Short Communication

Iodized salt supplementation and its effects on thyroid status amongst Orang Asli in Hulu Selangor, Malaysia

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Background: This research was performed to determine the prevalence of iodine deficiency disorder (IDD) and the effects of iodized salt supplementation on thyroid status amongst Orang Asli in Hulu Selangor, Malaysia. Methods: Study respondents were from three target groups, i.e. pre-school children (PSC), primary school-going children (SGC) and adult women. Each household was supplied with iodized salt fortified with iodate fortificant for a period of 12 months and the iodine levels in the salt ranged from 20 to 30 µg/L. Samples collected before and after 6 and 12 months of introduction to iodized salt were urine from all groups, as well as serum samples from adult women. Results: A total of 200 respondents were recruited; 58 (29.0%) PSC, 65 (32.5%) SGC and 77 (38.5%) adult women. The median urine-iodine concentration (mUIC) in all groups were of moderately low before the iodized salt intervention, but increased significantly in all study groups after 6 and 12 months of intervention. However, at the end of the study, there was an increase in severe iodine deficiency (mUIC <20 µg/L) from 7.5% to 12% and about 9% of PSC and SGC respondents had mUIC level of more than 300 µg/L while the adult women showed a significant increase in free triiodothyronine (fT3) levels. Conclusion: The study demonstrated that iodized salt supplementation was able to show an improvement in iodine level amongst Orang Asli. However, an increase in severe iodine deficiency and iodine excess indicated that the iodized salt programme needs to be carefully monitored.

Key Words: iodine deficiency disorder, Orang Asli, iodized salt, hyperthyroidism

INTRODUCTION

Iodine is an essential mineral for normal growth and development of the foetus, infant, child and for the normal physical and mental activity of adults. It presents in the body in minute amount, mainly in the thyroid gland.[1] Inadequate iodine can lead to insufficient production of thyroxine (T4) and triiodothyronine (T3) hormones, which may affect many different parts of the body, particularly the muscle, heart, liver, kidney and developing brain. These effects are collectively termed as iodine deficiency disorders (IDD) and are common in human being.[2] It was estimated that 1.9 billion people from 130 countries live in areas where the soil is iodine-deficient and iodine-rich seafood is unavailable or not part of the local diet. This can lead to thyroid function abnormalities, and if the deficiency is severe, it will lead to endemic goitre, cretinism, mental retardation, reduced fertility and increased perinatal and infant mortality.[3,4] When an iodine-deficient mother gives birth to an infant, the newborn will also be deficient in iodine and may develop neonatal chemical hypothyroidism which may be permanent suggesting that iodine supplementation is vital to all adult women.[5] Studies done from many countries around the world showed that children in iodine-deficient areas suffer from poor hand-eye coordination and have 10 to 15 intelligence quotient (IQ) points less than children who obtained enough iodine in the diet.[6]

A teaspoon of iodine is all a person required in a lifetime and iodized salt programme is one of the most common tools in the fight against IDD.[2] The recommended daily intake of iodine for children (6–12 years) and adults above 12 years are 120 ug/L and 150 ug/L, respectively.[7] Although majority of individuals can tolerate a wide range of dietary iodine levels, a small group of individuals may develop thyroid dysfunction and autoimmunity upon exposure to elevated or even normal levels of iodine. As a result, they will start making too much thyroid hormones, a condition called iodine induced hyperthyroidism or IIH.[8]

Over the past century, there have been sporadic reports on the high prevalence of iodine deficiency (ID), with occasional observation of cretinism among Orang Asli from remote areas in Malaysia.[9] The level of socio-

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economic development such as the building of highways and roads into the interior areas, had taken place over the last 20 years, and made outside food including seafood more widely available to communities living in once remote and inaccessible areas. However, pockets of mild IDD can still be found among these communities in a few states in Malaysia.[10,11]

In 1999, a study was conducted to determine the problem of ID amongst Orang Asli living in Hulu Selangor, Malaysia and revealed that this area is still being threatened with serious iodine deficiency problem despite their close proximity to urban areas.[12] This implies that there is a need to conduct additional research to better assess the present iodine nutrition problem in this area and to study the use of iodized salt supplementation as an potential alternative strategy to improve the IDD status amongst Orang Asli communities in Hulu Selangor. The objectives of this study are to determine the prevalence of ID and the effects of iodized salt supplementation on thyroid status amongst Orang Asli pre-school children (PSC), primary school-going children (SGC) and adult women in semi-urban areas in Hulu Selangor.

MATERIALS AND METHODS

This intervention study amongst Orang Asli was conducted from November 2005 to June 2007 in four selected areas in Hulu Selangor, Malaysia (Kg. Pertak, Kg. Gerachi, Kg. Kuala Kerling and Kg Sg. Jang). These areas are located between eight to 25 km from the Kuala Kubu Bharu town and approximately 90 km from Kuala Lumpur. This locations were selected based on high prevalence of IDD reported in a previous study.[12] For expedience, all Orang Asli pre-school children (≤6 yearr), primary school-going children (7–12 years) and adult women aged 15 years and above in these areas were recruited in the study.

The respondents were divided into three target groups, i.e. pre-school children (PSC) aged less than 7 years, primary school-going children (SGC) aged between 7–12 years and adult women aged more than 1 year. The iodized salt was fortified with fortificant in the form of iodate rather than iodide with an iodine level of 20 to 30 µg/L.[13] To prevent iodine lost, the iodized salt were delivered directly by specially arranged transportation from the iodized salt importer warehouse. The presence of iodine in salt was tested using Rapid Test Kits for iodate and iodide developed by the Institute for Medical Research, Malaysia.[14] Respondents were given iodized salt for their daily food preparation. Each household was provided with one kg of iodized salt every two months for a period of twelve months. All respondents were advised not to use salt other than those provided in their daily household use. During the study period, follow-up visits were done at two-monthly interval to ensure the iodized salt provided was being used.

The respondents were excluded from the data analysis if they were unable to participate in all the three data collection activities (at baseline, 6 months and 12 months of iodized salt supplementation) or if they became pregnant during the study period. The study protocol was reviewed and approved by the Medical Review and Ethics Committee, Ministry of Health Malaysia. Approval was also obtained from the Department of Orang Asli Affairs and cooperation was sought from the local health authority.

Data for adult women were obtained through interviews, analysis of blood and urine iodine, while the data for pre-school children (PSC) and primary school going children (SGC) were obtained through analysis of urine iodine. Spot casual urine samples were collected in screw cap test tubes while the 10 ml blood samples of venous blood were collected into plain tubes. The blood serum was separated in the Hospital Kuala Kubu Baru laboratory at the end of every single day. All samples were properly packed and labeled, kept in polystyrene boxes containing ice before transported to the laboratory in the Nutrition Unit at the Institute for Medical Research (IMR) for analysis. The samples were kept in a minus 20°C freezer prior to the analysis.

Iodine concentration in the urine was determined by the Sandell-Kolthoff reaction method, via digestion with ammonium persulfate at 90–110 °C and catalytic reduction of ceric ion by arsenite salt.[15] TSH status was determined using commercial DSL-10-5300 ACTIVE® TSH ELISA, with a sensitivity of 0.01 µIU/mL and normal reference range of 0.3–3.0 mU/L.[16] Free T4 status was determined by commercial DSL-10-40100 ACTIVE® free T4 EIA with a sensitivity of 0.05 ng/dL, and reference range of 9.0-24.0 pmol/L.[17] Commercial DSL-10-41100 ACTIVE® Free T3 EIA was used to determine free T3 status. The fT3 procedure has a sensitivity of 0.05 pg/mL and reference range of 2.5–5.3 pmol/L.[18]

Statistical analysis was carried out using SPSS version 18.0**.** Since data on urinary iodine, TSH, free T4 and free T3 were skewed, results were presented as median and inter quartile ranges. Differences in median urinary iodine, TSH, free T4 and free T3 before and after iodized salt supplementation were evaluated using Wilcoxon signed rank test and, differences in distribution of TSH, free T4, free T3 were evaluated using McNemar chi-square test. Two-tailed *p* values less than 0.05 were taken as significant.

RESULTS

A total of 200 respondents (57.8%) participated in the three data collection activities (before, 6 months and 12 months of iodized salt supplementation respectively). Out of these, 58 respondents (29.0%) were PSC, 65 (32.5%) were SGC and 77 (38.5%) were adult women. The means for age in the PSC, SGC and adult women were 4 (SD=1.4), 9 (SD=1.5) and 33 (SD=15) years respectively.

Prior to salt iodization intervention, the median urinary-iodine concentration (mUIC) amongst PSC, SGC and adult women were 49.2 µg/L, 47.8 µg/L and 39.0 µg/L, respectively. After 6 and 12 months of iodized salt intervention, the mUIC for all study groups had increased significantly ($p<0.05$). The results of the study showed the mUIC amongst adult women respondents at baseline was the lowest compared to PSC and SGC respondents. After 6 months of iodized salt supplementation, the mUIC increased significantly. The mUIC levels reduced drastically after 12 months, though it was significantly higher than the baseline mUIC. The distribution of low urine-iodine concentration (UIC <100 µg/L) amongst the respondents had decreased from 89% (at baseline) to about 70% after

Table 1. Median and distribution of urinary iodine concentration (μg/L) amongst PSC, SGC and adult women

Respondents	Urinary iodine concentration (μg/L)						
	Median (Q1– Q3)	Frequency (%)					
		<20 (severe)	20-49 (moderate)	50-99 (mild)	100-199 (optimal)	200-299 (excess)	≥300 (excessive)
PSC (n=58) †							
Baseline	49.2 (35.1 – 76.2)	2 (3.4)	27(46.6)	19 (32.8)	9 (15.5)	0 (0.0)	1 (1.7)
6 Months	64.2 (41.9 – 149)	2 (3.4)	18 (31.0)	17 (29.3)	13 (22.4)	7 (12.1)	1 (1.7)
12 months	69.8 (40.4 – 121)	5 (8.6)	17 (29.3)	16 (27.6)	11 (19.0)	4 (6.9)	5 (8.6)
SGC (n=65) †							
Baseline	47.8 (32.9 – 70.5)	2 (3.1)	33 (50.8)	24 (36.9)	6 (9.2)	0 (0.0)	0 (0.0)
6 Months	61.1 (39.0 – 114)	2 (3.1)	23 (35.4)	22 (33.8)	13 (20.0)	3 (4.6)	2 (3.1)
12 months	67.4 (34.8 – 136)	7 (10.8)	19 (29.2)	18 (27.7)	12 (18.5)	3 (4.6)	6 (9.2)
Adult women (n=77) †							
Baseline	39.0 (26.6 – 55.0)	11 (14.3)	43 (55.8)	17 (22.1)	6 (7.8)	0 (0.0)	0 (0.0)
6 Months	65.9 (43.4 – 107)	1 (1.3)	27 (35.1)	28 (36.4)	14 (18.2)	2 (2.6)	5 (6.5)
12 months	50.2 (27.9 – 95.6)	12 (15.6)	26 (33.8)	21 (27.3)	14 (18.2)	3 (3.9)	1 (1.3)
All Group (n=200)							
Baseline	45.1 (31.8 – 67.4)	15 (7.5)	103 (51.5)	60 (30.0)	21 (10.5)	0 (0.0)	1 (0.5)
6 Months	62.4 (42.0 – 115)	5 (2.5)	68 (34.0)	67 (33.5)	40 (20.0)	12 (6.0)	8 (4.0)
12 months	58.8 (34.4 – 116)	24 (12.0)	62 (31.0)	55 (27.5)	37 (18.5)	10 (5.0)	12 (6.0)

† Wilcoxon signed ranks test of median urinary iodine concentration:
Baseline vs. at 6 or at 12 months after iodized salt supplementation, $p<0.05$.

6 and 12 months of iodized salt intervention. Although the percentage of UIC <100 μg/L decreased in trend, the percentage of severe iodine deficiency (UIC <20 μg/L) and excessive iodine levels (UIC ≥300 μg/L) increased after 12 months of iodized salt supplementation amongst PSC and SGC groups. For the PSC respondents, the percentage of severe iodine deficiency and excessive iodine levels were 3.4% and 1.7%, at the baseline respectively. However, severe iodine deficiency level increased to 8.6% after one year of iodized salt supplementation. The similar pattern was observed in the SGC respondents. Amongst the adult women, the distribution of severe iodine deficiency and excessive iodine levels remained essentially unchanged during the study period (Table 1).

Table 2 shows the median of TSH, free T4 and free T3 levels amongst adult women, the median TSH level prior to salt iodization intervention was 1.3 mU/l and decreased significantly after 6 and 12 months of salt iodization supplementation. The medians of free T4 levels and free T3 at baseline were 13.3 pmol/l and 2.0 pmol/l, respectively; and increased significantly after 6 and 12 months of iodized salt supplementation.

Based on table 3, it was obvious that prior to salt iodization supplementation, only 1.3% of the adult women respondents had low TSH levels. However, after 6 and 12 months of the iodized salt intervention, the respondents with low levels of TSH had increased to 5.2% and 3.9%, respectively. There was a marked increase in free T4 level after 6 months of iodized salt supplementation in adult women. Nonetheless, this improvement of free T4 levels was not sustainable after another 6 months. However, the free T3 levels showed a significant increment after 6 and 12 months of the iodized salt intervention.

Table 2. Median of TSH, free T4 and free T3 amongst adult women (n=77)

hormone	median	Q1– Q3	*p*
TSH			
Baseline	1.30	0.8 – 2.1	
6 Months	0.92	0.6 – 1.3	0.001†
12 months	1.01	0.7 – 1.3	0.001‡
Free T4			
Baseline	13.3	12.2 – 14.3	
6 Months	18.5	14.6 – 21.1	0.001†
12 months	16.9	13.7 – 19.3	0.001‡
Free T3			
Baseline	2.0	1.6 – 2.6	
6 Months	2.4	1.7 – 3.2	0.001†
12 months	4.7	3.9 – 5.7	0.001‡

Wilcoxon signed ranks test of median TSH. free T4 and free T3:
†Baseline vs at 6 months after iodized salt supplementation
‡Baseline vs at 12 months after iodized salt supplementation

DISCUSSION

The study areas with an overall mUIC of 45 μg/L before the supplementation of the iodized salt would be classified as areas of moderate iodine deficient.[7] This level of mUIC was similar to those found in other Orang Asli communities close to town.[19] Although our study areas are not remote, the IDD prevalence was similar to the prevalence reported in most remote Orang Asli communities.[20] The levels of iodine intake amongst the respondents were low, which was exacerbated by low levels of iodine in drinking water in those areas.[21] Besides the lack of iodine in drinking water and food, these communities were also threatened by goitrogens in their daily food.[22]

Our study shows more than 70% of the population in Hulu Selangor remained iodine deficient and there was an increase in severe iodine deficiency from 7.5% to 12%. This might exert greater impact on mental and physical development amongst the population, especially in pregnant and lactating women.[7] Evidence is emerging that even mild iodine deficiency in pregnant women would affect child development in later life and the most serious consequences is the child being born a cretin.[23] The population in the study areas was exposed to inadequate iodine

Table 3. Distribution of TSH, free T4 and free T3 amongst adult women (n=77)

hormones	Sub-normal		Normal	*p*
	Low (%)	High (%)	Optimal (%)	
TSH	<0.3 mU/L	>3.0 mU/L	0.3–3.0 mU/L	
Baseline	1 (1.3)	8 (10.4)	68 (88.3)	
6 Months	4 (5.2)	5 (6.2)	68 (88.3)	1.000†
12 months	3 (3.9)	3 (3.9)	71 (92.2)	0.549‡
Free T4	<9.0 pmol/L	>24.0 pmol/L	9.0–24.0 pmol/L	
Baseline	1 (1.3)	1 (1.3)	75 (97.4)	
6 Months	1 (1.3)	9 (11.7)	67 (87.0)	0.021†
12 months	1 (1.3)	6 (7.8)	70 (90.8)	0.180‡
Free T3	<2.5 pmol/L	>5.3 pmol/L	2.5–5.3 pmol/L	
Baseline	56(72.7)	0 (0.0)	21 (27.3)	
6 Months	40 (51.9)	4 (5.2)	33 (42.9)	0.043†
12 months	8 (10.4)	23 (29.9)	46 (59.7)	0.001‡

McNemar chi-square test of thyroid hormones (normal vs sub-normal):
†Baseline vs at 6 months after iodized salt supplementation
‡Baseline vs at 12 months after iodized salt supplementation

intake even with the provision of iodized salt. The results from our study were comparable with a study from Thailand, which reported iodized salts programme failed to improve the iodine status among their community.[24] Nevertheless, it was contradictory with many other studies. The use of iodized salt appears to be an effective method of choice for IDD eradication programmes and many studies have demonstrated the rapid increase in UIC amongst the communities living in IDD areas.[25–27]

A number of reasons could be postulated to explain the inconsistent findings in the present study. Firstly, the lack of compliance amongst the respondents could one of the plausible reasons as it is very difficult to control intake of iodized salt individually. It was uncertain that whether respondents consumed their iodized salt as regularly as reported earlier.[28] Secondly, the iodine content of the salt may reduced by leaching under tropical conditions and humidity in the house.[29] Another possible factor leading to low levels of iodine in the urine could be attributable to the common habit of frequent cassava consumption amongst the Orang Asli communities.[30,31] Finally, the loss of iodine could also be due to the cultural habit (common in Asian societies) of adding salt to food during - rather than after cooking.[32]

In moderate to severe ID endemic areas, susceptible individuals may expose to the risk of developing iodine-induced hyperthyroidism (IIH) when their dietary iodine increase.[33] There have been reports of possible IIH in Zimbabwe and Zaire.[34,35] Our study shows that about 9% of the PSC and SGC respondents had excessive levels of iodine (mUIC >300 μg/L) at the end of the present study. The finding warranted follow-up for the development of IIH and if serum samples can be taken from the affected individuals, a more significant result may be seen.

The analysis of serum thyroid hormones amongst adult women failed to reveal any significant improvements in TSH and free T4. However, excess free T3 was found in 30% of respondents, although the majority of them had normal level of TSH. This suggested that iodine supplementation might have accelerated the development of hyperthyroidism and significantly increased the risk of hyperthyroidism though the iodine intake was fairly increased. The disagreement between the two IDD indicators need to be interpreted with caution by taking into consideration of the wide normal reference ranges.[36]

This was inconsistent with a study in Iran which reported that after ten years of iodine supplementation, the mUIC, serum T4, T3 and TSH concentration were within the normal range.[37] In Sri Lanka, after salt iodination was made compulsory in 1993, there was an increase in mUIC, free T4 and free T3 in all study respondents.[38]

Besides, these results were in agreement with the finding from a study that indicated the transition from IDD to sufficient or excess iodine intake was associated with the emergence of thyroid autoimmunity, especially in young women. This indicated that exposure to excess iodine may trigger thyroid autoimmunity.[39] Another study on the effects of iodine intake on the prevalence of thyroid dysfunction and autoimmune concluded that iodine supplementation in Turkey resulted in the elimination of IDD in Eastern Black Sea Region. However, this has been accompanied by an increase in the prevalence of autoimmune thyroiditis and thyroid dysfunction.[8]

Conclusion

The results of this study showed that the use of iodized salt had increased the iodine status amongst Orang Asli in Hulu Selangor, as indicated by mUIC levels. However, the unfavourable result of the study is that iodine intake was associated with a slight increase in severe ID and excess iodine level amongst the study population. This finding provides important evidence for an urgent need to investigate the circumstances leading to this situation. Also, our data will be useful for comparison purposes in future studies.

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ETHICAL CONSIDERATIONS

Ethical issues (Including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely observed by authors.

AUTHOR DISCLOSURES

The authors declare that they have no conflicting of interests.

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馬來西亞 Hulu Selangor 地區原住民碘鹽補充及其對甲狀腺狀況影響

背景：這個研究的執行是為了評估馬來西亞 Hulu Selangor 地區的原住民(Orang Asli)碘缺乏失調(IDD)的盛行率及碘鹽補充對於其甲狀腺的影響。方法：研究的受訪者來自於三個目標族群，即學齡前兒童、小學學童及成年女性。提供每個家戶為期 12 個月含碘酸鹽的強化碘鹽，食鹽中的碘含量從 20 至 30 μg/L。收集所有組別其碘鹽使用前及使用後 6 個月及 12 個月的尿液，成人女性加收血清樣本。結果：總共有 200 名受訪者被納入研究；58 名學齡前兒童(29.0%)、65 名小學學童(32.5%)和 77 名成年女性(38.5%)。在碘鹽介入前，所有組別的尿碘濃度中位數(mUIC)稍低，但是在介入 6 個月及 12 個月後，所有組別都顯著上升。然而，在研究結束時，嚴重碘缺乏(mUIC <20 μg/L)從 7.5%增加至 12%，但約 9%的學齡前兒童及小學學童受訪者 mUIC 超過 300 μg/L，同時成年女性游離三碘甲狀腺素(fT3)的量顯著增加。結論：此研究證實碘鹽的補充可以改善原住民的碘狀況。然而，嚴重碘缺乏及碘過多比例增加，顯示碘鹽計畫需要被小心嚴謹地監測。

關鍵字：碘缺乏失調、馬來半島原住民、碘鹽、甲狀腺機能亢進