The impact of an iodine deficiency disorders control program in West Sumatra, Indonesia

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A cross-sectional study on iodine deficiency disorders (IDD) status was conducted in a known endemic area where three types of IDD intervention (iodized oil capsule, iodized salt and iodinated water) were employed. A total of 238 children of 8–10 years of age from eight public elementary schools were included in the study. In addition to selected socio-economic and anthropometric data, output (iodine level in salt, iodine content in drinking water, iodized oil coverage) and outcome (goiter by palpation, urinary iodine excretion (UIE) concentration) were assessed. The total goiter prevalence (all were in grade 1) was 19% (mild IDD). The median UIE concentration was 193.5 μg/L (iodine-replete condition). Iodized oil capsule coverage was 61%, and 55% of those children received their latest capsule less than 1 year prior to the time of the study. Iodine level in salt was 14.4 ± 9 p.p.m. The iodine level in iodine supplemented drinking water was 11.7 ± 8.2 μg/L, while in surface water it was 12.2 ± 4.7 μg/L. Goiter was not associated with any of the three types of iodine supplementation, while UIE level was significantly associated only with iodized salt ($P < 0.001$), which suggested that, despite some problems in the universal salt iodization program, iodized salt was the most effective agent of the IDD control program at the community level. However, more research is needed to better understand the impact indicators of IDD control programs.

Key words: iodine deficiency disorder, Indonesia, iodized oil capsule, iodized salt, iodinated water, goiter, urinary iodine excretion.

Introduction

The broad range of iodine deficiency consequences was denoted as Iodine Deficiency Disorders (IDD), which include thyroid function abnormalities, and when iodine deficiency is widespread, in particular mental retardation impedes national human resource development. It is suggested that this problem still affects at least one billion people in the world, despite the known effective control measures. Therefore, iodine deficiency constitutes one of the most common preventable causes of mental deficiency in the world today.[1,2]

Iodine deficiency disorders are still prevalent in Indonesia, despite an IDD control program having been established for approximately 20 years. According to estimates, the iodine intake of about 42 million of Indonesia's inhabitants, which is approximately 17% of the population, is insufficient.[3] However, as part of the National Plan of Action for Nutrition, undertaken during the last 3 years in Indonesia, the IDD control program has received new input.[4] The program consisted mainly of three strategies: namely, the direct supplementation of iodized oil capsules in severe and moderate IDD areas; the universal iodization of salt for the whole population; and an iodinated drinking water program, which was introduced in some provinces.[3] However, the population has been exposed to these three strategies at varying levels of intensity. As a result, it is not known which one of these strategies is the most effective in reducing IDD. The following study aimed to describe the output and outcome of the Indonesian IDD control program in relation to school-aged children in an endemic area in which all three strategies of the program were employed.

Materials and methods

Study site and subjects

The study was carried out in Solok district of West Sumatra province. The study site is a hilly area at an altitude of 400–500 m above sea level. The area is 194.22 km^2 with 21 314 inhabitants in 4248 households. Agriculture is the main activity for living, with rice (*Oryzae sativa*), chilli (*Capsicum annum*), onion (*Allium cepa*) and candlenut fruit (*Aleurites moluccaua*) being the main produce. Public transportation facilities are available throughout the entire year.

The area was chosen based on the 1982 IDD mapping data, which showed a total goiter rate (TGR) among women in this area of 39–87%.[5] The most recent survey, conducted in 1993 as part of the baseline survey on the effectiveness of the iodized oil capsule and which covered five provinces, found that TGR in West Sumatra was the highest at 56.7%.[6] The IDD control program commenced at this site in 1974 using iodized oil injections for mothers. In 1992, iodized oil injection was replaced by iodized oil capsule with a wider target group, including school age children. The current IDD

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control program includes yearly distribution of iodized oil capsules for school children (two capsules with 200 mg iodine per capsule); iodized salt (current regulation is 40 ± 10 p.p.m. of KIO_3); and iodinated drinking water, achieved by adding one drop of iodine solution containing 250 µg iodine into 2 L of drinking water. Iodinated drinking water was introduced in early 1993. The surveyed villages did not receive the same type of IDD intervention program because they were involved in a study on the effectiveness of iodized oil capsules versus iodinated water (Table 1).

The study was designed as a cross-sectional survey involving 238 children aged 8–10 years from villages belonging to one health center catchment area. Eight out of the nine villages were chosen randomly and the existing public elementary school in each village was used for the recruitment of the children. In the case of the existence of more than one school in a village, a school was chosen randomly. The name and age of each child was obtained from the school's list, with the subjects being chosen randomly from the list.

Data collection and analysis

Goiter and urinary iodine excretion (UIE) levels were assessed to determine the IDD status of the population. Goiter was determined using the palpation method by one medical-trained assistant and graded according to WHO/ UNICEF/ICCIDD guidelines.[7] Urine samples were collected from casual urine samples and processed using the Sandell-Kollthof acid digestion method to determine the iodine level in urine, as described in Dunn *et al.*[8] Analysis of UIE was performed in the IDD laboratory at the University of Diponegoro, Semarang.

The coverage of the iodine supplementation was assessed by asking the children for administration of iodized oil capsules. Samples of drinking water and salt were obtained from each child and the iodine content was determined. Iodine concentrations in supplemented drinking water were assessed using a similar method to that of determining the UIE level and analyzed in the same laboratory. Furthermore, samples of surface water, such as from rivers, lakes, and wells, were collected to assess iodine content. Iodine concentration in salt was measured using the titration method.[9]

School children were weighed with an electronic platform-model weighing scale (SECA 770 alpha; SECA, Hamburg, Germany). The weight was recorded to the nearest 0.1 kg. Height was measured to the nearest 0.1 cm using a microtoise (CMS Weighing Equipment Ltd. London, UK). Anthropometric indicators of malnutrition were obtained by computing the Z-score of weight-for-height (WHZ), height-for-age (HAZ) and weight-for-age (WAZ) using the US National Center for Health Statistics reference data to correct for effects of sex and age on growth.[10] A result of –2 Z-score was considered as malnutrition, as recommended by WHO.[11,12]

Data processing and analysis

Data were entered and analyzed using SPSS for Windows 6.0.1 (SPSS Inc., Chicago, IL, USA). Anthropometric indices were calculated using EPI INFO 6.0 (CDC Atlanta, GA, USA), and values of more than – 3.9 WHZ, HAZ, and/or WAZ were excluded from the analysis. Differences in prevalence data between groups were tested using Chi-squared test, while differences in mean values were tested using ANOVA or Student's *t*-test. Urinary iodine excretion data were log-transformed in order to normalize distribution. Iodine levels in drinking water which were more than 50 µg/L were excluded from the analysis. A probability level of 5% was used to test for significant results. Multiple regression technique was used to create a model to predict UIE levels based on independent variables representing iodine supplementation (iodized oil capsule coverage) and iodine fortification (iodine level in salt and drinking water), anthropometric status, age, and gender. Categorical and ordinal data were put into dummy variables before entering regression

Table 1. Description of the iodine deficiency disorder control program related to the observed villages

Village	Type of intervention	Target group	Year of commencement
Murapi Utara	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	1991/1992
Murapi Selatan	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	1991/1992
Saning Bakar	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	1991/1992
Kampung Tangah	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	1991/1992
Gando	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	1991/1992*
	Iodized water	All population	1994/1995
Koto Baru Tambak	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	1991/1992*
	Iodized water	All population	1994/1995
Parumahan	Iodized salt	All population	1976
	Iodized oil capsule	Male (0–20 y) and female (0–35 y)	only in 1994/1995
	Iodized water	All population	1992/1993
Subarang	Iodized salt	All population	1976
	Iodized water	Male (0–20 y) and female (0–35 y)	only in 1994/1995
	Iodized oil capsule	All population	1992/1993

Note: all villages received an iodized oil injection between 1974–1991. *, Ceased at time of the study.

analysis. Components were entered in a stepwise fashion using 5% level of significance.

Ethical considerations

The international ethical guidelines for epidemiological studies were used for this study.[13] Study design and protocol were approved by the Committee of the SEAMEO-TROPMED Regional Center for Community Nutrition. Children as well as their parents and teachers gave informed consent, both oral and written. Participation in this study was voluntary. Consent was obtained from 238 participants and their guardians.

Results

Of the 238 children examined, 53% were boys and 47% were girls. The age distribution of 8, 9, and 10 years was 35, 30, and 35%, respectively. The prevalence of stunting, underweight, and wasting was 36, 31, and 5%, respectively. There was no statistically significant association between the anthropometric status and gender or age (data not shown).

Goiter prevalence and median UIE levels were similar among villages, while the three types of iodine supplementation were different. As presented in Table 2, according to palpation, 19% of children were identified as having goiter and all of these were classified as being Grade 1. Goiter was not associated with either age or gender of the subject ($P = 0.22$ and 0.10, respectively; Chi-squared test). Urinary iodine excretion concentrations indicated no significant public health problem, as shown by median value of 193.5 μg/L (range 36–528 μg/L), and the fact that only five subjects (2.1%) had UIE values below 50 μg/L. There was no statistically significant difference in the urinary iodine excretion between those with and without goiter ($P = 0.302$; Student's *t*-test).

In the households of 74% of the school children the iodized salt showed levels < 30 p.p.m. KIO_3 (17.8 p.p.m. of iodine), while in two of the households no iodine was detectable in the salt, according to the titration method. The mean value of iodine content in salt was 14.4 ± 9.0 p.p.m. Figure 1 shows that there were two salt samples from households with levels > 50 p.p.m.

In total, 61% of children responded to having taken iodized oil capsules (YODIOL; Kimia Farma, Jakarta, Indonesia). Among those who had received a capsule, 80 subjects (55%) received their latest capsule less than 1 year prior to the time of this study.

According to Table 3, the iodine level in surface water was 12.2 ± 4.7 μg/L and did not differ not significantly from the iodine supplemented drinking water ($P = 0.82$; Student's *t*-test). The survey area was included in the iodinated drinking water program, according to which a 125 μg KIO_3/L drinking water (KIO_3: LARUTAN IODIUM, Kimia Farma, Jakarta, Indonesia) was introduced directly into the household. Only four samples (1.7%) had concentrations above 125 μg iodine/L (Fig. 2). The average value of iodine levels in drinking water was 11.7 ± 8.2 μg/L.

There was no association between goiter and the iodine level in salt ($P = 0.16$; Student's *t*-test); goitre and receiving of an iodized oil capsule ($P = 0.64$; Chi-squared test); or goitre and the iodine level in drinking water ($P = 0.16$; Student's *t*-test). Multiple regression analysis was employed to investigate factors contributing to UIE level of subjects (Table 3). Independent variables included were the frequency of receiving an iodine capsule, the most recent time a capsule was taken, iodine content in drinking water and salt, gender, age, height, weight and goiter grade. Only iodine content in salt showed a low but significant statistical association to the

Table 2. Iodine deficiency disorders (IDD) indicators of the observed area

IDD indicator	Prevalence
Goiter rate (grade 1)	18.9%
Median urinary iodine excretion level	193.5 μg/L
Iodine level in salt (mean ± SD)	14.4 ± 9 p.p.m.
Claimed taking iodized oil capsule	61.3%
Iodine level in drinking water (mean ± SD)	11.1 ± 6 μg/L
Iodine level in surface water (mean ± SD)*	12.2 ± 5 μg/L

*, Samples were taken from rivers, lakes, springs and wells.

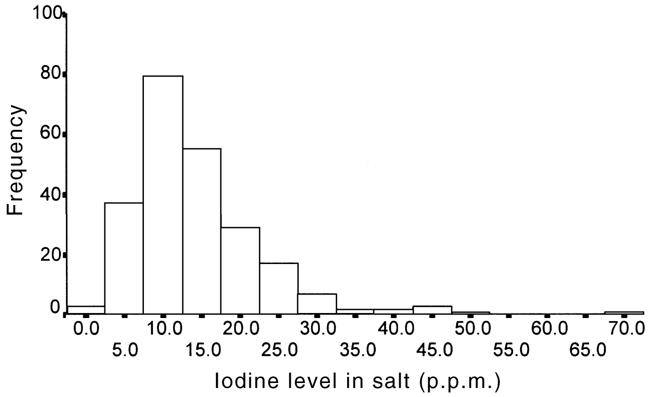

Figure 1. Frequency distribution of iodine level in salt (mean ± SD = 14.4 ± 9 p.p.m.; $n = 236$).

Table 3. Multiple regression for factors contributing to urinary iodine excretion level

Variable	Partial regression coefficient	*P*
Iodine content in salt	0.003	0.0022
(constant)	1.029	0.0000

Note: stepwise procedure, multiple $R = 0.21$.

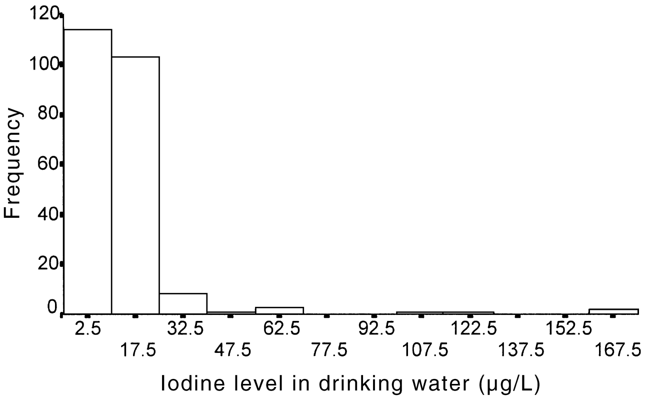

Figure 2. Frequency distribution of iodine level in drinking water (mean ± SD = 11 ± 6 μg/L; $n = 233$).

UIE level of subjects ($R^2 = 0.21$; $P = 0.002$; multiple regression analysis).

Discussion

Considering its TGR of 19% and median UIE level of 193.5 µg/L, the area studied can be categorized as having a mild IDD problem among school age children, according to WHO/UNICEF/ICCIDD standards.[7] The observed prevalence of goiter was lower than the national figure in 1990, which was 27.7%.[3] A study conducted in 1993 for the baseline data on the effectiveness of iodized oil capsules, which covered five provinces, showed that West Sumatra was in the highest rank of goiter prevalence among school age children 6–10 years, as shown by a TGR of 56.7%.[6] Although these studies are not fully comparable with that of 1993 due to differences in sampling, they may indicate a remarkable reduction of goiter prevalence after 3 years of the renewed efforts of the IDD control program.

The lack of an association between goiter and urinary iodine concentration can be explained by the fact that urinary iodine excretion reflects recent iodine ingestion and can change in days with a variation in dietary iodine, while goiter reflects a longer past exposure to iodine deficiency.[14] In particular, in countries in which the iodine status is improving due to an effective IDD control program, a higher discrepancy between both indicators must be expected.

Only 26% of the salt samples obtained from the households of surveyed children contained the amount of iodine recommended by the Indonesian government ($KIO_3 \geq 30$ p.p.m. or iodine ≥ 17.8 p.p.m.). This finding was lower than the results of the 1996 National Socio-Economic Survey, which showed that 58% of households had consumed salt with KIO_3 levels > 30 p.p.m.[15] However, the result of this study was even lower than that of a report of the Ministry of Industry,[16] which stated that 89.62% of households in West Sumatra consumed salt which was adequately iodized. The relatively high availability of labeled salt containing iodine in households indicated the high availability of labeled salt at market level and the high awareness of the population of IDD.

The iodine content in surface water was as high as in the iodine supplemented drinking water, suggesting that the implementation of the iodination program for drinking water had had a limited coverage. This fact was confirmed by the result of the multifactorial regression which showed a lack of effectiveness of the iodination of drinking water. Inadequate iodine concentration of iodine solution itself might be another reason for the low level of iodine in drinking water.

The lack of a statistical association between goiter or UIE and iodine supplementation with capsules may have been for several reasons. First, it is quite likely that the school children over-reported the reception and consumption of capsules. Second, the information about the time of administration of the capsules may have not been reliable. It may well have been that the time span was too long after the administration to be related to the urinary iodine content. Since most of the children were reported to have received a capsule more than 3 months prior to the time of the study, it is doubtful that the iodine status supplied by iodized oil would have been maintained constant in the body, if there were not other contributing iodine sources.[17]

The stepwise multivariate analysis revealed that only iodine content in salt showed a significant relationship with UIE level. Iodine is rapidly absorbed through the gut, and excess of iodine is excreted by the kidneys. As a result, the level of excretion correlates well with the level of intake.[18] When an adequate iodine level in the body is reached, any additional iodine intake will be excreted through the kidneys, as reflected by an increased UIE level. In this study, an increased urinary iodine concentration could only be explained by the intake of salt, but not by the iodination of water or the intake of iodine capsules.

Considering the iodine concentration in drinking water and salt, the contribution of water to the iodine intake was 28 µg/day (water intake of 2 L/day for school children), while the iodine intake from salt was 72 µg/day (with the assumption of salt intake of 5 g/day for school children), resulting in a total of 100 µg/day.[19] Given that the daily requirement of a school aged child on average can be met with this amount, it is quite reasonable that under these circumstances, UIE reflected a satisfactory individual supply of iodine.

Surprisingly, despite several iodine sources and higher urinary iodine levels, there is still a considerable goiter prevalence in the study area. Although iodine deficiency can be regarded as the major cause of endemic goiter, its degree does not always account for the severity of the endemia.[20] Three factors may be responsible. First, goitrogenic substances in food and bacterial pollution of water supply,[21,22] selenium deficiency,[23–26] vitamin A deficiency, and protein energy malnutrition[27,28] are additional factors that can contribute to goiter, in particular when the iodine supply is close to the borderline of the requirement. Second, given that the area was known as an IDD pocket with a higher percentage of goiter cases, it cannot be discounted that the surveyors had been biased in their assessments when palpating the children. Third and most likely, in high IDD areas where iodine intake is improving, it may well be that the iodine status reflected by urinary iodine content is changing faster than is indicated by goiter. Furthermore, it may well be that the iodine supply in preschool aged children is significantly lower than in school aged children, due especially to lower salt consumption in their diets. Therefore, the goiter prevalence may reflect a past situation not only in regard to the increased effectiveness of a iodination program among the whole population, but also within the life span of individuals.

Bearing in mind that this small study cannot represent the whole country, its findings suggest that, where conditions are similar to those of West Sumatra, salt iodination appears to be the most effective means of addressing IDD at the community level. However, more research is needed to understand the output and outcome indicators of IDD control programs.

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碘缺乏症控制規劃在
印度尼西亞Sumatra西部的效應

摘要

在實施三種碘缺乏症（IDD）預防措施（碘化油膠囊，碘化鹽和碘化水）的碘缺乏症流行區進行了碘缺乏症現狀的橫斷面調查。來自八所國立小學的238名8－10歲兒童參與了這項調查。除了調查選擇的社會經濟和人體測量方面的資料外，對碘缺乏症預防措施的實施（碘化鹽 和碘化水中碘的含量，碘化油的覆蓋率〕以及結果（甲狀腺腫大觸及程度，尿排泄碘（UIE）的濃度）也作了評價。總的甲狀腺腫大患病率（I度）是19%〔輕度IDD〕，尿排泄碘（UIE）濃度的中位數爲193.5毫克/升〔碘飽和狀態〕。碘化油膠囊的覆蓋率是61%，其中55%的兒童接收最后一次膠囊不超過一年。碘化鹽中碘的含量是14.4 ± 9 ppm。碘化飲水中碘的含量是11.7 ± 8.2μg/L，而地面水的含量則是12.2 ± 4.7μg/L。甲狀腺腫大患病率與上述三種碘補充實施没有任何聯系。可是尿排泄碘（UIE）的水平與碘化鹽 顯著相關（P<0.001）。盡管大衆化碘鹽規劃存在着一些問題，可這一調查則顯示了碘鹽是社區性控制碘缺乏症最有效的措施。當然，需要對控制碘缺乏症作用指征有進一步的認識以便 更好地理解其含義。

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