 0.00015 | 283.1 | 5 | 285 | 45 | 1,825 |
| 25 th percentile | 0.00023 | 299.4 | 15 | 353 | 51 | 5,475 |
| Median | 0.00027 | 311.3 | 24 | 365 | 57.5 | 8,760 |
| 75 th percentile | 0.00033 | 342.8 | 36.25 | 365 | 65 | 13,323 |
| 90 th percentile | 0.00036 | 385.6 | 55 | 365 | 70.2 | 20,075 |
| 95 th percentile | 0.00039 | 404.8 | 63 | 365 | 75 | 22,995 |
| 99 th percentile | 0.00041 | 437.5 | 68 | 365 | 80 | 24,820 |
| Max | 0.00042 | 469.4 | 71 | 365 | 90 | 25,915 |

risk equation. Risk characterization of non-cancer effects is evaluated by comparing an exposure level (ADD) with toxicity value (RfD). This ratio is called hazard quotient (HQ). If the calculated HQ is equal to or less than 1, the non-cancer adverse effect due to exposure pathway is assumed to be negligible while HQ more than 1 suggests that there may be concern for non-cancer effects. HQ is calculated as follows⁽¹²⁾:

$$HQ = \frac{ADD}{RfD} \quad (3)$$

For cancer effects, risk is estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen. Cancer risk (CR) is accepted in ranges 10^{-4} to 10^{-6} . In the present study, an acceptable risk of 1.0×10^{-4} was established for the population in the Ronphibun area that means only 1 of 10,000 people may have increased cancer effects. Cancer risk characterization can be estimated using the following equation⁽¹²⁾:

$$CR = LADD \times CSF \quad (4)$$

CR = Cancer risk

LADD = Life time average daily dose

CSF = Cancer slope factor

Results

Description of exposure factors

Concentrations of inorganic arsenic in duplicate meal samples ranged from 0.00014 to 0.00042 mg/g with a mean value of 0.00028 mg/g. The 95th

percentile of arsenic concentration, 0.00039 mg/g, was used for the estimated risk. An average amount of intake rate was 335.91 g/day (dry weight). Exposure duration was selected from an average value of 28 years with ranged 2 to 71 years and the average of exposure frequency was 350 days/year. U.S.EPA⁽¹²⁾ recommended a selected mean of body weight in calculation of risk for reason of toxicity evaluation. Mean body weight from the present data was 58.26, approximately 60 kg. Averaging time is fixed to 25,550 days for LADD estimation and ADD is equal to exposure duration multiplied by 365 days. These parameters characteristic are described in Table 2.

Evaluation of health risk

The ADD of inorganic arsenic via duplicate diet intake was 0.0021 mg/kg/day and LADD was 0.00084 mg/kg/day. Risk characterization of non-cancer effects from exposure to inorganic arsenic in term of HQ was 6.98. HQ greater than 1 indicted that risk is probably to result in any adverse health effects. The increased cancer risk of being exposed to inorganic arsenic by duplicate meal consumption according to equation 4 was 1.26×10^{-3} . In term of 1.26×10^{-3} means about 1 of 1,000 people may be at increased risk of cancer effects from the background. Cancer risk result exceeded the acceptable of 1 in 10,000 set in the present study. Daily intake by local residents can pose a potential health threat due to long term arsenic exposure. Table 3 summarizes the results of ADD, LADD, HQ, and CR. In conclusion, risk estimates of both non-cancer and cancer effects exceeded the risk level under study.

Table 2. Description of parameters used for estimating risk

| Parameter | Symbol | Units | Parameter characteristic |
|------------------------------------|--------|---------------------------|--------------------------|
| Concentration of inorganic arsenic | C | mg/g | 0.00039 |
| Ingestion rate | IR | g/day | 335.91 |
| Exposure duration | ED | years | 28 |
| Exposure frequency | EF | days/year | 350 |
| Body weight | BW | kg | 60 |
| Averaging time | AT | | |
| Cancer | ATc | days | 25,550 |
| Non-cancer | ATnc | days | 10,220 |
| Reference dose | RfD | mg/kg/day | 0.0003 |
| Cancer slope factor | CSF | (mg/kg/day) ⁻¹ | 1.5 |

Table 3. The outputs of exposure assessment and risk characterization

| Result | Non-cancer effects | | Cancer effects | |
|------------|--------------------|------|-------------------|-------------------------|
| | ADD | HQ | LADD | CR |
| Risk value | 0.0021 mg/kg/day | 6.98 | 0.00084 mg/kg/day | 1.26 x 10 ⁻³ |

Discussions

The major difference between the present study and previous studies in Ronphibun area is the use of inorganic arsenic contents in a duplicate diet rather than total arsenic to estimate exposure. Inorganic arsenic is considered to be the most toxic form and currently dose-response assessment was only based on exposure to inorganic arsenic. Water and foods are major potential sources of arsenic exposure in the arsenic-affected area but it is difficult to separate and specify the types of food and raw materials intake in the present study because the present study used replicate diet sampling for the purpose of actual intake. More research is needed to better understand the variation of inorganic arsenic in different types of food in this area. Three basic approaches for sampling food are used: individual food products; market basket studies; and duplicate diet portion. The duplicate diet approach is a direct sampling technique in which an exact duplicate of food being consumed is obtained and analysed⁽⁶⁾. Arsenic concentration may differ between cooked and uncooked food. Duplicate diet methods are considered to be more accurate at estimating personal exposures because they account for individual food and water sources, types and quantities of food items consumed, and cooking methods. It is important to note that the estimates

derived from duplicate diet studies depend on the dietary habits of participants in local areas and may not be generalized to other regions. In the present study, the impact of seasonal variation, the level of physical activity, or other factors on the intake rate in the population have not been adequately evaluated.

Table 4 shows a comparison of arsenic intake studied by the duplicate diet approach for seven days in various countries. Jorhem et al⁽¹⁶⁾ reported an average daily intake of total arsenic in 15 Swedish adults of $60.0 \pm 0.04 \mu\text{g/day}$. Mohri et al⁽¹⁷⁾ estimated daily intake of both total and inorganic arsenic in four Japanese adults living in Fukuoka being 27.0-376 and 1.8-22.6 $\mu\text{g/day}$, respectively. When compared to the values reported from other countries, the daily intake of total and inorganic arsenic by 20 adults residing in Ronphibun sub-district were relatively high. This can be explained that all of the reports from those countries were studied in uncontaminated areas.

The result of deterministic risk assessment in terms of HQ from exposure arsenic via duplicate diets was greater than 1 (HQ = 6.98) and CR was significantly greater than the safety levels of 1×10^{-4} (CR = 1.26×10^{-3}). This result only concerns the local residents in this area, not extended to people living in other regions of Thailand. However, the present results were similar to previous studies. The Ministry

Table 4. Arsenic daily intake by duplicate food approach for 7-consecutive days from other studies

| Country | n | Daily intake (µg/day) | | Reference |
|----------|----|--------------------------|---------------------------|------------------------------|
| | | Total | Inorganic | |
| Germany | 14 | 6.9 ± 12.4 (0.60-98.0) | nd | WHO ⁽¹⁵⁾ |
| Sweden | 15 | 60.0 ± 0.04 (<50-180) | nd | Jorhem et al ⁽¹⁶⁾ |
| Japan | 4 | 182.0 ± 114.0 (27.0-376) | 10.3 ± 5.5 (1.80-22.6) | Mohri et al ⁽¹⁷⁾ |
| Thailand | 20 | nd | 94.1 ± 23.5 (38.32-197.2) | Present study |

nd = not determined, Numbers in parentheses are ranges

of Public Health⁽¹⁸⁾ reported that the increased cancer risk from consumption of food and water in the Ronphibun site was 2.9×10^{-2} based on exposure duration of 20 years. Vitayavirasuk⁽¹⁹⁾ presented the cancer risk via drinking water in Ronphibun residents' 4.3×10^{-4} to 1.9×10^{-5} . In addition, Chantarawijit et al⁽²⁰⁾ had reported that the ranges of cancer risk from exposure to arsenic in drinking water was 4×10^{-2} to 5×10^{-4} and 4×10^{-3} to 8×10^{-4} from ingested arsenic via food. A health survey funded by Regional Office for South-East Asia, WHO (SEARO) in August 2000 estimated that approximately 6,120 of 24,566 potentially exposed subjects were showing symptoms of arsenicosis⁽²¹⁾. The metabolism of arsenic has an important role in its toxic effects. However, the exact mechanism of the action of arsenic is not known but several hypotheses have been proposed and the bioavailabilities of arsenic through consumption of cooked foods are not known. There is still a question about the risk to individuals who are exposed to arsenic, as well as the dose needed for adverse effects to develop. A definite understanding of the mechanism of action will allay any uncertainties associated with the risk assessment for arsenic⁽¹⁾.

Food crops may accumulate arsenic through root uptake from contaminated soil or water while animals can accumulate arsenic from contaminated feed, sediment and water. The possible mobilization of arsenic in the soil and subsequent leaching into ground or surface water or entry into the human food chain, should always be considered as a serious hazard. Most people in this area are agriculture workers. Agricultural foodstuffs were consumed by Ronphibun residents. Some foods may have highly accumulated arsenic and may thus represent a health risk. Bae et al⁽²²⁾ suggested that cooked food could be an important source of arsenic, if it is boiled with extensive arsenic contaminated water. Therefore, Ronphibun residents

should avoid drinking contaminants with a high level of arsenic in well water and they obtain drinking water and use water for cooking from other sources such as rainwater or bottled water. Numerous studies suggested that improvement of water quality, the rate of improvement in the symptoms and signs of arsenic poisoning in human beings may increase with a decrease in arsenic level in the drinking or cooking water source^(1,15).

The results of high risk estimate can be explained that the original problem of high arsenic accumulation in soil and water in this site has not been completely managed to solve the problem until today. Consequently, future generations of residents may be at risk because arsenic remains in the soil for hundreds of years. The suggestion for future studies could be as follows: economic of public health assessment and planning for uses of land to prevent the spread of arsenic to a wider environment.

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การประเมินความเสี่ยงต่อสุขภาพจากการได้รับสารหนูอนินทรีย์ของประชาชนที่อาศัยในร่อนพิบูลย์
ด้วยวิธี duplicate diet study

ปิยวัฒน์ สายพันธุ์, สุเทพ เรืองวิเศษ

วัตถุประสงค์: เพื่อประเมินความเสี่ยงต่อสุขภาพจากการได้รับสารหนูอนินทรีย์ที่ปนเปื้อนในอาหารของประชาชน
ในร่อนพิบูลย์

วัสดุและวิธีการ: เก็บตัวอย่างด้วยวิธี duplicate diet sampling จากประชาชนที่อาศัยในตำบลร่อนพิบูลย์ จำนวน
140 ตัวอย่าง วิเคราะห์ปริมาณสารหนูด้วยวิธีย่อยด้วยกรดและวิธี hydride generation-atomic absorption spectro-
metry คำนวณค่าความเสี่ยงตามวิธีประเมินของ United States Environmental Protection Agency (U.S. EPA)

ผลการศึกษา: ปริมาณที่ได้รับสารหนูเฉลี่ยและปริมาณที่ได้รับเฉลี่ยตลอดช่วงอายุ คือ 0.0021 และ 0.00084 มิลลิกรัม/
กิโลกรัม/วัน ตามลำดับ ค่าความเสี่ยงที่ประเมินได้มีค่า hazard quotient เท่ากับ 6.98 และ cancer risk เท่ากับ
 1.26×10^{-3}

สรุป: ความเสี่ยงจากการได้รับสารหนูอนินทรีย์ที่ปนเปื้อนในอาหารของประชาชน มีค่าสูงกว่าค่าความปลอดภัย
ที่กำหนด คือ hazard quotient เท่ากับ 1 และ cancer risk เท่ากับ 1×10^{-4}
