

Iodine deficiency in Europe

A continuing public health problem



Iodine Deficiency in Europe: A continuing public health problem

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This report is dedicated to the late François Delange who relentlessly dedicated his energy and expertise to combat iodine deficiency throughout the world and especially in Europe. He was eagerly anticipating the release of this report, which he believed would be critical in focusing the attention of the European public health community on the significance of iodine deficiency, the main cause of preventable cognitive impairment in children.

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Contents

Foreword	vii
Preface	viii
Acknowledgements	ix
Abbreviations	xii
Glossary	xiii
Executive Summary	1
1. Introduction	3
1.1 Iodine deficiency in Europe: overview and historical context	3
1.2 Global strategy for the prevention and control of iodine deficiency	4
1.2.1 Commitment of the international community	4
1.2.2 Salt iodization as the main strategy to control iodine deficiency	4
1.2.3 Sustainability of salt iodization programmes	5
1.2.3.1 Political commitment	5
1.2.3.2 Monitoring and evaluation	5
1.2.3.3 Partnership	6
2. Iodine deficiency, health consequences, assessment and control	8
2.1 The disorders induced by iodine deficiency	8
2.1.1 Definition of iodine deficiency disorders, epidemiology and magnitude	8
2.1.2 Health outcomes	8
2.1.2.1 Iodine deficiency in the fetus	8
2.1.2.2 Iodine deficiency in the neonate	9
2.1.2.3 Iodine deficiency in children and adults	10
2.2 Social and economic consequences	10
2.2.1 Cost-effectiveness and cost-benefit	10
2.2.2 Social benefits of the elimination of iodine deficiency	11
2.2.3 Economic benefits of the elimination of iodine deficiency	12
2.2.4 Conclusion	12
2.3 Assessment of iodine deficiency	13
2.3.1 Prevalence of goitre	13
2.3.2 Urinary iodine	13
2.3.3 Serum concentrations of TSH, thyroid hormones and thyroglobulin	14
2.4 Prevention and control	14
2.4.1 Salt iodization	14
2.4.2 Iodized oil	15
2.4.3 Other methods	15
2.4.4 Adverse effects associated with the correction of iodine deficiency	16
2.5 Monitoring and evaluation	18
2.5.1 Monitoring of salt iodization	18

2.5.2	Monitoring iodine status	18
2.5.3	Monitoring thyroid function	18
2.5.4	Indicators to monitor progress towards the international goal of elimination of iodine deficiency	19
3.	Iodine deficiency in Europe and its control: current status, progress and recent trends	20
3.1	Methods used to record information	20
3.1.1	Urinary iodine and goitre prevalence	20
3.1.1.1	Data sources	20
3.1.1.2	Selection of survey data	20
3.1.1.3	Population coverage	21
3.1.1.4	Classification of iodine nutrition	21
3.1.1.5	Proportion of population and the number of individuals with insufficient iodine intake	21
3.1.1.6	Total goitre prevalence	21
3.1.2	Serum concentrations of TSH, thyroid hormones and thyroglobulin	21
3.1.3	Salt iodization programmes	21
3.2	Epidemiology and severity of iodine deficiency	21
3.2.1	Urinary iodine	21
3.2.1.1	Population coverage	21
3.2.1.2	Classification of countries based on median UI	22
3.2.1.3	Proportion of population and number of individuals with insufficient iodine intake	23
3.2.2	Goitre prevalence	23
3.2.2.1	Population coverage	24
3.2.3	Assessment of iodine nutrition based on thyroid function	24
3.2.4	Progress and recent trends	24
3.3	Policy and legislation	25
3.4	Iodized salt	27
3.4.1	Access to iodized salt	27
3.4.2	Recent trends and obstacles to effective iodization programmes	27
3.4.2.1	Political and social changes	27
3.4.2.2	Increased exchange of food trade between countries	27
3.4.2.3	Changes in dietary sources of salt	28
3.4.2.4	Dietary sources of salt covered by regulations on iodization	29
3.4.2.5	Decreased levels of salt intake	29
3.4.2.6	Other factors	30
3.4.3	Quality assurance	31
3.4.4	Process indicators	31
3.4.5	Other iodine deficiency control measures	32
3.5	Economic consequences	32
4.	The major issue for EUROPE: sustained prevention and control	34
4.1	Main policy issues regarding the elimination of iodine deficiency	35
4.1.1	Assessment of iodine status	35
4.1.2	Implementation of USI	35
4.1.3	Focus on infants and pregnant women	36
4.1.4	Implementing alternative strategies to correct iodine deficiency	36
4.1.5	Monitoring and evaluation	36
4.1.6	Legislation	37
4.1.7	Economic impact	37
4.1.8	Advocacy and partnership	37
4.2	Challenges for the future	37
4.3	Conclusions	61

Annex A	The International Resource Laboratories Network for Iodine (IRLI): European laboratories members of the network	40
Annex B	General characteristics of countries included in the report	44
Annex C	The WHO Global Database on Iodine Deficiency	45
Annex D	Prevalence of iodine deficiency in school-age children and the general population based on urinary iodine data, by country	46
Annex E	Total goitre prevalence by country, level of survey and age group	50
Annex F	Findings and significance of currently available information on indicators of thyroid function	53
Annex G	Programmatic indicators for monitoring the elimination of iodine deficiency	59
References		61

Index of tables

Table 2.1	The Spectrum of iodine deficiency disorders	9
Table 2.2	Prevalence of, and number of individuals with, iodine deficiency in the general population (all age groups) and in school-age children (6–12 years), by WHO region, 2003	9
Table 2.3	Benefits of iodine intervention programmes	11
Table 2.4	Epidemiological criteria for assessing the severity of iodine deficiency based on the prevalence of goitre in school-age children	13
Table 2.5	Epidemiological criteria for assessing iodine nutrition based on median UI concentrations in school-age children	14
Table 2.6	Current global status regarding iodized salt consumption of households	16
Table 2.7	Tolerable Upper Intake Level (UL) for iodine (g/day)	17
Table 2.8	Criteria for monitoring progress towards sustainable elimination of iodine deficiency disorders	19
Table 3.1	Proportion of population, and number of individuals with insufficient iodine intake in school-age children (6–12 years) and in the general population (all age groups) in Europe, 2004	24
Table 3.2	Summary of regulations on salt iodization in Europe	26
Table 3.3	Penetration rate according to market segments	28
Table 3.4	Proportion (%) of sodium intake from various dietary sources in Finland, France and the UK showing change over time	29
Table 3.5	Total amount of dietary salt consumed (g/day) in some European countries at the end of the 1980s	30

Index of figures

Figure 1.1	Social process involved in a national iodine deficiency control programme	6
Figure 3.1	Type of UI survey data	22
Figure 3.2	Degree of public health significance of iodine nutrition based on median UI	23
Figure 3.3	Trends in Germany of iodized salt used by households and in the food industry following legislation to allow fortified salt in various processed foods	30

Foreword

Despite a worldwide application of successful iodine supplementation programs over the last four decades, iodine deficiency remains a major public health problem in Europe. In 2004, it was estimated that of the 2 billion people around the world at risk of iodine deficiency, 20 percent live in Europe, Eastern and Western Europe being both affected. While cretinism, the most extreme expression of iodine deficiency, has become very rare and even disappeared in Europe, of considerably greater concern are the more subtle degrees of mental impairment associated with iodine deficiency that lead to poor school performance, reduced intellectual ability, and impaired work capacity. For iodine-deficient communities, between 10 and 15 IQ points may be lost when compared to similar but non-iodine-deficient populations. Iodine deficiency is the world's greatest single cause of preventable brain damage. This fact is the driving force that led the international community and more specifically the World Health Assembly to adopt a resolution in 1990 to eliminate iodine deficiency. This resolution was reaffirmed in 1998, 2003, and 2007.

The main strategy for the control of iodine deficiency disorders (IDD) – salt iodization – was adopted by the World Health Assembly in 1993 and established as a UN General Assembly's Special Session on Children goal in 2002. Salt has been chosen as a vehicle because of its widespread consumption and the extremely low cost of iodization. However, where the prevalence of iodine deficiency is high and where salt iodization is not feasible, the alternative is to administer iodine directly, either as iodide or iodized oil, focusing on women and young children. In the early 1960s, only a few countries had IDD control programmes; most of them in the United States of America and Europe. Since then, and especially over the last two decades, extraordinary progress has been achieved by increasing the number of people with access to iodized salt and reducing the rate of iodine deficiency in most parts of the world. However, this has not been the case in several industrialized countries, especially in Europe. Compared to other regions in the world, iodized salt coverage is not as high in Europe, reaching only 27% of households. In addition, there is growing evidence that iodine deficiency has reappeared in some European countries where it was thought to have been eliminated. This underscores the need for sustaining current programmes. Furthermore, we should stress that salt iodization does not collide with the initiatives aimed at the reduction of overall salt consumption undertaken in Europe with the purpose to curb cardiovascular and particularly cerebrovascular disease rates in the region.

Given the magnitude of iodine deficiency in Europe, it is important to review this situation in order to assess the current strategy, identify the reasons why these programmes are not as effective as expected, and ultimately provide public health authorities with the information required to improve the iodine status of deficient populations. This is precisely the main objective of this report, and we hope that it will contribute to the goal of elimination of iodine deficiency in Europe.

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Preface

The health consequences of iodine deficiency for infants and children are well known: brain damage and irreversible mental retardation (1). Resulting mental deficiencies adversely affecting the workforce in turn negatively impact the economy. Reproduction is also affected. Yet, despite all that is known about iodine deficiency, it remains a public health problem in much of Europe.

In 1990, the international community adopted the goal of iodine deficiency elimination at the United Nations World Summit for Children. This goal was reaffirmed by the World Health Assembly and at several subsequent international forums. Putting the problem of iodine deficiency back on the national agenda of European governments is therefore the ultimate purpose of this report. Increased advocacy, which target both consumers and governments, is the first step. Following this, requisite needs include the implementation and monitoring of sustainable programmes that control and prevent iodine deficiency. Sustainability is a key issue. It is clear from the data contained herein that throughout parts of Europe, even in some countries with pre-existing iodine deficiency control programmes, the prevalence of iodine deficiency is re-emerging. Renewed efforts will only be successful if they can be sustained.

This has been recognized since 1993 (2) and was more recently further emphasized at a meeting of the Network for Sustained Elimination of Iodine Deficiency held in Gent (2002) (3). It was agreed at this meeting that an updated review of the iodine deficiency situation in Europe was needed – addressing not only the iodine status of national populations, but also the status and progress of intervention programmes – as a basis for planning further action.

The data contained in this review can also be used as the basis for more specific, national and subregional advocacy. The report includes the most recent data available from the 40 European countries made up of European Union Member States including applicant countries and those in the European Free Trade Association (EFTA).

The quality and availability of information on iodine nutrition has substantially improved over the past two decades. National data were used extensively as the basis of this review's conclusions. Where national data were not available, subnational data were used. As advocacy efforts continue, and monitoring and evaluation of programmes become widespread across Europe, it is hoped that the assessment of iodine status in every European country will follow. Thus, an even clearer picture of the iodine status across Europe will emerge. Based on the analysis and outcome of the data produced in this review, guidance aimed at sustainable elimination of iodine deficiency in Europe will be proposed. It is of course further hoped that this report will contribute to such change and to the necessary success of advocacy efforts to mobilize countries where needed.

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Abbreviations

CDC:	Centers for Disease Control and Prevention
EFTA:	European Free Trade Association
EU:	European Union
FAO:	Food and Agriculture Organization of the United Nations
ICCIDD:	International Council for Control of Iodine Deficiency Disorders
IDD:	Iodine deficiency disorders
IIH:	Iodine-induced hyperthyroidism
MI:	Micronutrient Initiative
NGO:	Nongovernmental organization
ppm:	Parts per million
RDA:	Recommended dietary allowance
RNI:	Recommended nutrient intake
SAC:	School-age children
T3:	Triiodothyronine
T4:	Thyroxine
Tg:	Thyroglobulin
TGP:	Total goitre prevalence
TSH:	Thyroid stimulating hormone
UI:	Urinary iodine
UL:	Tolerable upper intake level
UNICEF:	United Nations Children's Fund
USI:	Universal salt iodization
WHO:	World Health Organization

Glossary

Effectiveness refers to the impact of the intervention in practice. Compared to efficacy, the effectiveness of a fortification programme will be limited by factors such as non or low consumption of the fortified food for various reasons.

Efficacy refers to the capacity of an intervention such as fortification to achieve a desired impact under ideal circumstances. This usually refers to experimental, well-supervised intervention trials.

Essential micronutrient refers to any micronutrient that is normally consumed as a constituent of food, which is needed for growth and development and the maintenance of healthy life, and can not be synthesized in adequate amounts by the body.

Evaluation refers to the assessment of the effectiveness and impact of the programme on the targeted population. The aim is to provide evidence that the programme is achieving its nutritional goals.

Fortification is the practice of deliberately increasing the content of an essential micronutrient – vitamins and minerals including trace elements – in a food, so as to improve the nutritional quality of the food supply and provide a public health benefit with minimal risk to health.

Goitre refers to enlargement of the thyroid gland, most commonly due to iodine deficiency. A thyroid gland is considered goitrous when each lateral lobe has a volume greater than the terminal phalanx of the thumbs of the subject being examined.

Iodine deficiency disorders (IDD) refers to the spectrum of clinical, social and intellectual results of iodine deficiency.

Market penetration rate refers to the percentage of produced iodized salt that reaches households and/or food industry.

Market segment refers to different groups (segments) of customers on the salt market, including industry and households.

Mass fortification refers to the addition of micronutrients to foods commonly consumed by the general public, such as cereals, condiments and milk.

Monitoring refers to the continuous collection and review of information on programme implementation activities, for the purpose of identifying problems, such as non compliance, and taking corrective actions so as to fulfill stated objectives.

Nutrient requirement refers to the lowest continuing intake level of a nutrient that will maintain a defined level of nutriture in an individual for a given criterion of nutritional adequacy.

Processed foods are those in which food raw material has been processed into formulated products.

Quality assurance refers to the activities necessary to ensure that products or services meet quality standards. The performance of quality assurance can be expressed numerically by means of quality control.

Quality control refers to the techniques and assessments used to document compliance of a product with established technical standards, through the use of objective and measurable indicators.

Recommended dietary allowance (RDA) is equivalent to the recommended nutrient intake (RNI).

Recommended nutrient intake (RNI) refers to the daily intake which meets the nutrient requirements of almost all (97.5%) apparently healthy individuals in an age- and sex-specific population group. It is set at the estimated average requirement plus 2 standard deviations.

Salt penetration rate refers to the percentage of households consuming iodized salt.

Tolerable upper intake level (UL) refers to the highest average daily nutrient intake level unlikely to pose risk of adverse health effects to almost all (97.5%) apparently healthy individuals in an age- and sex-specific population group.

Total goitre prevalence (TGP) refers to total prevalence of goitre – both palpable but not yet visible and visible – in a population, usually schoolchildren aged 6–12 years. TGP is an indicator for past iodine deficiency.

Universal salt iodization (USI) refers to the addition of iodine to salt that is destined for both human and animal consumption.

Urinary iodine (UI) refers to urinary iodine concentration and is currently the best biochemical marker of recent dietary iodine intake.

Executive Summary

Every European nation endorsed the goal of eliminating iodine deficiency at the World Health Assembly in 1992. Globally, great progress has been made since that time. However, the World Health Organization's (WHO) European Region has been identified as having the lowest coverage of salt iodization of all the regions, most of which are considerably less fortunate in economic terms than the majority of European countries. As recently as 1993 there were only five countries in Europe where the iodine deficiency problem was under control. However, there has been impressive progress in iodine deficiency control over the past five to six years. The greatest challenge today is to sustain this progress. The goal of iodine deficiency elimination was re-affirmed in a special session of the United Nations in May 2002.

The primary purpose of this document is to review the current extent of iodine deficiency in the European Union (EU) Member States, applicant countries and those in the European Free Trade Association (EFTA). Its ultimate goal is the mobilization of all European governments to implement and monitor sustainable programmes to control and prevent iodine deficiency in their populations. Part one of the report gives the background, historical context and global strategies. The second part addresses the main issues related to iodine deficiency: its magnitude, the public health significance, and the health and economic consequences of iodine deficiency, and outlines the current strategies being used to reach the goal of iodine deficiency elimination. In the third part, the focus is on the iodine deficiency situation in 40 of the countries of Europe. The final part highlights the need for sustainable programmes and makes recommendations to help achieve this.

On the basis of the national medians of urinary iodine (UI) for the 40 European countries included in the review, it is estimated that the populations of 19 countries have adequate iodine nutrition, 12 have mild iodine deficiency, one moderate iodine deficiency, and eight countries have insufficient data. The data on goitre prevalence are limited and difficult to interpret due to different collection methods but vary markedly from country to country, ranging from 0 to 31.8%. Recent data on thyroid function are also lacking for many countries. Usually measured in the most susceptible groups, pregnant women and young infants, the data indicate alterations of thyroid function likely to lead to mild retardation in the intellectual development of infants and children.

Legislation pertaining to iodized salt, where enacted, varies from country to country. Of the 40 countries reviewed here with information available, only nine have coverage of iodized salt at the household level of at least 90%. Of the 32 with data, only 17 have a national programme; 23 have some legislation or regulation in place, although relatively rarely does this mandate universal salt iodization (USI). Monitoring of national programmes is currently insufficient, especially as it relates to measuring progress towards the goal of eliminating iodine deficiency.

Over the past decade, four main factors have led to the current situation. First, political and social changes have interrupted both the salt iodization process itself, and quality control measures. Second, the formation of common markets along with increasing globalization, have led to greater movement of foods across national barriers, some processed with iodized salt, some not.

Third, an increasingly smaller amount of salt is consumed as table salt (e.g. in the United Kingdom only 15% of all salt consumed – and a third of this is added at cooking). A similar situation has been described in Finland and France, and no doubt in other countries as well, given the clear trend towards a greater proportion of salt being “hidden” in processed foods which legislation in many countries does not cover. Finally, partly through concern about hypertension, salt consumption has gradually declined, although remains around 8–10 g/day. Current recommendations by WHO and other bodies are even lower (<5 µg/day), and if adopted by European populations would likely require some modification of iodine levels with which salt is being iodized in various countries. In the meantime, the considerable success seen over the past five or six years needs to be sustained, as remaining iodine-deficient sub-populations are addressed and the necessary public health goal of USI for all countries is achieved.

The review concludes that iodine deficiency remains a public health concern in Europe; the health, social and economic consequences of this are well established. Salt iodization remains the recommended strategy for eliminating iodine deficiency. Next steps towards this goal are noted in the recommendations in Chapter 4. Foremost among the challenges are:

- to strengthen monitoring and evaluation of national programmes for the prevention and control of iodine deficiency in the countries of an enlarged EU, including the surveillance of the iodine status of national populations;
- to ensure the sustainable implementation of USI in all countries of the enlarged EU, by harmonizing relevant legislation and regulations;
- to ensure adequate quality control and quality assurance procedures to strengthen the monitoring of foods fortified with iodine, especially salt iodization, from the producer to the consumer;
- to increase the awareness of political leaders and public health authorities on the public health and social dimensions of iodine deficiency and the need to implement and sustain programmes for its control;
- to educate the public on the need to prevent iodine deficiency by consuming iodized salt, and thereby also increase consumer awareness and demand; and
- to consider alternative iodine supplementation for the most susceptible groups – pregnant women and young infants – where there is insufficient iodized salt and to take into account public health policies to reduce salt consumption.

1. Introduction

1.1 Iodine deficiency in Europe: overview and historical context

Iodine deficiency, with endemic goitre as its main clinical manifestation, and brain damage and irreversible mental retardation as major public health consequences, is part of the history of the European continent. All European countries except Iceland have experienced this health and socio-economic scourge to a greater or lesser degree. Endemic cretinism, the most severe consequence of iodine deficiency, was extensively reported in the past, particularly from isolated and mountainous areas in Austria, Bulgaria, Croatia, France, Italy, Spain and Switzerland (4–7), and was so common that the term “cretin of the Alps” became part of the common vocabulary (8). Nevertheless, only limited attention has been paid to the public health consequences of iodine deficiency in Europe until recently.

Over a century and a half ago iodine deficiency had already been recognized. At the beginning of the 19th century, it was first suggested that the use of salt fortified with iodine would lead to good health in people living in mountainous regions (9). Switzerland was the first European country to introduce iodized salt on a large scale in order to eliminate iodine deficiency (10,11). After the pioneering work of Swiss doctors that demonstrated that iodine deficiency was indeed the cause of goitre, attempts began to locally iodize salt using a hand-and-shovel method. In 1922, the Swiss Goitre Commission recommended to the then 25 Swiss Cantons (provinces) that salt be iodized on a voluntary basis at a level of 3.75 mg iodine per kg salt (or 3.75 ppm). Non-iodized salt also remained available for sale. Due to the decentralized system of the Government of Switzerland the availability of iodized salt progressed slowly; the last Canton (Aargau) allowed the sale of iodized salt only in 1952. Today, over 90% of households consume iodized salt, and about 70% of the salt used in industrial food production is iodized (12). However, this example was not generally replicated by many other countries in Europe. Iodine deficiency control and prevention currently appear to be a public health issue of relatively low priority in Europe, presumably because of governments' perceptions that the health risks from iodine deficiency have been brought under control.

One of the first published references made to iodine deficiency in Europe was in the monograph “Endemic Goitre”. Published by the World Health Organization (WHO) over 40 years ago, it was an exhaustive review on iodine deficiency worldwide (13,14). It was only in the late 1980s that the European Thyroid Association re-evaluated the problem and clearly indicated that, with the exceptions of Austria, the Scandinavian countries, and Switzerland, European countries, especially in the southern part of the continent were still affected by iodine deficiency (15). The next crucial evaluation of iodine deficiency in Europe took place during a meeting titled “Iodine deficiency in Europe: a continuing concern” held in Brussels in 1992 (2). It concluded that iodine deficiency was under control in only five countries, namely Austria, Finland, Norway, Sweden and Switzerland but continued to persist in all other European countries, to some degree.

In 1993, WHO estimated that, based on the total goitre prevalence (TGP), 97 million people in the European Region of WHO were affected by iodine deficiency (16). For the 40 countries currently being reported on, iodine deficiency was considered a public health problem in 17 countries, two countries had iodine deficiency under control, 10 countries were likely to have iodine

deficiency under control and 11 countries had no data available. In 1997, a meeting was held in Munich on iodine deficiency attended by the representatives of 28 countries of eastern and central Europe (17). It revealed the severity of the problem, including the recurrence of goitre, and occasionally of endemic cretinism, in some countries in eastern Europe after the interruption of salt iodization programmes. This meeting triggered massive efforts in the implementation or restitution of iodized salt programmes, mainly by the United Nations Children's Fund (UNICEF) with the financial support of the service organization Kiwanis International.

Since then, an increasing number of studies have been carried out on various aspects of iodine deficiency and its control in Europe based on local, regional or national surveys. This included an evaluation of the iodine status of populations in 12 countries of central and western Europe by measuring goitre prevalence using ultrasound, and at the same time, urinary iodine (UI) levels (18). In 1999, the WHO Regional Office for Europe adopted the elimination of iodine deficiency as one of the targets in its nutrition action plan (19). A global report on the progress made towards the elimination of iodine deficiency was published by WHO and submitted to the World Health Assembly in May 2000 (20). It showed that of the 51 countries of the European Region of WHO, 32 were still affected by iodine deficiency and that salt iodization programmes were implemented in only 20 countries. In 2002, the data on iodine deficiency in Europe, and prevention and control programmes, were carefully reviewed again at the Annual Conference of the European Thyroid Association in Göteborg, Sweden. Evidence of a marked improvement in iodine nutrition was clearly shown (8,21). Information on current prevalence of iodine deficiency and the extent of salt iodization – necessary for the evaluation of a country's success in eliminating iodine deficiency and maintaining iodization programmes – has been updated and is presented in this report. Further information is available from the WHO Global Database on Iodine Deficiency (<http://www.who.int/vmnis>), the European Salt Producers' Association (www.eu-salt.com), the International Council for Control of Iodine Deficiency (ICCIDD) (www.iccidd.org) and the Global Network for Sustained Elimination of Iodine Deficiency (www.IodinePartnership.net).

1.2 Global strategy for the prevention and control of iodine deficiency

1.2.1 Commitment of the international community

Globally, the control and prevention of iodine deficiency has been largely successful over the past 75 years – even under a variety of conditions. Since the early 1960s, WHO has made a concerted effort to characterize the global extent of the problem of iodine deficiency, following which there was a period of integrating this information into national health systems. Yet the problem, while widely acknowledged, was not being addressed in any consistent way. ICCIDD, a multidisciplinary global network of specialists, formed in 1985 with support from UNICEF, WHO, and the Australian Government to act as a technical resource consultative group as well as advocate to national governments, international agencies and to a wide variety of health professionals and planners. In 1985 UNICEF also became more involved in the control of iodine deficiency. In 1992, the World Health Assembly took a pioneer step by adopting the goal of eliminating iodine deficiency as a public health problem (22). UNICEF in particular promoted this through its country offices with the technical support from WHO and ICCIDD. During the latter half of the 1990s, the global momentum increased further with the welcome addition of donor organizations such as the World Bank, the Micronutrient Initiative (MI) and Kiwanis International.

All these events helped build on the consensus achieved on what to do, as best expressed in the iodine elimination goals of the United Nations World Summit for Children (New York, 1990) and confirmed by 159 countries at the Joint Food and Agriculture Organization of the United Nations (FAO)/WHO International Conference on Nutrition (Rome, 1992). In May 2002 the Special session on Children of the United Nations General Assembly (New York) endorsed the goal of IDD elimination by the year 2005.

1.2.2 Salt iodization as the main strategy to control iodine deficiency

The relationship between good sources of iodine and the prevention of goitre goes back to antiquity, as in the use of seaweed in the Orient and marine fish in the Occident. The concept of adding iodine to salt began with the French chemist Boussingault, who, in the beginning of the

19th century stated “Je ne doute nullement qu’en répandant l’usage des sels faiblement iodifères, le goitre ne disparaisse complètement...”¹ (9). Since then, salt iodization has become progressively the main approach to control iodine deficiency throughout the world, as it has been proven an effective measure through rigorous monitoring and evaluation.

Following various international meetings during the 1980s, consensus was reached among national and international partners that universal salt iodization (USI) – iodization of all salt meant for human and animal consumption – should be the prime intervention to stop iodine deficiency. In 1993, WHO and UNICEF officially recommended USI as the main strategy to achieve the elimination of iodine deficiency (23). For implementation of this strategy collaboration with representatives of salt producer associations was initiated. Due to the recommendations by WHO and UNICEF, the preferred option of national programmes has been iodization of the salt supply, generally via USI. However, iodization of salt used in farming and food processing has often not been adopted by European national governments.

In some hard-to-access areas, iodine supplements such as iodized oil continues to be effective, particularly for the most susceptible groups: pregnant women and young children. Some countries have had success with other complementary interventions as well.

1.2.3 Sustainability of salt iodization programmes

The prevention and control of iodine deficiency is a continuous process. It requires monitoring to be sustainable. There are many examples throughout the world where iodine deficiency has re-emerged as a public health problem, where once it was under control. But it is in Europe where the problem is most dire. Overall, Europe has had the lowest salt iodization rate of all the WHO regions (20). Many of the countries of eastern Europe, which had good salt iodization coverage prior to 1990, saw a drastic drop in iodine status during the 1990s, although there have been encouraging increases recently (8,21,24).

The experience accumulated thus far has shown that the sustainability of a programme depends on several factors. These include a political commitment of the public health authorities and the decision-makers of governments, an effective and operational monitoring and evaluation system, a strong collaboration between the partners involved in the control of iodine deficiency, and public education. This is illustrated in Figure 1.

All partners must continuously remind themselves that the iodine deficiency problem is one that shifts with political and economic developments, and, most dangerously, complacency (25). National programmes will always require ongoing monitoring and adjustment by governments and their partners to protect their populations from this easily preventable scourge.

1.2.3.1 Political commitment

Clearly there must be technical conviction and political commitment to control iodine deficiency on the part of policy-makers within each country. This requires demonstration of the size of the iodine deficiency problem, its consequences, including the economic costs, and the proven effectiveness of programmes aimed at reducing it at the national level.

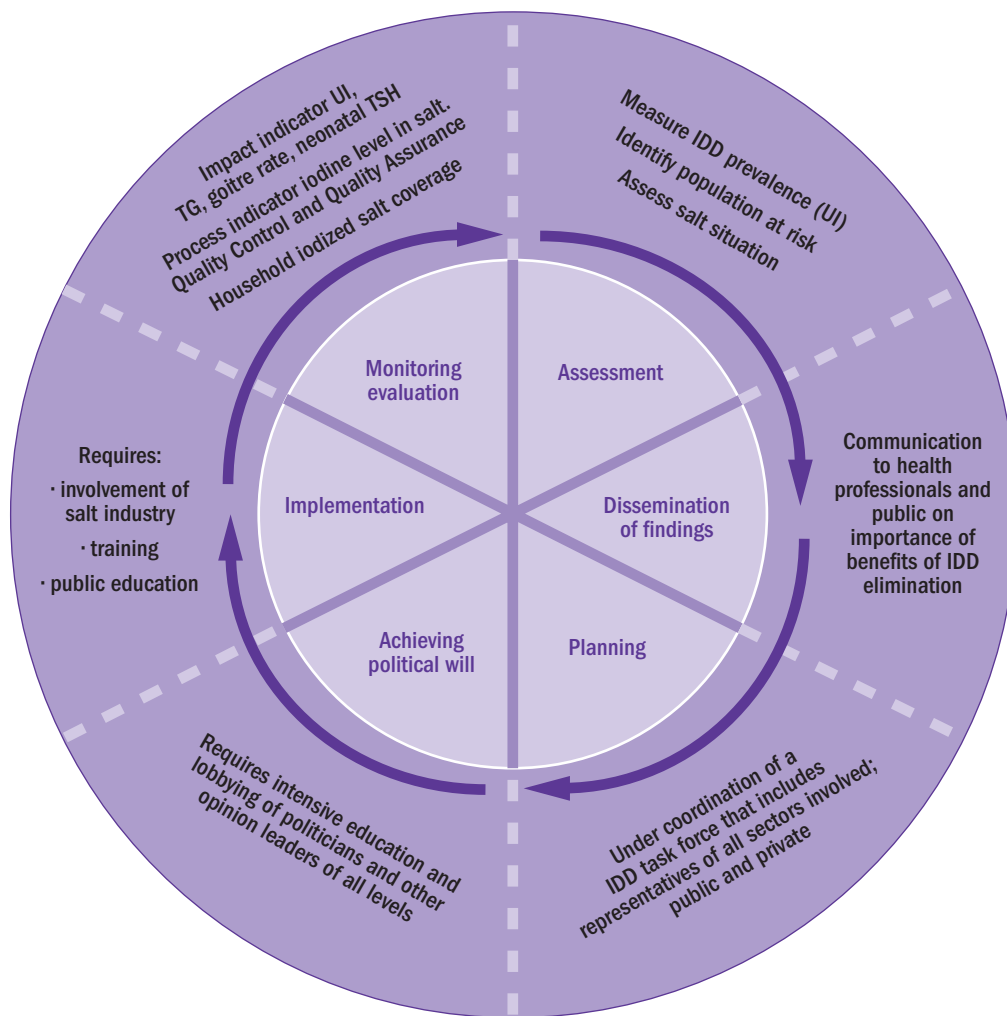
The key elements that demonstrate a political commitment include institutional support to facilitate the coordination between the different sectors involved, legislation on salt iodization and a policy document for controlling iodine deficiency. A survey conducted by WHO in 1999 (20) revealed that 80% of the countries where iodine deficiency is a public health problem had a National Intersectoral Coordinating Body Committee and a plan of action for iodine deficiency control, and 20 out of 51 countries had legislation in place. A detailed status of current legislation is given elsewhere in this review (Table 3.2 and Annex G).

1.2.3.2 Monitoring and evaluation

A major component in iodine deficiency prevention and control – and hence sustainability – is the monitoring and evaluation of national intervention programmes. Monitoring and evaluation

¹ “I have no doubt that increasing use of salt with a small amount of iodine added will lead to the complete disappearance of goitre...”

Figure 1 Social process involved in a national iodine deficiency control programme



Source: Adapted from Hetzel (26).

allows for the assessment of the effectiveness of a programme, as well as adjustment according to established objectives. However it became clear that many countries affected by iodine deficiency did not have the laboratory facilities to monitor the quality and levels of iodine in salt, nor the means to measure the iodine status of the populations being targeted. Consequently, an initiative was begun to set up a network of regional resource laboratories to assist countries in the surveillance of their programmes. The International Resource Laboratories for Iodine (IRLI) Network (iodelab@cdc.gov) was launched in Bangkok in May 2001 (http://www.cdc.gov/nccdphp/dnpa/impact/global/irli_network.htm). Co-sponsored by the Centers for Disease Control and Prevention (CDC), WHO, UNICEF, ICCIDD and MI, it is made up of a network of resource laboratories – one or a couple for each WHO region – which have as their main role the provision of technical support to national laboratories. There are two resource laboratories in Belgium and Bulgaria that provide support to the countries of western and central Europe (see details in Annex B). European laboratories that are members of the IRLI Network are listed in Annex A.

1.2.3.3 Partnership

The major lesson learned from the re-emergence of iodine deficiency in Europe has been that the apparently simple intervention of USI is actually quite complex and requires the full cooperation of many partners to be successful and sustainable. For example, close collaboration of partners at many levels is the major reason the prevention and control of iodine deficiency has been so success-

ful globally. In Europe, this collaborative process needs to be replicated, with particular emphasis on maintaining a strong partnership with the salt industry.

With iodization of the national salt supply being the most cost-effective and sustainable intervention in the prevention and control of iodine deficiency (27), the private sector has become a critical partner. Originally this was not a partnership at all: often the salt industry acted only in response to legislation imposed upon them by governments. Over the years, this relationship has improved, and has become a true partnership, culminating in the formation of the Network for the Sustained Elimination of Iodine Deficiency at the World Salt Symposium “Salt 2000” (The Hague, 2000).

An agreement was reached between United Nations (UN) agencies, representatives of the salt industry, academia and nongovernmental organizations (NGOs) to form a coalition of public, private, international and civil society organizations, to support and promote USI and thereby contribute to the goal of elimination of iodine deficiency. The Board of the Network currently includes representatives from UN agencies, academic and public institutions, donor agencies and salt industry.¹ The Network for the Sustained Elimination of Iodine Deficiency was officially launched at the UN Special Session on Children in New York in May 2002 (www.iodinepartnership.net).

¹ WHO, UNICEF, the CDC, ICCIDD, MI, the Emory University Rollins School of Public Health, the European Salt Producers' Association, the Salt Institute of the United States of America, the Chinese Salt Producers Association and Kiwanis International.

2. Iodine deficiency, health consequences, assessment and control

2.1 The disorders induced by iodine deficiency

2.1.1 Definition of iodine deficiency disorders, epidemiology and magnitude

Iodine is an essential micronutrient present in the human body in minute amounts (15–20 mg), almost exclusively in the thyroid gland. It is an essential component of the thyroid hormones, thyroxine (T_4) and triiodothyronine (T_3), with iodine comprising 65% and 59% of their weights, respectively. Thyroid hormones regulate metabolic processes in most cells, as well as playing a determining role in the process of early growth and development of most organs, especially that of the brain. In humans, most of the growth and development of the brain occurs during the fetal period and the first two to three years of postnatal life. Consequently, iodine deficiency, if severe enough to affect thyroid hormone synthesis during this critical period, will result in hypothyroidism and brain damage. The clinical consequence will be irreversible mental retardation (28).

The recommended daily nutrient intake of iodine is 90 μg (RNI)¹ for the age group 0–59 months, 120 μg for the age group 6–12 years, 150 μg for adolescents and adults and 250 μg during pregnancy and lactation, respectively (29,30). When these physiological requirements are not met in a given population, a series of functional and developmental abnormalities occur. They are grouped under the general heading of “iodine deficiency disorders” or IDD (31) as in Table 2.1.

Iodine deficiency represents a serious public health problem in the world: in 2003 WHO estimated that 54 countries are still affected by iodine deficiency as a public health problem and nearly 2 billion people have inadequate iodine nutrition (Table 2.2) (34). Iodine deficiency therefore represents the greatest cause worldwide of preventable mental retardation (27).

2.1.2 Health outcomes

As noted, the multiple impacts of iodine deficiency represent a range of effects, experienced throughout the life cycle, although the impacts are more devastating at some stages than others. As such they constitute a serious public health problem and a major impediment to the overall economic and social development of affected populations.

2.1.2.1 Iodine deficiency in the fetus

The consequences of iodine deficiency during pregnancy result from the impaired synthesis of thyroid hormones in both the mother and the fetus. In addition, maternal hypothyroxinaemia (low levels of thyroid hormone in the blood) results in a lowering in the maternal transplacental transfer of thyroxine during the early phase of fetal development – the first and second trimesters – during which the supply of thyroid hormones to the fetal brain comes almost exclusively from the mother. The long-term consequence of fetal hypothyroxinaemia occurring during early gestation is the development of a neurological syndrome, which includes severe mental retardation, spastic diplegia, hearing defects and squint. These symptoms correspond to what used to be called neurologic

¹ Recommended nutrient intake (RNI) refers to the daily intake which meets the nutrient requirements of almost all (97.5%) apparently healthy individuals in an age- and sex-specific population group. It is set at the estimated average requirement plus 2 standard deviations.

Table 2.1 **The Spectrum of iodine deficiency disorders**

Fetus	Abortions Stillbirths Congenital anomalies Increased perinatal mortality Endemic cretinism
Neonate	Neonatal hypothyroidism Endemic mental retardation Increased susceptibility of the thyroid gland to nuclear radiation
Child and adolescent	Goitre (Subclinical) hypothyroidism (Subclinical) hyperthyroidism Impaired mental function Retarded physical development Increased susceptibility of the thyroid gland to nuclear radiation
Adult	Goitre with its complications Hypothyroidism Impaired mental function Spontaneous hyperthyroidism in the elderly Iodine-induced hyperthyroidism Increased susceptibility of the thyroid gland to nuclear radiation

Source: Adapted from Hetzel (31), Laurberg et al. (32), Stanbury et al. (33).

Table 2.2 **Proportion of population, and number of individuals with insufficient iodine intake in school-age children (6–12 years), and in the general population (all age groups) by WHO region, 2003**

WHO region ^a	Insufficient iodine intake (UI <100 µg/l)			
	School-age children		General population	
	Proportion (%)	Total number (millions) ^b	Proportion (%)	Total number (millions) ^b
Africa	42.3	49.5	42.6	260.3
Americas	10.1	10.0	9.8	75.1
South-East Asia	39.9	95.6	39.8	624.0
Europe	59.9	42.2	56.9	435.5
Eastern Mediterranean	55.4	40.2	54.1	228.5
Western Pacific	26.2	48.0	24.0	365.3
Total	36.5	285.4	35.2	1988.7

^a 192 WHO Member States.

^b Based on population estimates in the year 2002 (35).

Source: WHO (34).

endemic cretinism. On the other hand, when fetal and neonatal hypothyroxinaemia occur later, after the first phase of maximal brain growth velocity, the long-term clinical consequence is severe thyroid insufficiency with stunted growth, myxoedema, delay in sexual development, but with a lesser degree of neurologic impairment and mental deficiency. This corresponds to the previous syndrome of myxoedematous endemic cretinism (28,36–38).

2.1.2.2 Iodine deficiency in the neonate

Iodine deficiency results in increased perinatal mortality, low birth weight on average and a higher rate of congenital anomalies (39). Even mild and moderate iodine deficiency, if occurring during the neonatal period, affects the intellectual development of the child (28). Neonatal thyroid stimulating hormone (TSH) is elevated. This indicator appears to be a particularly sensitive tool in the evaluation of the iodine status of a population and in the monitoring of iodine intervention programmes (40).

2.1.2.3 Iodine deficiency in children and adults

A high degree of apathy has been noted in populations living in severely iodine deficient areas (41). This affects the capacity for initiative and decision-making, indicating that iodine deficiency constitutes an hindrance to the social development of communities (41). It is also a major teratogen at the community level (42).

In addition to its impact on brain and neuro-intellectual development, iodine deficiency at any period of life, including during adulthood, can induce the development of goitre with mechanical complications and/or thyroid insufficiency. When iodine intake is abnormally low, adequate secretion of thyroid hormones may still be achieved by adaptive processes, including the stimulation of the trapping mechanism of iodide by the thyroid, as well as by the subsequent steps of the intrathyroidal metabolism of iodine leading to preferential synthesis and secretion of T_3 . This mechanism is triggered and maintained essentially by an increased secretion of TSH by the pituitary gland. The morphological consequences of prolonged thyrotropic stimulation are thyroid hyperplasia and goitre (43).

Another consequence of long-standing iodine deficiency in adults (32), but also in the child (44), is the development of hyperthyroidism in multinodular goitres with autonomous nodules in which thyrocyte proliferation occurs with scattered cell clones harbouring activation mutations of the TSH receptors. This mechanism is also responsible for the development of iodine-induced hyperthyroidism in case of sudden iodine overload in a previously severely iodine-deficient population (33,45). It is now accepted that hyperthyroidism in the elderly and iodine-induced hyperthyroidism are parts of the disorders induced by iodine deficiency.

2.2 Social and economic consequences

2.2.1 Cost-effectiveness and cost-benefit

Clearly, a programme for the control of iodine deficiency is worthwhile, in health economics terms, only if the cost of the programme is lower than the benefits which result from the correction of the deficiency. Previous studies have shown that fortification is both cost-effective (i.e. it has advantages over other methods as a way of increasing iodine intake), and has a high benefit–cost ratio (i.e. it is a good health investment) (46). Cost-effectiveness of an approach is defined as the cost of achieving some specified outcome, in this case, the cost of averting each case of goitre or iodine deficiency. While sometimes expressed in terms of cost per death-averted, this is a less useful calculation for iodine fortification, where effects on mortality are less well documented, and the important benefits include increased productivity instead (46). The key economic effects are assumed to be brain damage in infants. Likewise, cost-effectiveness, which is especially useful when comparing different programmes with the same outcome, is less useful in the design of iodine deficiency control and prevention programmes because there is broad consensus on the intervention of choice, salt iodization (27). Past studies, all now somewhat dated, and taken from different national settings, have attempted to compare the cost-effectiveness of iodized oil injections in Bangladesh, Indonesia, Peru and Zaire (14–46 1987 US cents), with water fortification in Italy (4 1987 US cents) and salt fortification in India (2–4 1987 US cents) (47–49).

Cost–benefit is where the monetary cost of an intervention is compared to the monetary value of the outcomes or benefits (46). In this case the benefits are increased productivity and reduced health costs, as well as the other costs of caring for those damaged by iodine deficiency. Cost–benefit estimations are useful for advocacy for increased resources and to compare health and nutrition interventions to the many other kinds of government spending (46). In calculating cost–benefit ratio, the proximate health intervention outcome, such as reduction in the prevalence of goitre, or change in median UI of the population, has to be converted to a financial outcome, usually derived from other studies. Thus, past studies that looked at the cost of productivity loss per birth to a mother with iodine deficiency, are used to estimate the cost of one person removed from goitre (as an intermediate outcome) (46).

WHO has looked at the cost–effectiveness and benefit–cost ratios of micronutrient interventions, especially fortification (46). Salt iodization programmes were again found to have “very high cost-benefit ratios” (46). If the cost of iodine fortification is \$0.10 per person per year (50), then the benefit : cost ratio of iodine fortification is about 40 : 1. If the costs are as low as \$0.01 per

person per year, as suggested for Central America (O. Dary, personal communication, 2002), then the programmes will have a considerably increased benefit : cost ratio of something like 400 : 1.

2.2.2 Social benefits of the elimination of iodine deficiency

The social benefits of the elimination of iodine deficiency have been extensively evaluated on the basis of reviews of data collected from several regions of the world: Bolivia, Ecuador, Germany, India, including Sikkim, and the USA (47,49,51,52). The complexity of studying the social and economic costs of iodine deficiency arise partly from the fact that the problem of iodine deficiency is no longer viewed as limited to goitre. Its most important effects are on the growth and development of the fetus, the neonate and the child. Potential benefits in preventing iodine deficiency are then much increased, including more and better education for children, greater productivity throughout life, and a better quality of life. Likewise the impact or effects of iodine deficiency interventions can be wide-ranging, and consequently not all will be captured and considered. An example of this is from China where a town with a traditionally high prevalence of iodine deficiency had virtually no out-marrying (due to perceived “stupidity” of young people from that town). After salt iodization, not only did the economic prosperity of the town go up but there was an observed increase in marriages with people from other towns (41).

The positive effects of mitigating iodine deficiency in individuals and communities include a reduction in mental deficiency, deaf mutism and hypo- and hyperthyroidism, as well as more subtle retention of mental potential, not easily measured on IQ tests. Particularly in the European context the main effects are consequences on brain development, rather than a reduction in more obvious effects such as deaf mutism. There is an often quoted estimation of the number of IQ points lost to infants born to mothers living in endemically deficient environments (53). While scientifically imprecise, this estimate of 10–15 IQ points lost does however have considerable advocacy strength for national policy-makers in terms of lost economic potential in their countries. In countries with a long history of iodine deficiency prevention and control programmes, such as in many European countries, these losses are subtle enough to be missed, but presumably are affecting the life chances of many children of unsuspecting parents, in an increasingly competitive and globalized world.

As USI mandates that iodized salt be used for livestock as well as for humans, the benefits to agriculture should also be factored in (Table 2.3). This review does not consider the literature from the agronomic side on the costs to primary industry of iodine deficiency on crop yields and animal productivity in Europe. However, improving the iodine nutrition of farm animals will result in an increase in live births and weight gained, as well as increases in general health and strength. Other positive effects, such as quality of wool in sheep, meat in cows and a reduction in congenital abnormalities, will also be seen (56,57). Farmers are more likely to be convinced of the need to

Table 2.3 **Benefits of iodine intervention programmes**

Physiologic benefits		Benefits to society
Humans	Reductions in: <ul style="list-style-type: none"> • Mental deficiency • Deafmutism • Spastic diplegia • Squint • Dwarfism • Motor deficiency • Goitre • Birth defects 	<ul style="list-style-type: none"> • Higher work output • Reduced costs of medical and custodial care • Reduced educational costs (because of less absenteeism and grade repetition)
Livestock	Increases in: <ul style="list-style-type: none"> • Live births • Weight and meat yield • Strength for work • Health (less deformity) • Wool coat in sheep 	Higher output of meat and other animal products and hence: <ul style="list-style-type: none"> • Profits • Higher work output of animals

Source: Levin et al. (49), Hetzel & Maberly (54), Levin (55).

supplement animal feed when such productivity increases are observed, and hence are potentially potent advocates for USI.

2.2.3 Economic benefits of the elimination of iodine deficiency

In India, the second most populous country in the world with an estimated population of 834 million (1991), the costs of the national salt iodization programme were estimated to be 25.9 million Euros, including land and buildings, iodization plants and equipment, potassium iodate, labour, supervision and administration, maintenance and electricity (47). The benefits of iodine deficiency control included were improved neurological, mental, auditory and speech capabilities as well as skeletal growth. The resulting higher work productivity, reduced costs due to reduced absenteeism, and higher achievements by students were also considered. The potential benefits of control of iodine deficiency in livestock populations were also considered as part of the overall benefit. Using various assumptions, the benefit of salt iodization in terms of productivity and of the money that would be saved on the management and support of those affected, resulted in a cost : benefit ratio of 1: 3.

Correa (58) reported that the estimated cost of salt iodization programmes in different parts of the world varied from US\$ 0.0025 to US\$ 0.1000 per person per year while the estimated cost for intramuscular iodized oil was US\$ 0.4338 per person per year (in the late 1970s). The study made assumptions based on some data from Chile that demonstrated the benefit of the iodine supplementation programmes on higher earnings associated with a reduction in mental deficiency. Since the increased IQ of the children would produce economic benefits only after several years, it was assumed that such an increase would begin to take place 15 years later but that after this initial delay, the benefit of the programme would be continued for as long as an iodine deficiency prevention and control programme was in place. According to this study, the benefit of reducing iodine deficiency among children, in terms of improvement in their lifetime earnings, also considerably exceeded the cost of the interventions.

In the USA, benefits of screening for iodine deficiency and treatment were estimated to be equal to three times the costs and included savings in treatment, long-term care and productivity losses (59). Choice of outcome measures is a key issue in economic evaluations of disease, and has helped preclude the effective advocacy of interventions aimed at iodine deficiency prevention and control. Nevertheless, it has been concluded in industrialized countries, such as the USA, that “cost-effectiveness of screening 35 year olds for serum TSH every five years favorably compared with other generally accepted preventive medical practices...” (60).

The economic benefits at the individual or household level are less well documented. The example from China above concerns whole communities. Other work in China has shown increased average incomes, export of cereal crops for the first time and the result that men became fit enough to join the army – presumably a benefit (49). In Ecuador, people with moderate iodine deficiency were consistently paid less for agricultural work (61). As noted, with the milder iodine deficiency seen in Europe, the gains in academic performance and individual and national productivity would be less, but, on a national basis, and especially for individuals, worthy of pursuing. From a national economic productivity perspective, as well as agricultural output and profitability, and extrapolating from modelling done by the Academy for Educational Development’s (AED) Profiles Project, preventing and controlling iodine deficiency would be expected to provide considerable economic benefit to countries, including those in Europe, having mild levels of iodine nutrition deficiency.

2.2.4 Conclusion

Because the calculations of cost-effectiveness and cost–benefit are a function of methodological differences, exchange rates, time, context, local price differences and the population base, these studies are of limited utility in terms of being able to be fully extrapolated (49). However together they do demonstrate “a high payoff from iodine interventions” (49). While more information and analysis are needed, and these studies are now somewhat dated, there is no reason to suppose the basic conclusions would have changed. It might even be argued that the increasing technology of the globalized world favours a higher benefit for avoiding neuro-intellectual damage. It appears

that the long-term correction of iodine deficiency is feasible at low cost and that the benefits of correcting iodine deficiency far outweigh its risks (62,63).

2.3 Assessment of iodine deficiency

The initial assessment of iodine deficiency at the population level in order to decide on the need and the nature of interventions for iodine correction is accomplished by a series of steps of increasing technological complexity. First, the problem is assessed by the prevalence and severity of goitre. If the enlarged goitres are small and hence difficult to diagnose, or grade, then urine is tested for amount of iodine excretion and graded against the standard prevalences signifying the seriousness of the public health problem (27). If more detailed information is needed, then evidence of abnormal hormonal levels (e.g. TSH) would be required.

The presence of iodine deficiency has been traditionally diagnosed where visible goitres (enlarged thyroid glands) are present. In past centuries, in some parts of Europe, goitres were so common that they were considered normal, and when of modest size, even to be cosmetically desirable, as in some of the portraiture in the Renaissance. High levels of goitre were generally found in mountain areas, alluvial plains and usually well away from the coastline. However, the greater availability of UI testing (see below) and other methods for diagnosing iodine deficiency, have demonstrated that the iodine deficiency disorders may occur in many other areas, including large cities, in areas where the prevalence of goitre as assessed by palpation has indicated no public health problem, and in industrialized countries such as many in Europe (27).

Goitre levels remain useful for initial assessment of the problem but are generally not suitable for monitoring purposes. Many countries continue to use TGP when describing their national problem, often because of an absence of other measures. In the past decade, however, an increasing number of countries have come to use UI – even for a first assessment.

2.3.1 Prevalence of goitre

The size of the thyroid gland changes inversely in response to alterations in iodine intake, with a lag interval that varies from a few months to several years (27,64), and so the prevalence of goitre is an index of the degree of long-standing iodine deficiency. Thyroid size has been traditionally determined by inspection and palpation but ultrasonography of the thyroid provides a more precise and objective method. It has already been used in several regions of the world, in particular in Europe. It is likely to be used more extensively in the future. The normative values for thyroid volume measured by ultrasonography are expressed as a function of age, sex and body surface area. The initially proposed values (18) have recently been re-evaluated by WHO (65). By definition, a thyroid gland is considered as goitrous when its volume is above the 97th percentile established for sex, age and body surface area in iodine-replete populations. The prevalence of goitre in iodine replete populations is below 5% (27). Table 2.4 shows the epidemiological criteria presently recommended for assessing the severity of iodine deficiency based on the prevalence of goitre in school-age children (i.e. children aged 6–12 years unless otherwise noted).

Table 2.4 **Epidemiological criteria for assessing the severity of iodine deficiency based on the prevalence of goitre in school-age children**

Indicator	Degree of iodine deficiency expressed as percentage of total number of children surveyed			
	None	Mild	Moderate	Severe
Total goitre prevalence	0.0–4.9	5.0–19.9	20.0–29.9	≥30

Source: WHO/UNICEF/ICCIDD (27).

2.3.2 Urinary iodine

Urinary iodine (UI) is a good marker of the recent dietary intake of iodine. It provides an adequate assessment of a population's iodine nutrition and is now the index of choice for evaluating the degree of iodine deficiency, and for monitoring and evaluating its correction (27). Because 24-hour samples are difficult to obtain it is emphasized that this is not necessary and that iodine concentrations can be measured in casual urine specimens of children or adults provided a

sufficient number of specimens (usually at least 30 schoolchildren per cluster) is collected (27). It is reported as micrograms/litre ($\mu\text{g/l}$) of urine. Relating UI to creatinine is expensive and unnecessary (27).

As the frequency distribution of UI is usually skewed towards elevated values, the median is considered more useful than the mean to indicate the status of iodine nutrition in a population. Table 2.5 shows the epidemiological criteria presently recommended for assessing iodine nutrition based on median UI concentrations in school-age children (27).

Table 2.5 **Epidemiological criteria for assessing iodine nutrition based on median UI concentrations in school-age children**

Median urinary iodine ($\mu\text{g/l}$)	Iodine intake	Iodine nutrition
<20	Insufficient	Severe iodine deficiency
20–49	Insufficient	Moderate iodine deficiency
50–99	Insufficient	Mild iodine deficiency
100–199	Adequate	Optimal
200–299	More than adequate	Risk of iodine-induced hyperthyroidism within 5–10 years following introduction of iodized salt in susceptible groups
≥ 300	Excessive	Risk of adverse health consequences (iodine induced hyperthyroidism, autoimmune thyroid diseases)

Source: WHO/UNICEF/ICCIDD (27).

In pregnant women a median UI <150 $\mu\text{g/L}$ indicates insufficient iodine intake, 150–249 $\mu\text{g/L}$ indicates adequate intake, 250–499 $\mu\text{g/L}$ reflects more than adequate intake and ≥ 500 $\mu\text{g/L}$ indicates intake for which no added health benefit is expected (29).

2.3.3 Serum concentrations of TSH, thyroid hormones and thyroglobulin

The serum concentrations of serum TSH and thyroid hormones further reflect the effects of iodine deficiency. Serum TSH typically increases in iodine-deficient populations, as does the serum triiodothyronine (T_3), while serum thyroxine (T_4) decreases. However, all of these may still remain within the normal range even in the face of iodine deficiency, and are therefore insufficiently sensitive to gauge levels of iodine deficiency, unless severe. Serum thyroglobulin represents a very sensitive index of a state of thyroid hyperstimulation (27).

In endemic areas, neonatal serum TSH is frequently elevated because of the hypersensitivity of the neonate to the effects of iodine deficiency, and, in these endemic areas, the recall rate for further investigation of infants under suspicion of congenital hypothyroidism in programmes of systematic screening for congenital hypothyroidism is elevated. Elevated neonatal TSH is the single indicator that best predicts brain damage and impairment of intellectual development. Systematic screening for congenital hypothyroidism, if already available, is a particularly sensitive tool in the evaluation of the iodine status of a population and in the monitoring of iodine supplementation programmes (40).

2.4 Prevention and control

Prevention and control activities basically aim to correct iodine deficiency in populations whose diets are providing insufficient iodine to meet requirements. As iodine has often been leached from the soil by glacial action and water washing out the soluble iodine, mountainous areas are particularly susceptible, but as already noted, by no means the exclusive sites of iodine-deficient populations. Traditionally, populations living close to the sea have been less at risk because marine foods, including seaweed, tend to be high in iodine. Other ways of replacing iodine have been through supplementation, tinctures of iodine, iodized oil, both intramuscular injections and oral, and by adding it to foods, especially salt but also water supplies and other food vehicles. Overwhelmingly, salt iodization is the intervention of choice, with the others ancillary to reach hard-to-access populations at high risk.

2.4.1 Salt iodization

The main intervention strategy for iodine deficiency control and prevention is universal salt iodization (USI). The key word is “universal” as it underlines the importance that all salt for human

consumption, including that used in processed foods, and that used for animal consumption, be iodized. This is often not the case in European countries. USI was first adopted by the World Health Assembly in 1992 (22) and established as a World Summit for Children mid-decade goal in 1995 (23). Salt iodization has been a remarkably successful intervention as it is feasible, cheap, safe, rapidly effective and widely accepted. There is also now considerable experience in many different countries, cultures and hence dietary practices. As noted, iodized salt is the most appropriate measure for iodine deficiency control because it is used by all sections of a community, is consumed at roughly the same level throughout the year and because its production is often confined to a few centres which facilitates quality control at the level of production (27,66).

Iodine can be added to salt as potassium iodide (KI), potassium iodate (KIO₃) or sodium iodide. Potassium salts are the most frequently used. Although slightly more expensive than iodide, the iodate salt is preferred, especially for moist, tropical conditions and where storage conditions are likely to be less than optimal, because it is less soluble and more stable than iodide. While most countries of the world use the iodate form, several countries in Europe (e.g. Switzerland) and elsewhere (e.g. Canada and the USA) use iodide.

The preconditions needed to ensure adequate salt iodization availability and consumption include:

- local production and/or importation of iodized salt in a quantity that is sufficient to satisfy the potential human demand (approximately 4–5 kg/person per year);
- 90% of salt for human consumption (whether local or imported) must be iodized according to government standards for iodine content;
- the percentage of food-grade salt with an iodine content of at least 15 ppm, in a representative sample of households, must be equal to or greater than 90%; and
- iodine estimation at the point of production or importation, and at the wholesale and retail levels, must be determined by titration; at the household level, it may be determined by either titration or certified kits (27).

Assuming an average salt consumption of 10 g of salt/capita per day, a loss of iodine of some 20% between production and retail and another 20% during food processing (67), to achieve the recommended level of iodine of 150 µg per person for adults, iodine in the salt should be in the range of 20–40 mg/kg (20–40 ppm) (68). The packaging of iodized salt is very important. To avoid losses as high as 75% over a period of nine months, waterproof packaging is required, as re-emphasized in the recent amended *Codex Alimentarius. Standard for Food Grade Salt* (69). Regulations and packaging information should specify the iodine content rather than that of KI or KIO₃.

Table 2.6 summarizes the latest global information of iodized salt consumption at the household level (70). Sixty-six per cent of households of the surveyed countries have access to iodized salt, as compared to 5 to 10% in 1990 (20).

2.4.2 Iodized oil

There continues to be an important role for iodized oil, especially in areas where iodized salt is still, for the meantime, particularly difficult to introduce. A variety of iodized oils have been used including most commonly, Lipiodol®, a poppy seed oil containing 40% iodine per weight. Over the past six or seven decades, iodized oil has been extensively used, initially in Papua New Guinea and thereafter in Africa, China and Latin America, and in many of the most severe endemic areas in the world (71), as well as in Europe (e.g. Romania) (72). More than 20 million doses of iodized oil have been administered since 1974, initially by injections and subsequently orally, with very few side-effects, including during pregnancy (73).

In the groups most susceptible to the effects of iodine deficiency, women of reproductive age, pregnant women and children less than 2 years, iodine supplements such as iodized oil are recommended where salt iodization coverage is inadequate (29).

2.4.3 Other methods

Bread fortified with iodine, often indirectly, has been utilized successfully in many countries, including countries of Europe, as well as Australia and the Russian Federation, in those areas where

Table 2.6 **Current global status of iodized salt consumption at the household level**

UNICEF region	Proportion (%) of households consuming iodized salt ^a
Sub-Saharan Africa	67
Middle East and North Africa	53
South Asia	49
East Asia and Pacific	80
Latin America and Caribbean	81
CEE/CIS and Baltic States	39
Industrialized countries	-
Developing countries	68
Least developed countries	54
Total	67

- No data.

CEE/CIS: central and eastern Europe/Commonwealth of Independent States.

^a Data refer to the most recent year available during the period 1997-2002. Source: Adapted, with permission, from UNICEF (70).

bread is a staple (39). Since 1942, the main carrier for iodine in the Netherlands has been the salt in bread. However, iodine intake remained inadequate, especially in Dutch women (74), and recently there has been an increase in the potassium iodide content of baker's salt to address this. Iodized water has been successfully used in several countries such as the Central Asian Republics, Italy (Sicily), Mali, and Thailand. A limiting factor of this approach, especially in terms of cost-effectiveness, is the question of availability of one single source of iodine for the whole population and for the livestock (75). Sugar has been iodized in pilot studies in Guatemala and the Sudan, and iodized tea has been used in China.

Indirect iodization has also been shown to be effective in correcting iodine deficiency. Adding potassium iodate into irrigation water has been reported as highly successful in Xinjiang, China: for example, the infant mortality rate dropped by 50% (76). Milk has adventitiously had its iodine content raised in many dairy-producing countries consequent to the use of iodophors in the dairy industry (e.g. to clean the teats). Iodine-rich milk thus became a major source of iodine in many countries in northern Europe, as well as in Australia, the United Kingdom and the USA (77). A change in dairy practice would reverse the situation and increase the likelihood of iodine deficiency in those populations. (Consequently it is of interest that iodophors are no longer permitted for this use in some countries in Europe.) Finland, besides fortifying their table salt for many years, has been fortifying animal fodder and the iodine content of foods derived from animal sources has increased. The salt licks used by animals in much of European stock-raising should be fortified with iodine but often are not. However, if the added iodine becomes part of the ecological system, cost-effectiveness is improved, and sustainability likely; quality control, however, may be harder to ensure.

These other options to increase iodine intake will become increasingly important within the next few years as a result of the policy adopted by many countries to reduce salt consumption intake to 5 g/day in order to prevent hypertension and cardiovascular diseases. This could potentially create a conflict between the two major public health goals of reducing average population salt intake and tackling iodine deficiency through salt iodization. Therefore, salt iodization should not promote salt consumption and countries should be encouraged to implement complementary measures to increase iodine intake (78).

2.4.4 Adverse effects associated with the correction of iodine deficiency

There are recognized adverse effects associated with the correction of iodine deficiency which can result in the development of thyroid function abnormalities. In public health terms, these are relatively minor; severe effects occur rarely. The benefits of the prevention of the neuro-intellectual damage from iodine deficiency far outweigh the side-effects that have been observed.

Iodine excess can follow over-correction of a previous state of iodine deficiency, and can also

impair thyroid function. The effect of iodine on the thyroid gland shows a U-shaped relationship between iodine intake and risk of thyroid diseases, as both extremes of low and high iodine intake are associated with an increased risk. Normal adults, at an individual level, can tolerate up to about 1000 µg iodine/day without any side-effects (79). However this upper limit is set considerably lower at the population level due to the range of individual variation and due to exposure to iodine deficiency in the past. The optimal level of iodine intake to prevent any thyroid disease may be within a relatively narrow range around the recommended daily intake of 150 µg.

In order to assess the risk of adverse effects from excessive intake of any nutrient, including iodine, intake can be compared to the tolerable upper intake level (UL), which is the highest average daily nutrient intake level likely to pose no risk of adverse effects to almost all individuals in the general population (80). WHO suggests a provisional maximal tolerable daily intake of 1 mg/day from all sources (81). The UL for iodine proposed by the European Commission's Scientific Committee for Food (EC/SCF) is 600 µg/day for adults and pregnant women (82). For children, since there is no evidence of increased susceptibility in children, the ULs are derived by adjustment on the basis of body surface area and ranges from 200 µg/day for 1–3 year olds to 500 µg/day for 15–17 year olds (82). In countries with long-standing iodine deficiency, the intake should not exceed 500 µg/day to avoid the occurrence of hyperthyroidism. In France, the Expert Committee on Human Nutrition has suggested an UL of 500 µg/day for that reason (83). In the USA, where the median intake of dietary iodine is about 240–300 µg/day for men and 190–210 µg/day for women, the UL is set at 1 100 µg/day, a value based on serum thyrotropin concentration in response to varying levels of ingested iodine while the recommended dietary allowance (RDA) for adult (men and women) is 150 µg/day (84). Table 2.7 shows that UL figures set forth by the Institute of Medicine (IOM) in the USA are generally higher than those of the European Commission, for all age groups (84).

Table 2.7 **Tolerable upper intake level for iodine (µg/day)**

Age group	EC/SCF, 2002	IOM, 2001
1-3 years	200	200
4-6 years	250	300
7-10 years	300	300
11-14 years	450	300
15-17 years	500	900
Adult years	600	1100
Pregnant women >19 years	600	1100

Source: European Commission/Scientific Committee on Food (82), Institute of Medicine (84).

When the iodine intake is chronically high (e.g. due to the environment) the prevalence of thyroid enlargement and goitre is high as compared to other populations, and the prevalence of subclinical hypothyroidism is elevated. The mechanisms behind this impairment of thyroid function are probably both iodine enhancement of thyroid autoimmunity and reversible inhibition of thyroid function by excess iodine (Wolff-Chaikoff effect) in susceptible subjects (85). However, this type of thyroid dysfunction has not been observed after correction of iodine deficiency, including in neonates after the administration of high doses of iodized oil to their mothers during pregnancy (73).

The possible side-effects of iodine excess include iodine-induced hyperthyroidism (IIH) as the main complication of iodine excess of public health significance. It has been reported in almost all iodine supplementation programmes (33). The disease has been observed to occur most frequently in individuals over 40 years of age with multinodular goitres, in autonomous nodules which have lost their mechanism of autoregulation against iodine excess. The most effective way to prevent IIH is to ensure effective monitoring of iodized salt quality and to train health staff to be alert in the identification of IIH (86).

Iodine in excess may also aggravate or even induce autoimmune processes in the thyroid resul-

ting in iodine-induced thyroiditis. However the mechanisms involved are still unclear (32,87). Some studies suggest also that iodine supplementation may be associated with a change in the epidemiological pattern of thyroid cancer, with a shift towards differentiated forms of thyroid cancer, which are generally detected earlier than less differentiated forms (88–90).

2.5 Monitoring and evaluation

Monitoring of iodine deficiency prevention and control programmes involves a series of well-established steps of increasing complexity (27) and are summarized, with cut-off points, in Table 2.8. These steps are carried out in a logical sequence starting with monitoring: i) the quality of iodized salt; then, ii) the adequacy of iodine nutrition; iii) the progressive disappearance of goitre; and, finally iv) the normalization of thyroid function.

Salt iodization is not an end in itself but only a means to achieve optimal iodine nutrition. It is why besides monitoring iodized salt quality, iodine status also needs to be monitored. As UI is a good marker of the recent dietary iodine intake, it is, therefore, the index of choice for evaluating correction of iodine deficiency. Changes in the prevalence of goitre after normalization are slow and often incomplete, and become more difficult to diagnose as the enlarged glands diminish in size, and hence goitre prevalence is less sensitive to the correction of iodine deficiency.

Finally, once the iodine content of salt has been consumed at a level that results in the normalization of UI, attention may shift to the evaluation of actual thyroid function by the determination of the serum levels of TSH, T_4 , T_3 and thyroglobulin, for confirmation, especially in more vulnerable groups.

2.5.1 Monitoring of salt iodization

Ensuring adequate iodization of salt is the first step in the process of eliminating iodine deficiency at a population level. Due to the strengthened partnerships with salt producers, the shift in monitoring is from quality control towards quality assurance. Quality assurance methods are well established and are the responsibility of the salt producers. Quality control will, of course, remain necessary for national assurance of the programme and for decisions on the successful elimination of the problem in a country.

The most reliable technique for making a quantitative determination of iodine level in salt is titration. This consists of measuring the free iodine liberated from iodate in a salt sample in the presence of sodium thiosulphate with starch as the external indicator. Government laboratories and the salt industry should have the facilities to carry out this method and use it for monitoring salt quality. Over the past decade, several types of rapid test kits have been developed for use in the field. They give qualitative results, indicating whether iodine is present or absent. While their reliability in determining the amount of iodine present has been questioned (91), their use as an effective tool for quality control and assurance, and hence for advocacy, remains valuable.

2.5.2 Monitoring iodine status

UI is recommended as the index of choice for monitoring the correction of iodine deficiency (27). The procedure and epidemiological criteria are discussed in section 2.3.2.

The total prevalence of goitre has been used for much of the recent past to assess and monitor iodine deficiency. However, as noted above, it is an index of long-standing iodine deficiency and, therefore, is less sensitive than UI in the evaluation of a recent change in the status of iodine nutrition (27).

2.5.3 Monitoring thyroid function

As already indicated, the final objective of programmes to correct iodine deficiency is the normalization of thyroid function. This is especially so in those most vulnerable to the consequences of the deficiency: pregnant and lactating women and young infants. The biochemical indicators to be used are listed and discussed in section 2.3.3. Normal serum levels of the free fractions of thyroid hormones and TSH indicate an adequate supply of thyroid hormones at the cellular level. A normalized serum level of thyroglobulin indicates the disappearance of a state of thyroid hyperstimulation, which occasionally occurs with some delay after normalization of serum T_4 and TSH.

2.5.4 Indicators to monitor progress towards the international goal of elimination of iodine deficiency

There has been outstanding unanimity on the goal of eliminating iodine deficiency and how this might be done. Since the adoption of the goal, in the 1990s, remarkable progress has been made. In many countries iodine deficiency is now under control and it is anticipated this progress will continue. Table 2.8 provides the criteria adopted by WHO and UNICEF for monitoring progress towards sustainable elimination of iodine deficiency disorders. These criteria include indicators related to salt iodization, urinary iodine status and to the programme itself. The last criterion is essential as it provides information on a programme's sustainability. As noted above, the control of iodine deficiency is a continuous process; it cannot be interrupted without the risk of re-emergence of iodine deficiency and its resulting disorders.

In addition to the components described in detail above (see section 1.2.3 and Figure 1) sustainability also includes the need to keep the general public well informed, and maintaining a partnership with the salt producers to ensure quality assurance. In brief, iodine deficiency is considered eliminated from a particular country when the following factors occur in combination: i) at least 90% access to iodized salt at the household level; ii) a median UI of at least 100 µg/l among at least 50% of a population; iii) less than 20% of the samples below 50 µg/l; and iv) implementation of at least eight of the 10 programme indicators listed in Table 2.8 (27).

Table 2.8 **Criteria for monitoring progress towards sustainable elimination of iodine deficiency disorders**

Indicators	Goals
Salt iodization	
<ul style="list-style-type: none"> Proportion of households using adequately iodized salt^a 	>90%
Urinary iodine	
<ul style="list-style-type: none"> Proportion of population with UI levels below 100 µg/l Proportion of population with UI levels below 50 µg/l 	<50% <20%
Programmatic indicators	
<ul style="list-style-type: none"> An effective, functional national body (council or committee) responsible to the government for the national programme for the elimination of IDD (this control should be multidisciplinary, involving the relevant fields of nutrition, medicine, education, the salt industry, the media, and consumers, with a chairman appointed by the Minister of Health) Evidence of political commitment to USI and elimination of IDD Appointment of a responsible executive officer for the IDD elimination programme Legislation or regulations on USI (while ideally regulations should cover both human and agricultural salt, if the latter is not covered this does not necessarily preclude a country from being certified as IDD-free) Commitment to assessment and reassessment of progress in the elimination of IDD, with access to laboratories able to provide accurate data on salt and UI A programme of public education and social mobilization on the importance of IDD and the consumption of iodized salt Regular data on salt iodine at the factory, retail and household levels Regular laboratory data on UI in school-age children, with appropriate sampling for higher risk areas Cooperation from the salt industry in maintenance of quality control A database for recording of results or regular monitoring procedures particularly for salt iodine, UI and, if available, neonatal TSH, with mandatory public reporting 	At least 8 of the 10

^a Adequately iodized salt refers to at least 15 ppm at household level.

3. Iodine deficiency in Europe and its control: current status, progress and recent trends

3.1 Methods used to record information

The information reported in this review is based on indicators of the European population's iodine status (UI, TGP and indicators of thyroid function) and status of the interventions carried out by health authorities to control iodine deficiency, mainly salt iodization (salt quality, including iodine content, and levels of salt consumption).

This review includes data from the 40 European countries made up of European Union Member States including applicant countries and EFTA countries. A detailed list of the countries included in the review is given in Annex B.

While writing this review, national experts from each of the 40 countries were contacted and asked to provide their most current data on the subject. Thus, the information contained herein is based on the best data currently available to WHO. Data are not necessarily the official statistics of countries.

3.1.1 Urinary iodine and goitre prevalence

For each country, the most representative data available on UI and TGP are selected and presented according to the methodology described previously (34).

3.1.1.1 Data sources

Data on UI and TGP have been extracted from surveys performed during the past ten years, 1994–2004, and compiled in the WHO Global Database on Iodine Deficiency (<http://who.int/vmnis>). In August, 2006, figures for UI were updated through the end of 2005 for five countries with new national data in school-age children. For more details on inclusion criteria for the database, see Annex C.

3.1.1.2 Selection of survey data

Surveys were first selected according to the administrative level. Surveys are considered as national level when they are carried out on a nationally representative sample of the population, or as sub-national level when they are carried out on a sample representative of a given administrative level: region, state, province, district or local.

Whenever available, data from the most recent national survey were used in preference to sub-national surveys. In the absence of national data, subnational data were used. When two or more subnational surveys of the same subnational level had been carried out in different locations of the country during the analysis period, the survey results were pooled into a single summary measure, using a weighted sample size for each survey.

WHO recommends that iodine deficiency surveys examine school-age children (6–12 years) (27). When data for this age group were not available, data of the next closest age group were used in the following order of priority: data from the children closest to school age, adults, the general population, preschool-age children, other population groups.

3.1.1.3 Population coverage

The population coverage of the data was calculated as the sum of the population of countries with data divided by the total population of Europe and expressed as a percentage. Population figures are based on population estimates for the year 2002 (35).

3.1.1.4 Classification of iodine nutrition

UI data were expressed in µg/l as recommended by WHO (27). Data expressed as µg/g creatinine were not included.

Countries were classified based on data of iodine nutrition: varying median UI levels correspond to different degrees of public health significance (Table 2.5). Median UI below 100 µg/l defines a population which has iodine deficiency. If a national median UI was not available, the methods were applied to derive a median from various available UI data (34).

3.1.1.5 Proportion of population and the number of individuals with insufficient iodine intake

National and regional populations (school-age children and general population) with insufficient iodine intake were estimated based on each country's proportion of population with UI below 100 µg/l as described previously (34).

3.1.1.6 Total goitre prevalence

In surveys where thyroid size was measured by palpation, goitre prevalence was reported as overall TGP, according to the WHO classification (27). For some countries, TGP was derived from thyroid size as measured by ultrasonography. The method used, including reference values applied, was carefully quoted for each survey and referenced.

3.1.2 Serum concentrations of TSH, thyroid hormones and thyroglobulin

Current available information on TSH, thyroid hormones and thyroglobulin in the literature were reviewed and summarized. Priority was given to TSH, especially during pregnancy and for neonates, as being the most sensitive indicator of a lack of thyroid hormones in the growing brain. Serum thyroglobulin was used as a particularly sensitive marker of the degree of stimulation of the thyroid gland.

3.1.3 Salt iodization programmes

Salt iodization, legislation, the proportions of households consuming iodized salt and other process monitoring data have been collected. As these data have come from national authorities and hence may have been collected from different formats, less rigorous criteria were used for their inclusion. In this case the data most similar to that in the tables were used and footnotes included. The data reported for legislation, permitted substances and market shares of iodized household salt by country were primarily based on the data collected by the European Salt Producers' Association (the European Salt Producers' Association, personal communication, 2003). For data on salt penetration rate (proportions of households consuming iodized salt) the main source of data was the database maintained by UNICEF on salt consumption (70). For the process indicators for monitoring progress towards elimination, the data collected by ICCIDD were used (8).

3.2 Epidemiology and severity of iodine deficiency

The data obtained from each of the 40 countries for UI, prevalence of goitre and indicators of thyroid function are summarized below and in Annexes D, E and F.

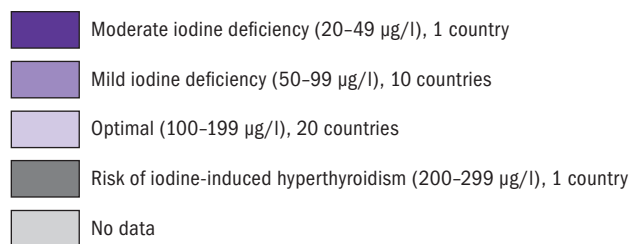
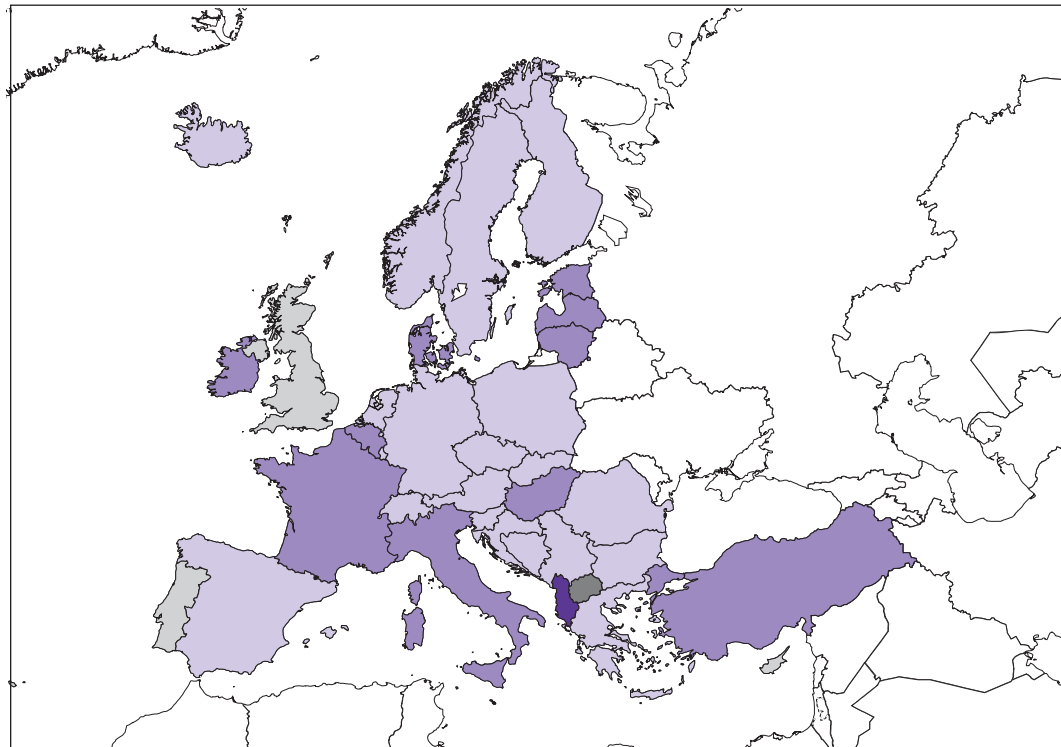
3.2.1 Urinary iodine

3.2.1.1 Population coverage

Data on UI collected between 1994 and 2004 were available from 32 of the 40 countries (Annex D). National data were available from 18 countries; 14 countries supplied subnational data (Figure 3.1). Eight countries had no data on UI.

Overall, the available UI data covered 90% of Europe's population 6–12 years of age. Fif-

Figure 3.2 Iodine nutrition based on median UI^a



The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement.

^a Refers to the most recent data available from 1994–2006.
Source: WHO Global Database on Iodine Deficiency.

severe iodine deficiency. The same is true for some countries classified as having adequate iodine nutrition on a national level, such as Bosnia and Herzegovina, and Serbia and Montenegro, where pockets exist in which people are still affected by iodine deficiency. Spain is a further example where the prevalence of iodine deficiency was previously known to vary markedly between regions (2), with limited areas of severe iodine deficiency complicated by cretinism (92). However, when pooling median UI from recent data, available from dispersed local and provincial areas, derived median UI levels indicate adequate iodine nutrition. Nonetheless, it is likely that Spain is still affected by some degree of mild iodine deficiency, considering the absence of a national intervention programme during the past decade. The country has recently announced the implementation of a national iodine deficiency programme.

3.2.1.3 Proportion of population and number of individuals with insufficient iodine intake

The proportion of the population and the number of individuals (school-age children and the general population) with insufficient iodine intake (defined as proportion of population with UI <100 µg/l) is presented by country in Annex D. The lowest proportion of the population with UI <100 µg/l is found in Bulgaria (6.9%) while the highest proportion is found in Albania (91.0%).

It is estimated that the iodine intake of 42.7% (22.2 million) of school-age children in Europe is insufficient (Table 3.1). Extrapolating the proportion of school-age children to the general population, it is estimated that 244 million individuals have insufficient iodine intake.

Table 3.1 **Proportion of population, and number of estimated individuals with insufficient iodine intake in school-age children (6–12 years) and in the general population (all age groups) in Europe, ^a 2004**

Insufficient iodine intake (UI <100 µg/l)			
School-age children		General population	
Prevalence (%) ^a	Total number (millions) ^b	Prevalence (%) ^a	Total number (millions) ^b
47.8	24.9	46.1	272

^a Based on data from 40 countries (Annex B and D).

^b Based on population estimates in the year 2002 (35).

3.2.2 Goitre prevalence

3.2.2.1 Population coverage

Data on TGP collected between 1994 and 2004 are available from 23 of the 40 countries (Annex E). They are nationally or subnationally representative in 13 and 10 countries, respectively. Nationally representative TGP data have been collected for 43.8% of Europe's population aged 6–12 years. No recent data are available in 17 countries. It should be noted that subnational data from selected area(s) only represent part of the country.

The national TGP, as measured by palpation, vary from 1.3% in Serbia and Montenegro to 47% in Slovenia. National TGP measured by ultrasonography varies from 0% in Switzerland and Croatia to 31.8% in Turkey.

TGP data should be interpreted with caution. They were collected using different methods. For example, TGP measured by ultrasonography applied different reference values in different cases, since international reference values were not yet available (65). Additionally, the age groups in which the data have been collected are not the same in all countries and TGP markedly varies with age. Finally, TGP is a poor index of the present status of iodine nutrition and changes very slowly subsequent to recent changes in iodine nutrition.

3.2.3 Assessment of iodine nutrition based on thyroid function

Indicators of thyroid function – serum concentrations of TSH, thyroid hormones and thyroglobulin – are not currently used to assess iodine status or monitor the impact of control programmes at the population level, although some of them, such as TSH, are likely to be used more extensively in the future. Rather they are used for individual assessment or for research purposes. In addition, the data currently available have been measured on small samples and are therefore difficult to compare to median UI or TGP as these are measured on a large scale at a national or subnational level. Finally, the present review is essentially based on data published in the scientific literature. This does not include the results of all the investigations that have been carried out, as many have not been published. Annex F summarizes the results of the indicators of thyroid function.

3.2.4 Progress and recent trends

From the above, it can be concluded that iodine deficiency and its many consequences are still a public health problem in much of Europe. Due to the use of varying methods and different indicators used in surveys (TGP versus UI) between the 1993 WHO estimates and this one (2,16), trend data are only pertinent when looking at country data individually. Data published by WHO in 1993 (16) were based on TGP and, at that time there were far fewer countries with such information, thus direct comparison is not feasible.

Briefly, the TGP data from the 1993 estimates for the 40 countries considered in this review reveal the following: iodine deficiency was a public health problem in 17 countries, two countries had iodine deficiency under control, 10 countries were likely to have iodine deficiency under control and 11 countries had no data available. Additionally, another review in 1993 concluded

that only five European countries had iodine deficiency under control (Austria, Finland, Norway, Sweden and Switzerland) (2).

Today, UI is the main indicator for assessing iodine status. The populations of 13 countries are considered iodine deficient, as indicated by UI; one country is moderately deficient and 12 mildly so (Figure 3.1). Nineteen countries have populations with adequate iodine intake. Iodine nutrition in these countries is considered as optimal. Another eight countries have no recent information available. As already noted, progress has clearly been made over the past five or six years. The greatest challenge now is to sustain and improve this progress.

3.3 Policy and legislation

It has been observed that the national commitment towards ensuring adequate iodine nutrition and its prevention is much weaker in most European countries than elsewhere in the world (8). Table 3.2 summarizes the data on regulations governing iodized salt and the penetration rate of iodized salt in households and, when permitted, in the food industry.

Iodine compounds used in fortification include potassium iodide (KI), sodium iodide (NaI) or potassium iodate (KIO_3). Many countries in Europe use KI instead of KIO_3 , as do other temperate zone countries such as Canada and the USA. KIO_3 is less soluble than KI and therefore more stable, but in temperate climates, with good quality salt, losses from KI are limited (93). Fifteen countries use KI exclusively, six use KIO_3 and 12 countries permit both and/or NaI. There are no available data for the remaining seven countries (Table 3.2). The concentration of the fortification levels range from 5 ppm (Norway) to 70 ppm (Sweden and Turkey). Albania's level is even higher (75 ppm).

Use of iodized salt is often permitted but not required in European countries. Table 3.2 shows that seven countries do not have legislation on iodine requirements. Information on the present state of legislation is available for 29 countries (Table 3.2). Use of iodized salt is voluntary in 16 countries and in 13 countries it is mandatory. In most countries, salt iodization is far from being universal. Iodized salt is permitted for industry only in Germany, the Netherlands and Switzerland.

There is currently a bewildering variety of legislations and regulations from one country to the next. Only Germany and Portugal exclusively mandate potassium iodate. In Denmark iodized salt is mandatory for bread and baking industry, but not allowed for other types of industry. Unfortunately, mandated levels of iodine differ between countries and are seemingly unrelated to the severity of iodine deficiency in a particular population. Therefore, those most in need of iodine supplementation are not necessarily receiving it. This situation is exacerbated in situations where monitoring of iodine levels has not been updated. Additionally, the present differences in national legislation constitute a significant problem for salt producers, and add to the market price that consumers pay. Currently the European Commission is developing a directive on micronutrient additives to foods which could act as an entry point for harmonized legislation on salt iodization.

The two examples that follow show how iodized salt can be introduced to consumers even in the midst of confusing legislation. In Italy, the low levels of purchase of iodized salt (and thus, low percentage of households using iodized salt) has led the Italian Government to pursue an agreement with the salt industry and scientists that might be described as a "semi-compulsory" compromise. Non-iodized salt must be specified on the label so that consumers know whether salt they buy is iodized or not. If a purchase is made without this specification, the retailer will automatically provide iodized salt. Underlying the agreement is a stipulation that all retailers in Italy must have iodized salt on the shelves. In the Netherlands, the compulsory use of iodized salt (with potassium iodate) was cancelled upon the insistence of a few bakers who argued for choice. A new rule states that if bakeries do not use iodized salt in bread baking, the bread and bakery products must be labelled specifying that it was prepared without iodized salt. The result is that in practice, almost no bread is for sale in the Netherlands that has been baked without iodized salt.

Unfortunately, the examples above are isolated cases; many European countries have not made iodine legislation a priority. This stems, in part, from perceptions about iodine on the part of consumers. Thus, their attitudes about iodine need to be more fully investigated, so that advocacy efforts can target incorrect assumptions, and inform the public on the need for USI.

Table 3.2 **Summary of regulations on salt iodization in Europe**

Country	Legislation	Year initiated and iodine amount (ppm)	Most recent review ^a	Permitted substances (iodate and iodide)	Iodine amount (ppm)	State of legislation ^b
Albania	No	1997 ^c	-	Iodide	75	-
Andorra	-	-	-	-	-	-
Austria	Yes	1963 (10)	1999	Both	15-20	M
Belgium	Yes	1990	1992	Both	6-45	V
Bosnia and Herzegovina	Yes	1953	1998	Iodide	20-30	M
Bulgaria	Yes	1958	1997	Iodate	22-58	M
Croatia	Yes	1953	1999	Iodide	25	M
Cyprus	-	-	-	-	-	-
Czech Republic	Yes	1950	1999	Iodate	27-42	V
Denmark	Yes	1999	2001	Iodide	13	M
Estonia	-	-	-	Both	-	-
Finland	No	1963 (25) ^d	1998	Iodide	25	V
France	Yes	1952 (10-15)	1997	Iodide	10-15	V
Germany	Yes	1981 ^e	2001	Iodate	15-20	V
Greece	Yes	1963	2000	Iodide	40-60	V
Hungary	No	-	1999	Both	10-20	V
Iceland	-	-	-	-	-	-
Ireland	No	-	1992	Iodide	25	-
Italy	Yes	1972 (15)	1997	Both	30	V
Latvia	Yes	-	-	Both	-	V
Liechtenstein	-	-	-	-	-	-
Lithuania	Yes	2003	2004	Both	20-40	M
Luxembourg	No	-	2000	Both	10-25	-
Malta	-	-	-	-	-	-
Monaco	-	-	-	-	-	-
Norway	Yes	-	1992	Iodide	5	V
Netherlands	Yes	1968 (3-8)	1998-99	Both	30-40	V
Poland	Yes	1935 (5)	1999	Iodide	30 ± 10	M,P
Portugal	Yes	1969 (20)	1996	Iodate	25-35	V
Romania	Yes	1956 (15-25)	2002	Iodate	34 ± 8	M
San Marino	-	-	-	-	-	-
Serbia and Montenegro	Yes	1951	2000	Iodide	20	M
Slovakia	Yes	1966 (19)	1999	Iodide	25 ± 10	M
Slovenia	Yes	1953 (10)	1999	Iodide	25	M
Spain	Yes	1982 (60)	2000	Both	51-69	V
Sweden	Yes	1936 (10)	1999	Iodide	40-70	V
Switzerland	Yes	1922 (1.9-3.75)	2002	Both	20-30	V
The former Yugoslav Republic of Macedonia	Yes	1999	1999	Iodate	20-30	M
Turkey	Yes	1999	2002	Iodide/Iodate	40-70/20-40	M
United Kingdom	No	-	1992	Iodide	10-22	V

- No data.

^a Refers to the most recent data available during the period listed.

^b V, voluntary; M, mandatory; U, uncertain; P, partial.

^c Decree prohibiting import of non-iodized salt.

^d Recommendation.

^e Declaration.

Source: John Dunn (93), Delange (8), ICCIDD (94), the European Salt Producers' Association, personal communication, 2003.

3.4 Iodized salt

3.4.1 Access to iodized salt

In 1999, it was estimated that of the 130 countries in the world having had a past iodine deficiency problem of public health significance, 68% of households had access to iodized salt as compared to five to 10 per cent in 1990 (20,95). However, at that time, the European Region of WHO was identified as having the lowest (27%) coverage of salt iodization of all the WHO regions, most of which are considerably less fortunate in economic terms than the majority of European countries.

The problem of iodine deficiency in European countries has been greatly underestimated for several decades, and so salt iodization programmes have suffered. After initially successful efforts to combat endemic goitre with iodized salt in the 1940s and 1950s, complacency took hold, and the issue of iodine deficiency was disregarded due to the emergence of other health problems in Europe. Recently there has been an increase in salt iodization coverage, which is encouraging.

All European countries have endorsed the goal of iodine deficiency elimination. The primary mode to do this is salt iodization, with the aim of eventually achieving USI. If USI is adopted and properly implemented, iodine adequacy will inevitably follow. The way the salt iodization programme is implemented is a key factor in the ability of a country to achieve the goal of iodine deficiency elimination. When appropriate levels of iodine concentrations are found in national salt systems, but the penetration rates of iodized salt into households is low, or if the salt used in food industries is not iodized, then the positive impact on public health will be muted.

Specific information by country on the current iodized salt distribution and coverage of households consuming iodized salt is shown in Table 3.3. Information is available from 27 countries. Of the information on penetration rate of iodized salt into households, only nine of the 27 countries with available information have a rate equal or greater than 90%, eight have a rate between 50 and 89% and 10 have a rate below 50%.

With regard to industry, data are reported only for three countries. The market segment of iodized salt at the national level is only 43% in Germany, 70% in the Netherlands and 60% in Switzerland. Data, however, should be treated with caution since they are not being systematically collected on a regular basis.

Overall, it can be concluded that, in the past five or six years, European countries, especially those of eastern Europe, have made great progress in establishing the conditions needed for effective salt iodization programmes, including legal provisions, public campaigns for the promotion of iodized salt and monitoring. Nevertheless, more progress towards USI is needed in the majority of countries.

3.4.2 Recent trends and obstacles to effective iodization programmes

Several factors influencing salt iodization programmes have led to the shift in the iodine nutrition situation over the past decade.

3.4.2.1 Political and social changes

Political and social changes have interrupted both the salt iodization process itself, and quality control measures. The changes that led to a resurgence of iodine deficiency in some countries, particularly in central and eastern Europe, have been largely resolved, and programme declines reversed. In fact, some of the most dramatic recent progress has been in countries of eastern Europe.

3.4.2.2 Increased exchange of food trade between countries

The enlargement of the EU market along with increasing globalization, have led to greater movement of food across national barriers, some processed with iodized salt, some not. Countries with a long history of iodization (e.g. Switzerland) have re-adjusted their salt levels in line with modern realities of the EU and increasing globalization, to maintain their “iodine deficiency under control” status.

Table 3.3 **Penetration rate according to market segments^a**

Country	Penetration rate (%) for the market segments (iodized salt >15 ppm) households/food industry
Albania	56
Andorra	Unknown
Austria	95
Belgium	10
Bosnia and Herzegovina	77
Bulgaria	98
Croatia	90
Cyprus	Unknown
Czech Republic	> 90
Denmark	Unknown / >90 ^b
Estonia	12
Finland	90
France	55
Germany	84/43
Greece	18
Hungary	90
Iceland	Unknown
Ireland	3
Italy	34
Latvia	3
Liechtenstein	Unknown
Lithuania	6
Luxembourg	Unknown
Malta	Unknown
Monaco	Unknown
Norway	Unknown
Netherlands	60/70
Poland	> 90
Portugal	1
Romania	53
San Marino	Unknown
Serbia and Montenegro	73
Slovakia	Unknown
Slovenia	Unknown
Spain	16
Sweden	Unknown
Switzerland	94/60
The former Yugoslav Republic of Macedonia	100
Turkey	70
United Kingdom	2

- No data.

^a Data are the most recent national data available.

^b Based on salt and bread samples from shops all over the country, 2001.

Source: John Dunn (93), UNICEF (70), ICCIDD (94), ICCIDD personal communication, 2004.

3.4.2.3 Changes in dietary sources of salt

An increasing amount of the proportion of total salt consumed comes from sources other than table salt. Table 3.4 illustrates the proportion of salt consumed from different sources with changes over time. Trends in three countries show a reduction in the consumption of table salt. A small amount of dietary sodium is consumed as table salt, for example, in the United Kingdom. This accounts for only 15% of all salt consumed – and a third of this is added at cooking (96). As well, there is a trend towards a far greater proportion (at least 60%, and often more) of the consumed salt being “hidden” in processed foods. Similar situations can be seen for Finland and France and the same trend is presumed to be occurring in virtually all European countries.

In countries of western and central Europe about 80% of salt is consumed in processed foods such as in bread, sausages, canned and other ready-to-eat foods, as so-called “hidden salt”. Consequently, if this hidden salt is not iodized, it is extremely difficult for a population to achieve

Table 3.4 **Proportion (%) of sodium intake from various dietary sources in Finland, France and the United Kingdom, with changes over time**

	Finland		France		United Kingdom	
	1980 ^a	1998 ^b	1950 ^c	2000 ^d	1985 ^e	1999 ^f
Average intake (g/day)	12.6	10.0	10.0	8.0	10.7	9.2
Discretionary salt						
• Table salt	38.0	12.0	55.0	15.0	15.0	9.0
• Cooking salt	-	-	-	-	17.0	6.0
Food salt						
• Natural food	12.0	10.0	14.0	12.0	12.0	14.3
• Processed food from industry	50.0	78.0	25.0	65.0	51.0	62.4
Other						
• Na salts added as food additives	-	-	6.0	8.0	-	-
• Salt in water	-	-	-	-	-	0.6

- No data.

Sources: Adapted from Information compiled by the European Salt Producers' Association (97) from various sources:

^a Pietinen (98).

^b H. Karppanen, Successful salt reduction in Finland, personal communication 2001.

^c B. Moinier, Evolution des ingesta sodés, personal communication 2002.

^d B. Moinier, personal communication, 2003

^e James et al. (99).

^f Edwards & Marsh (96).

adequate iodine intakes. The trend in some segments of the population to consume “cottage salts” and “plain” sea salt can also contribute to reducing the consumption of iodized salt.

3.4.2.4 Dietary sources of salt covered by regulations on iodization

Often only table salt or cooking salt is mandated to be iodized, and, as noted above, this represents an ever declining source of dietary salt (Table 3.4). Where iodization of this type of salt is still voluntary, this represents a real constraint to achieving the elimination of iodine deficiency.

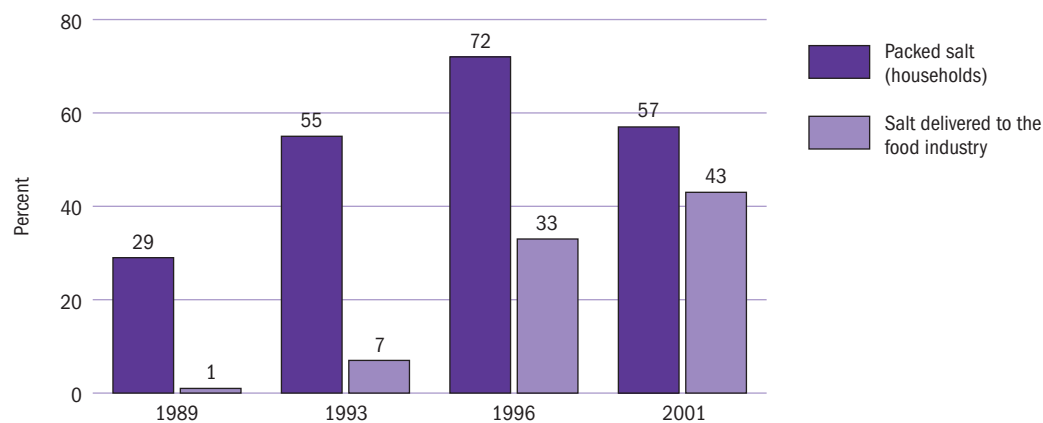
A few European countries require iodized salt in food processing but most do not. So far, only Denmark, Germany, the Netherlands and Switzerland have considered their national recent intake trends (in relation to iodine intakes) in how foods are being prepared and cooked at home, the increase in take-away foods and the supply and sources of processed food products in general, and amended their regulations accordingly (97).

Industry and national regulatory authorities have expressed some reservations about using iodized salt in some food processing, for example in meat products (curing and preservation with nitrited salt) and cheese. Nevertheless, when permitted, (e.g. in Germany since 1991 in meat products and in cheese since 1994) this has led to dramatic changes in the iodine penetration rate (Figure 3.3) (100). It also reflects a public health food processing policy more in line with population level prevention of iodine deficiency, and should become the norm in all countries.

3.4.2.5 Decreased levels of salt intake

Partly because of concern about hypertension, salt consumption has gradually declined, although it remains around 8–10 g/day at this time (Table 3.5). The national recommendations on salt intake range from 5–8 g/person per day (101). Several countries only recommend a limited salt intake but do not quantify this recommendation. If, as noted above (Table 3.4) packaged table salt is contributing only about 10 to 15% of the dietary salt intake, and the average consumption is only 8 g/day per person, then iodine intake for many people, in many European countries, depends on 1 g salt/day per person (100). Current recommendations on intake being considered by WHO and other bodies are even lower (<5 g/day), and if adopted for European populations, would likely require some modification of iodine levels with which salt is being iodized in different countries.

Figure 3.3 Trends of iodized salt usage by households and the food industry following legislation to allow fortified salt in various processed foods, Germany



Source: European Salt Producers' Association, personal communication, 2003 (100).

Table 3.5 Total amount of dietary salt consumed (g/day) in selected European countries at the end of the 1980s

Country	Intersalt	James et al.
Belgium	8.3–8.7	8.4
Denmark	8.2	7.4
Finland	9.0–10.0	7.0–11.1
France	–	7.9–8.4
Germany	7.2–8.9	9.7–10.1
Greece	–	8.2–10.3
Italy	9.8–10.9	9.0–11.0
Portugal	10.7	9.0–11.5
Spain	10.2–10.8	–
Netherlands	8.2–8.8	8.5
United Kingdom	8.3–8.8	–

– No data.

Source: Adapted from Intersalt (102), James et al. (99).

3.4.2.6 Other factors

The following factors also affect iodization programmes.

- The fact that iodine legislation is not mandatory throughout Europe could adversely affect consumption levels of iodine; some consumers may choose non-iodized salt for instance. Consequently, both consumers and policy-makers need to be fully informed about iodine deficiency, its consequences and its prevention and control.
- Many of the remaining problems with regard to iodine deficiency in European countries are due to inadequate controls on imported salt and a lack of resources for monitoring salt iodization effectively. The most important factor, however, is the lack of a clear commitment from governments, and ultimately, an insufficiently strong consumer demand for iodized salt.
- Instituting a standard for the level of iodine in salt across Europe would contribute to the removal of technical barriers to trade, and salt would then be distributed more freely among the countries of Europe. This lack of a standard is still a major problem in Europe: it unnecessarily restricts export/import and trade.
- The cost of iodized salt could potentially affect iodization programmes. Even when iodized salt is universally available, its price may be significantly higher than the price of the non-iodized form, which, combined with poor public awareness of the importance of iodine, could lead to low consumption (21). In most countries in Europe, the price of iodized salt is similar to that of plain salt (the European Salt Producers' Association, personal communication, 2003). [In chapter 2, the cost–benefit is clearly favourable. Wouldn't the price of iodized salt be subsidized somehow by governments, so as to keep it roughly equal with that of non-iodized salt?]

3.4.3 Quality assurance

The adequate production of iodized salt depends both on manufacturers capable of delivering food grade salt properly fortified with iodine, and on adequate legal provisions (legislation and more often, regulations) which stipulate quality standards for iodized salt (97). The salt manufacturers in Europe have agreed to do this, and to take responsibility for the quality assurance procedures that confirm that iodine levels are meeting local requirements during the process (batch) and before distribution (ex-factory). This is an important step in terms of sustainability and demonstrates the importance of a wider partnership. With the movement of iodized, but more importantly non-iodized, foods across national borders in Europe, widespread sustainability will only be achieved by increased harmonization of regulations, and comparability of quality assurance and control.

3.4.4 Process indicators

Most countries do not have strong units within the ministry of health (or elsewhere) with the responsibility of ensuring adequate iodine nutrition. Even if that were not the case, It has been noted by many observers that efforts to educate the government and citizens have been limited, given the many public health responsibilities of national governments.

Information on the progress towards iodine deficiency elimination can be seen in Annex G, which includes indicators necessary for successful USI programmes. Data are scarce for several of the process indicators designated for use in evaluating iodine deficiency control programmes, especially those concerning the presence of a national body, executive officer, adequate laboratories, quality assurance/control, cooperation with the salt industry and the presence of a national monitoring database. However, there appears to be relatively little correlation with the adequacy of iodine nutrition status of a country and these process indicators. From Annex G it can be seen that, of 21 countries with adequate or more than adequate iodine intake, about two thirds – 14 countries – have a national commitment (as expressed by having a national programme), and 17 countries have legislation or regulations in place. Thirteen countries regularly collect data on salt iodization and 11 countries regularly collect data on UI. For the countries with documented iodine deficiency, the number of countries with process indicators in place are less than half. These figures must be treated with considerable caution as it is likely that much of the missing information is, in fact, available at a national level. As each country evaluates its success towards the elimination of iodine deficiency, this information should emerge.

Encouragingly, there are several recent success stories regarding the elimination of iodine deficiency in Europe. In Germany, a multisectoral working group – *Jodmangel Arbeitsgruppe (Jodsiegel)* – has been active since 1997. This coalition brings together various stakeholders – government ministries, scientific experts, NGOs, the salt producers association, the pharmaceutical industry association and international agencies. German insurance companies were especially interested because the improvements of iodine nutrition would help to contain rising costs associated with diagnosis and treatment of thyroid disorders linked to iodine deficiency in the German population. This partnership was able to promote changes in regulations in Germany, after which households, the food industry and public catering were permitted to use iodized salt. They did this by sponsoring different activities designed to increase knowledge and awareness about iodine deficiency in Germany's population, thus stimulating the use of iodized salt, and motivating experts and industry to support salt iodization.

Denmark has also achieved success over the past few years by instituting USI. All table salt and salt for bread and bakery products is iodized, but at a relatively low level (13 ppm). Nevertheless, it is assumed to reach all households. However the use of iodized salt by the food industry is limited, which makes iodine nutrition adequacy at the national level somewhat problematic. As noted earlier, iodized salt used in bread making is also an important method of iodine supplementation.

Since 1942, in the Netherlands, the iodine in salt used by bakers for bread has been the main source of iodine. Recently national levels of iodine consumption were found to be still inadequate, and so the potassium iodide content has been increased from 55 to 65 mg/kg, as has that of table salt (from 23 to 29 mg/kg) (74). A recent study has reported normal values for the prevalence of goitre and UI in school-age children, including in a formerly iodine-deficient area (103).

3.4.5 Other iodine deficiency control measures

In countries such as Romania, where some endemic areas are not fully reached by iodized salt, alternative options need to be considered. Reinforcing the legislation or even making it compulsory is an option only applicable for areas with mild iodine deficiency. As areas in Romania were considered endemic, iodized oil was introduced and has been successfully used as a complement to iodized salt in schoolchildren to prevent iodine deficiency. The prevalence of goitre was 29% before the administration of the oil and was reduced to 9% one year later (72).

Finland has an innovative approach of adding iodine to animal fodder and fertilizer so that it enters the food chain (Pietinen, personal communication, 2003). Iodophores used in milking (sterilization of teats and milking machines) has been an adventitious source of iodine in the United Kingdom and many other western European countries for a long time (77), and as already noted, a change in dairy practice would reverse the situation and increase the possible likelihood of iodine deficiency. Consequently, it is of interest that iodophores are no longer permitted for this use in some countries in Europe.

Finally, iodized water has been used successfully in Italy (Sicily), but on a limited scale (104).

3.5 Economic consequences

Cost-effectiveness is a measure of the cost of the intervention and the amount of money saved because of the intervention. Both cost-effectiveness and benefit-cost ratio are defined in section 2.2.1. It is notoriously difficult to capture the total costs or the total benefits of health interventions. This might well be even truer of iodine deficiency, where the resulting effects can be very subtle and represent a continuum of clinical and subclinical sequelae. There appears to be little work on this issue in Europe.

What information there is comes largely from Germany where Pfannenstiel (105) reported that in 1981 the prevalence of goitre was 15%. Approximately 1.5 million patients were investigated each year for goitre and related disorders. The annual expenses incurred in the diagnosis and treatment was estimated to be about DM 770 million per year for thyroid disorders due to iodine deficiency. The author concluded that a reduction of DM 500–700 million per year should be possible by the introduction of a programme of USI. However, the cost of such a programme was not evaluated. More recent calculations have given health costs of approximately 2.1 billion DM (approximately 1 billion USD) (52). In their cost estimation of thyroid disorders in Germany, Kahaly and Dietlein (52) concluded that better prevention of iodine deficiency and its long-term consequences should effectively reduce direct as well as indirect costs and overall economic impact of endemic goitre as the most important thyroid disease in Germany. They note, however, that while sustainable elimination of iodine deficiency is technically possible, “it needs further commitment and support at all levels” (52).

In Switzerland, the figure of US\$ 0.07 per year per person is the quoted cost of the programme but it is unclear how this figure was derived (11). The total cost of the benefits accruing from iodine deficiency interventions in terms of health, economic productivity and agricultural productivity has not apparently been done in Switzerland, or indeed other European countries. In terms of useful advocacy, and the evidence-base for interventions, there is an urgent need for this to be attempted in some countries. Although the economic costs of not eliminating iodine deficiency are less well quantified in the context of an enlarged EU, it has been consistently shown to be a highly cost-effective national intervention where this has been addressed. It is also an intervention that has, in fact, been in place in some European countries for over 70 years and costs are presumably amortized over time.

The World Bank has made an estimate of the likely saving to countries of up to 5% GDP by investing only 0.3% GDP in micronutrient programmes (106). However, it is unclear that this would necessarily pertain to many countries in Europe. Likewise, the impacts and effects of iodine deficiency interventions can be wide-ranging, and often not very apparent. Consequently not all of the impacts can be captured and thus considered in the calculations of effectiveness, benefits and cost.

In most European countries the challenge is to identify benefits in often relatively healthy populations. One of the challenges is due to the fact that there are few florid cases of iodine deficiency.

Consequently, the health benefits are generally limited. This may be less so in some countries in eastern Europe. Another factor is the relatively resource intensive and greater expense of high technology approaches to screening, diagnosis and medical interventions (and health care costs in general), in most European countries. These factors considerably alter the cost-effectiveness of approaches to prevent and control iodine deficiency.

The picture is further complicated by time frames and changing trends. Many of the countries in central Europe that had previously adequate iodine status in the 1960s experienced precipitous declines in the 1990s. This was a direct result of changing public health systems, including a lack of resources being invested in prevention programmes such as those directed to iodine deficiency, and a breakdown of [quality assurance/quality control?] systems. This has been well documented (24). In many such countries this is now being reversed. Nevertheless, affluent European countries, often with a long history of salt iodization and with otherwise excellent public health systems, have also seen a decline in iodine levels in their populations – presumably a result of complacency.

4. The major issue for EUROPE: sustained prevention and control

This report clearly establishes that iodine deficiency remains a significant health and socioeconomic problem in Europe. Of the 40 countries reviewed, 11, including the more affluent countries such as Belgium, France and Italy, are still affected by well documented iodine deficiency, albeit only mildly. However, recent information is lacking for eight countries. In the countries where only subnational data are available, urgent action is needed to assess the status of iodine nutrition nationally.

Iodine deficiency has well established consequences in terms of public health and cost, especially in neuro-intellectual damage to infants born to even mildly iodine-deficient mothers, and to productivity, and hence, economic well-being. Health outcomes are basically the result of dysfunction of the thyroid, particularly among pregnant and lactating women and young infants. Additionally, iodine deficiency has adverse consequences for human fertility, is responsible for the development of often unrecognized hyperthyroidism in the elderly, and also increases the negative consequences, such as cancer, of irradiation of the thyroid gland in case of nuclear accident. The consequences for the agricultural sector due to the impact on farm animal productivity is also well documented.

The cost of the diagnosis and treatment of iodine deficiency in Europe has been estimated in only a limited number of countries. It has been attempted most precisely in Germany where costs of 1 billion Euros per year are needed by the curative medical sector to address the results of iodine deficiency and its disorders, whereas their prevention by programmes of salt iodization has been estimated to cost some 100 times less. The costs to European countries in terms of national productivity are likely to be significant but are as yet unknown.

Progress in ensuring adequate iodine nutrition has, nevertheless, greatly accelerated in recent years, and the review underscores the major improvement seen in the iodine status in European countries, especially within the past several years. Compared to the European situation of 10 years ago (2), when only five countries had achieved iodine sufficiency – Austria, Finland, Norway, Sweden and Switzerland – the present figure of 21 countries with well-documented iodine sufficiency represents real progress.

Mild iodine deficiency persists in 11 of the 40 countries reviewed, most of them in eastern Europe. In some of them (e.g. Turkey) moderate iodine deficiency persisted until recently. In several countries, there is still even the possibility of local foci of endemic cretinism in rare, severely affected areas. In others, iodine deficiency has not been severe for some time now and consequently there have been no obvious visible manifestations of the disease, such as a high prevalence of visible goitres or an alarming presence of endemic cretinism. It is probably the reason why, although the problem is particularly well documented, national measures aimed at its correction often remain inadequate in these countries.

This review establishes that iodization of salt has been the most appropriate response for the improvement of the situation. Major progress has been made in salt iodization, particularly in the private sector, which is taking increased responsibility in this area. Yet, a persistent constraint is the lack of commitment, including appropriate legislation and monitoring, to USI.

4.1 Main policy issues regarding the elimination of iodine deficiency

4.1.1 Assessment of iodine status

Eight countries have inadequate recent data on iodine status. Based on the experience of countries with long-standing iodization programmes, some of these eight are likely to be facing a public health problem with respect to iodine deficiency, especially in localized areas. Assessment is the preliminary step before any action can be taken. Where data show iodine deficiency, they can then be used to convince the authorities to revise an iodine deficiency control programme. Effective advocacy or the design of an appropriate intervention to complement USI cannot be done in countries with poor data. For example, dispersed regional data collected from generally iodine sufficient areas, and the absence of national programmes, are concerns for such countries.

Where no data on iodine status exist at all – the situation in eight of the 40 countries considered in this review – ongoing detailed evaluation is needed.

4.1.2 Implementation of USI

There is universal agreement that USI remains the intervention of choice for improving the status of iodine nutrition in Europe, as elsewhere in the world. The next step consequently is the implementation of USI programmes wherever iodine deficiency is documented, or where countries are only implementing a limited programme (e.g. only table salt is iodized). The second is to strengthen current programmes to ensure quality and sustainability. Most European countries have iodized salt available. All countries with a documented iodine deficiency problem have legislation on salt iodization. However, in many instances legislation is either not universal or not enforced.

Several factors have constrained efforts to reduce iodine deficiency over the past decade.

- In contrast to most other parts of the world, iodized salt is only voluntary in most European countries, which certainly diminishes the likely effectiveness of the programmes, although there are exceptions (e.g. Switzerland). For voluntary programmes to be effective, the public needs to be aware of the effects of iodine deficiency. Public awareness is missing in many countries where voluntary programmes exist.
- Political and social changes have interrupted national programmes, both the salt iodization process itself and the necessary quality control measures.
- The formation of common markets, along with increasing globalization, have led to greater movement of food across national barriers, some processed with iodized salt, some not.
- The percentages of salt coming from table salt and from processed foods have dramatically changed during the past 20 years. An increasingly smaller amount of salt is consumed as table salt and relatively more in processed foods. Overall, partly through the concern about hypertension, total salt consumption has gradually declined. Over the past 50 years consumption of salt dropped by an average of 2 g/day in a number of countries, bringing the average daily consumption to about 8–10 g/day. Consumption of smaller amounts of iodized salt reduces iodine consumption. In Europe, as elsewhere, the hidden salt in processed foods is becoming a major source of salt in the diet, which means that salt used in these foods should also be iodized. More information on the source and distribution of salt is required for most of the European countries.
- National level legislation on iodized salt show great diversity in terms of the required iodine concentration in salt, and even the compounds to be used, which creates major difficulties in the export and free movement of iodized salt from one European country to another. One of the major challenges for the future is the harmonization of the existing regulations and recommendations, and if necessary, the reinforcement and implementation of legislation on salt iodization. This includes standardization of techniques and consistent regulations on salt iodization in terms of the compounds that should be used, along with agreed ranges of iodization and harmonized regulations on trade. Some progress has been made: import duties on salt no longer exist within the European Economic Area (EU and EFTA) and value-added tax is now levied at the lowest rate for table salt.
- Advocacy efforts to promote two health objectives – decreasing salt intake as part of programmes for the prevention of cardiovascular diseases, and consuming iodized salt – have led

to the confusing message that consuming more iodized salt necessarily results in consuming more salt. This is untrue. Both objectives can be combined by adequately iodizing salt, that is, by limiting consumption to iodized salt. To do this, good data on levels of iodization and food consumption are needed.

4.1.3 Focus on infants and pregnant women

The groups most at-risk of the effects of iodine deficiency are pregnant women and young infants, because of their increased hypersensitivity to the effects of iodine deficiency during periods of rapid and new growth, and the risk of brain damage in the fetus and infant. Appropriately, the majority of recent investigations of thyroid function in iodine deficient countries have focused on pregnant women and neonates. It is particularly worrying to therefore note that so many neonates in Europe today still exhibit unquestionable biochemical signs of a lack of thyroid hormones in their developing brain (e.g. Belgium) (107). This clearly should alert health authorities to the continuing risk of brain damage and some likely degree of irreversible neuro-intellectual damage, and the resulting impact on national economies. Increased attention needs to be paid to these vulnerable groups. It may be that complementary alternative strategies to correct deficiency are needed, such as iodized oil (29). Additional efforts on monitoring of iodine status and thyroid function during pregnancy, in neonates and young infants, should be implemented in a series of countries, especially the ones for which these data are presently lacking, such as Bosnia and Herzegovina, and Portugal.

4.1.4 Implementing alternative strategies to correct iodine deficiency

It has been shown in many countries that USI will eliminate iodine deficiency if there is an effective programme in place and adequate time is given. However, in the European context, many countries are not implementing USI, or are doing so inadequately. In these cases, while work to strengthen the national programmes and private salt industry involvement continues, some susceptible groups are still receiving inadequate iodine. From this review, and cited evidence, this applies specifically to pregnant and lactating women and young infants, who are the groups most affected by the consequences of iodine deficiency. Thus, where iodine deficiency is identified in pregnant women and infants, alternative options to salt iodization should be considered, such as supplementation with iodized oil, physiological quantities of iodine through tablets, drops or multiple micronutrient supplements that include iodine (29). Based on results from Belgium (108) and France (109), such programmes need to be considered in iodine deficient countries as well. The temporary use of iodized oil in conditions of severe or moderate deficiency might also need to be considered in some remote areas not currently reached by iodized salt, such as Albania or Romania (72). Moreover the adoption of a policy to reduce salt consumption by a growing number of countries suggests that in addition to salt iodization, complementary strategies to increase iodine intake are needed (78).

4.1.5 Monitoring and evaluation

Monitoring is a key issue in the elimination of iodine deficiency. The defining criteria to be used are well-established (27), both for the process of salt iodization and for the monitoring of its impact on iodine nutrition and thyroid function. In monitoring iodine status and thyroid function, median UI remains the key indicator. As the ultimate desired outcome is normal thyroid function, countries should also consider neonatal screening of TSH (as an indicator of brain damage), which is already done in several countries (e.g. Belgium, Bulgaria, the Czech Republic, Germany, Poland and Spain).

Both monitoring and evaluation have improved recently but those improvements need to be sustained, especially at the national level. Yet gaps remain, and include inadequate data collection, and in many cases, actual lack of implementation of programmes. In several countries, the laboratory capacity to effectively monitor programmes is lacking, in particular with regard to iodine status. Cooperation between countries with well-equipped laboratories and those without is required. The IRLI Network, with reference laboratories in Belgium and Bulgaria, is an important step in this direction (see Annex A). A common challenge to all iodine deficient countries is the organization of quality control and assurance and effective monitoring of iodine supplementation

to ensure there is adequate iodization from the producer through to the consumer. In order to properly monitor iodized salt consumption, further information on household and food industry penetration with iodized salt is needed for all countries. Monitoring consumption not only shows iodine sufficiency, but can also reveal excess intake.

For example, possible side-effects of iodine supplementation, that is, potential iodine excess with possibly harmful health consequences (e.g. iodine-induced hyperthyroidism) has to be considered; uncontrolled fortification of just any food with iodine – often referred to as “wild” iodine fortification – could lead to unrecognized iodine excess. Thus, programmes monitoring hyperthyroidism are already being implemented in some countries (e.g. Croatia, Denmark and Poland). To date, iodine excess was not found in any country under review.

4.1.6 Legislation

In order to ensure USI sustainability, many iodine deficient countries need not only legislation on salt iodization, but also strengthened efforts implementing and monitoring that legislation, both of which are currently insufficient.

Attitudes of consumers also impact legislation efforts. In the Nordic countries and in France, for example, public opinion is decidedly anti-iodized salt, as compared with some other European countries. Moreover, a number of consumers prefer cottage salt products (containing no iodine), which are sold as being closer to nature irrespective of the product’s quality. There is also currently considerable public scepticism about government positions on food safety. As noted, if non-iodized salt is to be available, then considerable public advocacy is required to inform the public of the necessity of including iodine in the diet. Unfortunately, this is currently quite weak in most countries.

4.1.7 Economic impact

One of the clear outcomes of this review is highlighting the dearth of information available on the impact of iodine deficiency on the economic productivity and national neuro-intellectual capacities of the European populations concerned. What information does exist suggests that the impact is relatively significant. Additionally, data that do exist show that iodine deficiency control and prevention programmes are highly cost-effective.

4.1.8 Advocacy and partnership

A common challenge to all European countries is the need for communication and social mobilization: advocacy and training on iodine deficiency have to be maintained, reinforced and even initiated in some countries (110). This challenge is particularly relevant for countries in which iodine deficiency is particularly well established but where, in spite of sustained efforts, it has not been possible to institute a national policy to reduce it. The objective of educating all partners involved in iodine deficiency control to facilitate their collaboration remains valid, but needs considerably more progress if it is to be achieved. The main partners to be targeted include the public, national health authorities and the salt and food industry. The policy decision-makers need to be mobilized to increase their awareness of the public health importance of controlling iodine deficiency and its implications for health, education and economic development. The essential role of industry, both the salt and the food industries, is now established in the fight to control, and ultimately prevent, iodine deficiency. The public and their representatives in the consumer associations need to become enthusiastic advocates for consuming adequate levels of iodine to prevent iodine deficiency. The methodology of advocacy and social mobilization has been recently reviewed (110).

4.2 Challenges for the future

The Member States of the enlarged European Union and the EFTA countries face many challenges in the sustainable elimination of iodine deficiency. In the future the following issues will need to be addressed.

- Programmes for iodine deficiency prevention and control in countries with mild iodine deficiency or limited national commitment must be accelerated. This includes: i) ensuring

governments build on recent progress and continue efforts to eradicate iodine deficiency in their populations; and ii) gathering current data with which to accurately assess iodine status.

- All iodine supplementation programmes must become sustainable. Thus: i) ensuring governments institutionalize measures in place to ensure sustainability of adequate iodine nutrition for their populations; ii) Institutionalizing support so that national committees and similar bodies increase advocacy to governments; iii) increasing public education so that demand for adequately iodized salt is consumer driven; and iv) reinforcing iodine deficiency networks, especially the Network for the Sustained Elimination of Iodine Deficiency, to ensure effective cooperation between public and private partners.
- Commit to USI and strengthen or initiate measures to be taken to achieve USI.
- Increase focus on at-risk pregnant and lactating women, infants and young children (advocate iodine supplementation of pregnant and lactating women when required).
- Strengthen monitoring and surveillance systems. This includes: i) obtaining better information on the epidemiology of salt consumption, the techniques of salt production and salt iodization, and iodized salt in the diet; ii) maintaining the effort to strengthen cooperation with the salt industry to ensure salt quality control and assurance; iii) improving household salt consumption data by improving collection of information on household penetration rates, on the source of iodized salt used, household use, and intra-household distribution; iv) improving availability, and use, of information on commercial use of iodized salt in processed foods, and carrying out penetration rate studies for coverage of iodized salt in industry processing, retail outlets and household use; v) reinforcing laboratory performance and capabilities to get better monitoring of iodine status; vi) expanding the role of the iodine deficiency laboratory network, including publishing a list of national and regional laboratories with expertise in UI measurement and salt quality control; vii) substantially increasing the resources and efforts for monitoring the progress towards iodine deficiency elimination – governments should enlist national and international partners in evaluating national iodine deficiency programmes, using recommended indicators, including the regular monitoring of UI and USI; and viii) encouraging better information at the national level on thyroid function (e.g. screening of TSH levels in neonates) as resources permit.
- Enact legislation. This includes: i) enacting appropriate legislation and regulations in all countries; ii) continuing efforts to achieve harmonized salt iodization regulations, including levels of iodization, choice of iodine compound, and appropriate nutrition labelling; and iii) implementing cost-effectiveness and benefit–cost ratio studies.

4.3 Conclusions

Specific conclusions and new insights coming from this review follow below.

- The efforts, and results, in the fight to prevent and control iodine deficiency have markedly progressed in Europe during the past 10 years so that from only five European countries which were considered as iodine sufficient in 1994, there are, in 2004, a total of 21 countries which have reached iodine sufficiency.
- Iodization of salt has been the major intervention responsible for the significant improvement of the situation. However, in contrast to many other parts of the world, especially in less industrialized countries, it is clear that table salt now represents only a relatively small fraction of the salt intake in European populations. Therefore, the evaluation of the impact of programmes of iodized salt by estimating the access of iodized salt at the household level (as usually reported) provides only part of the information. In 1999, the access of iodized salt at the household level for Europe was the lowest regional average figure in the world at 27%. Because of the declining consumption of table salt, this probably did not reflect properly the access of households to salt, and consequently, potentially to iodized salt. Nevertheless, it also is clear that greater attention is urgently needed to iodize all sources of salt for human and animal consumption.
- In the meantime, the successful trends seen over the past five to six years need to be sustained, as remaining iodine-deficient sub-populations are targeted and the necessary public health goal of USI for all countries is achieved. This will most likely require increased efforts, as these areas

have not responded to initial progress. There do not appear to be trend data on increasing, or decreasing, levels of greater public and policy-maker awareness.

- Effective monitoring is critical to the sustained success of programmes, and requires both progress indicators and confirmatory urinary, and even clinical, indicators of successful implementation. Proper monitoring, as seen from several countries' experiences, is essential for sustained elimination of iodine deficiency.
- It is clear that there is very limited information available on the impact of iodine deficiency on thyroid function, and even less so on the impact on the economic productivity and national neuro-intellectual capacities of the populations concerned.
- In the course of the review, it was confirmed that pregnant women and neonates are the fraction of the population particularly susceptible to the effects of iodine deficiency. The single final objective of programmes of correction of iodine deficiency is to normalize thyroid function in all age groups but especially in pregnant women and young infants because of their hypersensitivity to the effects of iodine deficiency and the risk of brain damage in the progeny. Consequently, these groups must be properly assessed for possible further preventive and remedial action.

In conclusion, this review shows that iodine deficiency remains a significant public health problem in Europe. It provides an update on the current status of iodine deficiency and existing iodine programmes within the countries of Europe. The quality and availability of information on iodine nutrition has substantially improved over the past two decades. Thus, national data were used extensively as the basis of this review's conclusions. Where national data were not available, sub-national data were used.

This review must be a living document, that is, it will increase in relevance as more recent data become available. It is hoped that new data will reflect improvements in the national picture of iodine deficiency among countries in Europe; or, if not, they will show more clearly the gaps in coverage, so that efforts can focus on where they are most needed. It is further hoped that this review will contribute to the necessary success of the advocacy effort to mobilize countries towards the internationally agreed upon goal of the elimination of iodine deficiency.

ANNEX A

The International Resource Laboratories Network for Iodine (IRLI): European Laboratories members of the network

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ANNEX B

General characteristics of countries included in the report

Country	Total population (000) ^a	Total population aged 6-12 years (000) ^a	Annual growth rate 1991-2001b (%)	Life expectancy at birth (years) ^b	Mortality rate (per 1000 live births) ^b		Salt production
					Aged <1 year	Aged <5 years	
Albania ^f	3141	434	-0.5	69.5	25	32	-
Andorra ^f	69	6	5.0	79.5	4	5	-
Austria ^c	8111	650	0.4	79.0	4	5	Yes
Belgium ^c	10 296	846	0.3	78.0	4	5	Yes
Bosnia and Herzegovina ^f	4126	357	-0.2	72.8	14	17	-
Bulgaria ^e	7965	599	-1.0	71.5	14	17	Yes
Croatia ^f	4439	351	0.3	72.9	7	8	-
Cyprus ^d	796	87	1.3	76.9	6	7	Yes
Czech Republic ^d	10 246	810	0	75.4	4	5	Yes
Denmark ^c	5351	473	0.3	77.2	5	5	Yes
Estonia ^d	1338	112	-1.3	71.2	9	11	-
Finland ^c	5197	448	0.3	77.9	3	4	-
France ^c	59 850	5128	0.4	79.3	4	5	Yes
Germany ^c	82 414	6022	0.3	78.2	4	5	Yes
Greece ^c	10 970	759	0.4	78.1	6	7	Yes
Hungary ^d	9923	817	-0.4	71.7	8	10	-
Iceland ^f	287	32	0.9	79.8	3	4	-
Ireland ^c	3911	375	0.9	76.5	5	6	-
Italy ^c	57 482	3866	0.1	79.3	5	5	Yes
Latvia ^d	2329	200	-0.1	70.7	11	13	-
Liechtenstein ^f	33	3	n.a.	-	-	-	-
Lithuania ^d	3465	334	-0.1	72.9	8	10	-
Luxembourg ^c	447	40	1.4	78.5	3	4	Yes
Malta ^d	393	37	0.8	78.1	6	7	Yes
Monaco ^f	34	3	-2.9	80.3	4	5	-
Netherlands ^c	16 067	1395	0.6	78.3	5	6	Yes
Norway ^f	4514	427	0.5	78.8	3	4	Yes
Poland ^d	38 622	3437	0.1	74.0	8	9	Yes
Portugal ^c	10 049	774	0.1	76.5	5	7	Yes
Romania ^e	22 387	1836	-0.3	71.1	18	22	Yes
San Marino ^f	27	2	1.4	80.8	5	5	-
Serbia and Montenegro ^f	10 535	976	0.3	72.2	12	14	-
Slovakia ^d	5398	496	0.2	73.3	8	9	Yes
Slovenia ^d	1986	144	0.3	75.9	4	5	Yes
Spain ^c	40 977	2712	0.1	78.9	4	5	Yes
Sweden ^c	8867	807	0.2	80.0	3	4	Yes
Switzerland ^f	7171	576	0.4	80.2	5	5	Yes
The former Yugoslav Republic of Macedonia ^f	2046	213	0.6	71.8	13	14	-
Turkey ^e	70 318	10 119	1.7	69.0	36	43	Yes
United Kingdom ^c	59 068	5349	0.3	77.5	6	6	Yes

- No data.

^a Based on population estimates for the year 2002 (35).

^b Tabulation prepared for the *World Health Report 2002 (111)*.

^c European Union Member States.

^d New Member States 2004.

^e Applicant countries.

^f European Free Trade Association and other European countries

ANNEX C

The WHO Global Database on Iodine Deficiency

WHO's mandate with regard to iodine deficiency is to assess its global magnitude, monitor and evaluate the impact and effectiveness of WHO's prevention and control strategies, track progress towards the goal of iodine deficiency elimination, and identify emerging issues with regard to iodine deficiency. WHO manages the Global Database on Iodine Deficiency. The database compiles data on UI and TGP and presents it in a standardized and easily accessible format (<http://who.int/vmnis>).

Data sources are collected from the scientific literature and through a broad network of collaborators, including WHO regional and country offices, United Nations organizations, NGOs, ministries of health, other national institutions, and research and academic institutions.

MEDLINE and regional databases are systematically searched. Articles published in non-indexed medical and professional journals and reports from principal investigators are also systematically sought. Data are extracted from reports written in any language.

For inclusion in the database, a complete original survey report providing details of the sampling method used is necessary. Studies must have a population-based sample frame and must use standard UI and TGP measuring techniques (27).

Only TGP data measuring goitre by palpation are included. Until recently no international reference values for thyroid size measured by ultrasonography were available, and thus results from surveys using this technique have not yet been included (65).

When a potentially relevant survey is identified and the full report obtained, all data are checked for consistency as part of routine data quality control. When necessary, the authors are contacted for clarification or additional information. Final data are extracted, standardized and included in the database. The full archived documentation and correspondence are archived and available on request. The database contains data from 1960 to the present, and is continuously being updated as information from new surveys becomes available.

ANNEX D

Prevalence of iodine deficiency in school-age children and the general population based on urinary iodine data, by country^a

Country	Date of survey	Level of survey	Population group and age (years)	Survey data				Notes	References	Classification of iodine intake	Classification of iodine nutrition	Population affected	
				Sample size	Median UI (µg/l)	Proportion of population with UI <100 µg/l (%)	95% CI of proportion of population with UI <100 µg/l					Children aged 6-12 yrs (000) ^b	General population (000) ^b
Albania	2003	Local	SAC (5-14)	414	30	91	88.2-93.8	(112)	Insufficient	Moderate iodine deficiency	395	2858	
Andorra	-	-	-	-	-	-	-	-	-	-	0	0	
Austria	1994	Local	SAC (6-15)	589	111	50.3	46.3-4.3	(18)	Adequate	Optimal	327	4080	
Belgium	1998	National	SAC (-6-12)	2585	80	66.9	65.1-68.7	(113)	Insufficient	Mild iodine deficiency	566	6888	
Bosnia and Herzegovina	2005	National	SAC (8-10)	2309	157	22.2	20.5-23.9	(114)	Adequate	Optimal	79	916	
Bulgaria	2003	National	SAC (7-11)	809	198	6.9	5.2-8.6	(115)	Adequate	Optimal	41	550	
Croatia	2002	National	SAC (-6-12)	927	140	28.8	25.9-31.7	(116)	Adequate	Optimal	101	1278	
Cyprus	-	-	-	-	-	-	-	-	-	-	0	0	
Czech Republic	2002	National	All 0-98	1542	129.6	17.3	15.4-19.2	(117)	Adequate	Optimal	140	1773	
Denmark	1997-1998	Regional	Adults (18-65)	4616	61	74.2	72.9-75.5	(118)	Insufficient	Mild iodine deficiency	351	3970	

Estonia	1995	National	SAC (8-10)	1840	65	67	64.9-69.2	(119)	Insufficient	Mild iodine deficiency	75	896
Finland	1997	Local	Adults (30-42)	342	164	35.5	30.4-40.6	(120)	Adequate	Optimal	159	1845
							% < 100 µg/l calculated from median.					
France	1996	National	Adults (35-60)	12014	85	60.4	59.5-61.3	(121)	Insufficient	Mild iodine deficiency	3097	36149
							Medians from disaggregated data by sex pooled. % <100 µg/l calculated from median.					
Germany	1999	National	SAC (-6-12)	3065	148	27	25.4-28.6	(122)	Adequate	Optimal	1626	22 252
Greece	1996, 2001	Local	Adults (15-80), Adolescents (12-18)	1129	128.7	33.7	30.9-36.5	(123,124)	Adequate	Optimal	256	3697
							Median UI was calculated from % <100 µg/l for one survey. Medians from two local surveys pooled.					
Hungary	1994-1997	National	SAC (7-11)	2814	80	65.2	63.4-67.0	(125-128)	Insufficient	Mild iodine deficiency	533	6470
							% <100 µg/l from disaggregated data by county pooled.					
Iceland	1998 P	Local	Elderly (66-70)	89	150	37.7	27.6-47.8	(129)	Adequate	Optimal	12	108
							% <100 µg/l calculated from median.					
Ireland	1999	Local	Adults (22-61)	132	82	60.8	52.5-69.1	(130)	Insufficient	Mild iodine deficiency	228	2378
							% <100 µg/l calculated from median.					
Italy	1992-1994, 1993-1995, 1994P, 1997P, 1998P, 1999P	Regional, local	SAC (6-15)	11226	94	55.7	54.8-56.6	(131-139)	Insufficient	Mild iodine deficiency	2154	32018
							Medians from nine local and regional surveys pooled. % <100 µg/l calculated from median.					

Country	Survey data										Population affected		
	Date of survey	Level of survey	Population group and age (years)	Sample size	Median UI ($\mu\text{g/l}$)	Proportion of population with UI <100 $\mu\text{g/l}$ (%)	95% CI of proportion of population with UI <100 $\mu\text{g/l}$	Notes	References	Classification of iodine intake	Classification of iodine nutrition	Children 6-12 yrs (000) ^b	General population (000) ^b
Latvia	2000	National	SAC (8-10)	599	59	76.8	73.4-80.2		(140)	Insufficient	Mild iodine deficiency	154	1789
Liechtenstein	-	-	-	-	-	-	-	-	-	-	-	0	0
Lithuania	1995	National	SAC	2087	75	62	59.9-64.1	% <100 $\mu\text{g/l}$ calculated from median.	(141)	Insufficient	Mild iodine deficiency	207	2148
Luxembourg	2002	National	SAC (12-14)	498	148	38.3	34.0-42.6	% <100 $\mu\text{g/l}$ calculated from median.	(142)	Adequate	Optimal	15	171
Malta	-	-	-	-	-	-	-	-	-	-	-	0	0
Monaco	-	-	-	-	-	-	-	-	-	-	-	0	0
Netherlands	1995-1996	Local	SAC (6-18)	937	154	37.5	34.4-40.6	Thyromobile study. % <100 $\mu\text{g/l}$ calculated from median.	(103)	Adequate	Optimal	523	6025
Norway	1999	Local	Adults (23-64)	63	117	39.7	27.6-51.8		(143)	Adequate	Optimal	169	1792
Poland	1999-2001	Regional	SAC (7)	1499	103	47.2	44.7-49.7	Thyromobile study.	(144)	Adequate	Optimal	1622	18229
Portugal	-	-	-	-	-	-	-	-	-	-	-	0	0
Romania	2004-05	National	SAC (6-7)	2327	101	46.9	44.9-48.9	Median calculated from % <100 $\mu\text{g/l}$.	(145)	Adequate	Optimal	861	10500
San Marino	-	-	-	-	-	-	-	-	-	-	-	0	0
Serbia and Montenegro	1998-1999	Regional	SAC (7-15)	1515	158	20.8	18.8-22.8	Survey in Serbia and Vojvodina.	(146)	Adequate	Optimal	203	2191
Slovakia	2002	National	SAC (6-12)	1744	183	15	13.3-16.7		(147)	Adequate	Optimal	74	810

Slovenia	2002-2003	National	SAC (13)	676	148	22	18.9-25.1	1991-1994 national survey of 1740 SAC (13 years) reports median UI expressed in µg/g creatinine, median 82.9.	(148)	Adequate	Optimal	32	437
Spain	1995, 2000, 2000P, 2001P, 2002P	Provincial	SAC	3154	109	51	49.3-2.7	Medians from five regional and provincial surveys pooled. % <100 µg/l calculated from median. % <100 µg/l borderline, while median is 109 µg/l due to the equation used for the estimation (see methods section).	(44, 149-152)	Adequate	Optimal	1383	20 898
Sweden	2004P	Local	SAC (7-9)	61	194				(153)	Adequate	Optimal	0	0
Switzerland	2004	National	SAC (6-12)	386	141	24.0	19.7-28.3		(154)	Adequate	Optimal	138	1721
The former Yugoslav Republic of Macedonia	2005	National	SAC (7-12)	1200	228	8.7	7.1-10.3		(155)	More than Adequate	Risk of IHH in susceptible groups ^c	19	178
Turkey	2002	National	SAC (9-11)	11134	75	60.9	60.0-61.8		(156)	Insufficient	Mild iodine deficiency	6163	42 824
United Kingdom	-	-	-	-	-	-	-	1992-1993 survey with a nationally representative sample of 101 adults (38-93 years) reports median UI expressed in µg/g creatinine, median 102.	(157)			0	0

- No data.

P Published

^a Data refer to those most recently available for the period 1994-2004.

^b Based on population estimates for the year 2002 (35).

^c IHH – iodine-induced hyperthyroidism.

Source: WHO Global Database on Iodine Deficiency.

ANNEX E

Total goitre prevalence by country, level of survey and age group^a

Country	Date of survey	Level of survey	Age group (years)	Sample size	Survey data		Notes	References
					Palpation	Ultrasound		
Albania	2003	Local	SAC (5-14)	826	60.9	32.2 ^b	Purposive selection of endemic area. School children from Korçe and villages in the surrounding mountains.	(112)
Andorra	-							
Austria	1994	Local	SAC (6-15)	589		2.0-3.2 ^c		(18)
Belgium	1998	National	SAC (6-12)	2585		5.7 ^e		(18)
Bosnia and Herzegovina	1999	National	SAC (7-14)	9183	25.7		TGP from two surveys (Republica Srpska and Federation of Bosnia & Herzegovina, 23.5%, 27.1%) pooled.	(158,159)
Bulgaria	2003	National	SAC (7-11)	3939	13.4	4.3	TGR by ultrasonography from a subsample of 374 school-age children.	(115)
	2003	National	SAC (7-11)	374		2.1 ^e		(115)
Croatia	2002	National	SAC (6-12)	927		0 ^d		(116)
Cyprus	-							
Czech Republic	2001	National	SAC, adults (6-13, 14-18, 18-65)	NS			Thyroid volume 15.2 ml.	(8)
Denmark	1997-1998	Regional	Adults (18-65)	4649	12.1	18.6 ^e		(160)
Estonia	-							
Finland	-							

France	1996	National	Adults (35-60)	12 014	12.9	TGP from disaggregated data by sex (11.3[%?], 13.9%) Pooled.	(121)
Germany	1997	Regional	SAC (10-18)	255	0-6.0 ^c		(161-163)
	1997	Regional	SAC (7-17)	591	3.0 ^f		
	1997P	Local	SAC (3-15)	1080	4.0 ^g		
Greece	1996, 1999P, 2001	Local, Regional	SAC, Adults (9-80)	2342	10.2	TGP from three surveys (range 0-18%) pooled.	(123,124,164)
Hungary	1994-1997	National	SAC (7-11: male)	299 351 [correct sample size?]	11.6	TGP from disaggregated data by counties (range 3.6-23.0%) pooled.	(125-128)
Iceland	-						
Ireland	1989-1992	Local	Adults	311	8.2		(2,165)
Italy	1992-1994, 1993-1995, 1994P, 1998P, 1999P	Regional, Local	SAC	12 744	13.9	TGP from eight surveys (range 6.1-41.4%) pooled.	(132-140,166)
	-						
	-						
	-						
Latvia	-						
Liechtenstein	-						
Lithuania	-						
Luxembourg	-						
Malta	-						
Monaco	-						
Netherlands	1995-1996	Local	SAC (6-18)	937	1.8	TGP from two local sites in two different regions (palpation 0.8, 2.6%) pooled.	(103)
Norway	-				<5.0 ^h		
Poland	1999-2001	Regional	SAC (6-15)	1499	5.2 ^c		(167)
Portugal	-						
Romania	2001	National	SAC (6-16)		6.4-31.8		(144)

Country	Date of survey	Level of survey	Age group (years)	Sample size	Survey data		Notes	References
					Total goitre prevalence (%)			
					Palpation	Ultrasonography		
San Marino	-							
Serbia and Montenegro	1998	National (refugees)	SAC (9-18)	1421	1.3			(168)
Slovakia	1989-1995	Regional	SAC (6-15)	1923	4.4		TGP from disaggregated data by age and sex (range 2.0-7.7%) pooled.	(169)
Slovenia	2002-2003	National	SAC (13)	676	47	6.3 ⁱ		(148)
Spain	1995, 2000, 2002P	Provincial	SAC (6-16)	2745	10.4		TGP from three surveys (range 3.9-19.0%) pooled.	(44,149,150)
Sweden	-						Local study with a sample size of 60 is reported in Milakovic M et al, 2004.	(153)
Switzerland	1999	National	SAC (6-12)	610		0 ^c		(170)
The former Yugoslav Republic of Macedonia	2003	National	SAC (8-10)	1206	4.2	0.4 c	Sub-sample thyroid volume 3.72 ml	(171,172)
Turkey	1997-1999	National	SAC (9-11)	5948		31.8c		(173)
United Kingdom	-							

- No data.

P Published

^a Data refer to those most recently available for the period 1994-2004.

Criteria for defining goitre:

^b Zimmermann et al. 2004 (65).

^c Delange et al. 1997 (18).

^d Zimmermann et al. 2001 (174).

^e Gutekunst et al. 1988 (175).

^f Gutekunst & Martin-Teichert. 1993 (176).

Percentage above the age dependent "upper limits" (mean \pm 2SD).
>97 percentile.

>12.0 ml for boys, 13.1 ml for girls.

Source: Adapted from the WHO Global Database on Iodine Deficiency.

ANNEX F

Findings and significance of currently available information on indicators of thyroid function

Country	Age group	Variables	Findings and significance	Comments	References
Albania	-	-	-	-	-
Andorra	-	-	-	-	-
Austria	Adults	TSH, T ₄ , T ₃ , Tg	Optimal thyroid function for urinary I/creatinine ratio of 200–300 µg/g. [information missing from sentence?]	In 2972 euthyroid adults aged 60 ± 15 years and with serum TSH varying from 0.4 to 3.5 mU/l, the lowest serum TSH and low Tg values were found when the UI was between 201 and 300 µg/g creatinine (177). Increasing the level of salt iodization from 7.5 ppm to 15 ppm in 1990 transiently increased the incidence of Plummer's disease (autonomous nodule with suppressed TSH (<0.1 mU/l) and elevated T4 and/or T3) by 30% for a two-year period (178).	(177) (178)
Belgium	Adults Adolescents Pregnant women SAC Neonates	TSH, T ₄ , T ₃ , Tg	Normal thyroid function in adults and adolescents. Subclinical hypothyroidism in pregnant women and neonates.	Normal thyroid function (TSH-T4) in euthyroid adults and adolescents (179, 180). FT4 progressively decreases and TSH and Tg increase during pregnancy (180). Cord serum TSH and Tg higher in neonates than in their mothers (180). Thyroid function abnormalities in mothers and neonates corrected by iodine supplementation (150 µg/day) during gestation (108). Slightly elevated recall rate of neonates at the time of screening for congenital hypothyroidism based on primary TSH (181) and frequent subclinical hypothyroidism in neonates, especially in preterm infants (182). Mild iodine deficiency affecting thyroid function in pregnant women, neonates and young infants. Progressive and slight improvement by silent iodine prophylaxis.	(108,179–182)
Bosnia and Herzegovina	-	-	-	-	-
Bulgaria	Neonates	TSH	Persistence of elevated neonatal TSH (9.2% >5 mU/l).	No data except elevated neonatal TSH (9.2 % >5 mU/l). Mild impairment of neonatal thyroid function. (L. Ivanova, personal communication, 2002).	L. Ivanova, personal communication, 2002

Country	Age group	Variables	Findings and significance	Comments	References
Croatia	Neonates	-	-	No recent data.	-
Cyprus	-	-	-	-	-
Czech Republic	-	TSH, FT ₄ , FT ₃ , Tg	Tg and T3 clearly elevated in all age groups when UI <50 µg/l.	Repeated examinations in two regions (1995 vs. 2002 and/or 1997 vs. 2004) (8) showed a significant increase in median urinary iodine and the percentage < 100 µg/l and < 50 µg/l. No detectable increase of clinically relevant thyroid disorders due to increase of iodine supply and no consistent changes of parameters of thyroid function (TSH, FT ₄ , FT ₃) and/or of thyroid volume were recorded.	(183-186)
Denmark	Adults Adolescents Pregnant women SAC Neonates	TSH, FT ₄ , FT ₃ , Tg	Normal thyroid function in adults and adolescents; elevated TSH and Tg in pregnant women. Normal TSH and elevated Tg in neonates. Subclinical hyperthyroidism in the elderly.	Serum thyroglobulin appears as a good marker of iodine status in non-pregnant adults (183). Serum Tg and TSH increase during gestation and are elevated in cord blood?. These anomalies are prevented by iodine supplementation during pregnancy (184). The frequency of low or blunted TSH (0.40-0.01 mU/l) in the elderly is 9.7% in Jutland where the median UI is 38 µg/l, while it is 0% in Iceland where the median UI is 150 µg/l (183). Borderline hypothyroidism in pregnant women and neonates and subclinical or overt hyperthyroidism in the elderly (185).	(183-186)
Estonia	-	TSH	Frequency of elevated neonatal TSH (17.7% >5mU/l).	Neonatal thyroid screening starting in 1989 and including 20 021 neonates revealed a frequency of neonatal TSH > 5mU/l of 17.7% (187). Mild impairment of neonatal thyroid function.	(187)
Finland	-	-	-	-	-
France	Adults Pregnant women	TSH, FT ₄ , FT ₃ , Tg	Normal thyroid function in adults but subclinical hypothyroidism and elevated Tg during pregnancy.	Serum FT4 and TSH are normal in non-pregnant adults (188) but FT4 and FT3 decrease and TSH, Tg increase during pregnancy (109).	(109,188)
Germany	Pregnant women	TSH, FT ₄ , Tg	Normal thyroid function.	No recent data in non-pregnant adults. Serum FT4, Tg and TSH were normal in 70 pregnant mothers in early gestation (11 weeks) in spite of a median UI of 64 µg/l in the early 1990s (189).	(189)
Greece	Neonates	TSH	Elevated.	No recent metabolic data. The country, especially the mountainous northern part, used to be affected by mild to moderate iodine deficiency with marked alterations of thyroid function (190). Even after the implementation of iodized salt, the recall rate of neonates under suspicion of congenital hypothyroidism was still elevated (0.3% in 1994) and showed regional variations, probably related to regional differences in the iodine intake (191).	(190,191)

Hungary	Adults Pregnant women Neonates	TSH, FT ₄ , Tg Elevated T ₃ : T ₄ ratio during pregnancy; frequently elevated neonatal TSH (17.4% >5 mU/l).	The prevalence of (sub)clinical hyperthyroidism in the elderly (median age 81 years) is higher (3.4%) in an iodine deficient area (median iodine : creatinine ratio: 72 µg/g) than in an iodine replete area (median age 78 years; median iodine : creatinine ratio: 513 µg/g; prevalence of hyperthyroidism: 0%) (192). UI was below 100 µg/l in 57.1% of the 313 pregnant women investigated in an area supposed to be iodine sufficient. It was below 20 µg/l in 15.6% of them. Thyroid was enlarged in 19.2% of them and the serum T ₃ : T ₄ ratio was increased in 97% of them (193). In 2001, 17.4% of the neonates had serum TSH >5 mU/l (F. Peter, personal communication).	(192,193) F. Peter, personal communication, 2003.
Iceland	Adults Adolescents	Frequent hypothyroidism in the elderly. TSH, FT ₄ , FT ₃	In 100 randomly selected adults (mean age 68 years), 18% had elevated serum TSH levels (>4 mU/l), including 4% with values above 10 mU/l. None had a subnormal serum T ₄ . This biochemical picture represents subclinical hypothyroidism. All participants with TSH >10mU/l had serum levels of thyroid autoantibodies (Antiperoxydase TPO-Ab and antithyroglobulin Tg-Ab) (129).	(129)
Ireland	Adults	Hypothyroidism and aging in a rural coastal general practice TSH, T ₄	8.6% Hypothyroidism in females > 50 years. Functional consequences. Thyroid enlargement during pregnancy. Increased urinary iodine excretion starting at first trimester and continuing throughout gestation. Acute fall to nonpregnant levels at delivery.	(194-196)
Italy	(Sub)normal thyroid function in adults and adolescents with possibly (isolated) elevated Tg; frequent sub-clinical hyperthyroidism in the elderly; overt or biochemical hypothyroidism in pregnant women.	TSH, T ₄ , T ₃ , Tg	No alterations of thyroid function in the juvenile population in Sardinia (197). Slight decrease of serum T ₄ but normal TSH in juveniles in Sicily; serum Tg can be elevated in the absence of elevated TSH (131). Elevated prevalence (2.9%) of hyperthyroidism in the elderly (139). Overt or biochemical hypothyroidism in early and late gestation in 50-70% of pregnant women (198,199). Neonatal TSH is markedly elevated (14.4% >5 mU/l blood) in Calabria and is used as a monitoring tool of iodine deficiency control (200). Transient neonatal hypothyroidism may result in a loss of IQ points in childhood (201) and transient neonatal hyperthyrotropinaemia may result in sub-clinical hypothyroidism in early childhood (202). Clinically and biochemically euthyroid school children can exhibit mild retardation in psychoneuro-intellectual development, probably as the consequence of unrecognized neonatal hypothyroidism (203-205). Mild alterations of thyroid function in non-pregnant adults but definite alterations of this function in pregnant women, neonates and young infants with retardation in neuro-intellectual development of children as serious public health consequence.	(131,139, 197-205)

Country	Age group	Variables	Findings and significance	Comments	References
Latvia	-	-	-	-	-
Liechtenstein	-	-	-	-	-
Lithuania	Adults Adolescents	TSH, Tg	Frequency of elevated neonatal TSH (22% >5 mU/l) and 60% of thyroid volume in adolescents	-	Bartkeviciute R, personal communication, 2004
Luxembourg	-	-	-	-	-
Malta	-	-	-	-	-
Monaco	-	-	-	-	-
Netherlands	-	-	-	No recent data.	-
Norway	-	-	-	-	-
Poland	Pregnant women Neonates	-	Normal thyroid function during pregnancy. Elevated neonatal TSH. Normal after correction of iodine deficiency.	Thyroid function was normal at the time of delivery in a group of 46 women in spite of moderate iodine deficiency (mean UI 35 µg/l) and enlargement of thyroid volume (mean ± SD: 27.8 ± 15.3 ml) (206). Shift of neonatal TSH towards elevated values and progressive improvement with progress of the programme of salt iodization (207,208).	(206-208)
Portugal	-	-	-	No recent data.	-
Romania	SAC	TSH, FT ₄ , Tg	Normal thyroid function in SAC.	The serum levels of TSH, FT ₄ and Tg were normal in 214 schoolchildren 6-14 years of age in spite of a median UI of 42 µg/l (71).	(72)
San Marino	-	-	-	-	-
Serbia and Montenegro	-	-	-	-	-
Slovakia	-	-	-	-	-
Slovenia	-	-	-	-	-
Spain	SAC	TSH	Frequent suppressed TSH in iodine deficient children indicating subclinical hyperthyroidism.	The prevalence of suppressed serum TSH in healthy children is 2% when the median UI is 66.3 µg/l. It is only 0.3% when the median UI is 115.7 µg/l (43). This demonstrates that even in children, subclinical hyperthyroidism is higher in iodine-deficient than in iodine-replete areas.	(44)

Sweden	Adults Adolescents	TSH, FT ₄ , TPOAb	Expected prevalence of thyroid disease in adults. Three schoolchildren (15-17 years) of 59 screened needed medical attention or follow-up.	In 1154 randomly selected women, the prevalence of primary hypothyroidism (past and present) was 3.3% and of hyperthyroidism (past and present) 2.5%. The prevalence of visible goitre 2.1%.	(209)
Switzerland	Pregnant women	TSH	Normal thyroid function during pregnancy.	The incidence of toxic nodular goitre in adults used to be elevated but decreased after full correction of mild iodine deficiency (170). Median serum TSH was normal (0.6 mU/l) in 396 pregnant women who had a median UI of 138 µg/l (210) indicating iodine sufficiency.	(170,210)
The former Yugoslav Republic of Macedonia	Neonates	TSH	Frequency of elevated neonatal TSH (>5 mU/l) in 2002 - 4.3 %, and in 2003 - 5.9 %.		(172)
Turkey	SAC Neonates	TSH, FT ₄ , T ₄ , T ₃ , Tg	Subclinical hypothyroidism and elevated Tg in SAC; elevated neonatal TSH and exaggerated TSH response to TRH.	In 251 schoolchildren 9-11 of age years in four areas with mild (mean UI 56 µg/l) to moderate (mean UI 20.7-30.8 µg/l) iodine deficiency, serum FT4 was slightly low and TSH elevated (211). In a group of 73 healthy schoolchildren 7-12 of age years living in an endemic area of central Turkey (mean UI 39.1 µg/l), mean serum TSH, T ₄ , T ₃ and Tg were in the normal range but 64 and 23 children had serum Tg and T ₃ above normal, respectively, while 7 children had a free T4 below normal (212). Borderline neonatal hypothyroidism (elevated basal TSH and exaggerated response to TRH) with prolonged jaundice during the neonatal period was a common finding in Ankara. Iodine deficiency was a possible etiological factor (213). Neonatal thyroid screening in 30 097 newborns in Turkey (location not indicated) showed an abnormally high frequency of serum TSH above 40 m U/l (2.3%) among which only 1.6% had confirmed permanent congenital hypothyroidism. Iodine deficiency was a possible cause of this frequency of "false positives" (214).	(211-214)

Country	Age group	Variables	Findings and significance	Comments	References
United Kingdom	Pregnant women Neonates	TSH, FT ₄	In parts of Scotland, 40% of pregnant women had less than adequate UI and FT ₄ ; did not show the expected increase of FT ₄ during pregnancy; frequently elevated neonatal TSH.	Serum TSH, FT ₄ , anti-microsomal and anti-thyroglobulin antibodies and possibly T _s were measured in 1704 of the 1801 adults surveyed in 1992-1993. TSH was elevated (>5 mU/l) in 91 of them. It was above 10 mU/l in 31 of them. Of the latter, 87% had positive antithyroid antibodies. The mean incidence of hypothyroidism and hyperthyroidism in women were 3.5/1000 per year and 0.8/1000 per year, respectively. The incidence of hypothyroidism but not of hyperthyroidism increased in the elderly. Thyroid function was considered as normal in the other subjects. In Tayside (Scotland) the mean UI in pregnant women was 137 ± 104 (SD) µg/l but approximately 40% of them had UI below half the recommended value. The FT ₄ in pregnant women did not show the expected increase (215). In this region, neonatal thyroid screening revealed a substantial proportion of children with a transient rise in TSH (216). The incidence of hypothyroidism due to thyroid autoimmunity is elevated in the elderly.	(215,216)

- No data.

Notes: SAC, school-age children; Tg, thyroglobulin; TRH, thyroid releasing hormone; FT₃, free triiodothyronine; FT₄, free thyroxine.

ANNEX G

Programmatic indicators for monitoring the elimination of iodine deficiency

Country	Classification of iodine nutrition	National committee (year initiated)	National programme	Executive officer	Regulation	Public education programme	Regular salt monitoring	Regular UI monitoring	QA/QC with salt industry	Monitoring database	Adequate laboratories
Albania	Moderate iodine deficiency	Yes (2002)	Uncertain		No	Uncertain	Uncertain	Uncertain			
Andorra		-			-						
Austria	Optimal	No	Partial		Yes	No	Yes	Yes			
Belgium	Mild iodine deficiency	Yes (1993-1998)	No		Yes	No	No	No			
Bosnia and Herzegovina	Optimal	Yes (2000)	Yes		Yes	Partial	Yes	Yes			
Bulgaria	Optimal	Yes (1994)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Croatia	Optimal	Yes (1992)	Yes		Yes	Uncertain	Yes	Yes			
Cyprus		No	No		-	No	No	No			
Czech Republic	Optimal	Yes (1994)	Yes		Yes	Uncertain	Yes	Yes			
Denmark	Mild iodine deficiency	Yes (1994)	Yes		Yes	Uncertain	Yes	Yes	Yes	Yes	Yes
Estonia	Mild iodine deficiency	-			-						
Finland	Optimal	Yes	Yes		No	Uncertain	Yes	Yes			
France	Mild iodine deficiency	No	Yes		Yes	Uncertain	No	No			
Germany	Optimal	Yes (1984)	Yes		No	Uncertain	Yes	No			
Greece	Optimal	No	No		Yes	No	No	No			
Hungary	Mild iodine deficiency	Yes (1995)	Yes		No	No	No	No			
Iceland	Optimal	-	No		-	No	No	No			
Ireland	Mild iodine deficiency	-	No		No	No	No	Occasional			
Italy	Mild iodine deficiency	Yes (1985)	Partial		Yes	Partial	Partial	Partial			

Country	Classification of iodine nutrition	National committee (year initiated)	National programme	Executive officer	Regulation	Public education programme	Regular salt monitoring	Regular UI monitoring	QA/QC with salt industry	Monitoring database	Adequate laboratories
Latvia	Mild iodine deficiency	-	-	-	-	-	-	-	-	-	-
Liechtenstein		-	-	-	-	-	-	-	-	-	-
Lithuania	Mild iodine deficiency	No	Partial		-Yes	Yes	No	No	No	No	No
Luxembourg	Optimal	-	No		No	No	No	No	No	No	No
Malta		-	-	-	-	-	-	-	-	-	-
Monaco		-	-	-	-	-	-	-	-	-	-
Netherlands	Optimal	No	No		Yes	No	No	No	No	No	No
Norway	Optimal	-	No		Yes	No	No	No	No	No	No
Poland	Optimal	Yes (1991)	Yes		Yes	Partial	Yes	Yes	Yes	Yes	Yes
Portugal		No	No		Yes	No	No	No	No	No	No
Romania	Optimal	Yes (2002)	Yes		Yes	Uncertain	Yes	Partial	Partial	Partial	Partial
San Marino		-	-	-	-	-	-	-	-	-	-
Serbia and Montenegro	Optimal	Yes (2000)	Yes		Yes	Yes	Yes	Yes	Yes	Yes	Yes
Slovakia	Optimal	Yes (2000)	Yes		Yes	Uncertain	Yes	Yes	Yes	Yes	Yes
Slovenia	Optimal	Yes (1997)	Yes		Yes	Uncertain	Yes	No	No	No	No
Spain	Optimal	Yes	No		Yes	No	No	No	No	No	No
Sweden	Optimal	-	No		Yes	No	No	No	No	No	No
Switzerland	Optimal	Yes (1922)	Yes		Yes	No	Yes	Yes	Yes	Yes	Yes
The former Yugoslav Republic of Macedonia	Risk of IIH in susceptible groups	Yes (1997)	Yes		Yes	Yes	Yes	Yes	Yes	Yes	Yes
Turkey	Mild iodine deficiency	Yes (1994)	Yes		Yes	Partial	Yes	No	No	No	No
United Kingdom		No	No		No	No	No	No	No	No	No

- No data.

QA/QC: quality assurance/quality control.

IIH: iodine-induced hyperthyroidism

Source: WHO/UNICEF/ICCID (27), WHO/FAO (217), the European Salt Producers' Association, personal communication, 2003.

References

1. Hetzel BS. An overview of the elimination of brain damage due to iodine deficiency. In: Hetzel BS, ed. *Towards the global elimination of brain damage due to iodine deficiency*. New Delhi, Oxford University Press, 2004: 24–37.
2. Delange F, Dunn JT, Glinoe D, eds. *Iodine deficiency in Europe. A continuing concern*. New York, Plenum Press, 1993.
3. Network for Sustained Elimination of Iodine Deficiency. *Draft report of a meeting held in Ghent, Belgium, 12 July 2002*. 2002.
4. De Quervain F, Wegelin C, eds. *Der endemische Kretinismus [Endemic cretinism]*. Berlin, Verlag von Julius Springer, 1936.
5. König MP, ed. *Die kongenitale Hypothyreose und der endemische Kretinismus [Congenital hypothyroidism and endemic cretinism]*. Berlin, Springer-Verlag, 1968.
6. Merke F. *History and iconography of endemic goitre and cretinism*. Bern, Hans Huber Verlag, 1984.
7. Sanchez-Franco F et al. Alteraciones por deficiencia de yodo en las Hurdes. III Cretinismo [Disorders due to iodine deficiency in Las Hurdes. III Cretinism]. *Endocrinologia*, 1987, 34 (Suppl 2):88–93.
8. Delange F. Iodine deficiency in Europe anno 2002. *Thyroid International*, 2002, 5:3–18.
9. Boussingault JB. Recherche sur la cause qui produit le goître dans la cordillère de la Nouvelle Grenade [Research on the cause of goitre in the cordillera of Nueva Granada]. *Annales de Chimie et de Physique*, 1831, 48:41–69.
10. Bürgi H, Supersaxo Z, Selz B. Iodine deficiency diseases in Switzerland 100 years after Theodor Kocher's survey: a historical review with some new goiter prevalence data. *Acta Endocrinologica*, 1990, 123:577–590.
11. Delange F et al. World status of monitoring of iodine deficiency disorders control programs. *Thyroid*, 2002, 12:915–924.
12. Bürgi H et al. Thyroid volumes and urinary iodine in Swiss school children, 17 years after improved prophylaxis of iodine deficiency. *European Journal of Endocrinology*, 1999, 140:104–106.
13. *Endemic goitre*. Geneva, World Health Organization, 1960 (Monograph Series no. 44).
14. Kelly FC, Snedden WW. Prevalence and geographical distribution of endemic goitre. *Endemic goitre*. Geneva, World Health Organization, 1960: 27–234. (Monograph Series no.44).
15. Gutekunst R, Scriba PC. Goiter and iodine deficiency in Europe – the European Thyroid Association report as updated in 1988. *Journal of Endocrinological Investigation*, 1989, 12:209–220.
16. WHO, UNICEF, ICCIDD. *Global prevalence of iodine deficiency disorders*. Geneva, World Health Organization, 1993 (Micronutrient Deficiency Information System. MDIS Working Paper No.1).

17. Delange F et al. *Elimination of iodine deficiency disorders (IDD) in Central and Eastern Europe, the Commonwealth of Independent States, and the Baltic States*. Geneva, World Health Organization, 1998 (WHO/EURO/NUT/98.1).
18. Delange F et al. Thyroid volume and urinary iodine in European schoolchildren: standardization of values for assessment of iodine deficiency. *European Journal of Endocrinology*, 1997, 136:180–187.
19. WHO Regional office for Europe and Nutrition and Food Security Programme. *The first action plan for food and nutrition policy: WHO European region 2000–2005*. Copenhagen, WHO Regional Office for Europe, 2001 (EUR/01/5026013).
20. WHO, UNICEF, ICCIDD. *Progress towards the elimination of iodine deficiency disorders (IDD)*. Geneva, World Health Organization, 1999 (WHO/NHD/99.4).
21. Vitti P et al. Europe is iodine deficient. *Lancet*, 2003, 361:1226.
22. WHO. *National strategy for overcoming micronutrient malnutrition. Report to the Director General, 45th World Health Assembly*. Geneva, World Health Organization, 1992 (WHA 45.33).
23. *World Summit for Children – Mid Decade Goal: Iodine Deficiency Disorders*. UNICEF–WHO Joint Committee on Health Policy. Geneva, United Nations Children’s Fund, World Health Organization, 1994 (JCHPSS/94/2.7).
24. Gerasimov G. IDD in Eastern Europe and Central Asia. *IDD Newsletter*, 2002, 18:33–37.
25. Dunn JT. Complacency: The most dangerous enemy in the war against iodine deficiency. *Thyroid*, 2000, 10:681–683.
26. Hetzel BS, Pandav CS. *S.O.S. for a Billion. The conquest of iodine deficiency disorders*. Oxford, Oxford University Press, 1994.
27. WHO, UNICEF, ICCIDD. *Assessment of iodine deficiency disorders and monitoring their elimination: A guide for programme managers*. Geneva, World Health Organization, 2001 (WHO/NHD/01.1).
28. Delange F. Iodine deficiency as a cause of brain damage. *Postgraduate Medical Journal*, 2001, 77:217–220.
29. *Technical consultation for the prevention and control of iodine deficiency in pregnant and lactating women and in children less than two years old*. Geneva, World Health Organization, 2007. (To be published).
30. *Vitamin and mineral requirements in human nutrition: Joint FAO/WHO Expert Consultation on Human Vitamin and Mineral Requirements*. 2nd ed. Geneva, World Health Organization and Food and Agriculture Organization, 2004.
31. Hetzel BS. Iodine deficiency disorders (IDD) and their eradication. *Lancet*, 1983, 2:1126–1129.
32. Laurberg P et al. Thyroid disorders in mild iodine deficiency. *Thyroid*, 2000, 10:951–963.
33. Stanbury JB et al. Iodine-induced hyperthyroidism: occurrence and epidemiology. *Thyroid*, 1998, 8:83–100.
34. de Benoist B et al., eds. *Iodine status worldwide. WHO Global Database on Iodine Deficiency*. Geneva, World Health Organization, 2004.
35. *World population prospects: the 2002 revision*. New York, United Nations/Population Division, 2003.
36. Glinoe D, Delange F. The potential repercussions of maternal, fetal, and neonatal hypothyroxinemia on the progeny. *Thyroid*, 2000, 10:871–887.
37. Stanbury JB, ed. *The damaged brain of iodine deficiency*. New York, Cognizant Communications Corporation, 1994.
38. Morreale de Escobar G, Obregon MJ, del Rey FE. Is neuropsychological development related to maternal hypothyroidism or to maternal hypothyroxinemia? *Journal of Clinical Endocrinology and Metabolism*, 2000, 85:3975–3987.
39. Delange F, Hetzel BS. *The iodine deficiency disorders*. Hennemann G, DeGroot L, eds. Endocrine Education, Inc., 2003. Thyroid Manager (<http://www.thyroidmanager.org/Chapter20/chapter20.pdf>, accessed 20 September 2005).

40. Delange F. Screening for congenital hypothyroidism used as an indicator of the degree of iodine deficiency and of its control. *Thyroid*, 1998, 8:1185–1192.
41. Hetzel BS. The nature and magnitude of the iodine deficiency disorders. In: Hetzel BS, Pandav CS, eds. *S.O.S for a billion. The conquest of iodine deficiency disorders*. New Delhi, Oxford University Press, 1994: 3–26.
42. Hollowell JG, Hannon WH. Teratogen update: Iodine deficiency, a community teratogen. *Teratology*, 1997, 55:389–405.
43. Ermans AM. Intrathyroid iodine metabolism in goiter. In: Stanbury JB, ed. *Endemic Goiter*. Washington, DC, Pan American Health Organization, 1969: 1–13.
44. Garcia-Mayor RV et al. Effect of iodine supplementation on a pediatric population with mild iodine deficiency. *Thyroid*, 1999, 9:1089–1093.
45. Dremier S et al. Thyroid autonomy: Mechanism and clinical effects. *Journal of Clinical Endocrinology and Metabolism*, 1996, 81:4187–4193.
46. Allen L et al., eds. *Guidelines on food fortification with micronutrients*. Geneva, World Health Organization and Food and Agricultural Organization of the United Nations, 2006.
47. Pandav CS. The economic benefits of the elimination of IDD. In: Hetzel BS, Pandav CS, eds. *S.O.S. for a billion. The conquest of iodine deficiency disorders*. New Delhi, Oxford University Press, 1996: 129–145.
48. Population Health and Nutrition Department. *Bangladesh: food and nutrition sector review mission: cost-effectiveness of food and nutrition intervention programs*. Washington, DC, World Bank, 1985 (No. 4974-BD).
49. Levin HM et al. Micronutrient deficiency disorders. In: Jamison DT et al., eds. *Disease control priorities in developing countries*. New York, Oxford University Press, 1993: 421–451.
50. Mason JB et al. *The Micronutrient Report. Current progress and trends in the control of vitamin A, iodine, and iron deficiencies*. Ottawa, Canada, Micronutrient Initiative, 2001.
51. Hunt JM. Why countries and companies should invest to eliminate micronutrient malnutrition. In: Asian Development Bank, ILSI, MI, eds. *Manila Forum 2000. Strategies to fortify essential foods in Asia and the Pacific*. Manila, Asian Development Bank, 2000: 32–41. (Nutrition and Development Series).
52. Kahaly GJ, Dietlein M. Cost estimation of thyroid disorders in Germany. *Thyroid*, 2002, 12:909–914.
53. Bleichrodt N, Born MP. A metaanalysis of research on iodine and its relationship to cognitive development. In: Stanbury JB, ed. *The damaged brain of iodine deficiency*. New York, Cognizant Communication, 1994: 195–200.
54. Hetzel BS, Maberly G. Iodine. In: Mertz C, ed. *Trace elements in human and animal nutrition*. New York, Academic Press, 1986: 139–208.
55. Levin HM. Economic dimensions of iodine deficiency disorders. In: Hetzel BS, Dunn JT, Stanbury JB, eds. *The prevention and control of iodine deficiency disorders*. New York, Elsevier, 1987: 195–208.
56. Pandav CS, Rao AR. *Iodine deficiency disorders in livestock – Ecology and economics*. Delhi, Oxford University Press, 1997.
57. Hetzel BS, Clugston G. Iodine. *Modern nutrition in health and disease*. Baltimore, Lippincott Williams and Wilkins, 1999: 253–264.
58. Correa H. A cost-benefit study of iodine supplementation programs for the prevention of endemic goiter and cretinism. In: Stanbury JB, Hetzel BS, eds. *Endemic goiter and endemic cretinism*. New York, John Wiley and Sons, 1980: 567–588.
59. Barden HS, Kessel R. The costs and benefits of screening for congenital hypothyroidism in Wisconsin. *Social Biology*, 1984, 31:185–200.
60. Danese MD et al. Screening for mild thyroid failure at the periodic health examination – A decision and cost-effectiveness analysis. *Journal of the American Medical Association*, 1996, 276:285–292.
61. Greene L. Hyperendemic goiter, cretinism and social organization in Highland Ecuador. Malnutrition, behavior, and social organization. In: Greene L, ed. *Malnutrition, Behavior, and Social Organization*. New York, Academic Press, 1977: 55–94.

62. Braverman LE. Adequate iodine intake – the good far outweighs the bad. *European Journal of Endocrinology*, 1998, 139:14–15.
63. Delange F. Risks and benefits of iodine supplementation. *Lancet*, 1998, 351:923–924.
64. Gorstein J. Goiter assessment: Help or hindrance in tracking progress in iodine deficiency disorders control program? *Thyroid*, 2001, 11:1201–1202.
65. Zimmermann MB et al. New reference values for thyroid volume by ultrasound in iodine-sufficient schoolchildren: a World Health Organization/Nutrition for Health and Development Iodine Deficiency Study Group Report. *American Journal of Clinical Nutrition*, 2004, 79:231–237.
66. Mannar V and Dunn JT. *Salt iodization for the elimination of iodine deficiency*. Charlottesville, VA, International Council for Control of Iodine Deficiency Disorders, 1995.
67. Diosady LL et al. Stability of iodine in iodized salt used for correction of iodine-deficiency disorders. *Food and Nutrition Bulletin*, 1999, 19:240–250.
68. WHO, UNICEF, ICCIDD. *Recommended iodine levels in salt and guidelines for monitoring their adequacy and effectiveness*. Geneva, World Health Organization, 1996 (WHO/NUT/96.13).
69. *Codex Alimentarius. Codex Standard for Food Grade Salt*. Rome, Food and Agriculture Organization, 2001 (CODEX STAN 150–1985, revised 2001).
70. UNICEF. *The official summary of the state of the world's children 2004*. New York, United Nations Children's Fund, 2003.
71. Wolff J. Physiology and pharmacology of iodized oil in goiter prophylaxis. *Medicine*, 2001, 80:20–36.
72. Simescu M et al. Iodized oil as a complement to iodized salt in schoolchildren in endemic goiter in Romania. *Hormone Research*, 2002, 58:78–82.
73. Delange F. Administration of iodized oil during pregnancy: A summary of the published evidence. *Bulletin of the World Health Organization*, 1996, 74:101–108.
74. Brussaard JH et al. Iodine intake and urinary excretion among adults in the Netherlands. *European Journal of Clinical Nutrition*, 1997, 51:S59–S62.
75. [Anonymous]. Iodized water to eliminate iodine deficiency. *IDD Newsletter*, 1997, 13:33–39.
76. Delong GR. Iodine dripping into irrigation water: its role in correcting iodine deficiency. *IDD Newsletter*, 2002, 18:60–61.
77. Phillips DIW. Iodine, milk, and the elimination of endemic goitre in Britain: the story of an accidental public health triumph. *Journal of Epidemiology and Community Health*, 1997, 51:391–393.
78. WHO. *Report on the Expert Consultation on salt as a vehicle for fortification, Luxembourg, 21–22 March 2007*. Geneva, World Health Organization, 2007. (To be published).
79. WHO, UNICEF, ICCIDD. *Iodine and health: Eliminating iodine deficiency disorders safely through salt iodization*. Geneva, World Health Organization, 1994 (WHO/NUT/94.4).
80. Institute of Medicine. *Dietary Reference Intakes: applications in dietary assessment*. Washington, D.C., National Academy Press, 2001.
81. FAO and WHO. *Recommended nutrient reference values for food labelling purposes: report of a Joint FAO/WHO Expert Consultation on recommended allowances of nutrients for food labelling purposes (1988: Helsinki, Finland)*. Geneva, World Health Organization, 1988.
82. European Commission HaCPD-GSCoF. *Opinion of the Scientific Committee on Food on the tolerable upper intake level of iodine*. Brussels, European Commission, 2002 (SCF/CS/NUT/UPPLEV/26 Final).
83. AFSSA (Agence Française de Sécurité Sanitaire des Aliments). *Report from the AFSSA expert committee on human nutrition. Vitamin and mineral fortification of commonly eaten foods: meeting the nutritional and safety needs of the consumer*. 2001 (Case No. 2000-SA-0239).
84. Institute of Medicine. *Dietary Reference Intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc*. Washington, D.C., National Academy Press, 2002.
85. Wolff J. Iodide goiter and the pharmacologic effects of excess iodide. *American Journal of Medicine*, 1969, 47:101–124.

86. Todd CH. *Hyperthyroidism and other thyroid disorders. A practical handbook for recognition and management*. Harare, World Health Organization, International Council for Control of Iodine Deficiency Disorders, 1999 (WHO/AFRO/NUT/99.1).
87. Bournaud C, Orgiazzi JJ. Iodine excess and thyroid autoimmunity. *Journal of Endocrinological Investigation*, 2003, 26:49–56.
88. Williams ED. Dietary iodide and thyroid cancer. In: Hall R, Köbberling J, eds. *Thyroid disorders associated with iodine deficiency and excess*. New York, Raven Press, 1985: 201–207.
89. Feldt-Rasmussen U. Iodine and Cancer. *Thyroid*, 2001, 11:483–486.
90. Slowinska-Klencka D et al. Fine-needle aspiration biopsy of the thyroid in an area of endemic goitre: influence of restored sufficient iodine supplementation on the clinical significance of cytological results. *European Journal of Endocrinology*, 2002, 146:19–26.
91. Pandav CS et al. Validation of spot-testing kits to determine iodine content in salt. *Bulletin of the World Health Organization*, 2000, 78:975–980.
92. Escobar del Rey F, Morreale de Escobar G. Iodine deficiency in Spain: Update of a widespread and persisting problem. In: Delange F, Dunn JT, Glinoe D, eds. *Iodine deficiency in Europe. A continuing concern*. New York, Plenum Press, 1993: 395–402.
93. [Anonymous]. West and Central Europe assesses its iodine nutrition. *IDD Newsletter*, 2002, 18:51–55.
94. Aghini-Lombardi F, Antonangeli L, Pinchera A. Iodine deficiency in Europe. National reports on iodine status in West-Central European countries. First symposium of ICCIDD West-Central Europe. Goteborg, Sweden, September 7, 2002. *Journal of Endocrinological Investigation* 26[9 (Suppl.)], 1–62. 2003.
95. Delange F et al. Iodine deficiency in the world: Where do we stand at the turn of the century? *Thyroid*, 2001, 11:437–447.
96. Edwards DG, Marsh RA. The role of salt in food manufacture. In: Geertman RM, ed. *8th World Salt Symposium*. Amsterdam, Elsevier, 2000: 793–800.
97. Moinier B. Iodised salt production in West European countries. Presented at a meeting of the European Salt Producers' Association, Moscow, 2002.
98. Pietinen P. Sources of sodium in the Finnish diet. *Journal of the Scientific Agricultural Society of Finland*, 1981, 53:275–284.
99. James WPT, Ralph A, Sanchezcastillo CP. The dominance of salt in manufactured food in the sodium intake of affluent societies. *Lancet*, 1987, 1:426–429.
100. *Trends in Germany of iodized salt used by households and in the food industry*. European Salt Producers' Association. Trends in Germany of iodized salt used by households and in the food industry. European Salt Producers' Association, 2003.
101. WHO. *Food based dietary guidelines in the WHO European Region*. Copenhagen, WHO Regional Office for Europe, 2003 (EUR/03/5045414).
102. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. Intersalt Cooperative Research Group. *BMJ*, 1988, 297:319–328.
103. Wiersinga WM et al. A survey of iodine intake and thyroid volume in Dutch schoolchildren: reference values in an iodine-sufficient area and the effect of puberty. *European Journal of Endocrinology*, 2001, 144:595–603.
104. Regalbuto C et al. [Iodine deficiency and iodine prophylaxis experience in Sicily]. [Italian]. *Annali dell'Istituto Superiore di Sanita*, 1998, 34:429–436.
105. Pfannenstiel P. Direct and indirect costs caused by continuous iodine deficiency. In: Hall R, Köbberling J, eds. *Thyroid disorders associated with iodine deficiency and excess*. New York, Raven Press, 1985: 447–453.
106. World Bank. *World Development Report 1993: Investing in health*. New York, Oxford University Press, 1993.
107. Ciardelli R et al. The nutritional iodine supply of Belgian neonates is still insufficient. *European Journal of Pediatrics*, 2002, 161:519–523.
108. Glinoe D et al. A randomized trial for the treatment of mild iodine deficiency during pregnancy – maternal and neonatal effects. *Journal of Clinical Endocrinology and Metabolism*, 1995, 80:258–269.

109. Caron P et al. Urinary iodine excretion during normal pregnancy in healthy women living in the southwest of France: correlation with maternal thyroid parameters. *Thyroid*, 1997, 7:749–754.
110. ICCIDD and MI. *Ending iodine deficiency. Now and forever. A communication guide*. New Orleans, International Council for the Control of Iodine Deficiency Disorders, 1997.
111. WHO. *The World Health Report 2002 : Reducing the risks, promoting healthy life*. Geneva, World Health Organization, 2002 (WA 540.1 95WO 2002).
112. Zimmermann MB et al. Severe iodine deficiency in southern Albania. *International Journal for Vitamin and Nutrition Research*, 2003, 73:347–350.
113. Delange F et al. Silent iodine prophylaxis in Western Europe only partly corrects iodine deficiency; the case of Belgium. *European Journal of Endocrinology*, 2000, 143:189–196.
114. UNICEF Office for Bosnia-Herzegovina. *Iodine status of the population of Bosnia-Herzegovina*. Sarajevo, United Nations Children's Fund, 2006.
115. *Report of the results of the national representative survey on endemic goiter and iodine status in Bulgaria 2003*. Ministry of Health of Bulgaria, National Center of Hygiene MEaN, 2004.
116. Kusic Z et al. Croatia has reached iodine sufficiency. *Journal of Endocrinological Investigation*, 2003, 26:738–742.
117. Zamrazil V et al. The elimination of iodine deficiency in the Czech Republic: the steps toward success. *Thyroid*, 2004, 14:49–56.
118. Rasmussen LB et al. Dietary iodine intake and urinary iodine excretion in a Danish population: effect of geography, supplements and food choice. *British Journal of Nutrition*, 2002, 87:61–69.
119. Veinpalu M et al. Urinary iodine excretion in Estonian children. *European Journal of Endocrinology*, 1996, 135:248.
120. Valsta M et al. Iodine status of middle-aged subjects in Finland (abstract). *Spring Meeting of the Society for Nutrition Research*, 2003.
121. Valeix P et al. Iodine deficiency in France. *Lancet*, 1999, 353:1766–1767.
122. Hampel R et al. Urinary iodine excretion in German school children within normal range. *Medizinische Klinik*, 2001, 96:125–128.
123. Zois C et al. High prevalence of autoimmune thyroiditis in schoolchildren after elimination of iodine deficiency in northwestern Greece. *Thyroid*, 2003, 13:485–489.
124. Markou K et al. Iodine intake and thyroid function in villagers and city dwellers in Southwestern Greece. *Thyroid*, 1996, 1 (suppl):79.
125. Ildikó F, Mihályné S, Zoltán F. *Jelentés az 1997 évben I–IV osztályos fiútanulókon végzett tapintásos golyvagyakorlás és vizeletjód vizsgálatok eredményeiről [Report of 1997 survey of primary school boys, I–IV grade: goitre prevalence and urinary iodine excretion]*. Budapest, 'Johan Bela' National Public Health Institute, 1997.
126. Ildikó F, Mihályné S, Zoltán F. *Jelentés az 1996 évben I–IV osztályos fiútanulókon végzett tapintásos golyvagyakorlás és vizeletjód vizsgálatok eredményeiről [Report of 1996 survey of primary school boys, I–IV grade: goitre prevalence and urinary iodine excretion]*. Budapest, 'Johan Bela' National Public Health Institute, 1996.
127. Ildikó F, Mihályné S, Zoltán F. *Jelentés az 1995/1996 ös tanévben I–IV osztályos fiútanulókon végzett tapintásos golyvagyakorlás és vizeletjód vizsgálatok eredményeiről [Report of 1995/1996 survey of primary school boys, I–IV grade: goitre prevalence and urinary iodine excretion]*. Budapest, 'Johan Bela' National Public Health Institute, 1995.
128. Ildikó F, Mihályné S, Zoltán F. *Jelentés az 1994/1995 ös tanévben I–IV osztályos fiútanulókon végzett tapintásos golyvagyakorlás és vizeletjód vizsgálatok eredményeiről [Report of 1994/1995 survey of primary school boys, I–IV grade: goitre prevalence and urinary iodine excretion]*. Budapest, 'Johan Bela' National Public Health Institute, 1994.
129. Laurberg P et al. Iodine intake and the pattern of thyroid disorders: A comparative epidemiological study of thyroid abnormalities in the elderly in Iceland and in Jutland, Denmark. *Journal of Clinical Endocrinology and Metabolism*, 1998, 83:765–769.
130. *IDD status in Ireland*. Smyth PPA, 2002.
131. Rapa A et al. Iodine deficiency in Italy. *Lancet*, 1999, 354:596–597.

132. Frigato F et al. Epidemiological survey of goiter and iodine deficiency in Veneto region. *Journal of Endocrinological Investigation*, 1996, 19:734–738.
133. Pagliara S et al. Diffusione del gozzo endemico e della carenza iodica in provincia di Avellino [Widespread endemic goitre and iodine deficiency in the province of Avellino]. *Annali dell'Istituto Superiore di Sanita*, 1998, 34:417–421.
134. Vitti P et al. Thyroid volume measurement by ultrasound in children as a tool for the assessment of mild iodine deficiency. *Journal of Clinical Endocrinology and Metabolism*, 1994, 79:600–603.
135. Aghini-Lombardi F et al. Effect of iodized salt on thyroid volume of children living in an area previously characterized by moderate iodine deficiency. *Journal of Clinical Endocrinology and Metabolism*, 1997, 82:1136–1139.
136. Zini M et al. Indagine epidemiologica sulla prevalenza del gozzo e sulla escrezione urinaria di iodio nella popolazione scolastica della provincia di Reggio Emilia [Epidemiologic investigation on the prevalence of goiter and urinary excretion of iodine in the school population of the province of Reggio Emilia]. *Annali dell'Istituto Superiore di Sanita*, 1998, 34:383–387.
137. Cassio A et al. Prevalenza di gozzo e ioduria nella popolazione della scuola dell'obbligo in un'area dell'Appennino bolognese [Prevalence of goiter and urine iodine in a school population in an area of the Bolognese Apennines]. *Annali dell'Istituto Superiore di Sanita*, 1998, 34:389–391.
138. Lupoli G et al. Evaluation of goiter endemia by ultrasound in schoolchildren in Val Sarmiento (Italy). *Journal of Endocrinological Investigation*, 1999, 22:503–507.
139. Aghini-Lombardi F et al. The spectrum of thyroid disorders in an iodine-deficient community: the Pescopagano survey. *Journal of Clinical Endocrinology and Metabolism*, 1999, 84:561–566.
140. Selga G, Sauka M, Gerasimov G. Status of iodine deficiency in Latvia reconsidered: results of nation-wide survey of 587 school children in the year 2000. *IDD Newsletter*, 2000, 16(4).
141. [Anonymous]. IDD in the Baltics. *IDD Newsletter*, 2000, 16:53.
142. Ministe de la sante Ddls. *Etude sur l'evaluation de la iodurie effectivée en milieu scolaire 2002*. 2002.
143. Dahl L et al. Iodine intake and status in two groups of Norwegians. *Scandinavian Journal of Nutrition*, 2003, 47:170–178.
144. Golkowski F et al. Prevalence of goiter in schoolchildren – a study on the influence of adequate iodine prophylaxis in Poland. *Journal of Endocrinological Investigation*, 2003, 26:11–15.
145. Simescu M, Dimitriu I, Sava M. *Iodine concentration in spot urine samples of school children from 27 counties between 2000–2002*. Bucharest, Ministry of Health, 2002.
146. Simic M et al. *Republic of Serbia is free of iodine deficiency: results of comprehensive survey of school children, 2001*. 2001 (unpublished).
147. *Urinary iodine assessment in children in Slovakia*. Kostalova L, 2003.
148. Geberšček S et al. *Legislation and IDD status in Slovenia*. Ljubljana, University Medical Centre, Department for Nuclear Medicine, 2004.
149. Delgado Álvarez E et al. Nutrición de yodo en los escolares Asturianos tras 18 años de yodo-profilaxis con sal: erradicación del bocio endémico? [Iodine nutrition in Asturian schoolchildren after 18 years of iodine prophylaxis with salt. Eradication of endemic goitre?]. *Endocrinología Y Nutrición*, 2001, 48(Suppl 2):14.
150. Santiago P et al. Prevalencia de déficit de yodo en la provincia de Jaén [Prevalence of iodine deficiency in Jaén province]. *Endocrinología Y Nutrición*, 2002, 49(Suppl 1):77–78.
151. Serna Arnaiz MC et al. [The prevalence of antithyroid antibodies in Lleida]. *Anales de Medicina Interna*, 2000, 17:62–66.
152. Maudeno Car AJ et al. Prevalencia de bocio y deficiencia de yodo en población escolar de una zona básica de salud tradicionalmente endémica [Prevalence of goitre and iodine deficiency in a school population from a traditionally endemic health area]. *Atencion Primaria*, 2001, 27:258–262.
153. Milakovic M et al. Urinary iodine and thyroid volume in a Swedish population. *Journal of Internal Medicine*, 2004, 255:610–614.

154. Zimmermann MB et al. Increasing the iodine concentration in the Swiss iodized salt program markedly improved iodine status in pregnant women and children: a 5-y prospective national study. *American Journal of Clinical Nutrition*, 2005, 82:388–392.
155. Karanfilski B. *Report on the activities implemented by the National Committee for Iodine Deficiency for 2005*. Macedonia, Ministry of Health, 2005.
156. Erdođan G et al. *2002 yili 20 bölge türkiye iyot durumu monitörizasyon projesi kesin raporu [Final report of the iodine status monitoring project in 20 regions in Turkey, 2002]*. Ankara, Ankara University, Medical School, Department of Endocrinology and Metabolism, 2003.
157. Vanderpump MPJ et al. The Incidence of thyroid disorders in the community – a 20-year follow-up of the Whickham survey. *Clinical Endocrinology*, 1995, 43:55–68.
158. Tahirovic H et al. Assessment of the current status of iodine prophylaxis in Bosnia and Herzegovina Federation. *Journal of Pediatric Endocrinology & Metabolism*, 2001, 14:1139–1144.
159. Lolic A and Lolic B. *Iodine deficiency*. Banja Luka, United Nations Children's Fund, 1999.
160. Knudsen N et al. Goitre prevalence and thyroid abnormalities at ultrasonography: a comparative epidemiological study in two regions with slightly different iodine status. *Clinical Endocrinology*, 2000, 53:479–485.
161. Hampel R et al. Continuous rise of urinary iodine excretion and drop in thyroid gland size among adolescents in Mecklenburg-West-Pomerania from 1993 to 1997. *Experimental and Clinical Endocrinology & Diabetes*, 2000, 108:197–201.
162. Rendl J, Juhran N, Reiners C. Thyroid volumes and urinary iodine in German school children. *Experimental and Clinical Endocrinology & Diabetes*, 2001, 109:8–12.
163. Liesenkotter KP et al. Small thyroid volumes and normal iodine excretion in Berlin school-children indicate full normalization of iodine supply. *Experimental and Clinical Endocrinology & Diabetes*, 1997, 105:46–50.
164. Doufas AG et al. Predominant form of non-toxic goiter in Greece is now autoimmune thyroiditis. *European Journal of Endocrinology*, 1999, 140:505–511.
165. Smyth PPA et al. Maternal iodine status and thyroid volume during pregnancy: correlation with neonatal iodine intake. *Journal of Clinical Endocrinology and Metabolism*, 1997, 82:2840–2843.
166. Panunzi C et al. Prevalenza di gozzo ed escrezione urinaria di iodio in un campione di bambini in età scolare della città di Roma [Goiter prevalence and urinary excretion of iodine in a sample of school-age children in the city of Rome]. *Annali dell'Istituto Superiore di Sanita*, 1998, 34:409–412.
167. Wiersinga WM et al. A survey of iodine intake and thyroid volume in Dutch schoolchildren: reference values in an iodine-sufficient area and the effect of puberty. *European Journal of Endocrinology*, 2001, 144:595–603.
168. National Institute of Nutrition Italy. *The health and nutrition of the refugee population in the Federal Republic of Yugoslavia*. World Health Organization, 1998.
169. Podoba J et al. The effectiveness of iodine prophylaxis of endemic goiter in Slovakia from the viewpoint of physical and ultrasonographic examinations of the thyroid gland. *Bratislavske Lekarske Listy*, 1995, 96:622–626.
170. Hess SY et al. Monitoring the adequacy of salt iodization in Switzerland: a national study of school children and pregnant women. *European Journal of Clinical Nutrition*, 2001, 55:162–166.
171. Karanfilski B et al. Correction of iodine deficiency in Macedonia. *Journal of Pediatric Endocrinology and Metabolism*, 2003, 16:1041–1045.
172. Karanfilski B et al. *Macedonia success IDD elimination story*. Skopje, Institute of Pathophysiology, Nuclear Medicine and Medical Faculty, 2004.
173. Erdogan G et al. Iodine status and goiter prevalence in Turkey before mandatory iodization. *Journal of Endocrinological Investigation*, 2002, 25:224–228.
174. Zimmermann MB et al. Toward a consensus on reference values for thyroid volume in iodine-replete schoolchildren: results of a workshop on interobserver and inter-equipment variation in sonographic measurement of thyroid volume. *European Journal of Endocrinology*, 2001, 144:213–220.

175. Gutekunst R et al. Ultrasound diagnosis of the thyroid. *Deutsche Medizinische Wochenschrift*, 1988, 113:1109–1112.
176. Gutekunst R, Martin-Teichert H. Requirements for goiter surveys and the determination of thyroid size. In: Delange F, Dunn J, Glinoe D, eds. *Iodine deficiency in Europe: A continuing concern*. New York, Plenum Press, 1993: 109–118.
177. Buchinger W et al. Thyrotropin and thyroglobulin as an index of optimal iodine intake: Correlation with iodine excretion of 39,913 euthyroid patients. *Thyroid*, 1997, 7:593–597.
178. Mostbeck A et al. The incidence of hyperthyroidism in Austria from 1987 to 1995 before and after an increase in salt iodization in 1990. *European Journal of Nuclear Medicine*, 1998, 25:367–374.
179. Delange F, Ermans AM. Le métabolisme de l'iode à la puberté [Iodine metabolism in puberty]. *Revue Française d'Etudes Cliniques et Biologiques*, 1967, 12:815–821.
180. Glinoe D et al. Regulation of maternal thyroid during pregnancy. *Journal of Clinical Endocrinology and Metabolism*, 1990, 71:276–287.
181. Delange F et al. Regional variations of iodine nutrition and thyroid function during the neonatal period in Europe. *Biology of the Neonate*, 1986, 49:322–330.
182. Delange F et al. Increased risk of primary hypothyroidism in preterm infants. *Journal of Pediatrics*, 1984, 105:462–469.
183. Rasmussen LB et al. Relations between various measures of iodine intake and thyroid volume, thyroid nodularity, and serum thyroglobulin. *American Journal of Clinical Nutrition*, 2002, 76:1069–1076.
184. Pedersen KM et al. Amelioration of some pregnancy-associated variations in thyroid function by iodine supplementation. *Journal of Clinical Endocrinology and Metabolism*, 1993, 77:1078–1083.
185. Laurberg P et al. Iodine intake and thyroid disorders in Denmark. Background for an iodine supplementation programme. In: Delange F et al., eds. *Elimination of iodine deficiency disorders (IDD) in Central and Eastern Europe, the Commonwealth of Independent States, and the Baltic States*. Geneva, World Health Organization, 1998: 31–42.
186. Nohr SB, Laurberg P. Opposite variations in maternal and neonatal thyroid function induced by iodine supplementation during pregnancy. *Journal of Clinical Endocrinology and Metabolism*, 2000, 85:623–627.
187. Mikelsaar RV, Viikmaa M. Neonatal thyroid-stimulating hormone screening as an indirect method for the assessment of iodine deficiency in Estonia. *Hormone Research*, 1999, 52:284–286.
188. Barrere X et al. Determinants of thyroid volume in healthy French adults participating in the SU.VI.MAX cohort. *Clinical Endocrinology*, 2000, 52:273–278.
189. Liesenkotter KP et al. Earliest prevention of endemic goiter by iodine supplementation during pregnancy. *European Journal of Endocrinology*, 1996, 134:443–448.
190. Koutras DA et al. Endemic goiter in Greece – clinical and metabolic effects of iodized salt. *Journal of Clinical Endocrinology and Metabolism*, 1968, 28:1651–1656.
191. Mengreli C, Yiannakou L, Pantelakis S. The screening programme for congenital hypothyroidism in Greece: evidence of iodine deficiency in some areas of the country. *Acta Paediatrica*, 1994, 394:47–51.
192. Szabolcs I et al. Comparative screening for thyroid disorders in old age in areas of iodine deficiency, long-term iodine prophylaxis and abundant iodine intake. *Clinical Endocrinology*, 1997, 47:87–92.
193. Mezosi E et al. Prevalence of iodine deficiency and goitre during pregnancy in East Hungary. *European Journal of Endocrinology*, 2000, 143:479–483.
194. Laurberg P et al. Iodine intake and the pattern of thyroid disorders: A comparative epidemiological study of thyroid abnormalities in the elderly in Iceland and in Jutland, Denmark. *Journal of Clinical Endocrinology and Metabolism*, 1998, 83:765–769.
195. Bonar BD et al. Hypothyroidism and aging: the Rosses' survey. *Thyroid*, 2000, 10:821–827.
196. Smyth PP. Variation in iodine handling during normal pregnancy. *Thyroid*, 1999, 9:637–642.

197. Martino E et al. Endemic goiter and thyroid function in Central-Southern Sardinia – report on an extensive epidemiologic survey. *Journal of Endocrinological Investigation*, 1994, 17:653–657.
198. Vermiglio F et al. Maternal hypothyroxinemia during the first half of gestation in an iodine deficient area with endemic cretinism and related disorders. *Clinical Endocrinology*, 1995, 42:409–415.
199. Vermiglio F et al. Increased risk of maternal thyroid failure with pregnancy progression in an iodine deficient area with major iodine deficiency disorders. *Thyroid*, 1999, 9:19–24.
200. Costante G et al. The statistical analysis of neonatal TSH results from congenital hypothyroidism screening programs provides a useful tool for the characterization of moderate iodine deficiency regions. *Journal of Endocrinological Investigation*, 1997, 20:251–256.
201. Calaciura F et al. Childhood IQ measurements in infants with transient congenital hypothyroidism. *Clinical Endocrinology*, 1995, 43:473–477.
202. Calaciura F et al. Subclinical hypothyroidism in early childhood: a frequent outcome of transient neonatal hyperthyrotropinemia. *Journal of Clinical Endocrinology and Metabolism*, 2002, 87:3209–3214.
203. Vermiglio F et al. Defective neuromotor and cognitive ability in iodine-deficient schoolchildren of an endemic goiter region in Sicily. *Journal of Clinical Endocrinology and Metabolism*, 1990, 70:379–384.
204. Fenzi GF et al. Neuropsychological assessment in schoolchildren from an area of moderate iodine deficiency. *Journal of Endocrinological Investigation*, 1990, 13:427–431.
205. Vitti P et al. Mild iodine deficiency in fetal neonatal life and neuropsychological performances. *Acta Medica Austriaca*, 1992, 19:57–59.
206. Krzyczkowska-Sendrakowska M et al. Iodine deficiency in pregnant women in an area of moderate goiter endemicia. *Polish Journal of Endocrinology*, 1993, 44:367–372.
207. Ratajczak R, Rybakowa M, Tylek-Lemanska D. A mass screening program for congenital hypothyroidism as the best method of monitoring iodine deficiency. *Pediatrica Polska*, 1994, 6:459–461.
208. Delange F. Neonatal screening for congenital hypothyroidism: results and perspectives. *Hormone Research*, 1997, 48:51–61.
209. Petersen K et al. Thyroid disease in middle-aged and elderly Swedish women: thyroid-related hormones, thyroid dysfunction and goitre in relation to age and smoking. *Journal of Internal Medicine*, 1991, 229:407–413.
210. Baltisberger BL, Minder CE, Bürgi H. Decrease of incidence of toxic nodular goiter in a region of Switzerland after full correction of mild iodine deficiency. *European Journal of Endocrinology*, 1995, 132:546–549.
211. Erdogan MF et al. Endemic goiter, thiocyanate overload, and selenium status in school-age children. *Biological Trace Element Research*, 2001, 79:121–130.
212. Aydin K et al. Iodine and selenium deficiency in school-children in an endemic goiter area in Turkey. *Journal of Pediatric Endocrinology & Metabolism*, 2002, 15:1027–1031.
213. Siklar Z et al. Borderline congenital hypothyroidism in the neonatal period. *Journal of Pediatric Endocrinology & Metabolism*, 2002, 15:817–821.
214. Yordam N et al. Screening for congenital hypothyroidism in Turkey. *European Journal of Pediatrics*, 1995, 154:614–616.
215. Barnett CA et al. Inadequate iodine intake of 40% of pregnant women from a region of Scotland. *Journal of Endocrinological Investigation*, 2002, 25(Suppl 7):90.
216. Ray M et al. Audit of screening programme for congenital hypothyroidism in Scotland 1979–93. *Archives of Disease in Childhood*, 1997, 76:411–415.
217. WHO and FAO. *Diet, nutrition and the prevention of chronic diseases. Report of a Joint WHO/FAO Expert Consultation*. Geneva, World Health Organization, 2003 (WHO Technical Report Series, No. 916).

Every European nation endorsed the goal of eliminating iodine deficiency at the World Health Assembly in 1992. Globally, great progress has been made since that time. However, the World Health Organization's (WHO) European Region has been identified as having the lowest coverage of salt iodization of all the regions.

The primary purpose of this document is to review the current extent of iodine deficiency in the European Union (EU) Member States, applicant countries and those in the European Free Trade Association (EFTA). Its ultimate goal is the mobilization of all European governments to implement and monitor sustainable programmes to control and prevent iodine deficiency in their populations. Part one of the report gives the background, historical context and global strategies. The second part addresses the main issues related to iodine deficiency: its magnitude, the public health significance, and the health and economic consequences of iodine deficiency, and outlines the current strategies being used to reach the goal of iodine deficiency elimination. In the third part, the focus is on the iodine deficiency situation in 40 of the countries of Europe. The final part highlights the need for sustainable programmes and makes recommendations to help achieve this.

The report concludes that iodine deficiency remains a public health concern in Europe; the health, social and economic consequences of this are well established. Salt iodization remains the recommended strategy for eliminating iodine deficiency. Foremost among the challenges are (i) to strengthen monitoring and evaluation of national programmes for the prevention and control of iodine deficiency in the countries of an enlarged EU, including the surveillance of the iodine status of national populations; (ii) to ensure the sustainable implementation of USI in all countries of the enlarged EU, by harmonizing relevant legislation and regulations; (iii) to ensure adequate quality control and quality assurance procedures to strengthen the monitoring of foods fortified with iodine, especially salt iodization, from the producer to the consumer; (iv) to increase the awareness of political leaders and public health authorities on the public health and social dimensions of iodine deficiency and the need to implement and sustain programmes for its control; (v) to educate the public on the need to prevent iodine deficiency by consuming iodized salt, and thereby also increase consumer awareness and demand; and (vi) to consider alternative iodine supplementation for the most susceptible groups – pregnant women and young infants – where there is insufficient iodized salt and to take into account public health policies to reduce salt consumption.

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