Endemic goitre in a rural community of KwaZulu-Natal

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Objective. To quantify the prevalence of goitre and iodine deficiency.

Setting. Ndunakazi, a rural community of approximately 8 000 people in KwaZulu-Natal.

Design. A cross-sectional community-based survey and a school-based survey.

Participants. The 127 mothers and 114 children aged 6 - 11 years, selected during the cross-sectional survey, and 304 children aged 6 - 14 years, from the school-based survey.

Methods. Urinary iodine levels and thyroid size were determined and categorised according to guidelines proposed jointly by the WHO, UNICEF and the ICCIDD. Z-score anthropometric indicators were calculated, and mid-year exam marks of goitrous and non-goitrous pupils for Zulu and mathematics were compared.

Results. In school-aged children, both surveys demonstrated a goitre prevalence in the 20 - 29.9% range and a median urinary iodine level in the 2 - 4.9 µg/dl range, indicating iodine deficiency of moderate severity. Goitrous subjects scored consistently worse in their Zulu exam papers than those without goitre. Stunting was not more prevalent than in the rest of KwaZulu-Natal. Iodised salt was not available in any of the three community shops.

Conclusion. This level of iodine deficiency in children can adversely affect their neuropsycho-intellectual development. Factors contributing to deficient iodine intake in Ndunakazi are present in many rural areas, and South Africa cannot afford to be overly confident about the apparent absence of iodine deficiency as a public health problem.

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The World Health Organisation and the World Bank estimate that 1.6 billion people worldwide, concentrated mainly in developing communities, live in iodine-deficient environments.[1,2] Depending on the extent to which the physiological requirements of iodine are not met in a population, a whole spectrum of iodine deficiency disorders (IDD) can result. Iodine deficiency is considered the leading preventable cause of intellectual impairment, and even mild iodine deficiency can insidiously reduce IQ by 10 - 15%, without any clinical signs of IDD.[3,4] More severe levels of iodine deficiency can cause abortions, stillbirths, increased perinatal and infant mortality, cretinism, goitre, hypothyroidism and decreased fertility.[4] The cumulative effect of these conditions can eventually cause socio-economic stagnation.[5]

Goitre is usually the most obvious sign of iodine deficiency and is almost invariably the result of inadequate dietary iodine intake.[4-6] A total goitre rate of $\geq$ 5% in primary-school children (endemic goitre) increases the likelihood of other IDD in a community.[7] Median urinary iodine levels of < 10 µg/dl indicate iodine deficiency and a public health problem in need of correction.[5,7] Once goitre rates exceed 30% in school-aged children and pregnant women, and median urinary iodine excretion declines to less than 2 µg/dl, the risk of hypothyroidism, cretinism in the community, and mental and physical retardation becomes significant.[5]

In southern Africa, endemic goitre has been reported from Lesotho, Swaziland, Botswana,[8] Mozambique,[9] Zimbabwe[10] and Namibia[11] within the last decade. It is disturbing to note that the current prevalence of endemic goitre in South Africa is not known. The last extensive survey, published in 1955,[12] reported the existence of geographical 'pockets' of IDD ranging from the Western Cape across to the east coast to Mpumalanga, across the former Transvaal from east to west and down to the Northern Cape. Since then only one clinic-based[13] and one hospital-based[14] study during the 1970s have reported endemic goitre in South Africa.

The aim of this study was to quantify the prevalence of goitre and iodine deficiency in a cross-sectional community-based survey and in a school-based survey in a rural area of KwaZulu-Natal.

Subjects and methods

Ndunakazi is a rural community of approximately 8 000 people in the Valley of a Thousand Hills, KwaZulu-Natal. In the 1 000 households there are approximately 600 primary school attenders. This study was based on the observations of two consecutive surveys conducted in the same community by the National Research Programme for Nutritional Intervention of the Medical Research Council. In the first of these a cross-sectional survey was undertaken as part of a community nutritional survey of Ndunakazi. A 25% random sample of all mothers (or childminders) with children aged 6 - 11 years in their care was selected (127 mothers and 149 children). In the second survey all children aged 6 - 11 years attending Ndunakazi Primary School (N = 252), as well as an additional 30% of children aged 12 - 14 years (N = 54), were randomly selected. Although a

omprehensive assessment of the study population's utritional status was made, only data relevant to the ssessment of endemic goitre in school-aged children and eir mothers are reported here. The MRC Ethics ommittee, KwaZulu-Natal Department of Education, and e Ndunakazi Primary Health Care Committee approved is study and informed consent was obtained from all arents.

Trained nutritional monitors recorded the age, sex and ocality of all subjects. Calibrated electronic scales were tilised to assess body weight and anthropometers to etermine body height. Z-score anthropometric indicators were derived from the international growth reference.[15] In all subjects 6 years or older, iodine status was assessed on the basis of goitre prevalence and urinary iodine levels, according to the joint World Health Organisation, United Nations Children's Fund and International Council for the Control of Iodine Deficiency Disorders (WHO/UNICEF/ICCIDD) indicators.[7] A medical doctor palpated all subjects for an enlarged thyroid with the neck in a normal position. Thyroid size was classified as not palpable or visible (grade 0), palpable but not visible (grade 1) or visible (grade 2). Casual urine samples, obtained from all subjects, were analysed spectrophotometrically for iodine levels by means of a method using the Sandell-Kolthoff reaction.[16] The median urinary iodine level was calculated for both study samples. All urinary iodine values (in µg/dl) were categorised according to level of severity of iodine deficiency,[7] viz. mild (5 - 9.9), moderate (2 - 4.9) and severe (< 2.0).

Mothers were clinically examined for hypothyroidism. Venous blood samples for biochemical analysis of thyroid function were collected from those with symptoms and signs suggestive of hypothyroidism. Maternal venous blood collected in EDTA tubes was analysed for haemoglobin (Hb), mean corpuscular haemoglobin (MCH) and mean corpuscular volume (MCV). The concentrations of thyroid-stimulating hormone (TSH) and free thyroxin (FT4) were determined by means of the Becton Dickinson Simultrac kit and free tri-iodothyronine (FT3) concentrations were determined by means of the Kodak Amerlex-Mab kit. Haematological values were determined by means of a model STKS Coulter Counter. The body mass index (BMI) of all mothers was calculated according to the standard formula (weight (kg)/height (m)^2). Mean haematological values of mothers without goitre (grade 0), were compared with those of mothers with goitre (grades 1 and 2 combined) by means of an unpaired *t*-test.

The mid-year exam marks for the Zulu and mathematics papers of all children were obtained with permission of the school principal. A multivariate analysis of variance (MANOVA) test was used (Wilks Lambda test) to determine whether the exam marks of children with goitre differed significantly from those of children without goitre. Adjustment was made for age, sex and school standard effects.

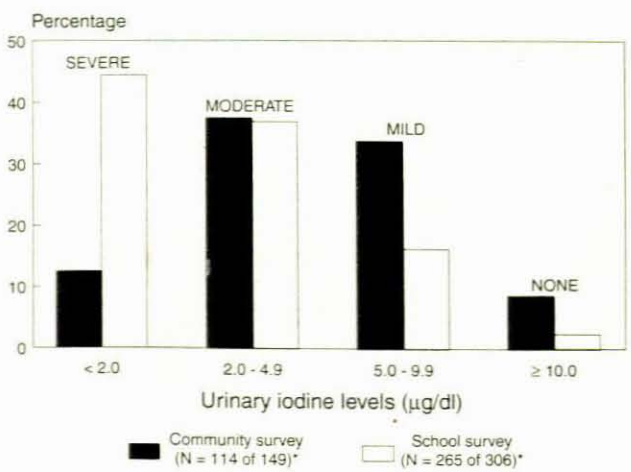
To establish the availability of iodised salt, all the shops in the area were visited and owners questioned to determine whether iodised salt was stocked.

The Epi Info 6 statistical package[17] and SAS version 6 were used for all statistical analyses.

Results

The cross-sectional survey revealed a goitre prevalence of 28.3% among mothers (36/127), of which 10.2% (13/127) was grade 1 and 18.1% (23/127) grade 2. The mean ages of the mothers of the children in grades 0, 1 and 2 were 42.0 (SD = 15), 40.1 (SD = 16) and 42.6 years (SD = 11) respectively. Forty per cent of mothers with goitre had nonspecific complaints of fatigue, muscle aches and pains and constipation. There was no oedema in mothers with goitre grade 0 (*N* = 77). However, mild oedema was present in 15.4% of mothers with goitre grade 1 (*N* = 2), and 30.4% of mothers with goitre grade 2 (*N* = 7). The percentage of obese mothers (BMI > 30) with goitre grade 0 was 43.2%, with goitre grade 1 42.4% and goitre grade 2 41.0%. Mean values for Hb, MCH and MCV were within normal limits and did not differ significantly in goitrous and non-goitrous mothers. All TSH, FT4 and FT3 levels were found to be normal.

The cross-sectional community study also revealed that children aged 6 - 11 years (*N* = 149) had a total goitre prevalence of 21.6% and a median urinary iodine level of 4.5 µg/dl. The distributions of goitre prevalence and urinary iodine levels are indicated in Table I and Fig. 1 respectively. The survey of Ndunakazi primary school children aged 6 - 14 years revealed a total goitre prevalence of 29.6%. The goitre prevalence (grades 1 and 2 combined) was in excess of 10% in all age groups and there was a significant increasing trend in the prevalence of goitre with increasing age ($P < 0.01$). The majority of goitres were grade 1 (palpable), but the grade 2 goitre prevalence (visible) increased with increasing age ($P = 0.07$). Girls tended to have higher goitre rates than boys. Table I summarises the sample size and the prevalence of grade 1 and 2 goitre for each survey. The median urinary iodine level in children aged 6 - 14 years was 2.4 µg/dl. The distribution is shown in Fig. 1. In the community-based survey, 85% of subjects and in the school survey, 97% had urinary iodine levels less than 10 µg/dl. The median urinary iodine levels in all age groups were below 5 µg/dl and were similar for all ages, sexes and grades of goitre. The percentage of children below –2 standard deviation of height for age, weight for age and weight for height did not differ significantly between those with goitre and those without.



* Not all urine samples were available for analysis.

Fig. 1. Categories of iodine deficiency.

Table I. Prevalence of goitre (%) per age group

Group	Survey	No.	Prevalence of goitre Grade 1	Grade 2
6 - 8 years	Community	66	9.1	4.4
	School	146	12.4	2.7
9 - 11 years	Community	83	14.8	13.2
	School	106	32.1	0.9
12 - 14 years	School	54	50.0	11.1
Mothers	Community	127	10.2	18.1

Fig. 2 indicates that the mid-year Zulu exam marks of goitrous children for the various school standards appeared consistently lower than those of children without goitre. Using the MANOVA and controlling for age, sex and school standard, differences were statistically significant between these two groups. Exam marks for mathematics were not significantly different in those with goitre and those without, for any school standard. Table II clearly shows that the presence of goitre had a significant detrimental effect on Zulu marks and that goitrous individuals scored on average 5.2% lower than children without goitre. For mathematics this figure was 2.7%, but not statistically significant. The MANOVA analysis of combined Zulu and mathematics scores showed a significant goitre effect ($P = 0.04$).

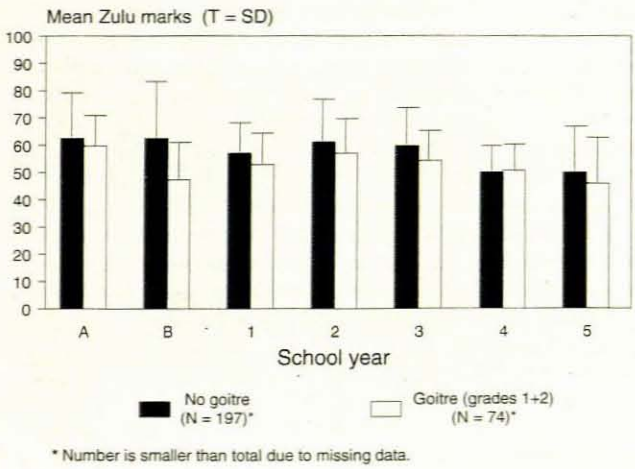


Fig. 2. Mid-year exam marks — goitre versus no goitre.

No iodised salt was available in any of the three shops in Ndunakazi. The only salt available was non-iodised coarse salt.

Discussion

School-aged children are a convenient test group because of accessibility. They reflect the current status of iodine nutrition in the community and are a major priority group for prompt correction of iodine deficiency. This study convincingly demonstrated, in a large rural community, the existence of iodine deficiency of moderate severity and a public health problem in need of correction.[7] At this level of iodine deficiency, Delange[4] reports that euthyroid schoolchildren born and living in iodine-deficient environments exhibit subtle or even overt neuropsycho-intellectual deficits when compared with controls from non-iodine-deficient environments, living in the same ethnic, demographic, nutritional and socio-economic system.[4] Low verbal IQ, perception and attentive functions have also been described with moderate iodine deficiency.[4] The onset of these effects is often insidious and most are irreversible after the second year of life.[18] The aforementioned facts, together with the continuum of mental retardation between the non-goitrous population and cretins (with severe mental retardation) reported by Lamberg,[6] explain to some extent why goitrous individuals performed significantly worse in their mid-term Zulu examinations than individuals without goitre. The cognitive profile of the study population is currently under further investigation.

The urinary iodine level is a good marker of dietary iodine, as 80 - 95% of the daily intake is excreted in the urine.[4,7] Since individuals' urinary iodine levels vary from day to day, values can only be used for a population estimate. The difference in median urinary iodine levels found in the two surveys, which took place 2 months apart, are probably the result of different sample sizes and varying iodine intakes in the sample population. Median urinary iodine values from 2 to 4.9 µg/dl indicate moderate iodine deficiency and, despite slight variation between the two surveys, the conclusion that moderate iodine deficiency exists in the study population remains constant. The distribution of urinary iodine levels seldom follows the bell-shaped normal curve, and it is therefore advisable to report median values and to characterise the distribution according to cut-off points. The low urinary iodine levels demonstrated in our surveys show that the most likely cause of endemic goitre in this community is low iodine intake. In view of the low urinary iodine excretion it appears unlikely that excessive consumption of goitrogens such as vegetables of the *Crucifera* family[4] (e.g. cabbage) contributed to the endemic goitre observed in this study. The substantial percentage of schoolchildren with urinary iodine levels less than 2 µg/dl (severe iodine deficiency) are at significant risk of developing hypothyroidism, and mental and physical retardation, should the iodine deficiency not be addressed.[5]

Table II. Analysis of variance for Zulu and mathematics scores in respect of goitre status

Parameter	Zulu scores Estimate	Standard error	*P*-value	Mathematics scores Estimate	Standard error	*P*-value
Age	–0.50	0.56	0.37	1.52	0.65	0.02
Sex (Male)	–7.27	1.76	< 0.01	–5.47	2.07	< 0.01
Goitre (None)	5.23	2.09	0.01	2.70	2.45	0.27
School standard	–1.57	2.77	0.57	20.66	3.25	< 0.01

In response to alterations in iodine intake, the size of the thyroid gland changes inversely, with a lag of 6 - 12 months.[7] In the absence of ultrasonography, which provides a more precise method for determining thyroid size, clinical classification of thyroid size into goitre grades 0, 1 and 2 provides an acceptable and simple alternative.[7] The authors are confident that misclassification of goitres did not influence the interpretation of the severity of iodine deficiency in the study population. Both the urinary iodine levels and the goitre prevalence indicate iodine deficiency of moderate severity. Results of thyroid palpation by the same clinician in both surveys showed a goitre prevalence that indicates moderate iodine deficiency (cut-off points: 20.0 - 29.9%). The increasing goitre prevalence with increasing age, is in accordance with the literature.[4]

Our results also show that despite the extent of iodine deficiency in this community, the prevalence of stunting is not significantly different from that reported for KwaZulu-Natal as a whole.[19]

The nonspecific symptoms and signs prevalent in goitrous adults are known to occur in hypothyroidism,[20] but all thyroid function tests proved to be within normal limits. In those over the age of 30 years, goitre and TSH levels are not reliable indicators of current iodine intake; thyroid function tests are often normal,[4,7] although the extra challenge of pregnancy could induce thyroid disorders in any euthyroid woman with goitre.[21] Thyroglobulins would have been a more sensitive indicator of insufficient iodine intake.[7] The higher prevalence of oedema in goitrous mothers could not be explained by the available data and needs further investigation.

It is increasingly realised that IDD can be prevented safely and cheaply, and eventually eliminated through iodisation of salt.[1] In view of the possible health effects of iodine deficiency, it is not surprising that the prevention of IDD is one of the most important achievable health goals of this decade, and will have an impact on a par with that of the global eradication of smallpox.[22] Prior to South African legislation's enforcing universal iodisation of salt on 1 December 1995, the distribution of iodised salt was determined mainly by public and trade demand.[23] Jooste *et al.*[23] concluded that because of factors such as low level of awareness of the benefits of iodised salt and price sensitivity, low-income people were inclined to purchase less expensive non-iodised salt; this especially put people in deep rural areas with a low dietary iodine intake at risk of developing iodine deficiency. Ndunakazi, where not a single shop stocked iodised salt and there was significant iodine deficiency, seems to be a case in point. With less than 30% of South African household salt iodised,[23] impoverished rural areas, areas previously known to be iodine deficient[12-14] and neighbouring countries with endemic goitre,[8-11] it seems likely that iodine deficiency could be prevalent in South Africa.

This study indicates that South Africa can ill afford to be overly confident about the absence of iodine deficiency. It is unlikely that changes in South African legislation, which enforce iodisation of table salt, will effectively protect communities living in iodine-deficient areas. Problems of poverty and price sensitivity, and the unknown quantities of non-iodised salt in circulation, are likely to remain for some time. It is also sobering to note that iodine deficiency is under control in only five European countries, despite long-standing compulsory iodisation of salt and committed efforts to eradicate iodine deficiency.[21] The probable causes of this phenomenon, viz. low levels of awareness of the problem by health authorities and the public and cultural factors causing low salt intake by some groups, are also present in South Africa.[23]

A multisectoral programme directed at the sustained elimination of iodine deficiency and other micronutrient deficiencies is currently being developed in Ndunakazi in co-operation with the community and can serve as a model for similarly afflicted communities.

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