Original Article

Primary school children from northeast Thailand are not at risk of selenium deficiency

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Selenium has important roles as an antioxidant, in thyroid hormone metabolism, redox reactions, reproduction and immune function, but information on the selenium status of Thai children is limited. We have assessed the selenium status of 515 northeast Thai children (259 males; 256 females) aged 6 to 13 years from 10 rural schools in Ubon Ratchthani province. Serum selenium (n=515) was analyzed by Graphite Furnace Atomic Absorption Spectrophotometry and dietary selenium intake by Hydride Generation Absorption Spectrophotometry from one-day duplicate diet composites, from 80 (40 females; 40 males) randomly selected children. Inter-relationships between serum selenium and selenium intakes, and other biochemical micronutrient indices were also examined. Mean (SD) serum selenium was 1.46 (0.24) µmol/L. Concentrations were not affected by infection or haemoglobinopathies, but were dependent on school (*P*<0.001), sex (*P*=0.038), and age group (*P*=0.003), with serum zinc as a significant covariate. None of the children had serum selenium concentrations indicative of clinical selenium deficiency (i.e. < 0.1 µmol/L). Significant correlations existed between serum selenium and serum zinc (r= 0.216; *P* < 0.001), serum retinol (r = 0.273; *P* < 0.001), urinary iodine (r = –0.110; *P* = 0.014), haemoglobin (r = 0.298; *P* <0.001), and haematocrit (r = 0.303; *P*<0.001). Mean (SD) dietary selenium intake was 46 (22) µg/d. Children with low serum selenium concentrations had a lower mean selenium intake than those with high serum selenium concentrations (38 ± 17 vs. 51 ± 24 µg/d; *P*<0.010). In conclusion, there appears to be no risk of selenium deficiency among these northeast Thai children.

Key Words: selenium, serum, diet, zinc, children, Thailand.

Introduction

Selenium is an essential trace element for animals and humans, with important roles in antioxidant defense, thyroid hormone metabolism, and redox control of enzymes and proteins.[1] Severe endemic selenium deficiency has been associated with two diseases: a cardiomyopathy known as Keshan disease[2,3], and an endemic osteoarticular disorder known as Kashin-Beck disease.[4] Selenium responsive conditions, including cardiomyopathy, muscle pain, and muscular weakness, have also been described in some patients receiving long-term total parenteral nutrition (TPN), but no selenium supplements.[5] However, not all TPN patients with a low selenium status develop clinical symptoms of selenium deficiency, suggesting other interacting factors may be involved.[6]

Among children in developing countries, including Thailand, selenium deficiency has been associated with protein energy malnutrition.[7] The deficiency has been attributed to co-existing dietary inadequacies of protein and selenium, exacerbated by an increased need for selenium and other antioxidants induced by malnutrition and infection. Inter-relationships with other micronutrients, notably iodine[8-11] and to a lesser extent, zinc and iron[12-14], have also been reported, although to date only the

mechanism for iodine has been firmly elucidated. These micronutrient interactions may be important among children in northeast Thailand because several micronutrient deficiencies (i.e. iodine, zinc, iron, and vitamin A) have been documented among school-aged children in this region[15-19]; the poorest region in the country, with 28% of the population living below the poverty line compared with only 7% in central Thailand.[20]

To our knowledge, there have been no studies on the selenium status of Thai children. Therefore, we have assessed the selenium status of a sample of primary school children attending ten schools in rural northeast Thailand, based on serum selenium concentrations (n=515), and dietary selenium intakes analyzed from one-day diet composites collected from a subsample (n=80) of the children. Inter-relationships between serum selenium and biochemical indices of iodine, zinc, iron, and vitamin A status were also examined.

Subjects and methods

Subjects

The school children in this study participated in the baseline data collection of an intervention trial conducted between June 2002 and March 2003 in Ubon Ratchathani province, northeast Thailand. The school with the largest roll in each of the ten sub-districts of Trakarn Phutphon, Ubon Ratchathani province was selected for participation in the trial. All the districts are of low socio-economic status. Details of the subject selection have been published earlier.[19] Of a total of 567 children aged 6.0 to 12.99 y who participated at baseline, serum samples from 515 of these children were available for selenium analysis. Only 80 children (40 males; 40 females) were randomly selected for the collection of duplicate diet composites for selenium analysis, after stratifying the 567 children by age (grades 1 to 3; grades 4 to 6), sex, and treatment group.

The Human Ethics committees of the University of Otago (New Zealand) and Mahidol University (Thailand) approved the study protocol. Permission from local school boards and Thai health workers was also given, following meetings in which the purpose and methods of the study were clearly explained by one of the principal investigators (PW). Informed written consent was obtained from the parents or guardians of the participating children. Details of the socio-demographic, anthropometric, health, and haematological status of the children, prevalence of haemoglobinopathies, and their biochemical iron, zinc, iodine and vitamin A status have been reported earlier.[19,21]

Blood collection and serum selenium analysis

The blood collection and separation procedure has also been described elsewhere.[19,21] Briefly, morning non-fasting venipuncture blood samples were taken for selenium analyses using trace-element free evacuated tubes (Becton Dickinson, Franklin Lakes, NJ). Blood was refrigerated immediately after collection and separated within one hour using trace-element free techniques.[22] Serum was frozen immediately at –20°C in trace-element free polyethylene vials, and later at –70°C for subsequent biochemical analysis.

Serum selenium concentration was analyzed in duplicate by graphite furnace atomic absorption spectrophotometry (AA-800, Perkin-Elmer Corp, Norwalk, Connecticut, USA) in the Trace Element Laboratory, Department of Human Nutrition, University of Otago, based on a modified method of Jacobson and Lockitch.[23] Accuracy and precision of serum selenium analysis was assessed during each batch of analysis using two certified reference materials (CRM): UTAK Reference Plasma (Cat #66816, UTAK Laboratories Inc, Valencia, CA), and Seronorm (Lot #MI0181 Seronorm[TM] Trace Element Serum, Laboratories of SERO AS, Billingstad, Norway). The means ± SD (%CV) were 1.55 ± 0.08 µmol/L (CV 5.2%) (n= 62) for UTAK and 1.04 ± 0.03 µmol/L (CV 2.9%) (n=59) for Seronorm, compared to the corresponding certified mean and expected range given by the manufacturers of 1.52 (1.14–1.90) µmol/L and 1.05 (0.97–1.13) µmol/L, respectively. Multiple aliquots of a pooled serum control were also included with each batch of analysis to assess precision. The mean (±SD) and CV (%) for aliquots of this pooled serum was 1.25 ± 0.06 µmol/L (CV 4.8 %) (n=65) for selenium.

Haematological variables and biochemical indices of iron, zinc, vitamin A, and iodine

Concentrations of ferritin, transferrin receptor, C-reactive protein (CRP) (as an index of acute infection and inflammation), and retinol were also analyzed in serum using methods described earlier.[19,21] A sample of EDTA-anticoagulated blood was used for the haemoglobinopathy analysis and complete blood cell counts.[19] Casual urine samples were also collected for urinary iodine analysis.[21]

Collection and analysis of diet composites

Research assistants collected diet composites from the 80 children in such a way that all days of the week were represented. The diet composites consisted of an exact duplicate portion of all food and beverage items consumed by each child on the pre-selected day. Each portion was weighed and transferred into a weighed polyethylene jar with a wide mouth, lined with a trace-element free polyethylene bag. Each collection included the child's breakfast, lunch-time meal consumed at school, evening meal, and any snacks eaten during the day or night of the designated 24-hour period. Diet composites were weighed, and then frozen as soon as possible after collection at –20°C. Money was given to the parents of the children to reimburse them for the cost of the food.

Duplicate diet composites were blended in an acid-washed blender to yield homogenous slurries. After homogenization, four 50 mL aliquots of each slurry were transferred into weighed, acid-washed polyethylene jars, reweighed, and then frozen at –20°C. Each frozen aliquot was later freeze-dried to constant weight, and ground to a fine powder using an agate ball and mill (Brinkman Model 2MN, Brinkman Instruments Division, Sybron, Canada). One of the freeze-dried diet composites was contaminated by mould, so that only 79 diet composites were analyzed for selenium.

Duplicate samples of each of the freeze-dried powdered diet composites (approximately 0.3g) were acid-digested using a modification of the method of Friel and Ngyuen[24] and others.[25-27] To avoid contamination from adventitious sources of selenium during the digestion and analysis, sterile powder-free disposable plastic gloves were worn, and all glassware was soaked overnight in 10% high grade nitric acid, and then rinsed thoroughly six times in distilled, deionized water.

After digestion, the solutions were quantitatively transferred into acid-washed 25 mL volumetric flasks, and adjusted to volume with 20% hydrochloric acid. Aliquots were then analyzed for selenium by flow injection hydride generation atomic absorption spectro-photometry using an atomic absorption spectrophotometer (AA-800, Perkin-Elmer Corp, Norwalk, Connecticut, USA) in combination with an MHA-FIAS-400 flow injection hydride generation system and AS-90 plus autosampler. Standard solutions were prepared from selenium stock standard solutions (BDH Laboratory supplies) in the following concentrations: 2.5, 5, and 10µg/L. To check on the accuracy and precision of the analytical procedures, a

National Institute of Standards and Technology Standard Reference Material (SRM) Rice flour (SRM 1568a) (n=21) was analyzed. The mean ± SD (%CV) for SRM 1568a was 4.81 ± 0.25 nmol/g (5.0%) compared with the certified value (mean ± SD) of 4.81 ± 0.51 nmol/g.

Collection and analysis of selected food samples

Subsamples (n=3) of 13 foods, frequently consumed by the NE Thai school children, were purchased from local markets and vendors in the Trakarn Phutphon district. Edible portions of each subsample were then combined to form one composite sample per food type, frozen at – 20°C, and then freeze-dried to constant weight. For the analyses, each composite sample was ground to a fine homogenous powder, after which duplicate samples were removed for acid digestion followed by selenium analysis by flow injection hydride generation atomic absorption spectrophotometry, as described for the diet composites.

Statistical analyses

Statistical analyses were carried out using SPSS version 12 (USD Inc, Stone Mountain, GA). Data were checked for normality by using the Komogorov-Smirnov test. The Student's two-tailed t-test for non-paired data was used to test for differences by sex, haemoglobinopathy status (i.e., AA versus AE haemoglobin type), and infection (i.e., serum CRP ≥10 mg/L), and to test for differences in the selenium intakes of children with high (above median) and low (below median) serum selenium concentrations. Correlations between serum selenium and biochemical indices of iodine, zinc, iron, and vitamin A status were examined using Spearman's Rank Correlations test. Analysis of variance (ANOVA) was used to investigate whether serum selenium concentrations were dependent on sex, school, and age group when serum zinc was treated as a covariate. A P value of <0.05 indicated statistical significance.

Table 1. Spearman's correlation coefficients for relationships between serum selenium (µmol/L) and other biochemical variables

	n	r	P
Haemoglobin (g/L)	467	0.276	<0.001
Haematocrit	467	0.277	<0.001
Serum zinc (µmol/L)	446	0.216	<0.001
Serum retinol (µmol/L)	464	0.250	<0.001
Log serum ferritin (µg/L)	468	–0.031	0.497
Mean cell volume (fL)	471	0.093	0.044
Log urinary iodine (µg/L)	464	–0.131	0.005

NB. All children with serum CRP ≥10 mg/L were excluded from the above analysis.

Results

Serum selenium concentrations

Mean (SD) serum selenium concentration was 1.46 (0.24)

µmol/L. Serum selenium concentrations correlated positively and significantly with serum zinc, haemoglobin, mean cell volume, and serum retinol concentrations but negatively with urinary iodine. In contrast, serum selenium concentrations were independent of serum ferritin (Table 1). No significant correlations were found between serum selenium and socio-economic status, haemoglobin type (AA or AE), or infection status (i.e., CRP < or ≥10 mg/L).

Analysis of variance (Table 2) shows that serum selenium concentrations were dependent on school (P <0.001), sex (P = 0.038), and age group (P = 0.003), with serum zinc concentrations as a significant covariate (P <0.001). Females had a significantly lower mean serum selenium concentration than males (1.45 vs. 1.49 µmol/L) and children less than 9 years of age had a significantly lower mean serum selenium concentration than those greater than 9 years of age (Table 2).

Dietary selenium intakes and selenium content of selected food samples

Mean (SD) dietary selenium intake was 46 (22) µg/d. Females tended to have a slightly lower mean intake of selenium than males (41 (3.5) vs. 51 (3.5) µg/d; P = 0.05, n = 79), but this tendency disappeared when intakes were expressed per kg body weight. No significant associations with school or age were apparent. The mean dietary selenium intake of children with low serum selenium concentrations (below median value) was significantly lower than the mean for children with high serum selenium concentrations (above the median value) (mean ± SD; 38 ± 17 vs. 51 ± 24 µg /day; P<0.010).

The selenium content of locally grown glutinous rice was very variable, and ranged from 2.8 to 28 µg/100g dry weight. Several foods rich in selenium were identified, including frogs (boiled) (Se concentration = 232 µg/100g dry weight), silkworms (fried) (Se concentration = 49.0 µg/100g dry weight), grasshoppers (fried) (Se concentration = 25.2 µg/100g dry weight), mushrooms (boiled) (Se concentration ranged from 62.4 to 608.6 µg/100g dry weight), and bamboo shoots (boiled) (Se content = 29.6 µg/100g dry weight). In contrast foods such as swamp cabbage (boiled) and hairy basil leaves (boiled) were low in selenium, with levels of 3.2 and 3.5 µg/100g dry weight, respectively.

Discussion

There have been very few studies on the selenium status of population groups in Thailand, and to our knowledge, this is the first report on the selenium status of Thai children. Our results indicate there is no risk of selenium deficiency among these northeast Thai children, based on selenium concentrations in serum. Indeed, for 61% of the children (n=312), serum levels were indicative of maximal activity of plasma glutathione peroxidase (GSHPx) and selenoprotein P, and for 38% (n=194), levels were above those suggested as necessary to protect against some cancers, based on the cut-offs for adequate selenium status by Thomson.[28] The mean serum selenium concentration of these NE Thai children (1.46 µmol/L) was higher than those reported for children in Poland[29] and from national surveys conducted in New Zealand[30] and

the United Kingdom[31] (Table 3). Dietary selenium intakes for NE Thai children were also higher than those for children in New Zealand[32], and the other countries[33], with the exception of the United States[34] (Table 4). It is of interest that despite the higher selenium intakes of the

Table 2. Univariate analysis of variance; dependent variable = serum selenium (µmol/L)

Source	Type III sum of squares	df	F	Sig
Corrected model	3.649	12	8.431	<0.001
Intercept	18.654	1	517.276	<0.001
School	2.236	9	6.891	<0.001
Sex	0.156	1	4.319	0.038
Serum zinc	0.781	1	21.658	<0.001
Age group	0.312	1	8.649	0.003
Error	15.615	443		

Adjusted r squared = 0.189

	Estimated marginal means*		
		95% Confidence interval	
	Mean	Lower bound	Upper bound
Sex = female	1.451	1.425	1.476
Sex = male	1.488	1.463	1.513
Age group = < 9y	1.443	1.416	1.469
Age group = > 9y	1.496	1.472	1.520

* Evaluated at serum zinc = 9.84 µmol/L

U.S.children, their mean serum selenium concentration[35] was slightly lower than those of the NE Thai children. It is possible that differences in the bioavailability of selenium from children's diets in the United States and NE Thailand may be responsible for this discrepancy. Selenium from seleno-methionine, the major source in plant foods, is better retained in body tissues than other forms of selenium due to its non-specific incorporation into tissue protein in place of methionine.[1,36] It is likely that the proportion of selenium from plant foods in the diets of NE Thai children is higher than that in US diets.

It is noteworthy, that despite the relatively small number of duplicate diet composites analyzed, a relationship between serum selenium and selenium intakes was noted: children with high serum selenium concentrations had a greater mean selenium intake than those with low serum selenium concentrations. Several[37-40] but not all[41,42] investigators have reported a positive relationship between serum/plasma selenium and selenium intakes.

In this study, a positive relationship was noted between school and serum selenium concentrations but not with school and selenium intakes. This discrepancy may be associated with variations in the major food sources of selenium by school, and thus bioavailability of selenium, despite similar intakes of total selenium. Unfortunately, knowledge of the bioavailability of selenium from selenium-rich indigenous foods such as frogs and insects analyzed in this study is limited. In general, absorption of selenium from food is high, about 80%, although there is some evidence that in mushrooms it may be poor.[43]

We also collected one-day weighed diet records from the children on the day of the diet composite collection. However, it was not possible to identify the major food

Table 3. Comparison of serum selenium concentrations of children from selected countries

Country	Reference	Methodology	Age (sample size)	Serum selenium concentration (µmol/L)	
				Mean	SD
Thailand	This study	Se concentrations determined in Northeast Thai children by GFAAS	6–12 years (n=515)	1.46	0.24
USA (NHANES III)	35	Se concentrations determined in NHANES III (1988–1994) population by AAS	4–16 years (n=5305)	1.48	0.18
UK (NDNS)	31	Se concentration determined in NDNS by ICP-MS	4–18 years (n=1127)	0.87	0.15
New Zealand (NCNS)	30	Se concentration determined in NCNS (2002) population by GFAAS	5–14 years (n=1547)	0.97	0.67
Poland	29	Se concentration determined in healthy Polish population (1990–1991) by the flurometric method	7–15 years (n=74)	0.46	0.09

GFAAS – graphite furnace atomic absorption spectrometry; HGAAS – hydride generation atomic absorption spectrometry; ICP–MS – inductively coupled plasma mass spectrometry; NHANES III – the National Health and Nutrition Examination Survey; NDNS – National Diet and Nutrition Survey; NCNS – National Children's Nutrition Survey

Table 4. Comparison of selenium intakes (µg/day) of children from selected countries

Country	Reference	Dietary methods	Age (sample size)	Selenium intake (µg/day)	
				Mean	SD
Thailand	This study	One-day diet composite	6–12 years (n=79)	45.9	22.4
USA (NHANES III)	34	24-hour dietary recall	6–11 years (n=3134)	96.0	1.7
New Zealand (NCNS)	32	24-hour dietary recall	5–14 years (n=3275)	37.1	48.1
Papua New Guinea	33	Interactive 24-hour diet recall	6–10 years (n=67)	20.0*	–
Malawi	33	3-day weighed intakes	4–6 years (n=62)	15.0*	–

* median;   NCNS – National Children's Nutrition Survey

sources of selenium from these diet records because of limited data on the selenium content of indigenous foods in northeast Thailand[44]; levels depend on local soil selenium levels. Instead we identified several foods rich in selenium, based on our analyses of foods frequently consumed by these NE Thai children. The richest food sources of selenium were certain species of mushrooms and frogs, followed by silk worms, bamboo shoots, and grasshoppers. The selenium content of the major food staple of these NE Thai children, glutinous rice, was very variable. Reasons for this discrepancy are uncertain. It is possible that when the glutinous rice is rinsed and then soaked overnight prior to steaming, some selenium is lost by leaching. Alternatively, some of the purchased rice samples may have been cooked at the same time with foods rich in selenium (e.g., mushrooms or frogs), and thus become contaminated with selenium.

Inter-relationships between selenium status and biochemical indices of zinc, iron, vitamin A, and iodine status
The existence of a strong positive correlation between serum selenium and serum zinc (r= 0.22; $P<0.001$) has been reported earlier in both animal and human studies.[45-48] The finding that the gene expression of GSHPx, a selenoprotein, can be up-regulated by zinc[49] supports the assumption implicit in the ANOVA shown in Table 2. Selenium may also be linked to the role of cellular Zn in redox reactions.[50,51] Selenium compounds regulate the delivery of zinc from metallothionein (MT) to zinc enzymes, specifically copper, zinc superoxide dismutase. Alternatively, the strong positive correlation between serum zinc and serum selenium may be because protein-rich foods contain high levels of both zinc and selenium.[52]

In this study we also noted positive correlations between serum selenium and haemoglobin (r=0.30; $P< 0.001$) and haematocrit (r=0.30; $P <0.001$), but not with the biochemical iron indices MCV and serum ferritin (Table 1). These findings suggest that a factor other than iron may be responsible for the positive correlation with haemoglobin. This factor may be vitamin A, because of the positive association between serum selenium and serum retinol (r=0.27; $P<0.001$) noted here, and our earlier observation of an inter-relationship between serum retinol and haemoglobin among these NE Thai children.[19] Very few other studies in animals or humans have observed any relationship between selenium and vitamin A

status (based on serum retinol), although in a study of adults in Singapore, a positive relationship between serum selenium and serum retinol concentrations was reported after adjusting for age.[53] In summary we suggest that there may be no direct relationship linking serum selenium and haemoglobin.

Both selenium and iodine are required for thyroid hormone synthesis, activation, and metabolism.[8] Hence, it is not surprising that some[54,55] but not all[56-59] researchers have reported a high prevalence of Iodine Deficiency Disorders among populations with a relatively low selenium status. In our study, however, we showed a negative correlation between serum selenium and log urinary iodine concentrations (r = –0.11; $P = 0.014$). In some animal studies, relatively high intakes of selenium have been associated with low urinary iodine concentrations,[60] but these findings have not been confirmed in human studies.

In conclusion, these NE Thai children were not at risk of selenium deficiency, and indeed for about a third of the children serum selenium levels were above those suggested to protect against some cancers. Some highly significant, positive inter-relationships were observed between serum selenium and zinc, haemoglobin, haematocrit, and retinol, although the mechanisms involved and the significance of these inter-relationships is uncertain.

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Original Article

Primary school children from northeast Thailand are not at risk of selenium deficiency

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泰國東北部的國小學童沒有硒缺乏的危險

不論是甲狀腺荷爾蒙代謝、氧化還原反應、生殖及免疫作用，硒都扮演抗氧化劑的重要角色，但是有關於泰國學童硒營養狀況方面的資訊卻相當有限。我們評估515名(259男生；256女生)6歲到13歲居住在泰國東北部Ubon Ratchthani城的十個鄉村學校的學童。血清硒使用石墨爐式原子吸收光譜法(Graphite Furnace Atomic Absorption Spectrophotometry) 分析， 飲食硒 攝取量的評估則是隨機選取80名學童(40名男生；40名女生)採用 Hydride Generation 收光譜分析複製一天飲食的混合樣本。血清硒、飲食硒攝取量及其他生化微量營養素指數之間的相關性也同時檢視。血清硒濃度平均值(標準差)為1.46(0.24) μmol/L，其濃度不受感染或是血紅素病變影響，但會受就讀學校($P<0.001$)、性別($P<0.038$)及年齡組別($P=0.003$)影響，血清鋅是顯著的共變項。沒有任何一個學童的血清硒濃度顯示有臨床上硒缺乏的現象(亦即$<0.1\mu mol/L$)。血清硒與血清鋅(r=0.216；$P<0.001$)、血清視網醇(r=0.273；$P<0.001$)、尿碘(r=-0.110；$P=0.014$)、血紅素(r=0.298；$P<0.001$)及血比容(r=0.303；$P<0.001$)有顯著的相關。飲食硒平均(標準差)攝取量為46(22) μg/天。 血清 硒濃度 較低的 學童比起血清硒濃度較高的學童其硒平均攝取量較低(38 ± 17 vs. 51 ± 24 μg/天; $P<0.010$)。總而言之，此研究顯示在泰國東北部的學童沒有硒缺乏的危險。

關鍵字：硒、血清、飲食、鋅、兒童、泰國。