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Radiodosimetry and preventive measures in the event of a nuclear accident

*Proceedings of an international symposium
organized by the
Polish Society of Nuclear Medicine
in co-operation with the
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and held in Cracow, Poland, 26–28 May 1994*



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FOREWORD

An international symposium on Radiodosimetry and Preventive Measures in the Event of a Nuclear Accident was held in Cracow, Poland, from 26 to 28 May 1994. The symposium was organized by the Polish Society for Nuclear Medicine, and co-sponsored by the IAEA. Over 40 experts from Belarus, Latvia, Lithuania, Germany, Poland, the Russian Federation, Sweden and Switzerland participated. The aim of the Symposium was to review models of iodine kinetics used in the calculation of internal radiation doses to the thyroid after the Chernobyl accident, to discuss internal and external radiation dose to the thyroid in terms of risk of thyroid cancer, and to present data on the incidence rate of thyroid cancer in the selected iodine deficient area in Poland. A part of the symposium was dedicated to the physiological basis of iodine prophylaxis and emergency planning for a nuclear accident. Recommendations of the IAEA on preventive measures in the event of a nuclear accident were also addressed. These proceedings contain the full text of the eight invited papers presented at the symposium.

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SUMMARY

The International Symposium held in Cracow from 26 to 28 May 1994 during the Congress of the Polish Society of Nuclear Medicine covered the following topics: reconstruction of radiation doses to the thyroid due to the Chernobyl accident, evaluation of the risk of thyroid cancer in low radiation areas after the accident, and iodine prophylaxis in the event of a nuclear accident.

In the event of a nuclear accident, radioiodine nuclides are the main source of internal and external exposure of the thyroid gland. The most effective and precise method of measurement of ^{131}I content in the thyroid is a direct measurement of its activity at the initial time after the accident. However, for many reasons, this technique was not widely available in many areas after the accident.

Another method of measurement is a dynamic dosimetric model based on an estimation of radioiodine intake to the human body from air, water including rainwater, and milk and other foodstuffs. Dosimetry performed after the Chernobyl accident was usually based on the Johnson formula, whereby radiation dose to the thyroid depends reciprocally and exponentially on the daily iodine intake, assuming, however, the 'reference man' daily iodine intake, i.e. $200\ \mu\text{g}/\text{day}$.

This is very far from the actual values observed in areas of iodine deficiency in the majority of European countries exposed to radiation following the accident. Two papers presented at the Symposium focused on this problem (Zvonova, I.A., St. Petersburg; Olko, P., Cracow) leading to the conclusion that calculation of the thyroid radiation dose in areas of iodine deficiency was underestimated and should be recalculated. An example is the Cracow region, where actual values of daily iodine intake were obtained from the results of a nationwide study on iodine deficiency.

To determine the true committed equivalent radiation dose to the thyroid after a nuclear accident, it would be necessary to use other techniques also, such as analysis of ^{137}Cs deposition and reconstruction of the meteorological conditions at the time of a nuclear accident. It is also possible that, in the future, evaluation of chromosomal abnormalities due to irradiation could help in reconstructing the radiation dose to the thyroid.

Reconstruction of radiation doses to the thyroid after a nuclear accident is crucial for evaluation of the risk coefficient of thyroid cancer due to irradiation. Ionizing radiation is a very well documented risk factor for thyroid cancer. However, at low doses of radiation, especially due to uptake of ^{131}I , a real risk coefficient of thyroid cancer is still unknown and information on potential carcinogenesis is scanty or conflicting (Hall, P.E., Stockholm).

Four years after the Chernobyl accident, a sharp increase in the rate of thyroid cancer in the Gomel region in Belarus has been observed and this phenomenon has again raised the problem of the risk coefficient of thyroid cancer due to irradiation. It is as yet impossible to plot a dose-response curve and to calculate the risk factor of thyroid cancer, which leads one to believe that other factors apart from radiation may play a role in carcinogenesis. This problem is taken up in one paper (Szybinski, Z., Cracow), leading to the conclusion that the best approach is to set up a standardized population based thyroid cancer register to monitor an incidence rate and to start a 'case control study' in the area of interest in order to detect all ethnological factors.

Evaluation of the risk coefficient of thyroid cancer due to irradiation after a nuclear accident is crucial for the creation of a system of protective measures in the event of such an accident. Two papers are dedicated to iodine prophylaxis in the event of a nuclear accident, one on the physiological basis for these countermeasures (Gembicki, M., Poznan) and one on its possible side effects (Höffken, H, Marburg). One paper (Niewodniczanski, J., Polish Atomic Agency) discusses the nuclear safety situation in Poland and a system of protective measures in the event of a nuclear accident.

On an international level, policy for intervention in the event of a nuclear accident is presented by Hedemann Jensen, Denmark and Crick and Gonzalez of the IAEA. The paper presents optimized intervention levels in the event of a nuclear accident for developing and developed countries for a wide range of accident situations. The measurable quantities include dose rate and activity concentrations in air, in foodstuffs and on ground surfaces.



INTRODUCTION

THYROID DOSIMETRY AFTER THE CHERNOBYL ACCIDENT AND THYROID CANCER IN IODINE DEFICIENT AREAS

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Of the radionuclides generated from ^{235}U and ^{239}Pu in a core of the nuclear reactor, radioiodines particularly ^{131}I , is the most significant in view of its huge quantities, easy dispersion and cumulation in the human thyroid in case of a nuclear accident. After nuclear accident in Chernobyl 20-50 million Ci of ^{131}I was released. Depending on the dose adsorbed to the thyroid, ^{131}I can cause a late appearance of a thyroid nodule or cancer and/or thyroid destruction leading to hypothyroidism. Thyroid irradiation may origin from two sources: external cumulative radiation mainly of gamma type and internal related to ^{131}I cumulation. So far most information on the risk factors of the thyroid cancer due to is related to from external radiation, but there is no scientific basis to believe that internal radiation cannot induce the thyroid cancer. Thyroid dosimetry after Chernobyl accident in near and far field is essential for calculation of the thyroid cancer risk coefficient due to radiation.

Internal thyroid dose used in radiation protection according to ICRP recommendation is a "committed thyroid dose equivalent" (ctde). Any calculation of the ctde must be developed according to a model for the kinetics of iodine whose origin dates back more than 30 years. The most recent variant of the multicompartiment dilution type kinetic model of iodine was created by Johnson and developed effectively by Turai and this model is a gold standard for dosimetry of radiation due to Chernobyl accident. Classic Johnson's formula assumed that parameters reflecting the transfer of iodine from the inorganic pool into the bladder are fairly independent of age, weight and are not greatly affected by changes in the dietary intake of iodine. This value approximates the average values summarised by ICRP's Task Group on Reference Man (70kg and 200 μg daily iodine intake) and Reference Female (58 kg and 166 μg respectively). This classic Johnson's formula has been generally accepted and used in all measurements performed after Chernobyl accident in the near and far field as well.

However, during last 10 years new information about daily iodine in the majority of European countries has been provided. Nation-wide study on iodine deficiency in Poland performed in 1992/1993 discovered that in the Sudeten and Carpathian areas and in the north-eastern part of Poland 40-80% of children 6-13 years old had urine iodine concentrations within 20-50 $\mu\text{g/l}$ and this region was classified as a moderate iodine deficient and an endemic goiter area. Kraków and Nowy Sącz districts lay in the Carpathian area, a one of the oldest endemic goiter areas in Europe. In this area concentration of iodine in potable water is within 0-3 $\mu\text{g/l}$, prevalence of goiter is 40-60% in the schoolchildren subgroup, concentration of iodine in the urine of over 50% of schoolchildren is within 20-50 $\mu\text{g/l}$ and this situation still persists because iodine prophylaxis was suspended in Poland in 1980 and up till now new obligatory model of prophylaxis has not been implemented.

Ionising radiation is a very well documented risk factor for the thyroid cancer, however, at the low dose radiation especially due to ^{131}I , a real risk coefficient is still unknown. In 1992 a report on sharp increase of number of thyroid cancer in children below 16 yr. of age in Homel region Bielorrussia, where thyroid dose was within the range of 1-5 Sv, was published. This finding raises a question about incidence rate in Poland - region of low dose radiation. However interpretation of any findings concerning of incidence of thyroid cancer must be caution because iodine insufficiency itself is a very well documented risk factor for the thyroid cancer.

In our Symposium two districts will be analysed: Kraków and Nowy Sącz. There are 2,0 mln people in this area, representing one of the deepest iodine deficiency in the country, and after Chernobyl accident it was included in the list of the most contaminated districts. In this area thyroid cancer register has been developed since 1976. These circumstances provided us with excellent opportunity to recalculate dose to the thyroid after Chernobyl in children and to answer the very important question whether thyroid cancer incidence rate increased in this part of the country.

The second task is to present the model of preventive measures in the case of nuclear accident in this part of Europe when in the south-eastern neighbouring countries 25 nuclear power units are still operating and at least 1/3 of them represent outdated 1. To make a review of the mathematical models being used in calculation of radiation dose for thyroid due to Chernobyl and risk factor of thyroid cancer in children exposed to low level radiation in iodine deficient area.

2. What was the real radiation dose to the thyroid after Chernobyl accident in Poland in iodine deficient area?
3. What is real risk for thyroid cancer - at low dose exposure i.e. "Far field"?
4. What kind of preventive measures in case of a nuclear accident should be undertaken in Poland - facing the real nuclear power status in this part of Europe?

I express the hope that our Symposium provides a chance for discussing many methodological problems, very important for nuclear safety in this part of Europe.

I wish everybody highly stimulating discussions.

TABLE I. NUCLEAR POWER STATUS IN THE COUNTRIES BORDERING UPON-EASTERN POLAND.

(According to International Data file IAEA Bulletin 4/1993)

Country	Nuclear power units		Distance between Warsaw and capitals (Km)
	in operation	under construction	
Czech Republic	4	2	500
Slovak Republic	4	4	540
Ukraine	15	6	650
Lithuania	2	1	380
Total	25	13	

RADIODOSIMETRY AND THYROID CANCER

(Session I)

Chairmen

Z. Szybiński, Poland
P.E. Hall, Sweden

**THE PRINCIPLES OF RADIOIODINE DOSIMETRY
FOLLOWING A NUCLEAR ACCIDENT**

XA9643027

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Abstract

Based upon the experience of radioiodine dosimetry after the Chernobyl accident main principals of radioiodine measurements and dosimetry in thyroid glands of population in case of a radiation accident are discussed in the report.

For the correct dose estimation following the radioiodine measurement in the thyroid one should know the "history" of radionuclide intake into the body of a contaminated person. So a measurement of radioiodine thyroid content should be accompanied by asking questions of investigated persons about, their life style and feeding after a nuclear incident. These data coincidently with data of radionuclides dynamic in the air and food (especially in milk products) are used for the development of radioiodine intake model and then for thyroid dose estimation.

The influence of stable iodine prophylaxis and other countermeasures on values are discussed in dependence on the time of its using.

Some methods of thyroid dose reconstruction used after the Chernobyl accident in Russia for a situation of thyroid radioiodine measurements lacking in a contaminated settlement are presented in the report.

1. INTRODUCTION.

The decades of using nuclear energy in the world showed that the radioiodine isotopes were the most dangerous factor of radiation influence on people among all the fission products. This is caused by the high yields of radioiodine nuclides during nuclear fission, its high volatility and high migration ability throw biological chains from fallout to pasture then to cow, then to milk and at last to man.

Radioiodine nuclides are the source of internal exposure of thyroid gland where they are concentrated after intake into human body and from the greatest organ absorbed dose. Furthermore, radioiodine nuclides deposited on soil are responsible for the major part of external exposure during the first days following a radioactive contamination of territories after a nuclear reactor accident or nuclear explosion. [1, 2].

Usually the exposure of thyroid to radioiodine is the most dangerous consequence of nuclear reactor accident. So at any radiation accident the problem of thyroid dose estimation for exposed people arises. There are several possibilities for estimating a thyroid dose following a release of radioiodine to the environment.

The first one is based on measurements of ^{131}I content in the thyroid of people if such measurements were organized and performed at initial post-accidental time.

Another way is model estimation of radioiodine intake to human body if data on radioiodine concentration in air, milk and other food-stuffs are available as well as data on ration composition for local population.

There may be a situation when data on direct radioiodine measurements in the human thyroid and data on environmental contamination are very limited in an area of interest. In this case it is necessary to investigate regularities of dose formation and correlations of received thyroid doses with various environmental parameters in some other region similar by conditions of contamination to the studied one and where such data were found.

All these situations were realized after the accident at a nuclear reactor at the Chernobyl NPP. Let us discuss the main principals and difficulties of thyroid dose estimations using the experience of such work on contaminated territories of Russia after the Chernobyl accident.

2. REQUIREMENTS TO THE MEASUREMENTS OF ^{131}I IN THYROID.

In contrast to the radionuclide diagnostics for medical purposes, radioiodine measurements in human thyroid after a nuclear accident are to be performed over the background of human body contamination by other radionuclides. For example, spectrometric measurements of inhabitants from the contaminated areas of Ukraine, Belorussia, Russia made at the Institute of Radiation Hygiene, St Petersburg during May-June of 1986 (Lebedov O.N. et al [3]) showed the presence of ^{131}I , ^{134}Cs , ^{137}Cs , ^{103}Ru , ^{106}Ru , sometimes ^{132}Te , ^{140}Ba , ^{95}Zr radionuclides. Special control upon surface contamination of skin and clothes of the measured people should be provided to avoid mistakes in results during the measurements.

To reduce the influence of extrathyroid activity and other negative factors on the ^{131}I measurements in the thyroid, the technique of a measurement should satisfy the following requirements:

- the spectrometric regime of measurements is desirable;
- the detector should be placed into a leaden collimator;
- for the estimation of the contribution of extrathyroid activity the measurement of the thyroid should be accompanied with the measurement of a thigh (in the position 5 cm over the knee where its muscles mass and circumference close to the neck);
- it is necessary to choose an accommodation for measurements with minimal background;
- cleanness of body skin and people's clothes should be thoroughly controlled by the staff.

Equipment for radionuclide diagnostics is the most suitable for such measurements. But sometimes it is necessary to organize thyroid mass radiometry among people living in contaminated territories with nonspecific transportable instruments. In this case special calibration procedure of the devices is necessary with standard solution of ^{131}I and phantom of neck. Calibration may be also performed by comparison of the measurement results of the same persons with a calibrated radiodiagnostic equipment and with used nonspecific instruments.

The ^{131}I -I activity in thyroid is calculated on the equation:

$$G = (N_n - 0.9 N_t) \cdot K, \text{ Bq}, \quad (1)$$

where N is the count rate from the neck (counts/s); N_t is the count rate from the thigh (counts/s) ; K is the calibration coefficient Bq/s/counts). Numerical coefficient appeared from experimental investigations in radionuclide diagnostic with ^{131}I and when devising method of ^{131}I -I measurement after Chernobyl accident [4].

During a measurement the following information on the investigated person should be obtained and recorded: name, sex, year of birth, residence, the length of residence in the zone of radioactive contamination, stay in the open air during the first days of the accident, data on consumption of locally-produced milk and green-stuff, consumption of stable iodine drugs. Data on location and time of the cattle pasture may be useful too. Such questionnaire can be modified according to local

life style of inhabitants and season of an accident . Such questionnaire allows to determine the dose in thyroid of the person with the least error.

Many problems of following interpretation of the measurement results would disappear if all the mentioned requirements concerning thyroid measurements procedure had been fulfilled.

3. DOSIMETRIC MODEL

a. Data for dosimetric estimations.

Individual thyroid dose of a person can be estimated by the equation:

$$D = K E_{ef} \int_{1/m} R(t) dt , \quad (2)$$

where $R(t)$ - function of radioiodine content in the thyroid (Bq);

E_{ef} - effective energy (MeV transf⁻¹);

m - thyroid mass (g);

K - the system coefficient,

$$K = 13.8 \mu\text{Sv g d}^{-1} \text{ transf Bq}^{-1} \text{ Mev}^{-1}.$$

If the retention function of radioiodine in the thyroid after single intake of activity is described by a single exponent then the variation of radioiodine content in the thyroid with time can be shown by the equation:

$$\frac{dR(t)}{dt} = -\lambda R(t) + f_2 \ln(t) , \quad (3)$$

where: λ - the effective constant of radioiodine excretion from the thyroid (d⁻¹)
 λ is equal to the sum of radioactive decay (λ_d) and biological (λ_b) constants : $\lambda = \lambda_d + \lambda_b$.

This parameter may be also presented by the half-time of radioactive decay (T^*) and biological (T_b) half-time of radioiodine excretion from the thyroid:

$$\lambda = \ln 2 \frac{T_b + T^*}{T_b T^*}$$

f_2 - the fraction of radioiodine entered to the body that is deposited in the thyroid;

$In(t)$ - the function of radioiodine intake into the human body.

We can see that while counting the dose the parameters are used dependant on nuclear decay characteristics (E_{ef} , T^*), from biological characteristics of iodine metabolism in the human body.

(m , T_b , f_2) from ecological circumstances of radioactive contamination of areas, that form iodine intake function into human body ($In(t)$).

In table I there are shown nuclear decay characteristics for the most important radioiodine nuclides from the point of view of their radiation danger in accident situations that occur with fission products. There are the energy and the yield of the main type of radiation, the half-time of decay, the effective energy for the thyroid of adults [5, 6].

Iodine metabolism parameters, especially mass of thyroid gland and biological half-time of iodine's secretion with thyroid hormones, differ with age. In table II there are shown the values of these parameters, received due to analysis and generalization of literary data [7].

While choosing numerical definitions of iodine metabolism parameters it is important to remember that thyroid mass, fraction of iodine uptake in the thyroid f_2 and in the lesser measure T_b , depend greatly on the average daily intake of stable iodine with food ration of inhabitants. According to the analysis of available information on this problem in [8] the fraction of iodine in thyroid f_2 for people with the average daily iodine consumption In ($\mu\text{g}/\text{day}$) gets approximated by the equation:

$$f_2 = \frac{85}{In + 85} \quad (5)$$

TABLE I. DECAY CHARACTERISTICS OF RADIOIODINE NUCLIDES [5, 6].

Mass number	Half-time of decay,	Main energies (meV) and yield (Bq s) ⁻¹		Effective energy, MeV transf ⁻¹
		β	γ , X-ray	
129	1.57.10 ⁷ y	0.0049 (1.00)	0.0050 (0.79)	0.068
		0.0033 (0.46)	0.0298 (0.37)	
		0.0042 (0.25)	0.0295 (0.20)	
		0.00081 (1.39)		
131	8.04 d	0.191 (0.894)	0.364 (0.821)	0.20
132	2.3 h	0.242 (0.128)	0.668 (0.987)	0.56
		0.422 (0.190)	0.773 (0.762)	
		0.608 (0.127)		
		0.841 (0.176)		
133	20.8 h	0.441 (0.835)	0.530 (0.863)	0.44
134	0.88 h	0.463 (0.311)	0.847 (0.954)	0.70
		0.583 (0.160)	0.884 (0.653)	
		0.687 (0.111)		
		0.968 (0.118)		
135	6.61 h	0.358 (0.218)	1.132 (0.225)	0.42
		0.535 (0.241)	1.260 (0.286)	

Thyroid mass and f_2 react equally upon the variations of the level of stable iodine in diet, satisfying the correlation:

$f_2 / m = (0.018 \pm 0.003)$ [8]. These relationships may be useful during thyroid doses estimations in inhabitants of different areas with different level of daily stable iodine intake.

Table II presents also the thyroid dose equivalent values per unit activity of ¹³¹I deposited in thyroid (h_1) or intaken into the body (h_2). The h_2 value was calculated with two-exponential accumulation-excretion function of the iodine in the gland [9] taking into account the fact that the ratio of a iodine thyroid uptake

TABLE II. AGE-DEPENDENT PARAMETERS OF IODINE METABOLISM AND DOSIMETRY IN HUMAN BODY.

Age years	Thyroid mass, g	T_b , days	h_1 , $\mu\text{Sv/Bq}$	h_2 $\mu\text{Sv/Bq}$
New-born	1.6	16	13.3	4.00
0.08-0.50	1.7	16	12.6	3.77
0.50-0.99	1.8	18	12.3	3.70
1-2	2.5	20	9.2	2.75
3-4	3.8	25	6.4	1.92
5	4.8	30	5.3	1.58
6-7	6.5	35	4.0	1.21
8-9	8	40	3.3	1.00
10	9	50	3.1	0.92
11	9.7	55	2.9	0.87
12	10.5	60	2.7	0.81
13-14	12	65	2.4	0.71
15	13	70	2.2	0.66
Adults	20	100	1.5	0.53

to its mass is constant for different regions: $f_2/m = 0.018$ [9]. The effective energy absorbed in thyroid per one decay of the radionuclide is formed basically by beta-radiation and therefore does not practically depend on the age. Differences in the effective energy of newborn and adults are less than 10 % for all radioiodine nuclides.

Under the deposition of the same activity in thyroid of children and adults, thyroid dose for new-born children appears to be 11 times higher than for adults. Under the intake of the same activity into the body the thyroid dose in the newborn is as 8.5 values for adults.

b . Intake function

Determination of radioiodine intake function into human body is the main question when thyroid dose should be estimated based on a single measurement of $^{131}\text{-I}$ content in the thyroid.

Short-lived nuclides ($^{132}\text{-I}$, $^{133}\text{-I}$, $^{134}\text{-I}$, $^{135}\text{-I}$) enter into a human body during the first hours after an accident. However due to the short half-life the most part of them could not be measured in thyroid. For example, 4 to 7 days after the

Chernobyl accident in the thyroids of the inhabitants evacuated on 27.04.86 from the town of Pripyat there could be measured only $^{131}\text{-I}$, $^{133}\text{-I}$ and the predecessor of $^{132}\text{-I}$ - ^{132}Te . By alimentary way with milk and foods there came only $^{131}\text{-I}$ that could be measured in the inhabitants of the most contaminated areas up to the middle of June, 1986.

The $^{131}\text{-I}$ intakes into human body with inhaled air during the first days after the accident, and with food products, predominantly with milk, at subsequent weeks. Calculations show that under conditions of permanent residence in contaminated area, consumption of local food, absence of protective actions, the contribution of $^{131}\text{-I}$ to the total thyroid dose is the greatest among all nuclides of I, up to 95-98%. Only 2 to 7% of this value occur from inhalation of $^{131}\text{-I}$ [1,7].

In the cases when protective actions aimed at interruption of radionuclides intake by people were used ratio of inhaled and alimentary induced fractions of thyroid dose changes apart from the rise of the inhaled fraction. In the case of inhalation of fresh fission products without consumption of contaminated food, the absorbed thyroid dose from all short-lived iodine isotopes approximately is equal to the dose from $^{131}\text{-I}$.

In every concrete case for setting an intake function it is necessary to analyse carefully the whole available information upon the dynamics of area radioactive contamination, upon the changes in iodine concentration in air and food stuffs, upon the behaviour regime and diet of local population. When there is enough reliable information upon all the named items, we can create the model of ^{131}I intake by different ways of radioiodine intake (with air, milk, water and other components of diet). If we do not have enough information, then we should simplify the model, avoid detalization and base upon the main regularities of radioiodine intake into body. Such way was chosen in Russia after the Chernobyl accident for thyroid dose estimation for inhabitants of contaminated areas.

After the Chernobyl accident it was found that $^{131}\text{-I}$ concentration in the air decreased with half-time of 0.5 to 0.6 days [10, 11, 12]. At the same time $^{131}\text{-I}$ concentration in milk increased reaching the maximum in 2-5 days and stayed without changing for 10 to 15 days. Then $^{131}\text{-I}$ concentration in milk reduced with the effective half-life about 5 days [7, 12].

Taking into account all the patterns of radioiodine intake to inhabitants of contaminated locations it was hypothesised that the daily $^{131}\text{-I}$ intake was constant

during first 15 days, and then decreased with the half-time of 5 days in conjunction with the concentration in milk. In the first days, when milk contamination gradually increased, inhalation component and consumption of green-stuff supplemented the intake with milk. So intake rate in equation (3) was described as:

$$I(t) = I_0, \quad (\text{Bq/day}) \quad \text{for } t \leq \tau = 15 \text{ days}; \quad (6)$$

$$I(t) = I_0 \cdot \exp(-k(t-\tau)), \quad (\text{Bq/day}) \quad \text{for } t > \tau = 15 \text{ days}, \quad (7)$$

where k is the effective constant of the ^{131}I concentration decrease in milk,

$$k = 0.14 \text{ day}^{-1}.$$

The solution of equation (3) under conditions (6) and (7) has the form :

$$G(t) = \frac{I_0 f_2}{\lambda} (1 - e^{-\lambda t}) \quad \text{under } t \leq \tau, \quad (8)$$

$$G(t) = \frac{I_0 f_2}{\lambda} (1 - e^{-\lambda t}) e^{-\lambda(t-\tau)} + \frac{I_0 f_2}{k - \lambda} [e^{-\lambda(t-\tau)} - e^{-k(t-\lambda)}] \quad (9)$$

under $t > \tau$

We used equation (8) and (9) for calculation of I_0 and of the total ^{131}I intake into the body of the investigated person I_Σ before the moment of measurement t :

$$I_\Sigma = I_0 \tau + \frac{I_0}{k} [1 - e^{-k(t-\tau)}], \quad \text{Bq} \quad (10)$$

If the ^{131}I incorporation continued also after the measurement, the total intake into the body I_∞ was determined in the following way :

$$I_\infty = I \left(\tau + \frac{1}{k} \right), \quad \text{Bq} \quad (11)$$

Expressions (10) and (11) are true, when the $^{131}\text{-I}$ intake into the body $I(t)$ satisfy conditions (6) and (7) and are not deformed by protective measures. When stable iodine prophylaxis was performed, or consumption of local milk was interrupted or people were relocated from the accident zone at the moment θ the accumulation of $^{131}\text{-I}$ ceased. This is expressed formally by addition of condition:

$I(t > \theta) = 0$, where θ is the time of beginning the protective measure. The corresponding analytic solutions analogous to (8), (9) and (10).

The proposed calculation model is based on minimum of actual data and does not specify the role of separate pathways of $^{131}\text{-I}$ incorporation into the body, because such excess specification would introduce more errors than refinements in the presence of many uncertainties in the available data.

In practice, we applied the following algorithm for calculating the thyroid dose based on the $^{131}\text{-I}$ measurement in the thyroid $G(t)$ at t days after the radioactive fallout:

- a) calculation of daily $^{131}\text{-I}$ intake into the body I_0 from equations (8) or (9);
- b) calculation of the total $^{131}\text{-I}$ incorporation into the body I_{Σ} according to (10) or (11) in dependence of incorporation duration;
- c) calculation of expected dose equivalent in thyroid H (Sv) by multiplication of I_{Σ} or I_{∞} by the dose coefficient h_2 for the corresponding age from Table II.

If the $^{131}\text{-I}$ intake into the body had been artificially ceased at the moment θ , the calculation of I_0 and I_{Σ} was modified as it had been described above.

4. APPROACH FOR THYROID DOSE RECONSTRUCTION

For radiation accidents such situations were frequent when during the first period of accident's development necessary measurements of people and of environment were not carried out. In such case it is necessary to use different methods of dose reconstruction, in particular for $^{131}\text{-I}$ irradiation.

One of the ways of the thyroid dose reconstruction is creating ecological and metabolic models, which describe radioiodine's behaviour in nature and in human organism from the time of the accident release up to thyroid accumulation. Such models are verifying by experimental data on different

stages of the radioiodine migration in environment atmosphere transfer, fall outs upon plants and soil, transfer from plants to cattle and then with milk to human body Discussion of these models gets out of the limits of this article

Another way is the analysis of the relation between thyroid dose and parameters of environmental contamination in the areas, where measurements of ^{131}I content in thyroid were performed in time and where there are enough information for basing the radioiodine intake function Next step is transfer of the exposed regularities to other territories and settlements, which had similar conditions of radioactive contamination, but where direct thyroid measurements of inhabitants were not carried out Such a way was chosen while reconstructing thyroid doses in Russian contaminated areas, where measurements of the ^{131}I in the thyroid of inhabitants were not performed in the critical period May-June 1986 This problem was solved in two stages first, mean dose values were determined for a given settlement, then individual doses were estimated in this settlement [3]

The first regularity used for dose reconstruction was the real dependence of thyroid dose on age, realised in contaminated settlements

It was found that these relationships differed largely in town and village populations Table III presents results of this analysis for six age groups under a year 1-2, 3-6, 7-11, 12-17 years, adults

TABLE III. RATIO OF THE MEAN DOSE IN THE SPECIFIED AGE INTERVAL TO THE MEAN DOSE IN ADULTS IN SETTLEMENTS OF TOWNS AND VILLAGE.

Settlement	Age, years					
	<1	1-2	3-6	7-11	12-17	≥ 18
Town	13± 3	9± 4	6± 2	5± 0.8	0.5± 1.0	1
Village	5±3	5± 2	3± 1	2.2± 1.0	3± 2	1

An average value for adults was taken as unity for each type of settlement The observed differences are attributable not only to age-related differences in iodine metabolism, but also to social factors and nutrition habits of town and village inhabitants such as greater milk consumption by village inhabitants, higher incidence of breast-feeding of babies in villages than in town, greater level of fodder additions to cattle diets on the collective farms that resulted higher radionuclide concentration in privately produced milk than in milk from collective farms All

these factors caused an average dose among adults of villages 1.5 to 2.5 times higher than in the towns for a given level of soil contamination.

Age relations of the dose from Table III were used for evaluation of the mean dose in those age groups for which the ^{131}I measurements in thyroid were not available.

The analysis of meteorological data on atmospheric transfer of the released activity from the destroyed reactor showed that "spots" of the greatest radioactive contamination on the territories of Bryansk, Tula, Kaluga and Oryol regions of Russia were formed due to the same radioactive cloud [13, 14] So the same regularities in the connection between thyroid dose and parameters of environmental contamination on these territories could be waiting. Such regularities can be taken from one area to another.

Linear regression analysis was used for the investigation of the relationships between the mean thyroid dose in the age group of 3-7 years and some parameters of radioactive contamination of the environment, surface ^{137}Cs activity on soil, kerma rate in the air on May 10-12 1986, mean ^{131}I concentration in milk in the period of May 5-12 1986.

In fig. 1-3 you can see the results of the regression analysis for the settlements of Bryansk and Tula regions of Russia, the most contaminated regions due to the Chernobyl accident [7] On the ordinate the average value of thyroid dose for

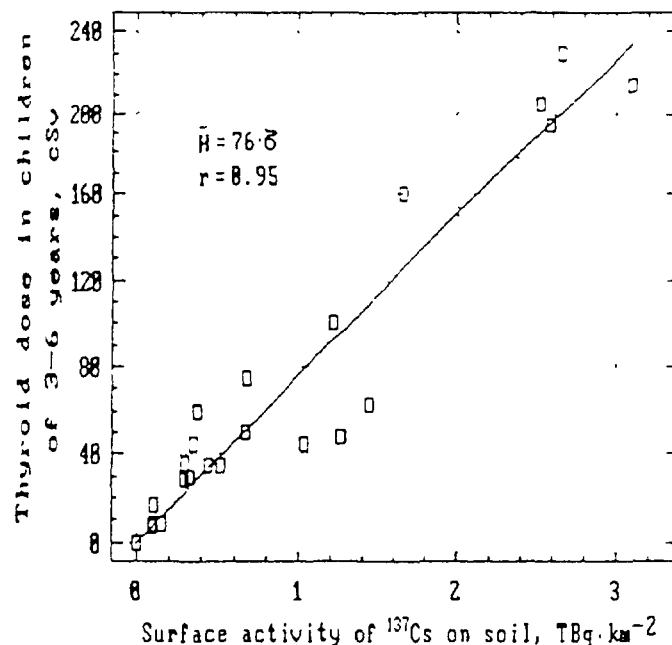


Fig 1 . Correlation of mean thyroid dose in children of 3-6 years of age with the surface ^{137}Cs activity in the settlement.

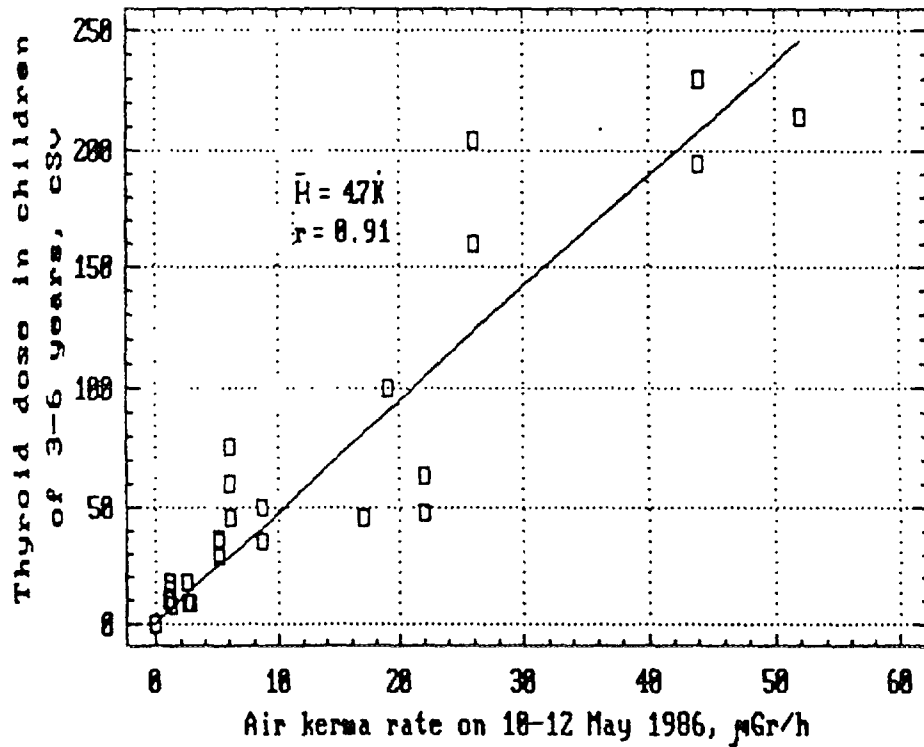


Fig. 2 . Correlation of the mean thyroid dose in children of 3-6 years of age with the mean air kerma rate in settlement on May 10-12, 1986.

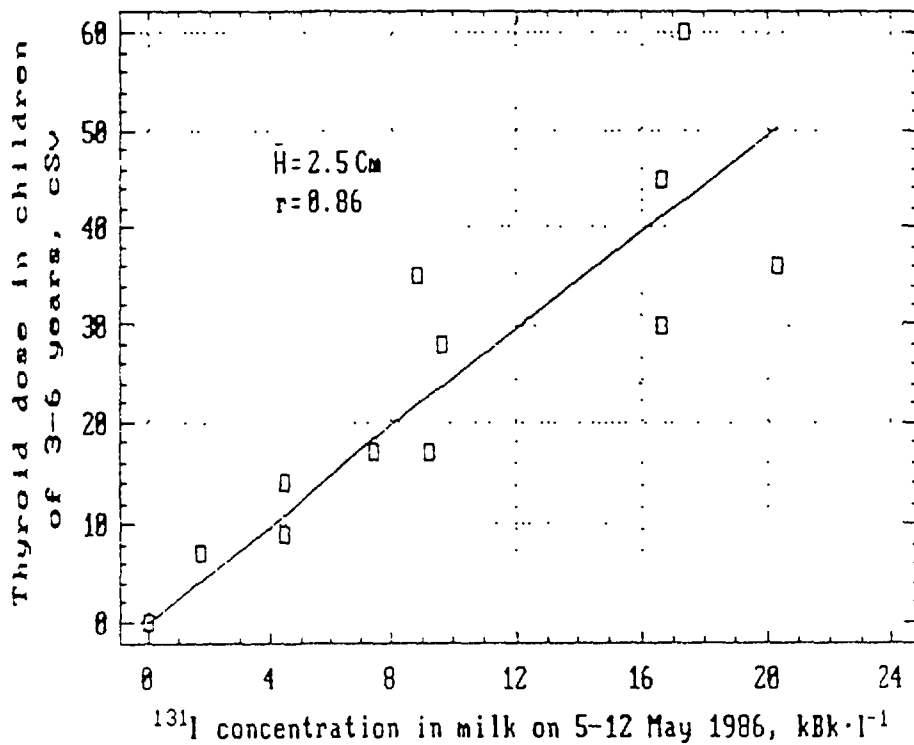


Fig. 3 . Correlation of the mean thyroid dose in children of 3-6 years of age with the mean body ^{131}I -I concentration in milk in settlements on May 5-10, 1986.

children in the age from 3 to 7, H , is plotted. These values are the maximum doses that could be realised in the settlements without protective actions. The abscissa is the average for the settlement value of the studied parameter.

The connection between parameters was highly reliable in all cases: correlation coefficients lie from 0.86 to 0.95.

We also investigated the correlation between the mean dose in thyroid of children of 3 to 7 years and the mean content of cesium radioisotopes in adults measured in August-September 1986 (fig.4). The fact that cesium radionuclides intake to inhabitants was stopped at summer 1986 in both regions was the precondition for such analysis. In the Tula region intake of I and Cs radionuclides took place only during the time when surface contamination of vegetation was present. Further migration of Cs through ecological chains was suspended because of the extremely low transfer coefficient of Cs from soil into plants on chernozem soils. In the Bryansk region Cs transfer coefficients were significantly higher due to podzol and sandy soils that are typical for the area. However, this process was interrupted by the implementation of protective actions in summer 1986.

Excretion of Cs from the body of adults occur slowly (with a half-time of 100 days), and therefore the measurements taken in July-September reflected Cs intake during the initial stage of the accident when intake of I also took place. The rate of

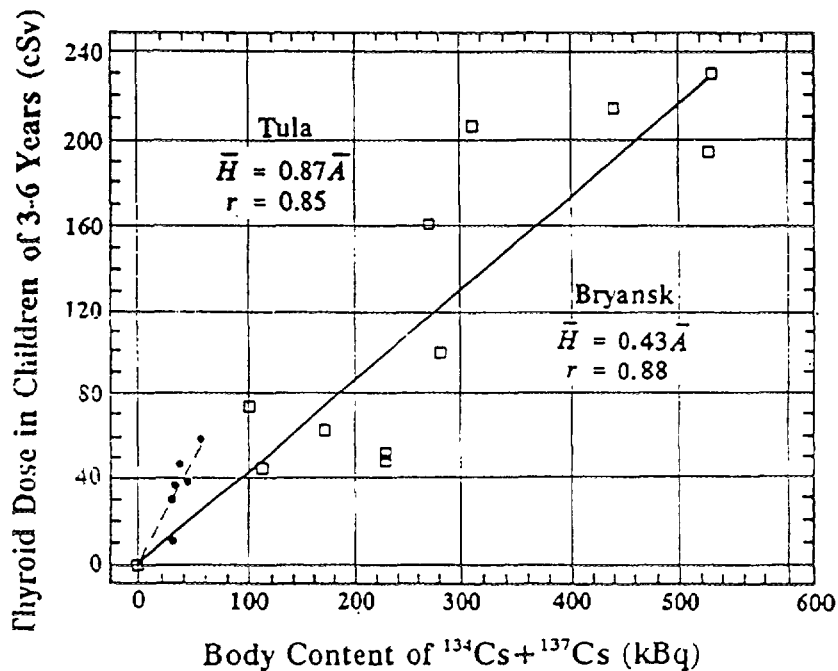


Fig. 4. Correlation of the mean thyroid dose in children of 3-6 years of age with the mean body $^{134}\text{Cs} + ^{137}\text{Cs}$ content in adults of the same settlements in August-September 1986.

the excretion for children is considerably higher, therefore Cs content in children several months after the accident is less reliable as correlating factor with thyroid dose.

The regression equation obtained for the Bryansk region can be applied to other areas with poor podzol, sand or peat soils, while the equation for Tula region can be applied to other areas with chernozem soils where ratio of I/Cs in fallout was the same as in investigated areas.

Finally the mean thyroid dose was evaluated by comparing values obtained with different regression equations, taking into account the reliability of initial parameters and introducing the correction for the performed countermeasures.

In the post-Chernobyl situation in Russia there also appeared a problem of the individual dose reconstruction due to the lack of ^{131}J measurements in thyroid. The basis for individual thyroid dose reconstruction was the developed correlation between the individual thyroid dose and Cs content in human's body, measured later - in August-September 1986, and between the individual dose and individual milk consumption in May 1986. The analysis of such connections was carried using data of ^{131}J and $^{134}\text{Cs} + ^{137}\text{Cs}$ measurements in the body and the questionnaire of the inhabitants of several settlements in Bryansk region [7, 15].

The deviation of the reconstructed with such methods individual thyroid dose from real value could be by a factor of 2 to 3, that can be consider acceptable in some situations.

We should examine particularly the questions upon thyroid dose reconstruction in new-borns that were fed with mother's milk or were irradiated by iodine radioisotopes in utero.

Analysis of simultaneous measurements of $^{131}\text{-I}$ in thyroids of breast-fed infants and their mothers performed after the Chernobyl accident showed reliable linear correlation between baby's and mother's $^{131}\text{-I}$ content in thyroid and their thyroid doses [7].

Corresponding regression equations has the from:

$$g_{inf} = 0.22.G_m, \text{ kBq} \quad (13)$$

$$H_{inf} = 3.6.H_m, \text{ cSv} \quad (14)$$

where g_{inf} and H_{inf} are the $^{131}\text{-I}$ activity in the baby's thyroid and the dose in it, G_m and H_m are the corresponding parameters for his mother.

Linear relationship between the parameters for the baby and the mother is highly significant: the correlation coefficient of the $^{131}\text{-I}$ activity in thyroid of the mother and her baby is 0.93, and for thyroid doses is 0.97. The obtained relationship (14) was used for the evaluation of the thyroid dose in baby when mother's thyroid dose was estimated due to direct $^{131}\text{-I}$ measurement or with methods of reconstruction.

Some correction is necessary to be made if the child consumes both breast and another milk. Such correction should take into account quantity of daily consumption of different sorts of milk and relationships between $^{131}\text{-I}$ concentration in breast milk, milk from private cows, goats, or from shops.

Thyroid dose estimation in fetus was performed by calculation method. The model of iodine metabolism in the body of a mother and her fetus suggested by H. Johnson for the single intake [16] was used for the reconstruction of the fetal thyroid

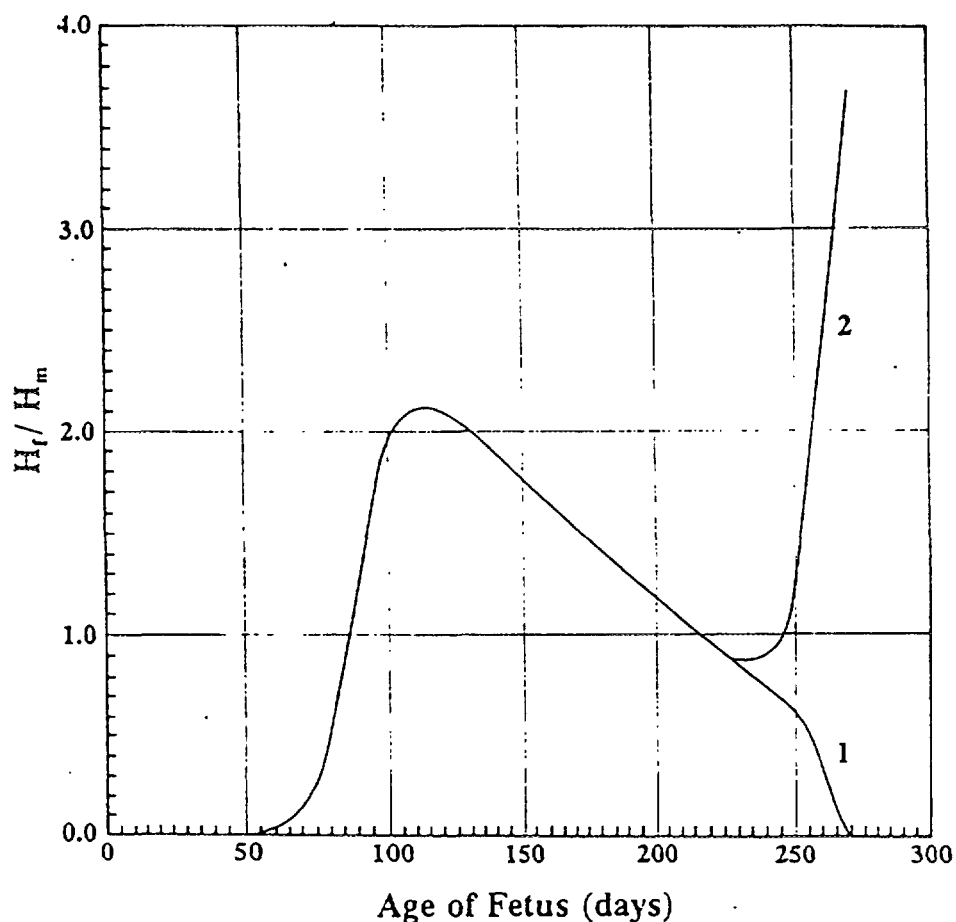


Fig. 5. Ratio of the fetal thyroid dose to the mother's one (1) in dependence on the fetal age at the time of the accident 2-the same ratio with the addition of dose due to $^{131}\text{-I}$ intake with breast milk after the birth.

dose from the prolonged intake into mother's body described with the equations (6) and (7). Fetal thyroid dose depends on the term of pregnancy at the time of the accident as it is seen in fig. 5 [7].

If the accidental intake has begun earlier than the fetal thyroid began functioning (90 days), the dose in the fetus is determined only by that part of activity, which entered into mother's body after 90 days of pregnancy. The maximum difference between thyroid dose of the fetus and its mother (on a factor of 2) occurs at a fetal age of 110 to 120 days and decreases continuously until childbirth.

If the accident occurred at the last stage of the pregnancy, then the dose received by the new-born child was determined partly by the in utero intake before birth and partly by radioiodine intake with mother's milk after birth (curve 2 on fig. 5). The latter part is determined according to the equation (14), where H_m is the dose in mother's thyroid formed because of the intake during the period after the birth.

Such methods of thyroid dose reconstruction in fetus or infants could be realized, if dose in mother's thyroid was available.

CONCLUSIONS

Main principals of radioiodine dosimetry in a human body after a nuclear accident have been discussed in the report. Dose estimations essentially depend upon the radionuclide intake function. Season features, pathways of intake to a human body, radioiodine migration in the environment, food habits and rations of people, countermeasures should be taken into account deducing analytical from of the intake function.

Measurements of ^{131}I in thyroids of people should be accompanied by simultaneous measurements of extrathyroidal tissue (thigh) for taking into consideration irradiation of other nuclides in the body. Questionnaire of measured people about milk consumption, duration of stay in contaminated area, countermeasures etc. is necessary for making more accurate dose estimations.

Methods of thyroid dose reconstruction in the absence of direct measurements of ^{131}I in human body are based on the analysis of radiation situation in the contaminated area and using of empiric correlation's of thyroid dose in measured people with parameters of environment contamination (^{131}I concentration in milk, contamination of soil with long-lived radionuclides, kerma rate in the air) in the settlements with similar radionuclides fallout conditions and similar life style of inhabitants.

Some examples of using the described methods of thyroid dose estimations in people exposed with radioiodine nuclides after the Chernobyl accident in Russia are presented in the report.

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RECALCULATION OF THYROID DOSES AFTER THE CHERNOBYL ACCIDENT IN AN IODINE DEFICIENT AREA

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Abstract

The thyroid doses were estimated in Poland shortly after the Chernobyl accident with assumption of stable iodine consumption for the reference man and areas with "standard" stable iodine consumption. These estimates are not representative for southern part of Poland which is known as the iodine deficient area. Therefore the thyroid doses were recalculated basing on the real and differentiated stable iodine intakes for people groups of different age without and with thyroid blockade after the accident.

1. INTRODUCTION

An assessment of thyroid dose after the Chernobyl catastrophe in Poland was performed by Zarnowiecki and Krajewski [1-3] using a dosimetric model of iodine metabolism in human body, developed by Johnson [4]. The assessments were performed under assumption that the consumption of stable iodine in Poland can be approximated by values used by Johnson for a Reference Man [5] i.e., about 200 μg of iodine per day for a 70 kg adult man and correspondingly less for children e.g. 116 μg per day for 10 years' old children. The Johnson's model demonstrates that stable iodine intake significantly influences thyroid doses because it dilutes radioactive μg per day for 10 years' old children. The Johnson's model demonstrates that stable iodine intake significantly influences thyroid doses because it dilutes radioactive iodine in blood and in thyroid compartments thus the estimates made till now underestimated significantly the thyroid doses on some areas.

South part of Poland, heavily-contaminated after the Chernobyl accident, is known as an area of iodine deficiency. Well documented data obtained in the last

years [6], clearly indicate that stable iodine consumption among schoolchildren and adults in Carpatien endemic is 2-3 times lower than those used in the previous estimates. This was the reason for recalculation of thyroid doses for inhabitants of this area basing on input data more reliable than those used in the first estimates.

The aim of this work was:

-to study the influence of stable iodine intake on thyroid doses basing on the recently obtained data on stable iodine intake and revised data on ^{131}I -I contamination in the south-Poland taking as an example two administrative areas called voivodities i.e. Krakowskie and Nowosadeckie.

-to estimate the efficiency and the possible strategies of administration of stable iodine to block thyroid in the iodine deficient areas.

2. SOURCE DATA AND METHODS

Johnson's model and calculation procedure

The most important features of the model are well known therefore they are only listed in the present paper. More details are in the original paper [4] as well as in [1-3]. Johnson's model is a dosimetric, 5-compartment model which is designed to study the kinetics of iodine transfer to different organs and is oriented on calculation of the radiation dose absorbed in these organs. In practice, in the case of radioactive iodine uptake, only thyroid doses are of interest. iodine is assumed to come to blood from lungs or gastrial tract (see Fig.1). Then, it is partly transferred to a bladder and excreted with urine and partly absorbed in a thyroid. The rate of thyroid uptake is assumed to be proportional to the ratio of radioactive and stable iodine concentration in the inorganic compartment (blood). iodine that is incorporated in the thyroid is assumed to be mixed with iodine already present in the compartment. Then, iodine leaves thyroid with a given rate, inversely proportional to the thyroid mass.

During this study the calculations were performed by using a code MODELAR, prepared by P. Krajewski [2]. The code enables to calculate, in a single run, thyroid dose for a given set of data such as time distribution of ^{131}I -I activity intake, stable iodine intake and the subject characteristic (age and sex). When dosimetric calculations are performed, activity of radioiodine in thyroid, integrated over the time scale is recorded. Then, this integrated activity in the source organ is recalculated into

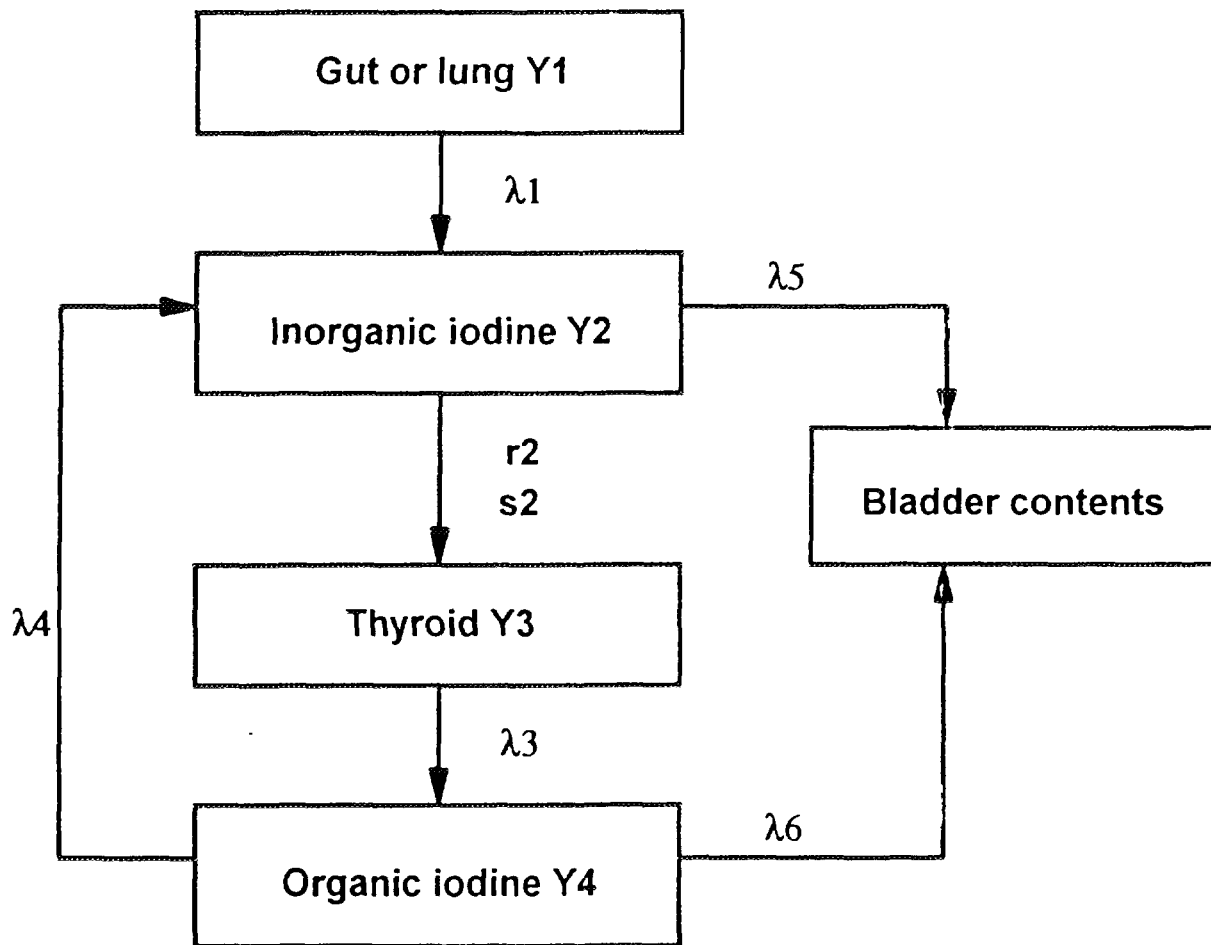


Fig.1. Metabolic model for iodine in man

committed absorbed dose, (which numerically is equal to equivalent dose in the thyroid), with the use of dosimetric factor converting integrated activity in the source organ to dose [4].

Consumption of stable iodine

The intake of radioactive iodine to thyroid is dependent on the concentration of stable iodine in the blood. Therefore, consumption of stable iodine influences radioactive iodine intake and, in consequence, thyroid doses.

In 1993 a large, representative cohort of schoolchildren in south-eastern Poland in age from 6 to 13 year was investigated for iodine concentration in urine and the results were published by Rybakowa et all. [6]. In Figs. 2 and 3 the frequency distribution of concentration of iodine in urine for children in three age groups in Krakowskie and Nowosadeckie voivodities are given respectively. For the purpose of this study it was assumed that children excrete one litre of urine per day and that amounts of excreted and consumed iodine are equal. These data were used for

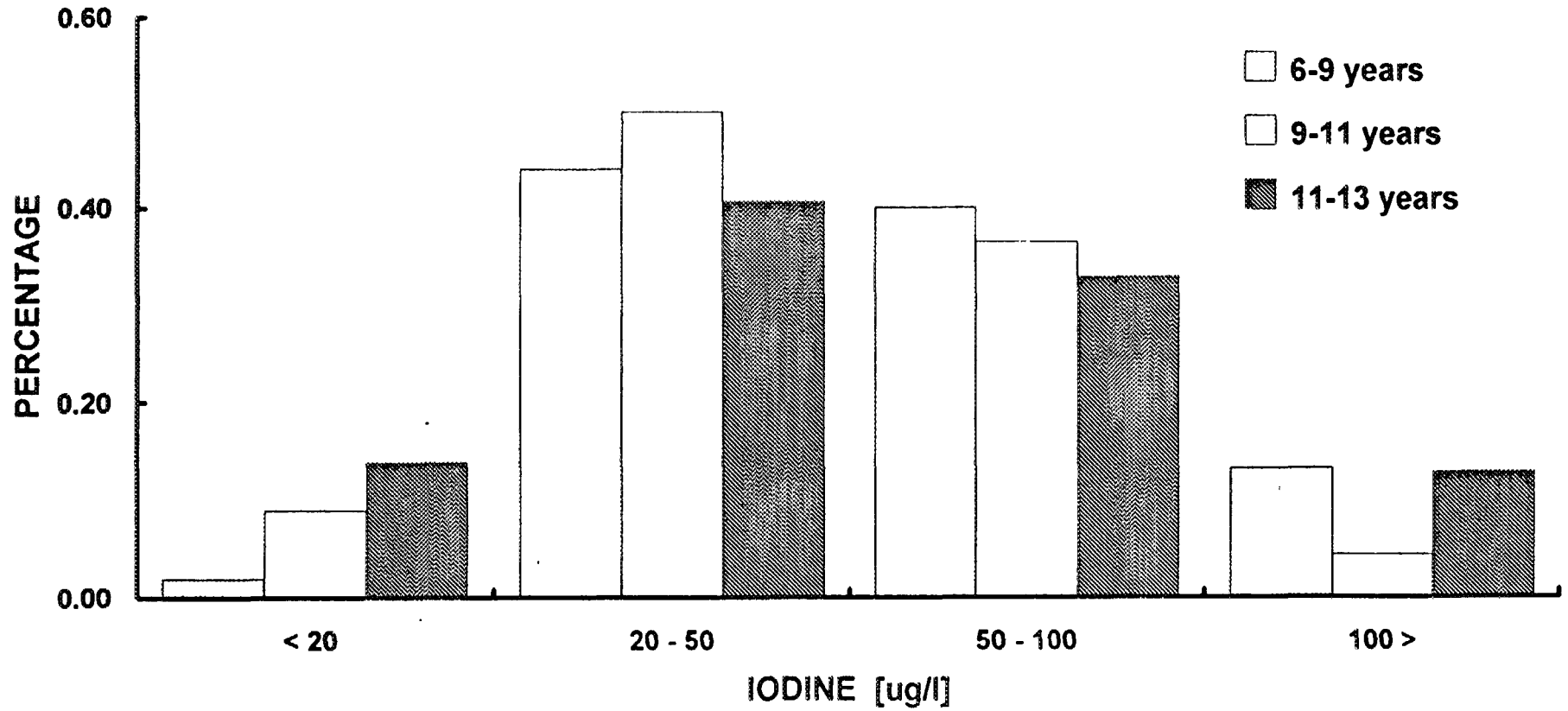


Fig.2. Frequency distribution of stable iodine in Krakowskie voivodity for children between 6 and 9 years, between 9 and 11 years and between 11 and 13 years [6].

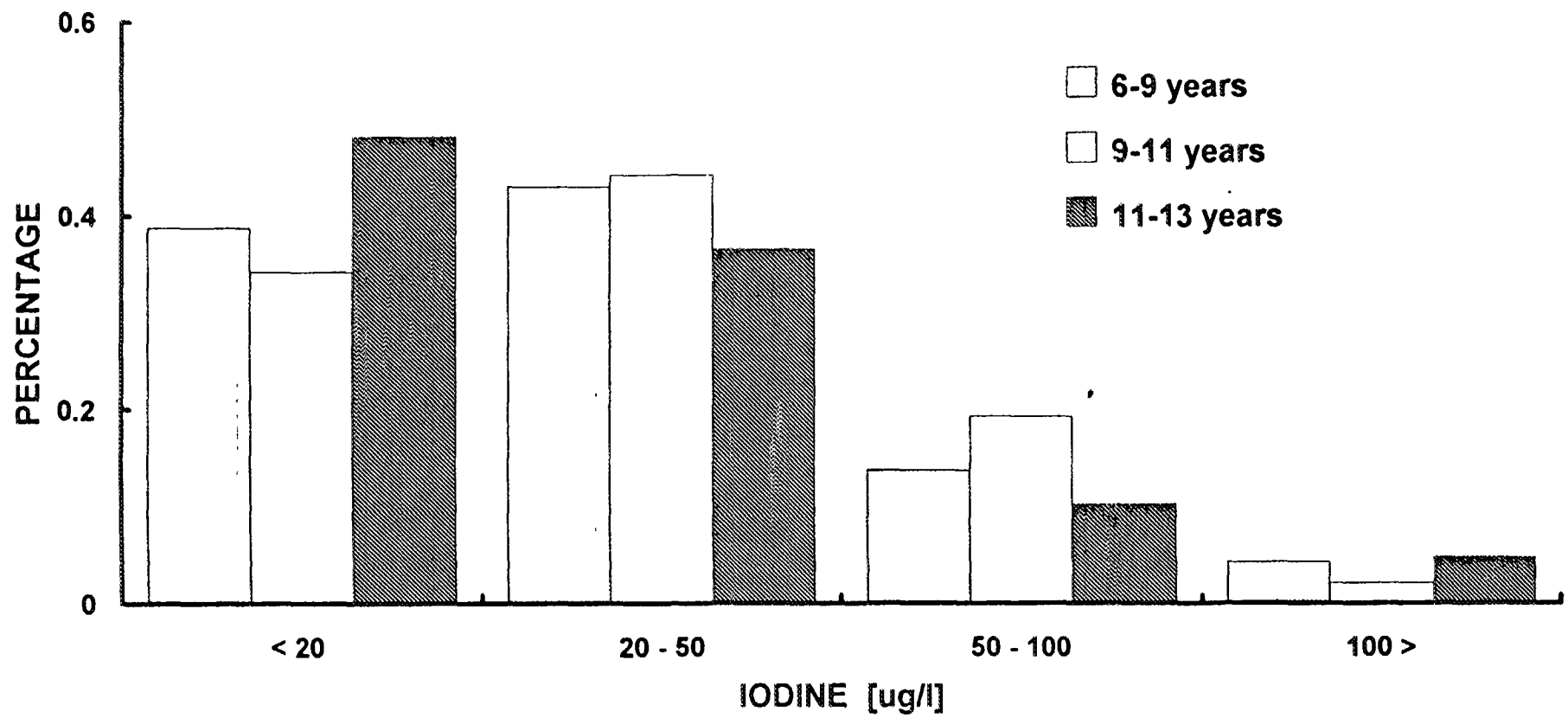


Fig.3. Frequency distribution of stable iodine in Nowosadeckie voivodity for children between 6 and 9 years, between 9 and 11 years and between 11 and 13 years [6].

children while for adults the daily iodine consumption was calculated on the base of a knowledge of dietary habits of population in Krakowskie and Nowosadeckie and of an average concentration of iodine in some basic food products [7, 8].

Intake of radioactive iodine

Thyroid doses, calculated in the present work, result principally due to $^{131}\text{-I}$ intake. The air which arrived from Chernobyl was contaminated also by other iodine isotopes mainly by $^{132}\text{-I}$ [9]. However, $^{132}\text{-I}$ contributed to thyroid dose more than one order of magnitude less than $^{131}\text{-I}$ and therefore was not taken into account in this study.

There are two main pathways of radioactive iodine intake i.e. inhalation and ingestion. The inhalation dominated during the first few days while the ingestion with milk, milk products, water and vegetables lasted practically to the end of May.

Inhalation

The amount of inhaled radioiodine was assumed to be dependent on time distribution of $^{131}\text{-I}$ activity in the air after the accident and on age-dependent breathing rate. For the ongoing calculations the results of measurements of $^{131}\text{-I}$ activity in air in Kraków obtained by Niewiadomski and Ryba (10) were used. The results, together with those used by Krajewski [3] are shown in Fig. 4. The diagram shows two activity waves, first one reached reached Kraków on April 29, a day later than Warszawa. Breathing rates taken from ICRP Report [5], which were used in the calculations, are shown in Tab.I. It was assumed in addition that 66"/a of $^{131}\text{-I}$ activity (inhalation class D), which entered the lungs during breathing, was transferred to the blood.

TABLE I. BREATHING RATE AND TOTAL ACTIVITY INTAKE DUE TO INHALATION FOR DIFFERENT AGE GROUPS [5].

Subject	Breathing rate m3/h	Activity [Bq]
Reference Man	0.98	3444
Reference Woman	0.88	3093
10 years' child	0.616	555
5 years' child	0.4	1406
1 year child	0.158	2165

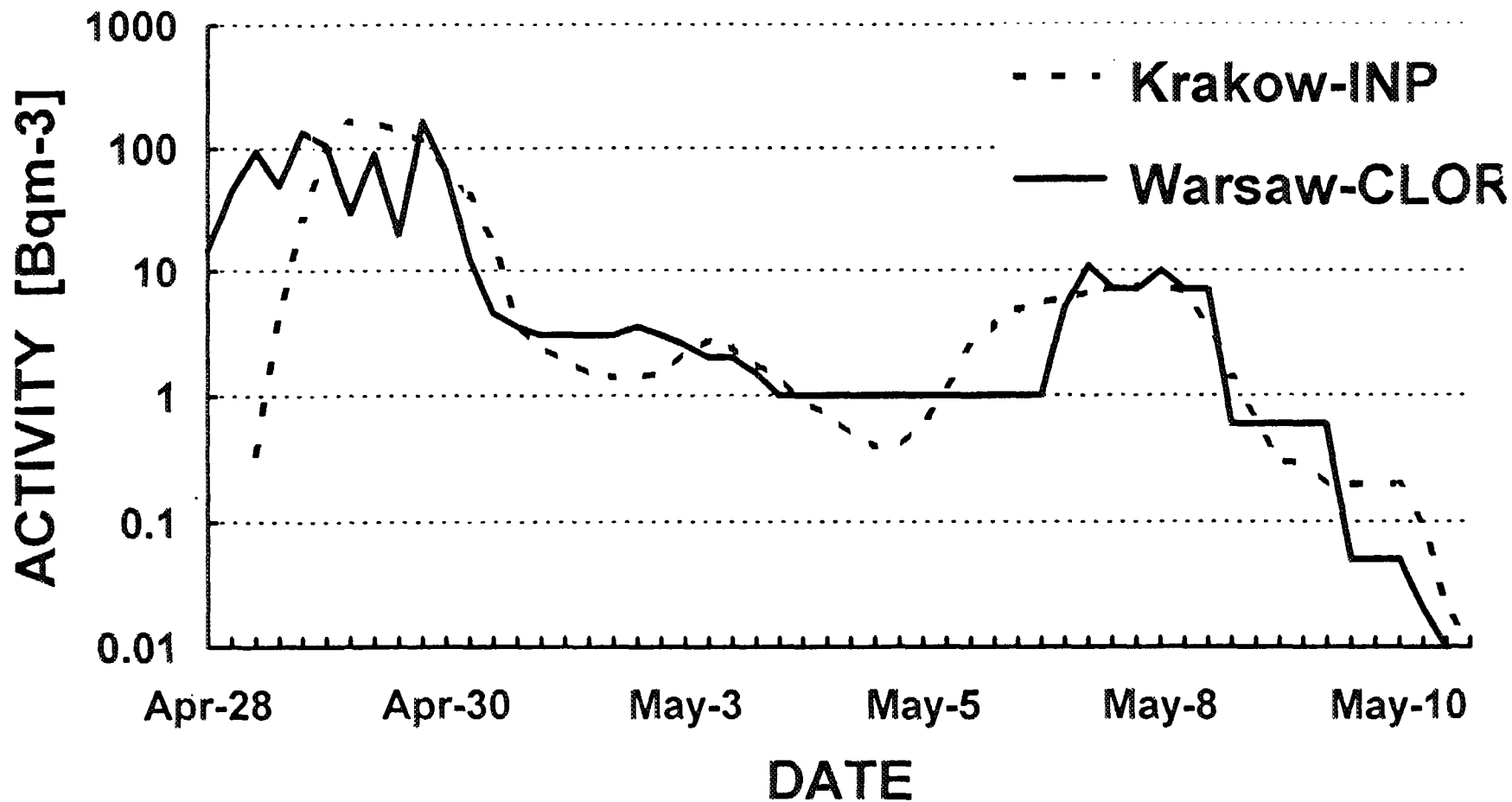


Fig.4. ^{131}I activity in air measured in laboratories Kraków (Institute of Nuclear Physics, INP) [11] and Warsaw (Central Laboratory for Radiological Protection, COLOR) [2].

Ingestion

The dietary intake of radioiodine was mainly due to water, milk and milk products (cheese) consumption. The intake with contaminated vegetables could be neglected because, at the time of the accident, people were warned not to consume fresh vegetables. Changes in activity of radioiodine in water, as measured in rivers Wisla and Raba as well as in Kraków drinking water, are presented in Fig. 5 [9, 11]. Water in Wisla was assumed as representing drinking water for population of Krakowskie voivodity while this in Raba for Nowosadeckie voivodity. The data show an evident lower contamination in Raba river than in Wisla. Activity of ^{131}I in drinking water in Kraków-town, measured in the AGH followed that in Wisla river therefore it was assumed that similar situation was as well on the territory of the whole voivodity as in Nowosadeckie.

Milk activity as measured by Cywicka-Jakiel et al [11] (Kraków-IFJ and Nowy Sacz) and Florkowski [9] (Krakow-AGH) is shown in Fig. 6. Milk in Kraków was measured in three large milk-factories, which collect milk from the distance of 10-20 km. The curves represent average values, because a short, heavy rain, which occurred on May 1 st, deposited differentiated local ^{131}I contamination's, which caused the significant variations of milk activity.

The amounts of the most important diet components, which were used in the activity intake calculations, are presented in Tab.II.

TABLE II. THE AMOUNTS OF WATER AND FOOD WHICH WERE USED FOR THE CALCULATIONS OF ACTIVITY INTAKE [12].

Diet [kg]	Men	Wome n	1 year	5 years	10 years'
Milk	0.35	0.4	0.65	0.6	0.6
Water	2	2	1.0	1.5	2.0
Milk products	0.1	0.15	0.04	0.045	0.06

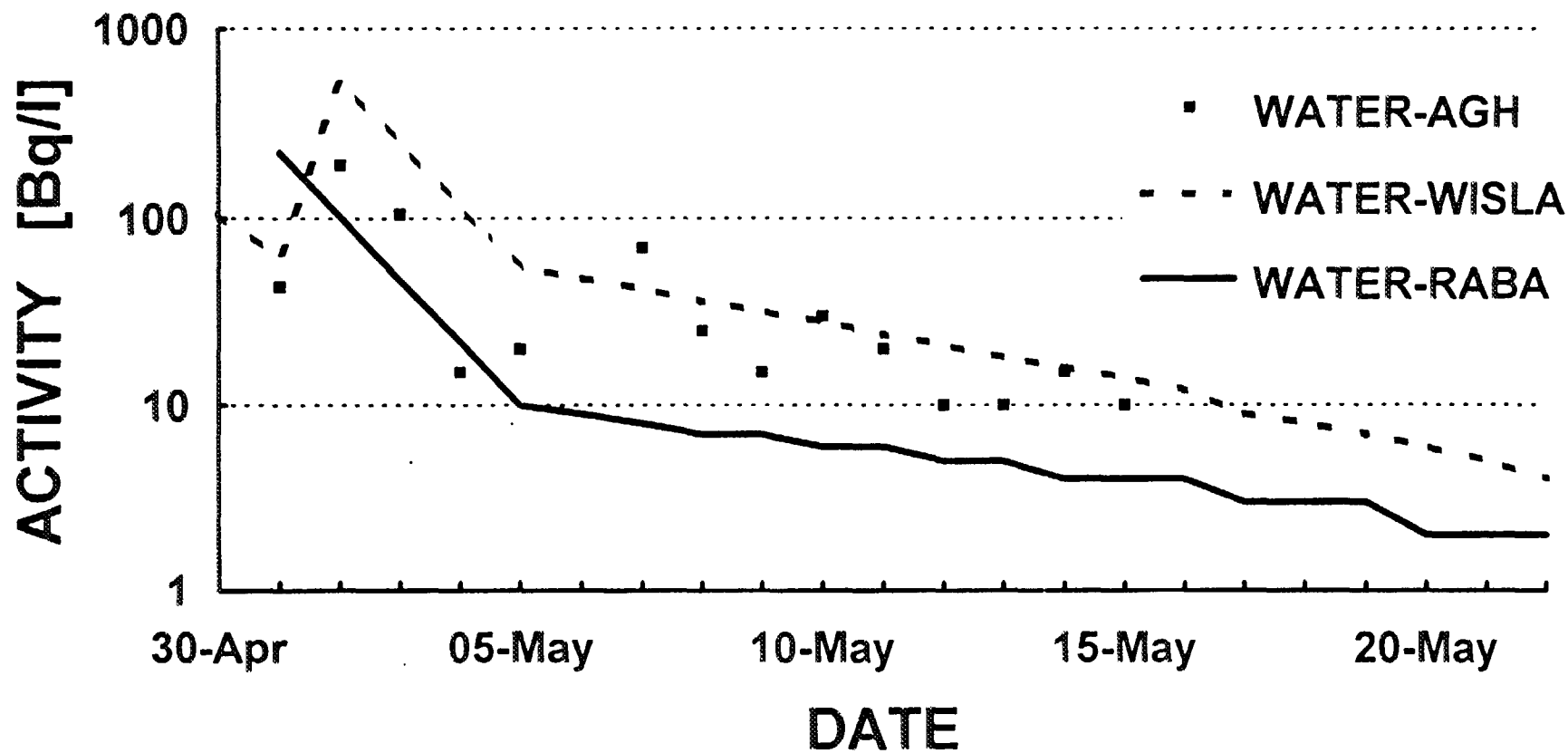


Fig.5. ^{131}I activity in water in rivers supplying waterworks in voivodities Krakowskie (Wisła) and Nowosądeckie (Raba) as measured in the INP [11] and in drinking water in Kraków measured in the INT at the AGH [9].

Cumulative activity intake

¹³¹I cumulative amounts since April 30 to June 3 were calculated by integration over this period ¹³¹I activities by taken into account diet components listed in the Tab.II. and unit activities as presented in Figs. 5 and 6. For Krakowskie the data on water from Wisla and milk measured in IFJ were used whereas for Nowosadeckie the data on water from Raba and milk measured for this area. No difference for inhalation was assumed in both districts. The results of calculations are shown in Table III.

TABLE III. CUMULATIVE ¹³¹I INTAKE (INGESTION, INHALATION AND TOTAL) FOR DIFFERENT GROUPS OF PEOPLE IN THE PERIOD APRIL 30 - JUNE 3

INGESTION of ¹³¹I [Bq]

Voivodity	Men	Women	1 y.child	5 y.child	10 y.child
K	6920	7378	7191	7600	8504
N-S	2592	2739	2742	2845	3165

INHALATION of ¹³¹I [Bq]

Voivodity	Men	Women	1 y.child	5 y. child	10 y. child
K	3444	3093	555	1406	2165
N-S					

TOTAL ¹³¹I INTAKE (Bq)

Voivodity	Men	Women	1y.child	5 y.child	10 y.child
K	10364	10471	7746	9006	10669
N-S	6036	5832	3297	4251	5330

Higher ¹³¹I intake for people living in Krakowskie than in Nowosadeckie (2. 6 times) was caused by difference in contamination of water and milk in Krakowskie due to the heavier rain on this area on May 1 st.

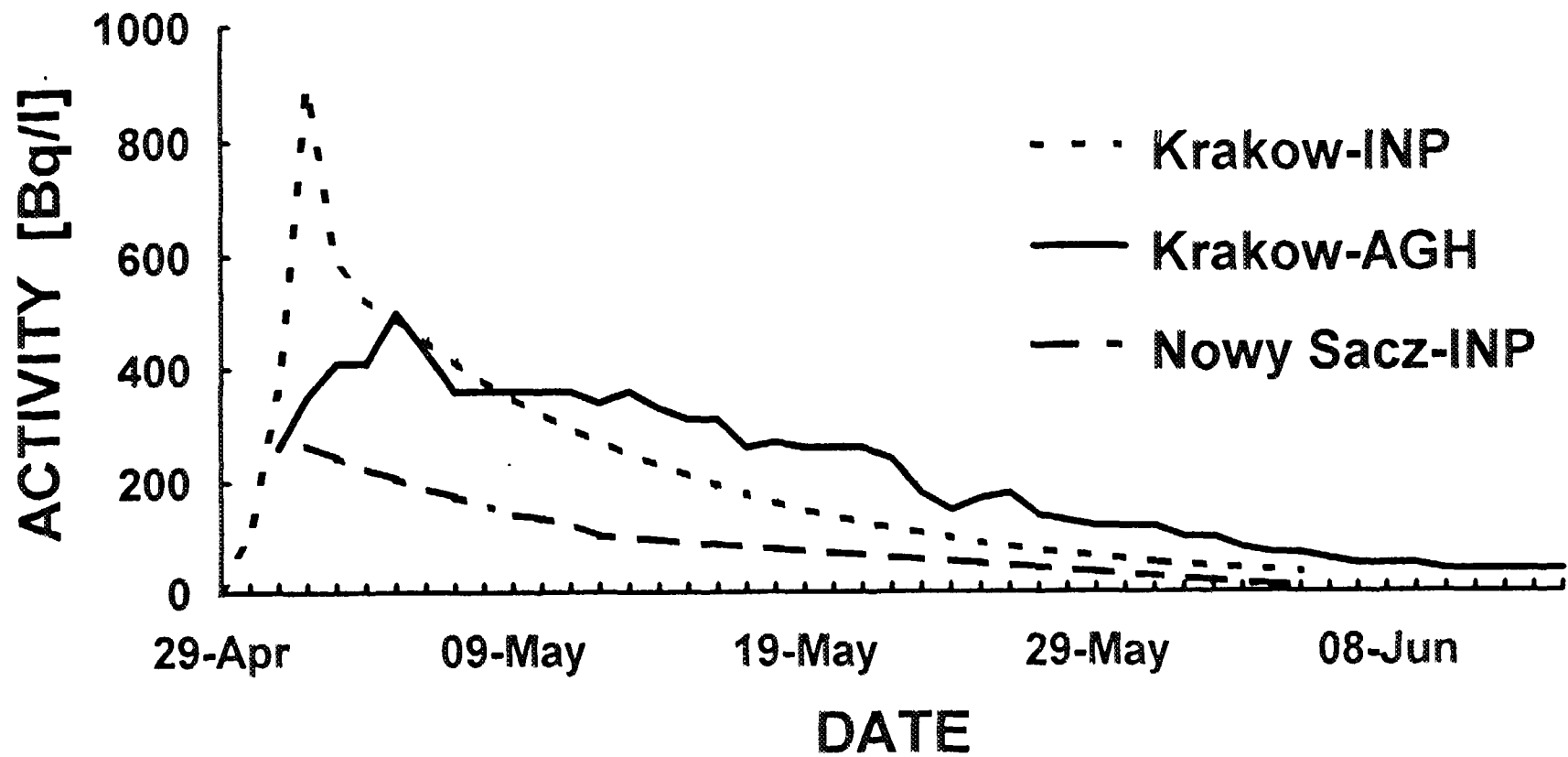


Fig.6. Activity in milk from different suppliers from Krakowskie and Nowosądeckie voivodities as measured in INP [11] and the INT at the AGH [9].

3. RESULTS AND DISCUSSION

Changes of doses due to different stable iodine intake

Since one of the aims of this work was to estimate differences in thyroid dose due to various stable iodine intake before the radioiodine exposure therefore the population was divided into groups according to this parameter. Calculations for children were performed starting with calculations of thyroid doses for several single values of the stable iodine intake. Then the doses were weighted over the distribution presented in Fig. 2 to obtain the average thyroid dose for the given group of children. For the adults the estimates of average iodine consumption are only available. Therefore, the calculations were performed for some discrete values of intake i.e. 35 (range 25-50), 75(50-100), 150(100-200) and 200 $\mu\text{g}/\text{day}$.

Committed thyroid doses for 10 years old children, calculated for several values of stable iodine intake are presented in Fig. 7. The calculated doses for the lowest intakes, about 20 $\mu\text{g}/\text{day}$ amounted to 19.6 mSv in Nowosadeckie and 39 mSv in Krakowskie. The corresponding doses calculated for intake used by Johnson for 10 years old children (116 $\mu\text{g}/\text{day}$) are about 3 times lower i.e. 13.4 and 6.8 mSv respectively. Similar differences between doses for "standard" and really observed iodine consumption were also obtained for the other age-groups. E.g. for 11-13 years children in Nowosadeckie the assumption of stable iodine consumption of 136 $\mu\text{g}/\text{day}$ leads to 3.2 mSv dose. However, about 50% of these children consumed daily less than 20 μg of iodine thus the received doses were as high as 9.7 mSv.

Differences in doses between voivodities Krakowskie and Nowosadeckie are caused by two factors acting in opposite directions. The lower stable iodine intake in Nowosadeckie increases dose while the lower ^{131}I contamination lowers them. While in Krakowskie only 9% of kids consume below 20 μg of iodine, in Nowosadeckie 34%, the weighted thyroid doses for Krakowskie (26 mSv) and Nowosadeckie (15 mSv) differ by the factor of 1.7.

The calculations performed for the Reference Man demonstrate a similar regularity (see Fig. 8). Thyroid doses for Krakowskie ranged from about 5 to 16 mSv. For the most probable stable iodine consumption between 50 and 100 $\mu\text{g}/\text{day}$, the thyroid dose for this area was 6.5 mSv.

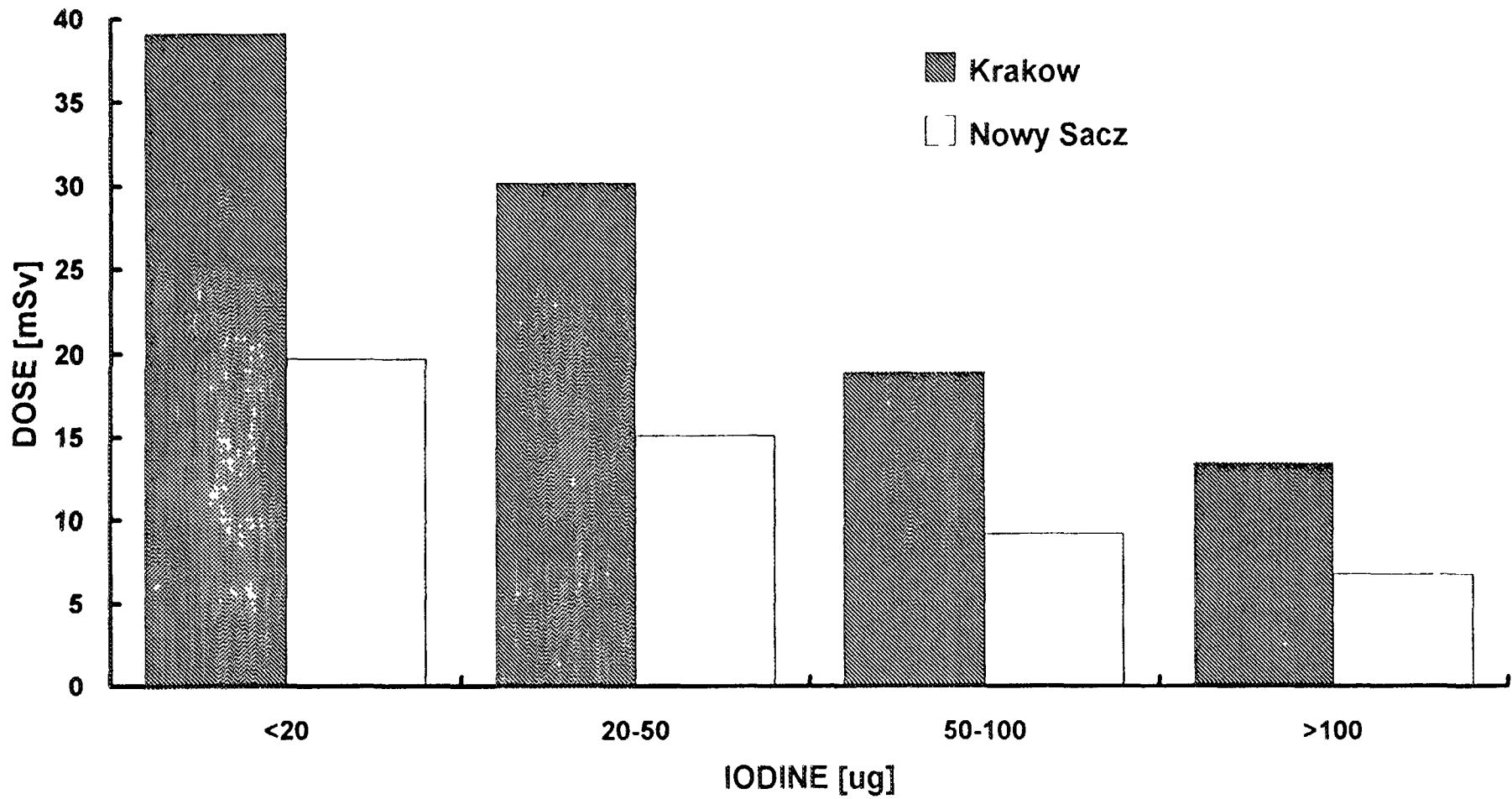


Fig 7 Committed thyroid dose for children of 10 years old in Kraków and Nowy Sacz in dependence on stable iodine intake.

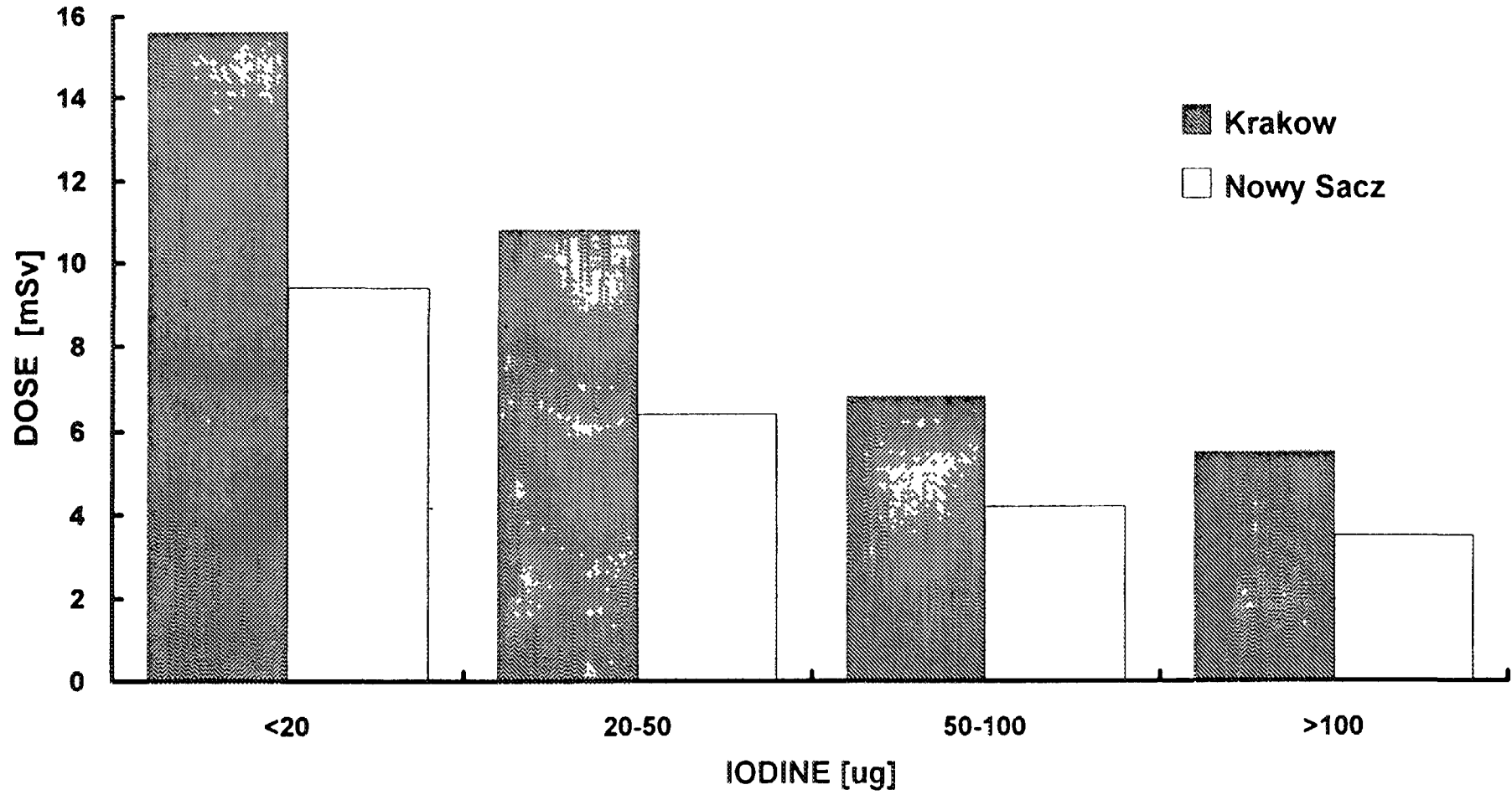


Fig.8. Committed thyroid dose for reference man in Kraków and Nowy Sącz in dependence on stable iodine intake

The variation of thyroid dose is discussed in this paper with regard to stable iodine consumption using average radioiodine intake for given area. It is worth to mention that the variations of dose due to differences in $^{131}\text{-I}$ intakes were surely much higher. The reason for that are local (not recorded) contaminations due to the rain of different intensity and variations in the diet.

Effects of stable iodine administration (blocked)

One of the discussed problems during the iodine phase of Chernobyl catastrophe was usefulness and strategy of administration of prophylactic doses of stable iodine. This action was started in Poland on April, 29, 1986 in eleven most contaminated districts, also in Krakowkie and Nowosadeckie. Stable iodine doses, in amount of 15 to 60 mg were recommended and administered to infants and children up to 16 years.

The result of this action i.e. the remaining dose fraction after administration of a single dose of 15 mg iodine to 1 year old child, in the function of administration day is presented in Fig. 9. It can be seen from the figure that the action had limited effect and that the highest dose reduction (68%) could be obtained if the action would start on May 2nd. The reason for that is that the maximum intake of $^{131}\text{-I}$ occurred in south-Poland after a heavy rain on May 1 st which resulted in a strong milk contamination during the next days. These high levels of milk contamination were significant over the whole May. A single prophylactic dose of iodine is however effective for about one week only. While the most $^{131}\text{-I}$ intake happened later, mainly from milk and water consumption, single prophylactic iodine doses had little impact on decreasing children thyroid doses.

The amount of stable iodine is also not a critical parameter for dose reduction, as can be seen in Fig. 10. Of course, the situation could be quite different if the $^{131}\text{-I}$ intake by ingestion is reduced e.g. by introducing milk powder on the market. Then, thyroid blockade is much more effective [3] because the most serious inhalation intakes lasts about one week. It is difficult to predict a single, best day for a thyroid blockade with a high iodine dose. It would be better to administer (from the radiation protection point of view because medical point of view can be different) low-doses of iodine eg. 1 mg per day, but over a period of a month.

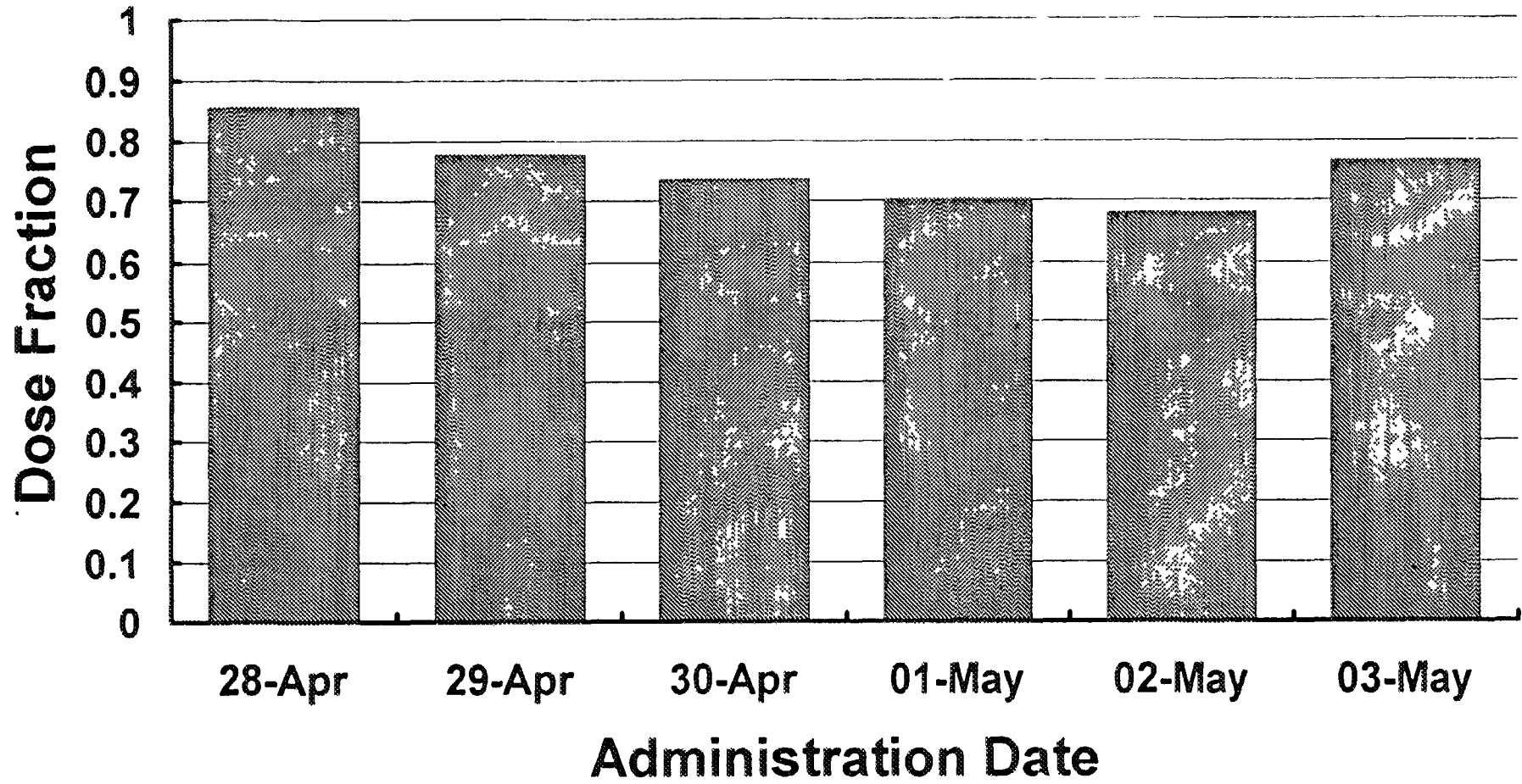


Fig 9 Effect of thyroid blockade: 1 year child, by 15mg stable iodine administered on different days, daily iodine intake 20 μ g.

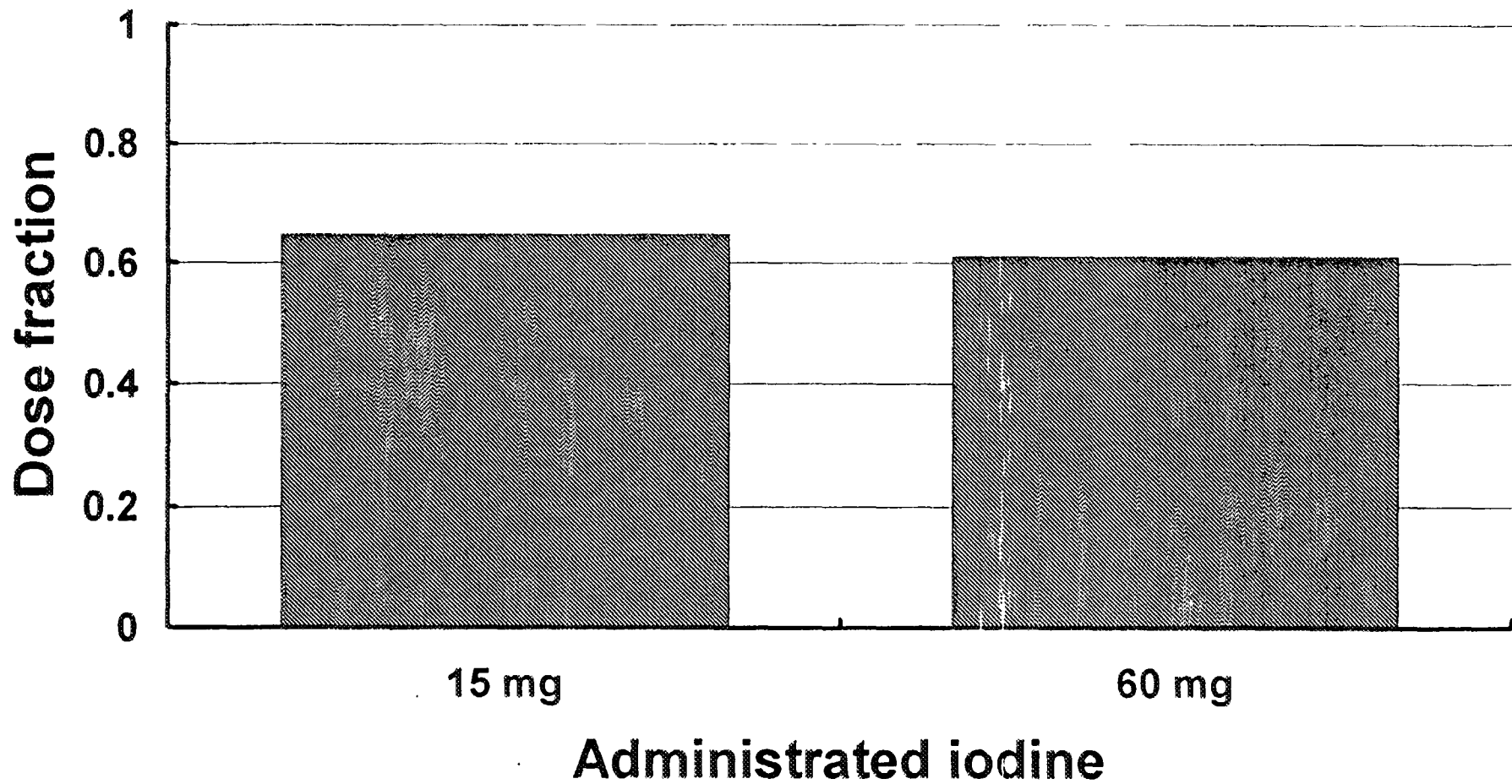


Fig.10. Effect of thyroid blockade: 1 year child, 15 and 60 mg of stable iodine administered on May 2nd.

CONCLUSIONS

It was demonstrated that thyroid doses after accidental ^{131}I intake are seriously dependent on the state of saturation of thyroid by stable iodine. The stable iodine concentration is low on some areas where a lack of this element in water and food occurs. An example of such area is the territory of south Poland. Calculations for inhabitants of this territory, based on the Johnson's of iodine metabolism in human body has shown that for 10 years old children who consume daily 20 μg thyroid dose is three times higher than the dose for children with "standard" 116 μg intake and with the same ^{131}I consumption with air, food and water.

The estimates of the optimal day of thyroid blockade by administration of stable iodine to children has shown that this blockade is effective for one week only, thus the day of administration should be chosen in dependence of anticipated situation of radioiodine supply. Since this is rather difficult it often would be better to administer low doses of stable iodine e.g. 1 mg per day over the period of one month instead of the single high dose. As far as situation in south Poland after the Chernobyl catastrophe is concerned it was found that the amount of the prophylactic stable iodine was not a critical parameter for thyroid dose reduction thus the lower dose could be recommended as this which results lower medical complications in the future life of the person who was the subject of this type of recovery operation.

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**THYROID CANCER INCIDENCE AMONG SWEDISH PATIENTS
EXPOSED TO DIAGNOSTIC DOSES OF IODINE-131:
A PRELIMINARY REPORT**



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Abstract

The level of risk associated with I-131 is not well defined but appears lower than equivalent doses of x-rays. To provide quantitative data on the risk of thyroid cancer following I-131 exposure, 34,104 patients surviving ≥ 5 years after I-131 administration between 1950-69 for diagnostic purposes were studied. The mean thyroid dose was estimated to be 1.1 Gy (range 0-40.5). A significantly increased risk of a subsequent thyroid cancer was found, however, the excess rates were based entirely on patients referred because of a suspicion of a thyroid tumor. There was no suggestion of an increasing risk with increasing dose or time since exposure. No significant excess risk was found among those less than 20 years of age at exposure. The absence of a risk among those over age 20 is consistent with studies of A-bomb survivors and implies that any type of exposure to ionizing radiation later in life is associated with a *minimal* cancer risk.

1. INTRODUCTION

The correlation between high-dose rate ionising radiation and thyroid cancer was first suggested more than 50 years ago (1, 2) and thyroid cancer has since then been convincingly linked to ionising radiation only after childhood exposure (3-7). In the most recent follow-up of A-bomb survivors, thyroid cancer risk was increased only among individuals under age 20 years at exposure (Table 1) (8). Age at exposure thus appears to be the most important determinant of future risk, and differences in reported risk estimates might merely reflect differences in age distribution.

No increased risk of thyroid cancer has been found among hyperthyroid patients treated with I-131 (9-11). The advanced age of the population treated and the fact

TABLE I. FITTED EXCESS RELATIVE RISK ESTIMATES AT 1 SV AND NUMBER OF THYROID CANCERS, WITHIN PARENTHESIS, BY SEX AND AGE AT EXPOSURE AMONG A-BOMB SURVIVORS (8).

Sex	Age at exposure, year				All
	0-9	10-19	20-30	≥40	
Male	9.39 (7)	2.60 (7)	-0.23 (10)	-0.23 (12)	1.08. (36)
Female	9.47 (26)	3.12 (44)	0.42 (65)	-0.23 (54)	1.15 (189)
All	9.46 (33)	3.02 (51)	0.34 (75)	-0.23 (66)	1.15 (225)

that cellular destruction predominates over cell transformation at average organ doses of more than 60 Gy are probably the two most important explanations for the lack of increase.

Fallout from nuclear weapons tests has resulted in increased risk of thyroid cancer among Marshall Islanders, but most of the dose was delivered from gamma rays and short-lived radioiodines and not I-131 (12). In an extended follow-up of 4,818 children exposed to fallout from nuclear devices at the Nevada Test Site an increased risk of thyroid was found after a mean thyroid dose of 0.17 Gy (13). A recent registry evaluation reports high rates of thyroid cancer in children living in the Chernobyl area (14). No childhood study has found an excess of thyroid cancer within 5 years of exposure, even following high-dose radiotherapy (5). The earliest thyroid neoplasm diagnosed among Marshall Islanders occurred 9 years after exposure (12), and 13 years among infants given radiotherapy for a supposed enlarged thymus gland (15). The remarkable increase seen in Belarus within 5 years of the accident probably, to some extent, reflects increased screening procedures and increased awareness as shown by Ron et al. (16), rather than exposure to ionising

radiation. Against this background, we extended the follow-up of a large series of patients administered I-131 diagnostically (17). Individual radiation dose to the thyroid was computed for the first time. The aim was to provide quantitative data on the risk of thyroid cancer after exposure to relatively low dose and dose-rate exposures.

2. METHODS

The cohort has been described elsewhere (17, 18). Patients were examined with I-131 during the period 1950-1969, less than 75 years of age at exposure, and had not received external radiotherapy. The cohort consisted of 34,104 patients (80% women and 20% men) with a mean age of 43 years (range 1-75 years) at first exposure, and a mean follow-up of 24 years (range 5-39 years, Table 2). A total of 2,408 individuals were exposed before 20 years of age and 316 before the age of 10 years.

A total of 10,785 (32%) patients were referred under the suspicion of a thyroid tumor and 23,319 patients for other reasons (mainly possible hypo- or hyperthyroidism). The characteristics of the patients examined because of a suspicion of thyroid tumor and of those examined for other reasons differed mainly in administered I-131 activity (Table 2). We estimated absorbed thyroid dose using the individual administered I-131 activity and 24 hour thyroid uptake of I-131, as well as ICRP tables (19). The follow-up period started at the time of first exposure or if exposed prior to 1958, at January 1, 1958, and lasted until thyroid cancer diagnosis, death, emigration, or December 31, 1990. The first 5 years at risk were excluded in order to reduce the number of thyroid cancers that were related to referral or to increased medical surveillance and not to the I-131 exposure. All thyroid cancers observed within the first five years of followup were excluded for the same reason.

The cohort was matched with the Swedish Cancer Register (SCR), using the personal 10-digit identification number, for the period 1958-90 to identify thyroid carcinomas. The expected number of thyroid cancers were calculated using incidence data from the SCR and indirect standardisation with adjustment for sex, attained age at exposure, and calendar period. The standardised incidence ratios (SIR) were calculated as the ratio between observed and expected numbers of thyroid cancers. The 95% confidence intervals (CI) were calculated assuming the observed number of cancers being Poisson distributed. Trends for SIR were calculated using the formulas suggested by Breslow and Day (20).

TABLE II. CHARECTERISTICS OF PATIENTS EXPOSED TO I-131 IN RELATION TO REASON FOR REFERRAL. THE FIRST 5 YEARS AFTER EXPOSURE WERE EXCLUDED.

	Reason for referral		All
	Suspicion of thyroid tumor	Other reasons	
No. of patients	10,785	23,319	34,104
Males/Females %	14/86	22/78	20/80
Mean age at exposure (range), years	44 (1 -75)	42 (1 -75)	43 (1 -75)
Patients <20 years of age at exposure, %	6	8	7
Mean follow-up period (range), years	23 (5-38)	24 (5-39)	24 (5-39)
Mean 24-hour thyroid uptake (range), %	40	40	40
Mean administered activity (range), MBq	2.4 (0.04-37)	1.6 (0.04-37)	1.9 (0.04-37)
Mean dose to the thyroid (range), Gy	1.3 (0.0-25.7)	0.9 (0.0-40.5)	1.1 (0.0-40.5)

3. RESULTS

The absorbed dose to the thyroid gland was 1.3 Gy among those referred under the suspicion of a thyroid cancer and 0.9 Gy among those examined for other reasons (Table I).

Between 1958 and 1990, a total of 67 thyroid cancers were found more than 5 years after exposure. Forty-two were referred under the suspicion of a thyroid cancer, and 25 for other reasons. The mean time from exposure to I-131 and diagnosis of the thyroid cancer was 15 years.

The overall risk for thyroid cancer more than 5 years after exposure was 1.35 (95% CI 1.05-1.71 ; Table III). A significantly higher risk was seen for those 10,785 patients referred under the suspicion of a thyroid cancer (SIR=2.86; 95% CI 2.06-3.86) compared to those referred for other reasons (SIR=0.75; 95% CI 0.48-1.10).

When patients were divided into different dose categories no trend of increasing risk with increasing dose was noticed regardless of reason for referral (Table III). The highest risk were seen during the period 5 to 9 years after exposure regardless of reason for referral (Table III). For both referral categories a non-significantly higher risk was seen for the period 20 years or more after exposure compared to 10 to 19 years after exposure.

The risk of a thyroid cancer was highest among those exposed before the age of 20 years (SIR=1.69; 95% CI 0.35-4.93), although based on 3 thyroid cancers only (Table IV). Among the 1,764 exposed children not referred under the suspicion of a thyroid tumor, 2 thyroid cancers were found, giving a non-significantly elevated SIR of 1.38. The three individuals developing thyroid cancers were exposed between the age of 15-19 years.

DISCUSSION

The thyroid gland of children appears to be one of the most susceptible organs to radiation carcinogenesis with relative risk estimates at 1 Gy ranging from 4 to over 30 (21). As 93% of our patients were over age 20 years when I-131 was administered, the absence of an overall effect might be attributable to the lower sensitivity of the adult thyroid gland. No trend of increasing risk with increasing dose was suggested, neither was an increasing risk seen with time since exposure, i.e. no risk was apparent after 10 years, arguing against a relationship for I-131 and thyroid cancer in this series.

A recent report from cancer registry data in Belarus purports high rates of thyroid cancer to be associated with radioactive fallout from the Chernobyl accident (14). This opinion was shared by an expert panel formed by the Commission of the European Communities, although they emphasised that the influence of screening should be carefully considered in assessing the results (22). The time between exposure and appearance of the thyroid cancer is surprisingly short and the dramatic increase in thyroid cancers most likely is, at least in part, related to the intense screening increased awareness and changed referral routines (16). In this context it is

TABLE III . OBSERVED NUMBER OF THYROID CANCERS, SIR, AND 95% CI, IN RELATION TO DOSE AND YEARS AFTER EXPOSURE. THE FIRST 5 YEARS AFTER EXPOSURE WERE EXCLUDED

Dose, Gy	Years after exposure											
	5-9			10- 19			≥20			All		
	Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI	Obs	SIR	95% CI
All												
≤ 0.25	4	1.72	0.47-4.40	4	0.77	0.21-1.97	3	0.93	0.19-2.73	11	1.03	0.51-1.83
0.26-0.50	5	2.67	0.87-6.24	6	1.41	0.52-3.07	5	1.93	0.63-4.51	16	1.84	1.05-2.98
0.51-1.00	1	0.50	0.01-2.77	4	0.86	0.23-2.20	4	0.95	0.26-2.44	9	0.46	0.38-1.57
>1.00	11	2.95	1.47-5.28	8	0.93	0.40-1.84	12	1.70	0.88-2.98	31	1.60	1.09-2.27
All	21	2.11	1.31-3.23	22	0.97	0.61-1.47	24	1.41	0.90-2.10	67	1.35	1.05-1.71
Referred for suspicion of thyroid tumor												
≤ 0.25	2	4.88	0.59-17.62	2	2.17	0.26-7.85	2	5.71	0.69-20.64	6	3.57	1.31-7.77
0.26-0.50	5	7.94	2.48-18.52	4	2.78	0.76-7.11	3	4.17	0.86-12.18	12	4.30	2.22-7.51
0.51-1.00	0	0.00	0.00-6.15	3	2.14	0.44-6.26	1	1.14	0.03-6.33	4	1.39	0.38-3.56
>1.00	7	3.87	1.55-7.97	6	2.17	0.79-4.71	7	2.53	1.02-5.21	20	2.72	1.66-4.20
All	14	4.06	2.22-6.81	15	2.30	1.29-3.79	13	2.75	1.47-4.71	42	2.86	2.06-3.86
Referred for other reasons												
≤ 0.25	2	1.04	0.13-3.76	2	0.47	0.06-1.69	1	0.35	0.01-1.95	5	0.55	0.18-1.29
0.26-0.50	0	0.00	0.00-2.97	2	0.71	0.09-2.57	2	1.07	0.13-3.86	4	0.68	0.18-1.73
0.51-1.00	1	0.71	0.02-3.95	1	0.31	0.01-1.71	3	0.90	0.19-2.64	5	0.47	0.20-1.46
>1.00	4	2.08	0.57-5.33	2	0.46	0.06-1.65	5	1.17	0.38-2.73	11	1.04	0.52-1.86
All	7	1.08	0.43-2.22	7	0.48	0.19-0.98	11	0.89	0.45-1.60	25	0.75	0.48-1.10

TABLE IV. OBSERVED NUMBER OF THYROID CANCERS, SIR, AND 95% CI, INRETATION TO AGE AT EXPOSURE AND SEX. THE FIRST 5 YEARS AFTER EXPOSURE WERE EXCLUDED.

Age years	Men			Women			All		
	Obs.	SIA	95% CI	Obs.	SIR	95% CI	Obs.	SIA	95% CI
All									
≤20	0	0 00	0 00-23 06	3	1 85	0 38-5 41	3	1 69	0 35-4 93
21 -50	6	2 33	0 86- 5 08	34	1 23	0 85-1 72	40	1 32	0 94-1 80
>50	6	3 14	1 15- 6 84	18	1 17	0 69-1 84	24	1 38	0 88-2 05
Referred for suspicion of a thyroid tumor									
≤20	0	0 00	0.00-184 85	1	3 24	0 08-17 97	1	3 04	0 08-16 88
21-50	6	10 71	3 93- 23 32	19	2 13	1 28-3 32	25	2 63	1 70-3 88
>50	6	11 54	4 23- 25 11	10	1 81	0 87-3 33	16	2 65	1 51-4 30
Referred for other reasons									
≤20	0	0 00	0.00- 26 35	2	1 53	0 18-5 52	2	1 38	0 17-4 98
21-50	0	0 00	0 00- 1 84	15	0 80	0 45-1 32	15	0 72	0 40-1 19
>50	0	0 00	0 00- 2 65	8	0 81	0 35-1 59	8	0 71	0 31-1 39

interesting to note that the incidence of childhood leukemia in Belarus, an entity known to be increased 2-3 years after exposure to ionizing radiation, was not higher during the period 1986-1991 than in the preceding period 1979-1985 (23).

CONCLUSION

In conclusion, it is in some sense reassuring that the careful examination of about 34,000 patients who received substantial radiation doses to their thyroid glands from I-131 did not identify a significant increased risk of thyroid cancer. While it is impossible to exclude the possibility of a low risk associated with this exposure, nonetheless, it appears clear that exposures in adult life are associated with minimal risk. Contrasting studies of childhood exposures, I-131 appears considerably less effective in inducing thyroid cancer than acute exposure to x or gamma rays.

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**THYROID CANCER INCIDENCE IN IODINE DEFICIENT AREAS
EXPOSED TO RADIATION AFTER CHERNOBYL ACCIDENT**

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Abstract

In two districts of Southern Poland; Krakow and Nowy Sacz (2 million people), standardised thyroid cancer incidence rate IR was evaluated from 1976 to 1992. This area was classified as a moderate iodine deficiency endemic goiter region and belonged to the most contaminated areas in Poland after Chernobyl accident. Recalculated - in terms of real iodine intake-thyroid commitment equivalent dose in the youngest age groups reached 100 mSv. The aim of the study was to evaluate, 6 years after Chernobyl accident, thyroid cancer incidence rate and histotype. Age and sex specific incidence rate in Krakow for male was 0.827 and for female 3.093 and 0.93 and 2.164 for Nowy Sacz respectively. There was no increase of IR in the group of age 0-19 yrs, however in the group over 44 yrs in females in the period of time 1989-1992 significant increase of follicular cancer was observed. In the histotype, the follicular cancer predominated over the papillary one: 42,9% against 33,6%. Predominance of the follicular cancer is typical for iodine deficient area. Significant increase of the follicular cancer may be due to the increase of iodine deficiency.

1. INTRODUCTION

Iodine deficiency and endemic goiter are regarded as risk factors of thyroid malignant neoplasm's-mainly of the thyroid cancer. It is also generally accepted that the thyroid gland is very sensitive to ionizing radiation especially when exposure appears during childhood. The Department of Endocrinology of the Jagiellonian University and Institute of Oncology in Kraków have been developing since 1976 thyroid cancer register in two districts: Kraków and Nowy Sacz. This area with about 2.0 mln people represents a very well-defined iodine deficient part of the Carpathian

endemic goiter, subjected to iodine prophylaxis up to 1980 when, because of economic reasons, iodization of table salt was suspended. Through the 80-s, two events took place: slow increase of incidence of goiter due to lack of iodine prophylaxis, and in 1986 radiation exposure due to ¹³¹I fallout after Chernobyl accident, enhanced by increased thyroid uptake in general population [2, 9, 12, 13, 14].

In 1992 in Nature [6, 11] report on great increase in the frequency of thyroid cancer in children up to 13 years old in Gomel district in the south-eastern part of Belarus was published. This increase started only 4 years after Chernobyl accident-surprisingly short time by comparison with other studies and this phenomenon motivated us to start a comparative study on thyroid cancer incidence in Kraków and Nowy Sącz region. This area is comparable to Gomel district in terms of population (2.0 and 2.5 mln people respectively) and iodine deficiency, because in WHO report in 1960 a southern part of Belarus was indicated as endemic goiter region. The contrasting difference is the level of radiation dose to thyroid in comparable age group of children 0 - 0,1 Gy in Kraków and Nowy Sącz against 1-5 Gy-s in Gomel district. Therefore the aim of our study is as follows:

1. To evaluate a thyroid cancer incidence rate in well defined iodine deficient area before and after exposure to radiation dose due to Chernobyl accident.
2. To carry out a stratified analysis of thyroid cancer incidence by its histotype, age and sex in the period of time relevant to radiation exposure.
3. To test the hypothesis that low dose radiation in iodine deficient area may result in detectable increase of thyroid cancer incidence 6 years after Chernobyl accident.

2. MATERIAL AND METHODS

The thyroid cancer register was developed on the basis of two independent sources: regional register of the Institute of Oncology in Kraków and registers of all the histopathologic laboratories in the area. The register of the Oncology Institute was based on the reports on autopsy or microscopic examination coming from hospitals. Therefore to enter the main register created in the Department of Endocrinology at least one of the two criteria had to be fulfilled: death certificate based on autopsy or microscopic examination according to WHO classification [5, 10]. Incidence rate was calculated as a number of newly-diagnosed thyroid neoplasm's in calendar year adjusted to age group per 10⁵. The observation time was divided into three

Tab.1.

Number of thyroid malignant neoplasms by age and sex in Kraków and N.Śącz districts (1976-1992)

	< 19	19 - 44	> 44		Total	
	N	N	N	N	%	F / M
KRAKÓW						
M	1	9	71	81	14,1	3,9
F	7	56	255	318	55,4	
N. ŚĄCZ						
M	2	6	43	51	8,8	4,0
F	1	15	109	125	21,7	
Total	11	86	478	575		
%	1,91	14,96	83,13		100	

Tab.II.

**Age specific incidence rate per 10⁵ of thyroid malignant neoplasms in Kraków
and Nowy Sącz districts by sex (1976-1992)**

	years	<19(N=11)	20-45(N=86)	>45(N=478)	Total (N=575)	Total		
KRAKÓW	1976-1985	0.056	0.174	2.101	0.672	0.827		
	M	1986-1989	0.000	0.320	2.396		0.806	
		1990-1992	0.000	0.283	4.366		1.350	
		1976-1985	0.235	1.083	5.452		2.206	
	F	1986-1989	0.140	1.495	7.296		3.125	3.093
		1990-1992	0.370	2.410	13.780		5.770	
<hr/>								
N. SĄCZ	1976-1985	0.156	0.098	3.302	0.939	0.936		
	M	1986-1989	0.000	0.210	2.384		0.673	
		1990-1992	0.000	1.080	3.549		1.273	
		1976-1985	0.082	0.851	5.922		2.122	
	F	1986-1989	0.000	0.000	6.280		1.920	2.164
		1990-1992	0.000	1.662	6.783		2.612	

Tab.III.

**Histopathological verification of thyroid malignant neoplasms in
Kraków and Nowy Sącz districts (1976 - 1992)**

	1976-1985	1986-1989	1990-1992	1976-1992	
	Number of cases	Number of cases	Number of cases	Subtotal	Total
Follicular cancer	64	33	61	158 (43,9%)	158 (27,5%)
Papillary cancer	49	34	19	102 (28,3%)	102 (17,7%)
Anaplastic cancer	28	9	11	48 (13,3%)	48 (8,3%)
Medullary cancer	3	1	1	5 (1,4%)	5 (0,9%)
others	1	18	28	47 (12,7%)	47 (8,2%)
subtotal	145	95	120	360 (100%)	360 (62,6%)
non verified	109 (42,9%)	48 (33,6%)	58 (32,6%)		215 (37,4%)
total	254	143	178		575

Tab.IV.

Age specific incidence rate (per 10⁵) of microscopically verified
thyroid malignant neoplasms in Kraków and N.Sącz districts
by sex (1976-1992)

	years	<19(N=6)	20-45(N=71)	>45(N=283)	Total (N=360)	Total		
KRAKÓW	1976-1985	0.000	0.087	0.891	0.283	0.439		
	M	1986-1989	0.000	0.320	1.198		0.467	
	1990-1992	0.000	0.142	2.977	0.900			
	F	1976-1985	0.176	1.039	3.162		1.453	
	1986-1989	0.140	1.175	6.042	2.551		2.198	
	1990-1992	0.370	1.985	9.086	3.987			
N. SĄCZ	1976-1985	0.000	0.098	1.143	0.324	0.441		
	M	1986-1989	0.000	0.210	0.894		0.299	
	1990-1992	0.000	0.540	3.155	0.979			
	F	1976-1985	0.000	0.567	2.763		1.031	1.160
	1986-1989	0.000	0.000	3.489	1.067			
	1990-1992	0.000	1.662	3.700	1.679			

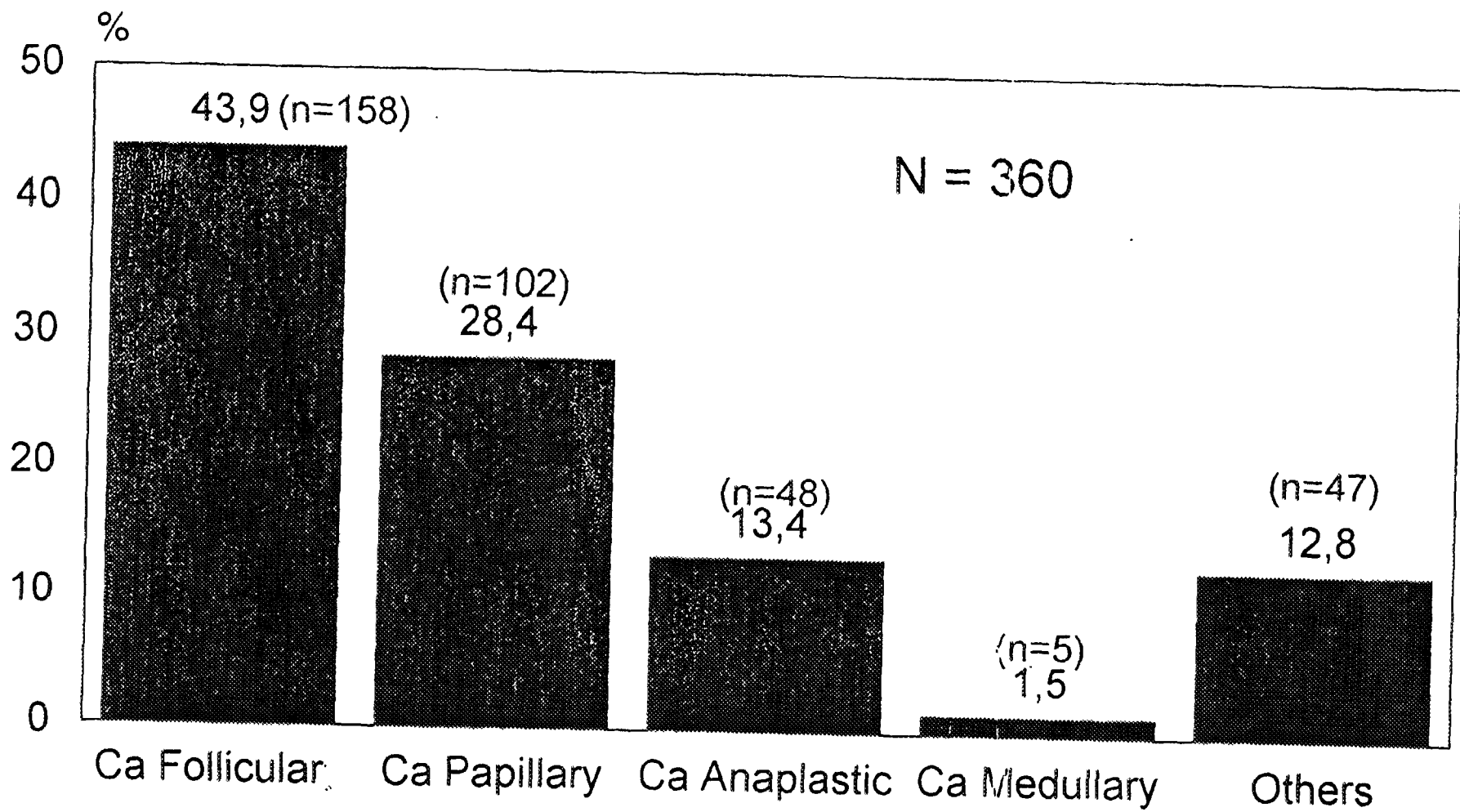


Fig.1
 Histopathological verification of thyroid cancer in Kraków and
 Nowy Sącz districts (1976-1992)

Tab.V.

**Case studies of the youngest group (0-19) of thyroid cancer in
Kraków and N.Śącz districts**

	n(11)		age (years)		diagnosis	
	Kraków	N. Śącz	Kraków	N. Śącz	Kraków	N. Śącz
1976	1	1	18	14	N	P
1977	1	1	11	9	N	N
1978						
1979						
1980						
1981						
1982						
1983	1		18		N	
1984	2		18,17		P,P	
1985		1		1		N
1986	1		14		P	
1987						
1988						
1989						
1990	1		<1		N	
1991	1		19		F	
1992						

N- Non-verified

P- Papillary cancer

F- Follicular cancer

intervals: 1976-1985 - before Chernobyl accident, 1986-1989 and 1990-1992 after the accident. Apart from incidence rate a stratified analysis by histotype, age and sex was performed.

All data were collected on data base files using IBM computer and D-Base Packet. The results were verified statistically the aim of Pearson-Chi square test.

3.RESULTS

In period of the time 1976-1992, 575 newly diagnosed thyroid malignant neoplasms in Kraków and Nowy Sacz districts were registered. Tab.I shows the number of all the registered thyroid neoplasms by sex and age. The group of age 0-19 years comprises 2% of the total number of registered thyroid neoplasms while the majority (83,13%) it is the age group over 44 years-predominantly females (F/M ratio is about 4). Tab.II illustrates a specific incidence rate adjusted to age and sex in the intervals of time before and after Chernobyl accident. Statistically significant increase of incidence rate is observed in the group of age over 44 years-mainly after 1990 in females in Kraków region. There is no significant increase in the youngest group of age 0-19 years. In our material 62,6% of cases is verified in terms of their histotype (tab.III). Tab.IV shows specific incidence rate of microscopically verified thyroid neoplasm's by age, sex, and intervals of the observation time. The prevailing type of thyroid cancer is the follicular cancer in females over 44 years (Fig.1.), where incidence rate has increased statistically significantly since 1986.

To answer the question if thyroid cancer incidence has increased since 1986 in the youngest group of age, we have analyzed case by case since 1976 (tab.V) and no increase after 1986 was observed.

DISCUSSION

Ethiology of the thyroid cancer is multifactorial (tab.VI). [3,4,7] Environmental factors like iodine deficiency, radiation and diet associate with oncogenes and growth factors. In the investigated area two leading factors were well defined: iodine deficiency and exposure to radiation after Chernobyl accident. Kraków and Nowy Sacz districts are situated in the part of Carpathian mountains where iodine deficiency and endemic goiter were observed through centuries. In the nation-wide investigation on iodine deficiency in Poland completed in 1993, in this region moderate degree of iodine deficiency and prevalence of endemic goiter-according to ICCIDD criteria -

Etiologic factors of thyroid cancer

- **Exposure to ionizing radiation**
- **Diet**
 - Iodine deficiency**
 - Vit D, Calcium**
 - Ethanol**
- **Hormonal factors**
 - TSH stimulators**
 - Female reproductive hormones**
 - Epidermal Growth Factor**
- **Genetic factors**
 - Families of oncogenes**
 - Intracellular signal transduction (c-ras)**
 - Gene transcription (c-myc)**
 - Growth Factors (sis)**
 - Epidermal Growth Factors receptors (erb B)**
 - Inherited Medullary Thyroid Cancer**
- **Pharmacological compounds**
 - Contraceptive**
 - Lactant suppressors**
 - Barbiturates**
 - Antihistamines**
 - Spiroinolacton**
 - Griseofulvin**

were found [14]. Determination of iodine concentration in urine and thyroid volume by means of USG in children 6-13 years old, allowed to recalculate the dose to thyroid due to Chernobyl accident. Using Johnson's model Olko and Niewiadomski evaluated radiation dose to thyroid in children within the range 100-200 mSv assuming, that endemic situation has not been changed significantly since 1980 when iodine prophylaxis was suspended in Poland. This dose level is still within the range of the low level radiation but much far from mean dose evaluated for Poland after Chernobyl accident [4, 9]. It is much more close to the dose calculated during Polish programme MZXVII [9].

Iodine deficiency has been recognized as a risk factor for thyroid cancer. In the investigated area thyroid cancer register has been developed on the population base since 1976 and is characterized by two features: about two times greater incidence rate with comparison to a mean value in the country and predominating follicular cancer in females over 40 years [13].

The Kraków and Nowy Sącz regions represent "far field" - 800 km from Chernobyl and was exposed to the low level radiation. This area is comparable to Gomel region in Bielorussia in terms of population size (around 2 millions) including subpopulation of children 0-5 years at time of accident, and similar prevalence of endemic goiter recognized during WHO investigation in 1958. The main differences are: radiation dose level to thyroid about 10 times greater in Gomel region (0,75 - 2,0 Gy), and increase of prevalence of papillary cancer in children which was found to be relatively aggressive with distant metastases to the lungs [6, 11].

An analysis of our data shows, that there was no apparent difference in the incidence rate of thyroid cancer in the youngest age group 0-19 years in the period before and after Chernobyl accident. When the data were subdivided into the different types of tumors there were no differences observed after 1986: still predominating type of thyroid cancer it was follicular cancer mainly in women over 40 years. However significant increase of incidence rate in this group was observed especially in Kraków region.

Interpretation of these results should be careful. There is no reason to regard radiation as a causative factor of the increased number of follicular cancer when it represents different group of age and different type cancer as well - in comparison to known effect of radiation on thyroid cancer in humans. The most probable causative factor is the increased iodine deficiency in Poland due to suspension of iodine

prophylaxis in 1986. However, the surprising increase of number of thyroid cancer in the Gomel region 4 years after exposition needs a prolonged observation in the "far field".

Comparative studies in the areas representing different levels of thyroid dose due to Chernobyl accident seem to be valuable for evaluation of the risk coefficient of thyroid cancer due to ionizing radiation. However, this type of study raises new methodological problems. First of all, in endemic goiter areas representing well defined iodine deficiency, radiation dose to thyroid should be recalculated, because so called "mean population radiation dose to thyroid" after Chernobyl may be far from realities. Secondly, calculation of the risk coefficient should be based on the incidence rate of thyroid cancer - instead of on mortality rate. Life expectancy in the case of thyroid cancer depends on many other factors having no interrelationship with radiation dose absorbed in the thyroid. Thirdly, classification of thyroid malignant neoplasms must be unified according to WHO classification. Evaluation of the risk coefficient of thyroid cancer due to radiation after Chernobyl accident in the areas with different levels of exposition may provide a rational basis for a limit dose system and for establishing of intervention level in case of a nuclear accident [1, 8].

CONCLUSION

1. In the period of time from 1986 to 1992 no increase in thyroid cancer incidence in the age group from 0 to 19 was observed.
2. In the period of time from 1989 to 1992 statistically significant increase in follicular thyroid cancer in females over 44 years old in Kraków region was observed.
3. Increase of incidence rate of follicular cancer in females over 44 may be caused by increase of prevalence of goiter due to suspension of iodine prophylaxis in Poland in 1980.

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**PREVENTIVE MEASURES IN THE EVENT OF A
NUCLEAR ACCIDENT**

(Session II)

Chairmen

M. Gembicki, Poland

A. Gonzalez, IAEA



RADIATION EMERGENCY PLANNING IN POLAND

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Abstract

The paper presents a schematic outline of the radiation emergency policy in Poland, rather from the point of view of logistics of the problem than discussing details of existing or proposed procedures.

1. INTRODUCTION

There are no power plants in Poland; the Polish nuclear power programme was interrupted in 1990 when the Polish Parliament decided that construction of the nuclear power plant in Zarnowiec (two WWER-440/213 units had been constructed since 1982, two other were planned) was not to be continued. However, nonexistence of nuclear power plants or nuclear fuel processing installations on Polish territory does not mean that radiation emergency situation cannot occur in Poland and that radiation emergency planning is not needed.

According to the Atomic Energy Act [1], the National Atomic Energy Agency (NAEA), being a governmental nuclear regulatory body in Poland, is responsible not only for licensing and inspection of all the activities involving atomic energy (including application of radiation sources in industry, medicine and R&D), but also for radiological protection of the whole population. Therefore, a proper planning and execution of intervention procedures needed in case of radiation emergency situation, based on monitoring system, early warning information network and decision supporting programmes, play a most important role in the NAEA legislative and executory activities.

2. POTENTIAL SOURCES OF RADIATION EMERGENCY

Radiation emergency in Polish reality may be caused by the incidents in:

- nuclear (research) installations in Poland or nuclear installations in neighbouring countries;
- radioisotope laboratories and radiowaste management system;
- transportation of radioactive materials within the country and declared or undeclared (illegal) trans-border transportation.

Polish nuclear installations consist of two research reactors, 10 and 30 MWth of power, and of a spent fuel interim storage facility containing about 5000 spent fuel elements (of about 2×10^{16} Bq activity), all located in a research centre at Swierk, 20 km from Warsaw.

There are about 2750 isotope laboratories in research institutions, hospitals and clinics and in industry; 30 laboratories use radioisotopes in field works. More than 420 utilise open, unsealed sources (445 of the lowest, 60 of the medium, and 15 of the highest category/class, and more than 15 in the field). Radioisotopes of different kind and for various purposes (mainly radiopharmaceuticals) are offered by 15 producers through more than 70 dealers. The biggest manufacturer of radioisotopes in Poland is OBRI (R&D Centre of Isotopes) at Swierk which in 1993 produced and sold 4000 sealed sources and 82000 portions of unsealed radioactive material (2×10^{15} Bq and 5×10^{13} Bq, respectively). This list of users of radioactive materials does not include all the applications of radioisotope (Pu or Am) smokedetectors (about 1 mln detectors in more than 6000 institutions). [2]

Polish radioactive waste immobilization and deposition system consists of the waste treatment installation at Swierk and the landfill low and medium activity waste (RMI type) repository at Rozan (100 km from Warsaw); annual amount of wastes processed and finally stored being about 175 m^3 of liquids and about 100 m^3 of solids, the total activity deposited in 1993 was about 10^{14} . [2]

Within the range of 200 km of a Polish border there are 7 nuclear power plants with 17 active units of total installed capacity of 14 GW(e). If the 600 km range is considered, the total number of nuclear power plants is 26 (50 reactor units), including the Chernobyl-type RBMK reactors and the oldest versions of WWER reactors.

Potentially all the mentioned above installations and radioisotope users can create an emergency situation and can cause a need for intervention. Signals in such situations may come to the decision making centre directly from the user (or from a special service operating at the site, e.g. police, fire brigades etc.), through special monitoring stations (contamination measuring units) or from the exchange-of-information contact point (due to the international and bilateral obligations and agreements signed in Poland).

3. RADIATION MONITORING

The radiation monitoring system as considered from the point of view of emergency planning is now under reconstruction. Generally, it has to consist of two subsystems

-a core subsystem, depending (administratively and financially) entirely on the National Atomic Energy Agency (NAEA), containing about 10 field stations spread throughout Poland,

-auxiliary system, i.e. other stations belonging to other Polish institutions (under ministries of health, of environmental protection, of defence etc.), providing data for the purposes of early notification on emergency situations, if those data correspond to the requirements and standards set up by the NAEA

The radiation monitoring system can operate at four different levels of activity, depending on the degree of emergency situation. These levels can be defined as

-normal (all readings correspond to the background within the agreed level of discrepancies),

-elevated (there exists a possibility of an increase of the radioactive contamination due to the accident reported to the centre),

-emergency (the reading exceeds significantly an agreed background level or there are confirmed in reports on the accident which may create the emergency situation),

-post-emergency (during the period of time between the occurred emergency situation and the return to the normal = background level readings)

The measurements performed by field station consist of the following elements

-gamma-ray dose (in air),

-radioactivity of precipitation,

-radioactivity contamination of suspended air particulates,

-radioactivity of other environmental samples (soil, water, plants, foodstuff and feedingstuff),

including spectrometry, if identification of radionuclides is needed. Different levels of the measuring system generally mean the differences in timing regime of the measurements or of reporting on the results [3, 4, 5]

4. DATA ANALYSIS AND INTERVENTION

All the information coming from the radiological monitoring system (in real time or as the post-measurement reports, e.g. on the mean value of air contamination by

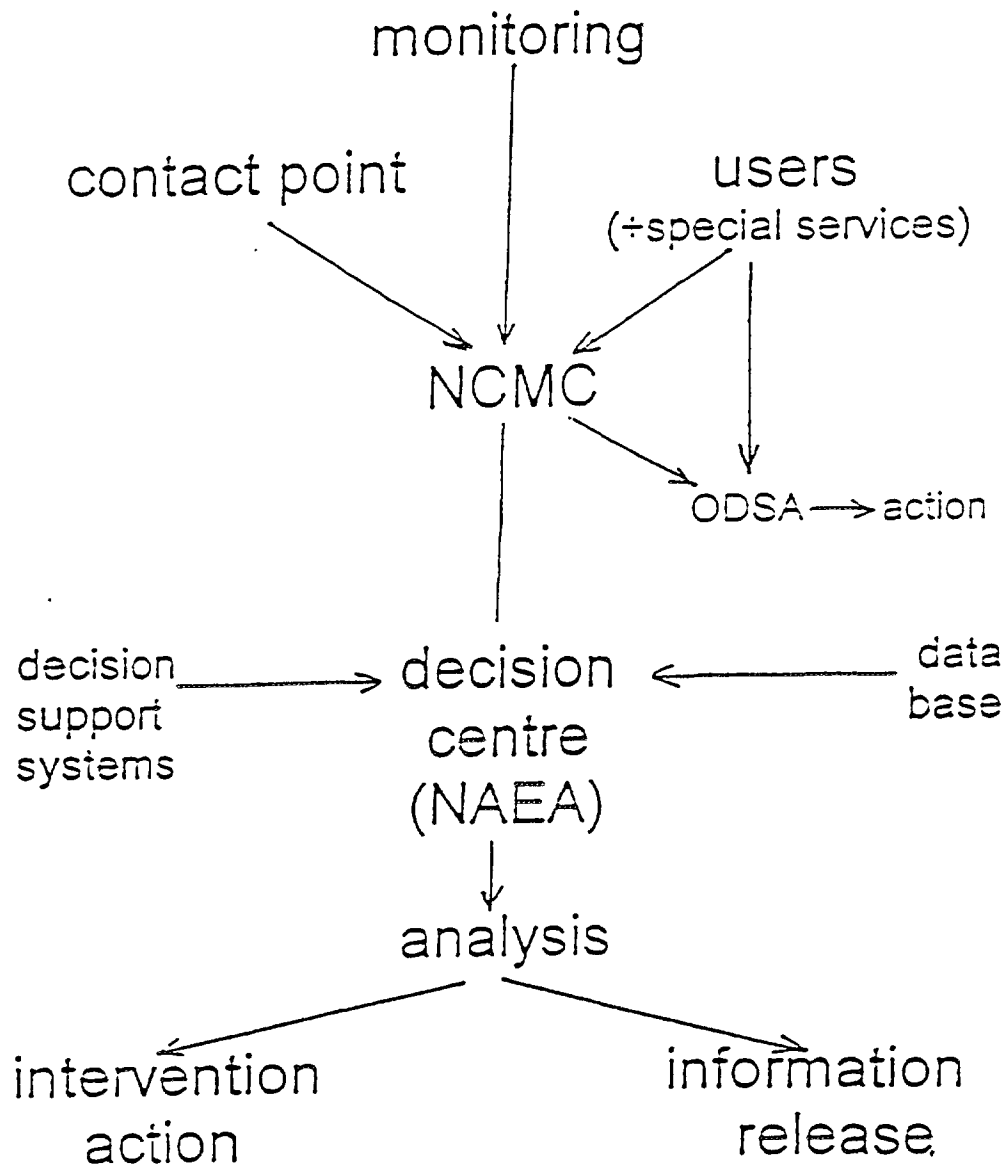


FIG. 1. Radiation emergency system scheme.

different isotopes in a given period of time, etc.), together with information obtained by the contact point from the IAEA in Vienna and from neighbouring countries as well as information on radiation incidents from radioisotope users and special services, are collected and analysed by the National Contamination Measurement Centre (NCMC), operating under the NAEA. If an immediate action is needed in case of accident in laboratories and on the roads or other emergencies of very limited character, a special action squad (so called ODSA) is sent to handle the situation (co-operating, if needed, with local police and/or fire brigades). If the emergency situation is of more serious character (from the point of view of radiation hazard or of number of people potentially affected) - the decision on the action is left to the decision centre

at the NAEA, assisted by the elaborated decision support computerised programmes and by the access to the databases with information on all domestic and foreign potential sources of radiation emergency situations.

The decision centre, after proper analysis of all the data, undertakes an intervention action, according to the size of the problem, through the specially established action teams and scenarios and releases information to mass media, government officials, co-operating institutions in Poland and abroad, etc.

5. FINAL REMARKS

Different scenarios of intervention actions depend on a scale of an event. Necessity of co-operation of various institutions and services requires preparation and consequent implementation - of a whole of very precise and detailed set of regulations. Actions involving transboundary effects have to be co-ordinated with the neighbouring countries, having in mind obligations resulting from international conventions or bilateral agreements signed by Poland. All procedures, standards, methods of measurements etc. have to be in accordance with international (e.g. IAA, WHO etc.) and other (e.g. AEU) recommendations. The National Atomic Energy Agency is trying at present to improve the existing profile to make it more functional and meeting the mentioned above conditions.

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Abstract

After the Chernobyl accident the stable iodine was used in millions of children and adults for the protection of the thyroid against accidental irradiation after the uptake of radioactive iodine isotopes.

To understand better how stable iodine acts, the development of the thyroid and the synthesis and excretion of the thyroid hormones were described.

The thyroid gland of adult men needs regular daily supply with about 150-200 μg of stable iodine. Iodine under the form of iodines is trapped by the thyroid from the circulation, is concentrated and create intrathyroidal pool of iodine which is large and has long biological half time.

The radioactive iodine isotopes emitted to the atmosphere during nuclear accident were inhaled or digested and later on trapped by the thyroid gland, irradiating the thyroid.

The uptake of radioactive iodine may be prevented in nearly 100% if the stable iodine will be in sufficiently large quantity before the release radioactive isotopes to the atmosphere. It may also, at least partially reduce the thyroid uptake of radioactive isotopes even if given later.

The quantity of stable iodine recommended differs, however most of the data indicate that the dosis of 100 mg given to the adult persons might nearly totally block the thyroid iodine uptake.

The prophylactic action carried out in Poland after the Chernobyl disaster when about 12 million children and 6 million adults received the stable iodine allowed to collect well documented data in this matter.

It was stated that 70 mg of stable iodine is blocking the thyroid uptake with the mean of 82% after 24 hours, 39% after 48 hours and only 26% after 72 hours.

Therefore it seems that the blocking dosis (which may be even less than 70 mg) should be given every 24 hours for a few days hoping that the most serious danger of contamination with radioactive iodine will pass.

The foetus and new-born are especially sensitive to the irradiation because of the small size of their thyroid gland and its ability to accumulate radioactive iodine. They should be protected in the first place, together with pregnant and lactating women as well as children and adolescents.

The possible side effects were discussed but according to the literature and Polish experience they do not play a significant role.

Finally, the importance of a regular, proper alimentation with stable iodine was underlined stating that the thyroid of the person from iodine deficient areas has always higher iodine uptake and is therefore much more exposed to the internal irradiation in case of a nuclear catastrophe.

1. INTRODUCTION

The Chernobyl nuclear plant accident in 1986 and emission to the atmosphere of a large quantity of different radioactive iodine isotopes stimulated scientists and practitioners as well as different international organizations to consider how much the use of stable iodine may protect the thyroid gland against accidental irradiation.

The important role of the thyroid gland in the development, growth and well being of man is well known and no one in fact have doubts that the thyroid should be protected.

The stable iodine seems to be the most important and most effective element which can protect or at least partly protect the thyroid gland against the accidental radioiodine uptake during nuclear catastrophe.

The stable iodine was in fact widely used as the thyroid blocking agent after the Chernobyl accident, undoubtedly on the largest and well organized scale in Poland [4,8]. Nevertheless it was initiated as well in various countries by some groups of people or many individuals. As it happened quite often when the worst threat had gone the discussion started if the prophylactic use of stable iodine following nuclear accident was a proper way to protect the thyroid and if so how to do that and how to limit or even better to eliminate the possible side effects both early and late.

2. IODINE REQUIREMENT AND METABOLISM

It is clear that before using the stable iodine for the protection of the thyroid gland it is necessary to know the ways of the thyroid development, growth and its physiological functions.

The thyroid needs for the production of hormones, which are thyroxine (T4) and triiodothyronine (T3) a regular supply with sufficient quantity of iodine.

This quantity recommended by the WHO is 150 - 200 mcg (as iodide) daily [10].

The other institutions like the Food and Nutrition Board and the National Academy of Sciences, National Council of USA and European Society of Pediatrics Gastroenterology and Nutrition recommended the following daily intake for different groups of people [5,10]:

30 mcg for children from 0 - 6 months

45 mcg for children from 6 - 12 months

60 - 100 mcg for children from 1 - 10 years

100 - 150 mcg for those over 10 years

125 - 150 for pregnant and lactating women

The total iodine contents in the body of a healthy adult person is about 15-20 mg and the quantity of the iodine utilized during the whole human life is about 4-5 g [5].

This indispensable element is delivered to the men and animals with the food and water. Additional source of iodine supplementation may be the iodized salt and different iodine containing medicines (fig 1.)

Figure one shows the model of iodine metabolism. The iodine in the inorganic form of iodides is absorbed from the digestive tract or inhaled from the contaminated air in case of a nuclear accident and create in the circulating blood extrathyroidal iodine pool containing 40-60 mcg of iodine. The iodides are trapped from the circulation mostly by the thyroid. The thyroid pool contains about 10000-15000 mcg of iodine predominantly as iodothyrosines and iodothyronines.

The smaller quantity of iodine is also trapped from the extrathyroidal pool by the salivary glands, stomach, placenta, and breast.

The rest of plasma inorganic iodine is excreted in the major part with urine and in small quantity with the bile and feces.(fig.2.)

Figure 2 shows the iodine turnover in man. It is worth to note that all the phases of iodine turnover are stimulated by thyrotropin (TSH). The inhibiting substances are different at different stages of synthesis and secretion of thyroid hormones.

200 $\mu\text{g}/\text{d}$

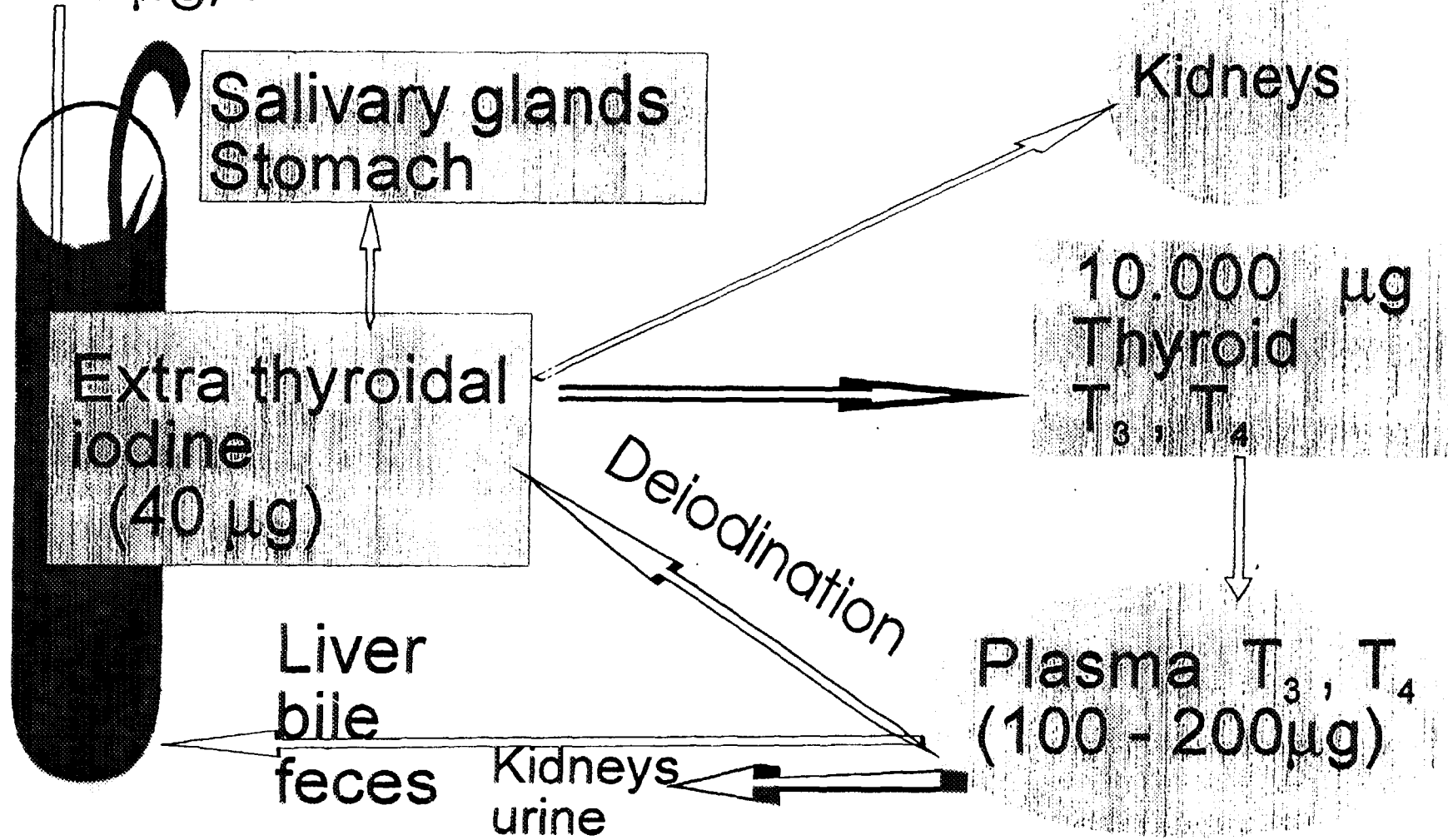


FIG. 1. Model of iodine metabolism.

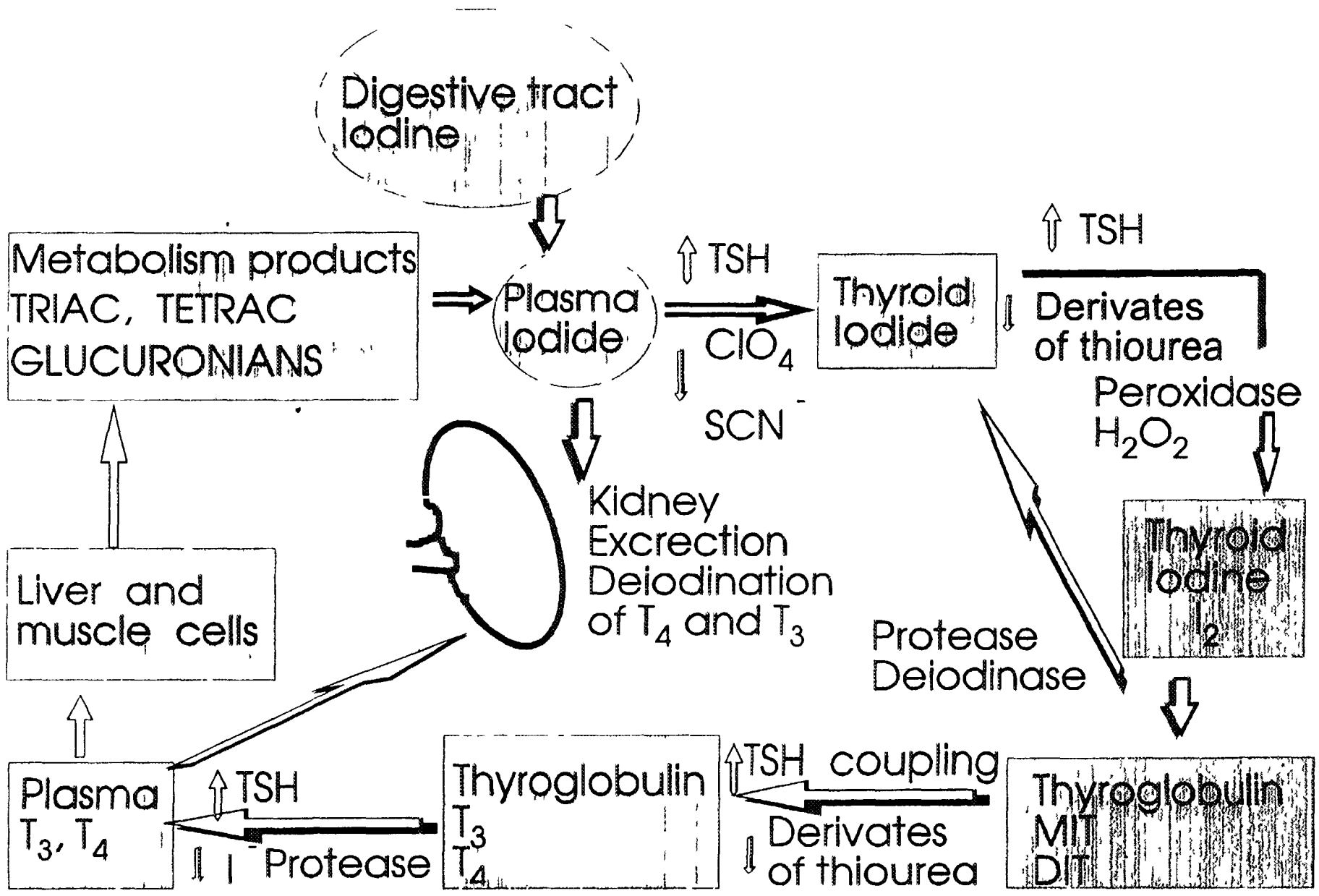


FIG 2 Iodine turnover in men

The trapping process is inhibited by perchlorate and thiocyanate, the oxidation of the iodides to elementary form while organification is inhibited by derivatives of thiourea similarly as coupling of thyrosines. The releasing of T4 and T3 from the thyroglobulin and excretion to the circulating blood may be inhibited by the iodine.

The products of the thyroid hormones metabolism like triiodo and tetraiodoacids (TRIAC and TETRAC) and others under the form of glucuronians are excreted with bile and feces.

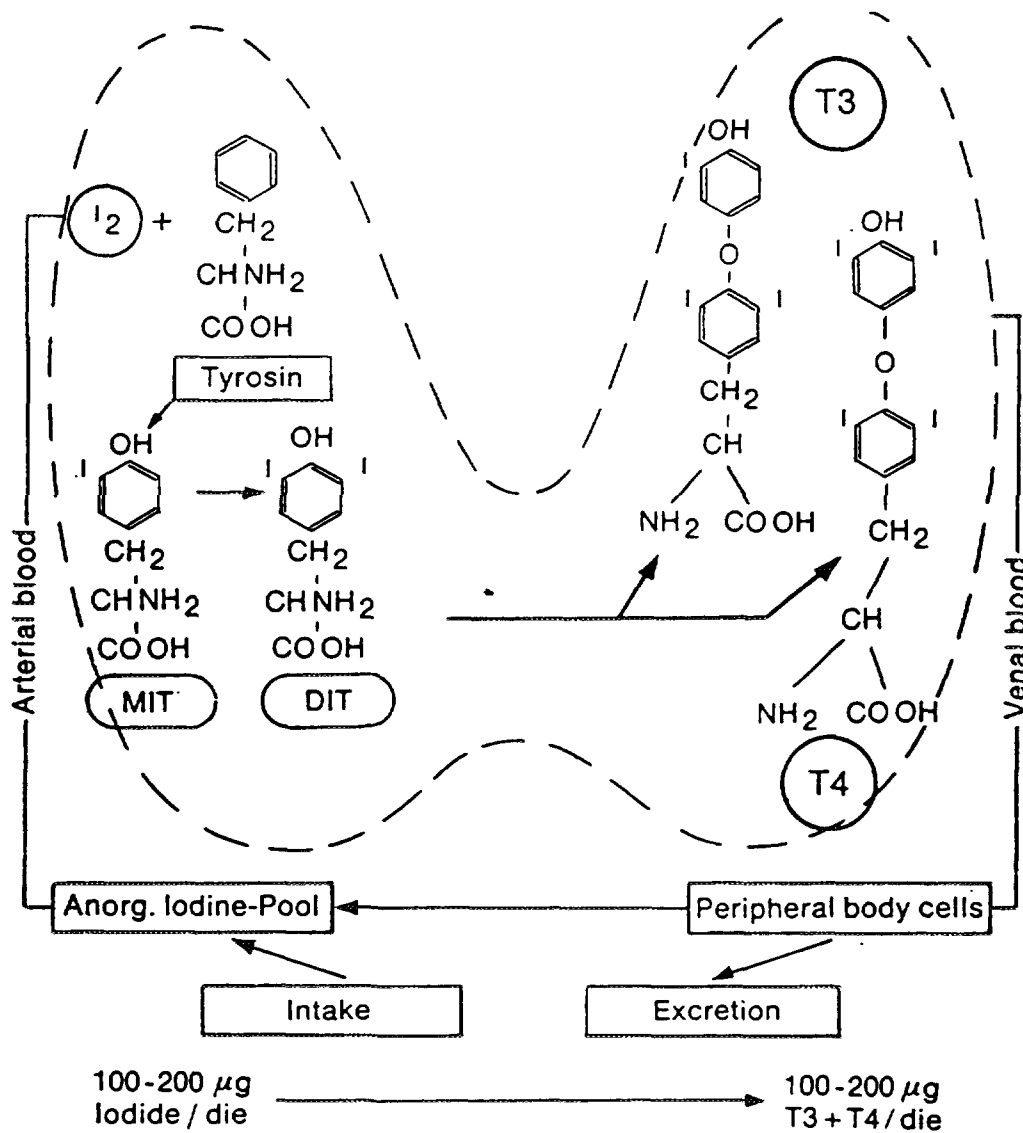
As the majority of the plasma iodides are excreted by kidneys, it is considered that iodine urine excretion is in very close relation to the daily iodine intake and is generally accepted as an index of sufficient or insufficient alimentation with iodine.

The plasma iodine clearance occurs mainly through thyroid iodine trapping mechanism and kidneys excretion. Both processes are fast and as the result of that the plasma iodine turnover has half time of 6-8 hours.

On the contrary the thyroid gland iodine clearance is much slower with biological half time of about 80-100 days. As it was mentioned before, the thyroid iodine content is about 10000-15000 mcg [2]. Considering the daily intake of 100-200 mcg of iodine and the daily excretion of about 130mcg of iodine in the form of 100-200 mcg thyroid hormones (see figure 3), the slow metabolic rate of thyroidal iodine pool is understandable (fig.3.).

The large quantities of stable iodine accumulated by the thyroid gland remain there for a long time. Radioactive iodine trapped by the thyroid is disappearing faster with the so called effective half time which is the combination of both biological and physical half time. However, even this shorter time is long enough for the irradiation of the gland that is why the protection of the thyroid against radioiodine accumulation is so important.

According to Stanbury the ratio of iodine in the thyroid to that in plasma (the T/S ratio) is constant and if the iodine plasma concentration increases the organification of the iodine also increases. However this ratio may fall in case when the plasma concentration of iodine increases above 15-28mcg/dl [11]. Such a situation is known as the Wolff-Chaikoff phenomenon [1,7,13,14]. This phenomenon concerns the inhibition of the organification process which after trapping iodides is the next step in the thyroid hormones synthesis.



(Diagnosis of Thyroid Diseases -
P. Pfannenstiel
Byk-Mallinckrodt Serie - 1975)

FIG. 3. Schematic drawing of the intrathyroidal hormone synthesis.

In the majority of cases this inhibition is rather transient with the duration of few hours or maximum few days. However in some persons this inhibition may persist and develop hypothyroidism or even goiter. The Wolff-Chaikoff phenomenon is independent of the hypothalamo-pituitary control and is an important side effect in case of the use of very large doses of iodine as an thyroid protecting agent. The model of iodine metabolism described above is applicable to the well developed

thyroid in adult person. However the different stages of the thyroid gland development create several other aspects which it is necessary to take into consideration before the application of stable iodine for the thyroid gland protection.

3. THE DEVELOPMENT OF THE THYROID GLAND

The thyroid gland appears early and may be visualised at about 16-17 days of gestation [6]. At that time the thyroid of the fetus is not able to accumulate the iodine. Such a possibility begins after 12-14 weeks of gestation. In case of contamination with radioactive iodine at the time of fetal life the thyroid being very small (1-3 g at terms) but trapping and concentrating the iodine is especially exposed to the radiation. According to Escobar [6] the radioiodine uptake is especially high per gram of tissue at mid gestation and sometimes may be 510 times higher than adult thyroid gland.

Knowing that it is obvious that, first of all the pregnant women and new-borns should be protected just as lactating mothers because of the transfer of the iodine with mother milk to the new-born.

4. POSSIBILITY OF THE PROTECTION OF THE THYROID AGAINST RADIOACTIVE IODINE

The radioactive iodine from the biological point of view, behaves as the stable one. It is trapped, accumulated, concentrated by the thyroid gland and is irradiating the gland. Therefore, it is worth to know how to reduce or eliminate the danger. According to Stanbury it may happen in several ways [11].

1. "By saturating iodine transport and diluting the radioiodine entering the thyroid."
2. "By eliciting the Wolff-Chaikoff effect."
3. "By permitting an increased storage of iodine leading to attainment of a new steady state at a lower level of radioiodine uptake."
4. "By impeding recirculation of radioiodine or by impeding or enhancing the release of stored iodine."
5. "By the schedules by which the radioactive and stable iodine enter the body."

The first way which may be realised by application of proper quantities of stable iodine seems to be most useful and most effective.

The second looks less promising because this is in fact the unwanted effect and its nature is transient.

The third is very important but not applicable in the emergency situation because quite long time is needed to obtain the reduction of the iodine uptake by the thyroid. However, this way has to be taken into consideration and should be advisable as the long-term prophylaxis in case of atomic plant workers or people living in the neighbourhood.

The fourth is difficult to realise because it will be necessary to accelerate the thyroid gland metabolism and make the effective half time of the accumulated radioiodine very short.

The fifth is a crucial one because the prophylactic activities should start immediately after releasing the radioiodine to atmosphere and such time relations determine the success of the whole action.

According to Stanbury the inverse relation exists between the thyroid uptake of radioiodine and supply of iodine in the diet [11].

It is known from different publications that there are still large groups of world population living in the iodine deficient areas. If in such places the daily supply of iodine is below 100mcg/day the thyroid uptake of the radioactive iodine will be high reaching sometimes 40-60%.

In the areas with high daily iodine intake, for example 150 - 500 mcg/day (like in the USA and Japan) the thyroid radioactive uptake will be about 20%.

It is evident that the exposure of the thyroid gland to the radioactive iodine in case of nuclear accident is much bigger in cases with higher thyroid radioiodine uptake than with a low one.

5. QUANTITY OF STABLE IODINE BLOCKING THE RADIOACTIVE IODINE THYROID UPTAKE

The time when the stable iodine was given is a very important factor, because the blocking effect starts with the moment when the sufficient quantity of iodine reaches the thyroid. If the stable iodine is given before the radioactive one the protective effect will be, of course, much more effective than when it is done after inhalation or digestion of radioactive iodine.

There are several observations and investigations done in connection with this matter [4,11,12].

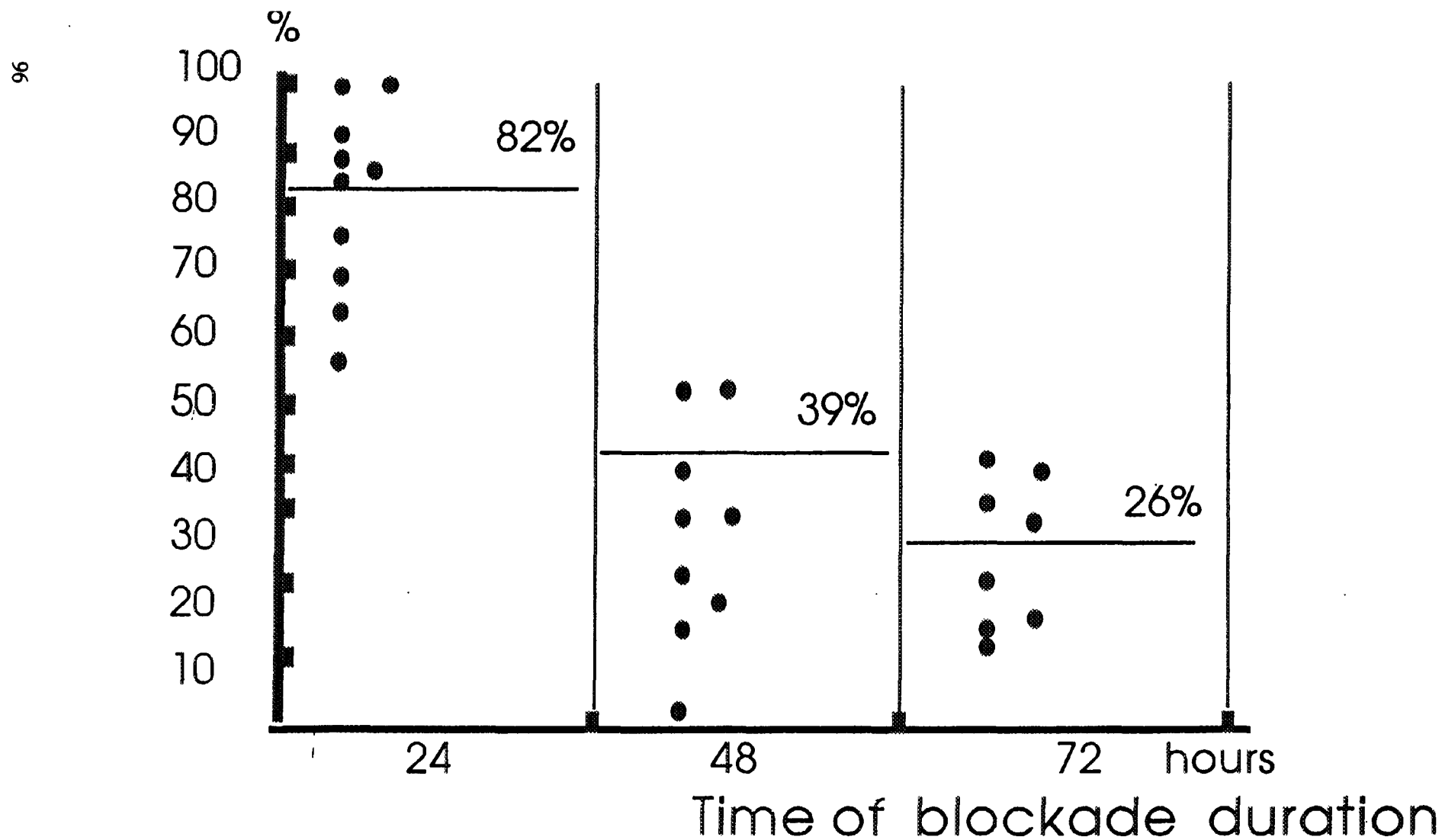


FIG. 4. Thyroid iodine uptake blockade efficacy.

Stanbury says that the dose of 100mcg of stable iodine will be effective up to 60% in 24 hours and 25% after 40 hours [11]. However if the radioactive iodine is trapped by the thyroid gland before the stable iodine was given (this happened during the last catastrophe in Chernobyl) the blocking effect up to 50% of iodine uptake may still occur considering the stable iodine was given no later than three hours after the emission of the radioactivity to the atmosphere. If stable iodine was taken later, for example 10 hours after an accident, immediate blocking effect would be much lower because of rapid accumulation of radioactive iodine prior to blocking.

It is important to remember, however, that the plasma iodine concentration of 10 mcg/dl is blocking further uptake of radioactive iodine which may be released to the atmosphere later on in case of prolonged emission of radioactivity (as it happened during Chernobyl catastrophe).

This is especially important because it is known that there are 22 different radioisotopes of iodine. Majority of them have so short physical half time of desintegration that it is possible to skip them. However, some of them, especially those inhaled soon after the emission of the radioactivity to the atmosphere might be responsible, beside ^{131}I , up to 40-50% for the total irradiation of the thyroid. It is also possible that they are more responsible than ^{131}I for the carcinogenic effect occurring in the thyroid.

The problem of the size of the dose of stable iodine used as a blocking agent is presented differently by the authors performing the investigations. Most of them observed nearly complete blocking of the thyroid radioiodine uptake after the dose of 100 mcg iodine or more.

Our own investigations done on healthy adult volunteers shows that after a single dose of 70 mcg of stable iodine (in the form of Lugol's solution) blocked the thyroid radioiodine (^{131}I) uptake with the average of 82% after 24 hours, 39% after 48 hours and 26% after 72 hours (3). The dose of 70 mcg and Lugol's solution were used because they corresponded to the quantity and form of stable iodine applied in Poland for the thyroid protection after the Chernobyl accident. The results are shown in figure 4.

5. POSSIBLE SIDE EFFECTS AFTER THE PROPHYLACTIC USE OF LARGE QUANTITIES OF IODINE

The application of stable iodine in large quantities may beside the expected blocking of thyroid iodine uptake cause also several side effects as well. One of them is Wolff-Chaikoff phenomenon already described. This one concerns the intrathyroidal disturbances in organification of the iodine.

The others may affect digestive tract causing stomach pains, vomiting and diarrhoeas as well as skin rash, breathlessness or pains of the thyroid.

The Polish experience in this matter shows that with the dose of 70 mcg of stable iodine given to adults and lower doses in case of children (reduced according to age) such complications were rare and very seldom needed medical assistance. The Wolff-Chaikoff phenomenon was not noted either in children or adults.

There are suggestions that the iodine, especially when given in large quantities, may cause autoimmune thyroid disorders and disturb the thyroid gland functions.

Four years after Chernobyl catastrophe and distribution of 30-70 mcg of iodine to several millions of children and adults in Poland, the antithyroglobulin and antimembrane antibodies were elevated only from 1.8 to 7.0% in different age groups. The 7.0% was found in adult women's group consisting of more than 12 thousand investigated persons. All these figures are well corresponding to the values observed before this prophylactic action.

Our observations are in agreement with the statement of Pinchera that "there is no evidence that excess iodine may de novo elicit thyroid autoimmunity in normal subject, particularly when given for short period" [9].

CONCLUSIONS

1. The physiological properties of the iodine metabolism of the thyroid gland create suitable conditions for the protection of the thyroid gland against contamination with radioactive iodine and internal irradiation.
2. The intake of stable iodine in sufficiently large quantities is saturating the thyroid gland and if it happens before the inhalation or ingestion of radioactive iodine the thyroid radioiodine uptake may be blocked significantly and quite often up to 100%.
3. The stable iodine given as the protective agent after inhalation or ingestion of the radioactive iodine is not very efficient, however still might reduce the further uptake of the iodine, taken with contaminated food, considering the iodine plasma concentration will be in the range of 10-15 mcg/dl.

4. The small size of the thyroid of fetus and newborn, its high radiosensitivity and possibilities of radioiodine uptake create a special danger of very high irradiation. Therefore, the pregnant women, newborns and lactating mothers should be treated as a group needing in the first place prophylaxis with stable iodine in case of a nuclear accident.

5. On the basis of Polish experience it seems that better protection of the thyroid gland might be achieved when the prophylactic dose, even smaller than 70 mcg, will be repeated every 24 hours for a week. The blockade of the radioiodine uptake will be more complete and the thyroid gland will be better protected against internal irradiation.

6. From the point of view of the thyroid gland physiology the important protective factor against the irradiation in case of a nuclear catastrophe is a proper supplementation of the iodine in everyday diet prior to the accident. The daily delivery of about 200 mcg of stable iodine will reduce the thyroid iodine uptake significantly and diminish in this a way the danger of internal irradiation.

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**COMPLICATIONS OF HIGH DOSE IODINE THERAPY
IN THE CASE OF REACTOR ACCIDENTS**

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Abstract

In the case of a reactor accident pharmacological doses of iodine should be applied in order to reduce iodine 131 thyroid uptake. The risks may be twofold: iodine goiter only in a minority of predisposed persons or iodine induced hyperthyroidism in people with autonomously functioning thyroid tissue (AFTT). The risk of iodine induced hyperthyroidism in patients with AFTT increases with the amount of AFTT and depends on the amount of administered iodine, too. Small doses of iodine up to 100 μ g/day do not induce hyperthyroidism in these patients. But the optimisation of the daily iodine supply with 200 μ g/day is already sufficient to induce latent hyperthyroidism in 14% and clinically manifest hyperthyroidism in 4% of the patients with AFTT. The amount and the activity of autonomous thyroid tissue can be estimated by the determination of the Tc-99m-pertechnetate uptake during TSH-suppression (TcTU_{supp}), because TcTU_{supp} and the volume of autonomous thyroid tissue (V_a) correlate linearly. All patients with got hyperthyroid had a TcTU_{supp} higher than 3,3%. From that the "critical" volume of autonomous thyroid tissue can be calculated to be between 8 and 12 ml, which equals a solitaire nodule with about 2.8 cm diameter. On the other hand an enhanced risk of hyperthyroidism can be excluded with a high probability in those patients whose TcTU_{supp} is less than 2%.

1. INTRODUCTION

In order to protect the population as effectively as possible from the sequelae of a reactor accident, the storage of radioactive iodine isotopes in the thyroid must be prevented. The storage of radioiodine can be prevented by administering large amounts of nonradioactive stable iodine prior to any uptake of radioactive iodine. Due to the limited storage capacity of the thyroid, saturation of the iodide pump and autoregulatory inhibition of iodine uptake, only a minimal amount of radioactive iodine can then be stored in the thyroid [24]. The remainder is excreted via the kidneys with a biological half life of about 6 hours [15, 24].

Potassium iodide tablets are most effective if they are taken a few hours before or during exposure to radioiodine. If the tablets are taken in time, the iodine load in the thyroid will be less than 1%, corresponding to a reduction of the radiation dose by more than 97% [15]. If, however, tablet intake is delayed by about 5 hours, as much as 50% of the absorbable radioactive iodine isotopes has already been stored in the thyroid. Eight hours after absorption, the ingestion of potassium iodide tablets has no further influence on the storage of radioactive iodine in the thyroid and thus on the radiation load. To achieve that the uptake of radioactive iodine isotopes is blocked as completely as possible, a high plasma concentration of stable iodine has to be reached. For this reason a relatively high initial potassium iodide dose, at 200 mg for adults, is necessary. This high plasma concentration of stable iodide must be maintained as long as there is a risk of radioactive iodine isotopes being taken up. Therefore it is recommended to take a further dose of 100 mg potassium iodide dose every 8 hours. The dose given to children should be half of the dose being recommended to adults. New-borns during the 1st month of life only receive one single dose of up to 25 mg potassium iodide. After birth the neonate should be subjected to careful clinical and laboratory-technical examination to detect hypothyroidism. Since in the third trimester the fetus reacts particularly sensitively to high iodine doses through the placenta, the administration of potassium iodide should be restricted in pregnant women to 2 daily doses [24]. Iodine may act either as an antithyroid drug and induce hypothyroidism with goiter, or serve as a thyroid hormone precursor and induce hyperthyroidism. In large pharmacological doses of more than about 2 mg iodide acts as an antithyroid drug due to the Wolff-Chaikoff effect [31], but this effect is only temporary. Pharmacological doses of iodine are usually required to induce iodide goiter. On the other hand, even small physiological doses may induce hyperthyroidism in those patients with some autonomy of thyroid function, either primary due to the thyroid itself, or secondary due to a thyroid stimulating agent. Iodide induced thyroiditis or extrathyroidal side-effects like sialadenitis, gastrointestinal symptoms, iodide fever or allergic reactions are very rare [24, 32]. Thus the main risks of high iodide doses may be twofold: iodide goiter in predisposed persons or iodide induced hyperthyroidism in persons with autonomously functioning thyroid tissue.

2. IODIDE GOITER

Acting as an antithyroid drug, iodine may have several effects inducing blocking of iodine organification [31]. Not everybody exposed is likely to develop iodide goiter, probably because of the "escape phenomenon" [6]. Hypothyroidism and iodide goiter have been considered an uncommon effect of iodine administration, occurring only in a minority of predisposed patients [1]. Patients with autoimmune thyroiditis are especially prone to develop iodide myxedema [5], because iodine organification defects are already present in this condition [7].

3. IODINE-INDUCED HYPERTHYROIDISM

The normal thyroid gland adjusts its iodine clearance in a reciprocal way to the plasma inorganic iodine concentration, i.e. diminishes it in the case of an iodine excess. At the same time organic iodine binding decreases [20]. Iodine induced hyperthyroidism may therefore be considered as a breakdown of the regulatory mechanisms caused by an autonomy of the gland. Autonomous function of the thyroid gland increases during the long term development of goiter and contributes significantly to thyrotoxicosis in iodine deficient areas [11, 16, 18, 20, 21]. Iodine-induced hyperthyroidism has been first described by Coindet [8] as a side-effect of iodine prophylaxis and treatment programs for the eradication of endemic iodine deficiency goiter. The best documentation proving the reality of iodine induced hyperthyroidism has been obtained in Northern Tasmania [9, 30]. Following the addition of potassium-iodide to bread, the incidence of thyrotoxicosis increased 3.8 times that occurring before in persons 50 years old or older and 1.6 times in persons younger than 40 years. There was no increase in the cases of endocrine exophthalmus! The incidence of thyrotoxicosis in patients with pre-existing nodular goiters reached a peak and displayed a gradual fall thereafter [9, 30].

In these patients with autonomously functioning thyroid tissue (AFTT) due to a failure of the regulatory mechanisms iodine elevates the serum hormone levels and exacerbates the clinical picture to hyperthyroidism [12, 22]. The risk of iodine induced hyperthyroidism in patients with AFTT increases with the amount of AFTT and depends on the amount of administered iodine, too. In order to define patients at risk, it is first necessary to find a method to quantify the volume of autonomous tissue in the individual thyroid gland and to define a borderline amount of iodine which can still be tolerated by these patients without getting hyperthyroid [3]. Small doses of

iodine up to 100µg/day do not induce hyperthyroidism in patients with different amounts of autonomous tissue [17, 18, 19]. But even medium doses up to 500µg/day may lead to hyperthyroidism [12, 14, 22], and as expected the number of patients getting hyperthyroid rises with further increase of the administered iodine dose into the range between milligrams and grams (10, 29). Besides the height and the duration of the iodine-exposure, the second condition for the induction of hyperthyroidism is an adequate amount and activity of functionally autonomous tissue: Thus no hyperthyroidism could be induced in patients with small unifocal autonomies by medium or even large doses of iodine [21, 23]. In countries with more than sufficient iodine supply, the experience was made, that in patients with unifocal thyroid autonomy with a diameter less than 2.5 cm no hyperthyroidism occurred. Hyperthyroidism would be induced only, if the autonomously functioning nodule had a diameter of more than 3 cm [4, 13, 25,26]. Our results (17, 18) confirm the assumption of a causal connection between the prevalence of hyperthyroidism and the amount of autonomously functioning thyroid tissue. The latter can be determined only approximately by sonography, but only in definitely outlined autonomous nodules and not in the multifocal or disseminated form of functional autonomy. The amount and the activity of autonomous thyroid tissue, however, can be estimated by the determination of the Tc-99m-pertechnetate uptake during TSH-suppression (TcTUsupp), because TcTUsupp and the volume of autonomous thyroid tissue (Va) correlate linearly: $Va = 0.09 + 2.88 * TcTUsupp$; $r^2 = 0.90$ [17]. The TcTUsupp can be determined routinely by quantitative thyroid scintigraphy. Thus the degree of functional thyroid autonomy can be quantified [19], and the "critical" amount of autonomous thyroid tissue can be determined by the "critical" TcTUsupp, above of which the development of hyperthyroidism will have to be expected, if the height and duration of iodine exposure is sufficient [16, 18]. This "critical" TcTUsupp ranges between 2% [21] and maximal 30% [18].

Is an estimation of the individual risk of hyperthyroidism possible?

Functionally autonomous thyroid tissue usually grows slowly [4, 27], and the prevalence of hyperthyroidism is judged to be low: during up to 12-year follow up studies, only 20% of patients with autonomous thyroid nodules became hyperthyroid. Even knowing the volume of the autonomous tissue does not allow any prognostic statement without knowledge of additional parameters: there seems to be no correlation between Va and the peripheral hormone concentration, but only a

tendency of an increase of the concentrations of FT4 and total-T3 with increasing amount of autonomous thyroid tissue [2]. But on the condition that the serum-iodide concentration is $> 1.0\mu\text{g/dl}$ the FT4 - concentration increases proportionally to the volume of autonomous tissue [17]. This iodide concentration cannot be reached by the administration of low iodine doses up to $100\ \mu\text{g/day}$ so that hyperthyroidism cannot be induced even at high amounts of autonomous tissue [17, 18, 19]. But the optimisation of the daily iodine supply with $200\ \mu\text{g/day}$ is already sufficient to induce latent hyperthyroidism in 14% and clinically manifest hyperthyroidism in 4% of the patients with AFTT [28]. There was a plain difference between the patients with a TcTUsupp less than 2.5% and those whose TcTUsupp was higher than 2.5%: in more than two thirds of the latter group of patients the bTSH concentration declined beneath $0.1\ \text{mE/l}$, but in only one third of the patients with a TcTUsupp less than 2.5%. An increase of FT4 was found only in those patients, whose TcTUsupp was higher than 3%. All the patients who developed hyperthyroid had a TcTUsupp higher than 3.3% [31]. From that the "critical" volume of autonomous thyroid tissue can be calculated to be between 8 and 12 ml, which equals a solitaire nodule with 2.8 cm diameter but without any regressive changes and in the case of multifocal or disseminated autonomy it correlates to a TcTUsupp between 2% and 3%. The risk of getting hyperthyroid in the case of long term administration of iodine doses increases with increasing V_a , but an individual prognosis whether a patient with a TcTUsupp higher than 3% necessarily becomes hyperthyroid in the case of the administration of excessive iodine doses is not possible. On the other hand an enhanced risk of hyperthyroidism can be excluded with a high probability in those patients whose TcTUsupp is less than 2%.

CONCLUSIONS

The risk of hyperthyroidism rises with increasing age and increasing amounts of functionally autonomous thyroid tissue and depends on both the height and the duration of the additionally applied iodine dose, at which the individual iodine supply has to be taken into account. Then the serum-iodide concentration is decisive: an initial increase of the FT4-concentration being proportional to the amounts of autonomous tissue may be only expected above a serum-iodide concentration of $1.0\ \mu\text{g/dl}$ [16, 18, 19]. The supply of additional low doses of $0.1\ \text{mg}$ iodide is not

sufficient to raise the serum-iodide concentration to the required level in patients living in an iodine deficiency area [17, 18, 19], but the supply of 0.2 mg/day may be sufficient in some persons [19, 28]. Thus the critical amount of iodide being sufficient to induce hyperthyroidism may be in the range between 0.1 and 0.2 mg/day. The iodine doses being necessary to minimize the iodine-131 thyroid uptake in the case of a reactor accident are considerably higher than the "critical" dose causing an enhanced risk of hyperthyroidism.

The estimation of the "critical" amounts of autonomous tissue is reliably possible only by determining the TcTUsupp: above a threshold between about 2% and 3% an additional iodine-load of sufficient height and duration can induce hyperthyroidism. If the TcTUsupp is higher than 3% iodine-induced hyperthyroidism is probable but in individual cases this risk cannot be predicted with certainty [19, 28].

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INTERNATIONAL POLICY ON INTERVENTION IN THE EVENT OF A NUCLEAR ACCIDENT

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Abstract

Criteria for taking particular protective actions with the aim of preventing or reducing radiation exposures to the population or to workers in the event of a nuclear accident or radiological emergency can be established on the basis of radiological protection principles for intervention situations. It is of utmost importance that pre-established intervention levels for different protective measures form an integral part of an emergency response plan. Generic optimized intervention levels and their derived operational quantities based on the principles given in this paper are judged to provide protection that would be justified and reasonable optimized for a wide range of accident situations although they can only be used as guidelines. Any specific optimization would lead to intervention levels that might be either higher or lower than those emerging from a generic optimization.

1 Introduction

In the event of a nuclear accident or radiological emergency, there is a need for criteria for taking particular protective actions with the aim of avoiding or reducing radiation exposures to the population or to workers. Such criteria can be established on the basis of radiological protection principles for intervention situations. The effectiveness of measures to be taken to protect a general public will depend heavily upon the adequacy of emergency plans in which these criteria are specified. There is, therefore, an important role for planning in the establishment of *intervention levels* for different protective measures. It is of utmost importance that pre-established intervention levels form an integral part of an emergency response plan.

2 Basic Principles

2.1 Practices and interventions

In most situations in which there is a need to consider controls over people's exposure to radiation, the source of radiation is deemed to provide a net benefit to society, for which an increased radiation exposure can be justified. This is the case for all normal exposures as a result of industrial processes utilizing radiation sources. These situations are defined as *practices* by the ICRP (ICRP91).

There are, however, a small number of situations in which the source of radiation exposure does not provide a net benefit. The aim of radiological protection in these circumstances is to reduce the exposure by taking some protective or remedial action. The two most easily identifiable examples of these situations are exposures resulting from the natural occurrence of radionuclides in the environment and exposures resulting from the release of radionuclides following an accident. These situations are defined as *intervention* by the ICRP (ICRP91).

Current radiation protection philosophy clearly distinguishes between a *practice*, which causes either actual exposures or probabilities of exposure and therefore will *add* radiation doses to the existing background, and *intervention* situations, in which radiation exposures can be reduced only by intervention in order to put exposed people in a better position. The radiation protection systems for practices and interventions are completely *separate systems*.

2.2 Principles for intervention

In existing exposure situations, i.e. existing at the time when control procedures are being considered, the choice of action is limited. The most effective action, that applied at the source, is rarely available and controls have to be applied in the form of intervention.

The system of radiological protection for intervention is based on the following general principles of *justification* and *optimization*:

- (a) *All possible efforts should be made to prevent deterministic effects.*
- (b) *The intervention should be justified, in the sense that introduction of the protective measure should achieve more good than harm.*
- (c) *The levels at which the intervention is introduced and at which it is later withdrawn should be optimized, so that the protective measure will produce a maximum net benefit.*

Dose limits used in the radiation protection system for practices do *not* apply in the case of intervention.

The process of justification and optimization *both* apply to the protective action, so it is necessary to consider them *together* when reaching a decision. *Justification* is the process of deciding that the disadvantages of each component of intervention, i.e. of each protective action or, in the case of accidents, each countermeasure, are more than offset by the reductions in the dose likely to be achieved. *Optimization* is the process of deciding on the method, scale and duration of the action so as to obtain the *maximum net benefit*. In simple terms, the difference between the disadvantages and the benefits, expressed in the same terms, e.g. monetary terms, should be positive for each countermeasure adopted and should be *maximized* by refining the details of that countermeasure's implementation.

The benefit of a particular countermeasure within a programme of intervention should be judged on the basis of the reduction (*dose subtraction*) in dose achieved or expected by that specific countermeasure, expressed as an *avertable dose*.

2.3 Factors entering optimization

The factors entering the optimization process can be divided into those describing *benefits* from the countermeasure and those describing *harm*. In analysing the inputs to the decision on the introduction of countermeasures, it is necessary to decide on the relative importance of each factor. The most relevant factors are summarised below.

Benefit	Harm
<ul style="list-style-type: none"> Avertable individual risk Avertable collective risk Reassurance 	<ul style="list-style-type: none"> Individual physical risk Collective physical risk Monetary costs Social disruption Individual disruption Countermeasure anxiety Worker risk

The weightings to be attached to each of these factors are necessarily subjective and it has been difficult to agree internationally upon their exact values. In any case the importance of some of the factors will vary with the site and nature of the accident, thus making it hard to generalize. Nevertheless, the dominant factors are those related radiological protection principles, and to psychological and political factors.

Socio-political and psychological factors indeed may well contribute to, or even dominate, some decisions. The competent authorities responsible for radiation protection should therefore be prepared to provide the radiation protection input (justification and optimization of the proposed protective actions on radiological grounds) to the decision making process in a systematic manner, indicating all the radiological factors *already considered* in the analysis of the protection strategy. In the decision process the radiological protection and the political factors should *each* be taken into account *only once* to avoid the same political factors being introduced in several places.

2.4 Generic and specific intervention levels

In the management of accidents, there are two distinct phases in which optimization of protective measures should be considered. In the phase of planning and preparedness, prior to any actual event, a *generic* optimization of protective actions should be studied, based on a *generic* accident scenario. This should result, for each protective measure and each selected scenario, in an optimized *generic* intervention level, which is meant to be the first criterion for action to be used immediately and for a short time after the occurrence of an accident.

Some time after a real event, specific information on the nature and likely consequences of the accident would become available. In this case, a more precise and *specific* optimization analysis can be carried out on the basis of actual data and efficiency of protective measures. This could result in a *specific* intervention level for each protective measure, to be used as a criterion in the medium and long term. However, in many cases the optimization will be constrained by sociopolitical factors, which may make it difficult to alter the generic intervention levels unless there are overriding reasons.

3 Selection of International Generic Intervention Levels

3.1 Working Premises

Intervention levels for urgent and longer term protective actions can be based on the justification and optimization principles and the following premises:

- national authorities will spend the same resources on radiation health risks as on other similar health risks;
- physical risks from the action are taken into account;
- disruption to individuals, such as livelihood or to resources, is considered;
- 'good' and 'harm' of psychological nature are excluded (although unpredictable, these are taken to result in a null net benefit);
- political, cultural, and other social factors (such as disruption) are excluded (because they will be considered separately).

The above relatively simple premises are considered appropriate to assist the selection of internationally applicable generic intervention levels. The premises have been used in Safety Series No. 109 (IAEA94a) for the development of generic intervention levels. A variety of decision aiding techniques are available to assist in questions of social risk

management, including cost-benefit theory, decision theory and social choice theory (MERK87). Cost-benefit theory was adopted in (IAEA94a) as an appropriate rationale for assisting in the selection of generic intervention levels. This rationale was first adopted for the purposes of countermeasure decisions by (BEN86). The problem can be conceptualized in cost-benefit terms whereby the net benefit of a proposed action compared with taking no action can be expressed as:

$$B = \Delta Y - R - X - A_i - A_s + B_c \quad (1)$$

where the six terms are expressions respectively of the radiological detriment averted by taking the action; the detriment associated with the physical risk of the action itself; the resources and effort need to implement the action; individual anxiety and disruption caused by the action; social disruption; and the reassurance benefit provided by the action. An intervention level (IL) for a countermeasure can be selected if principles (b) and (c) for intervention are satisfied. This can be achieved by conceptualizing them as conditions that B must be greater than zero, and that $dB/d(IL) = 0$ respectively, and resolving the above expression accordingly.

3.2 Simplistic analysis

For clarity of expression and understanding a simplistic analysis was performed in the Annex of Safety Series No. 109 (IAEA94a), expressing the terms in Eq.(1) in a way consistent with the premises described above. The two terms expressed quantitatively were the financial costs (X) and the radiological detriment averted (ΔY). For illustrative purposes, the analysis for temporary relocation is considered below. The financial costs of temporary relocation can be expressed as the sum of one-off transport costs (away and return), loss of income per month, rental of substitute accommodation per month and depreciation/maintenance costs per month. The *average* cost per person was evaluated as between about \$400 and \$900 for the first month of relocation, and between about \$200 and \$500 for subsequent months (IAEA94a).

The radiological detriment averted by temporary relocation was expressed simplistically in (IAEA94a) as the product of the collective dose averted by the action and an α -value representing the resources allocated to averting unit collective dose. Several methods have been developed to assess how much value is placed by individuals and society on avoiding health detriment, including the human capital approach, legal compensation approaches, insurance premium analogies, implied or revealed preference approaches and willingness to pay approaches. There are flaws in all of these methods. Nevertheless it is possible to arrive at a credible range of values for α . The method used in (IAEA94a) is based on the human capital approach used in (IAEA85) modified to take account of the 1990 Recommendations of the ICRP (ICRP91). The average loss of life expectancy associated with 1 manSv of collective dose is estimated as 1 year. On a purely economic basis, a minimum value to be associated with a statistical year of life lost is the annual GDP per head. This was used in (IAEA94a) to estimate a value for α of \$20,000 per manSv saved. A factor of two uncertainty in the risk per unit dose was explicitly used in considering a range in the α -value from \$10,000 to \$40,000 per manSv. (NB All monetary costs are expressed for a highly developed country. The argument is not significantly different for less developed countries.)

On this basis the temporary relocation of people will be justified for more than one month if the avertable dose in that month exceeds IL_{rel} :

$$\frac{\$400 \text{ to } \$900 \text{ in first month}}{\$10,000 \text{ to } \$40,000 \text{ per manSv}} \approx \text{ten to several tens of mSv in the first month} \quad (2)$$

The optimum return time is when the avertable dose in a following month falls below IL_{ret} :

$$\frac{\$200 \text{ to } \$500 \text{ in the month}}{\$10,000 \text{ to } \$40,000 \text{ per manSv}} \approx \text{a few to a few tens of mSv in the month} \quad (3)$$

3.3 Sensitivity analysis

In support of the guidance in Safety Series No. 109 (IAEA94a) more extensive sensitivity analyses were performed to consider explicitly the influence of the other relevant terms in Eq. (1). Moreover, several objections are raised and consequently modifications are often made to the basic value of α . Firstly, it takes no account of pain, grief and suffering associated with a premature death. Secondly, because people show an aversion to higher levels of individual risk, and because society is normally willing to allocate relatively more resources to protect people at higher risks, a modification is often used whereby α is increased according to the level of risk. Thirdly, an argument is made that because there is an inherent social time preference to speed up the receipt of desirable outcomes and postpone undesirable ones, a reduction factor should be applied to account for the delay between exposure and the occurrence of the effect. These three factors, which in some way counteract each other, could be assessed by willingness to pay methods. An example is given below of the influence the second two factors have on the range of intervention levels for temporary relocation.

Several national authorities provide guidance on the use of multipliers (so-called β -term) to apply to a baseline α -value to account for the level of individual dose received. (CEC91) presents several schemes for such β -terms. One formulation can be expressed as:

$$\beta = \beta_0 E^\nu \quad \text{where } \beta_0 \text{ and } \nu \text{ are parameters} \quad (4)$$

Moreover a discount factor, F_d to account for the time delay between the dose received and the time of appearance of a cancer can be applied, of the form $(1+r)^{-T}$ where r is a discount rate (typically between 0 and 10% per annum) and T is the time delay. The detriment saved by invoking a countermeasure can be expressed then as :

$$\Delta Y = \alpha_0 \beta_0 F_D \left(E_{tot}^{\nu+1} - E_{res}^{\nu+1} \right) \quad (5)$$

where E_{res} and E_{tot} are the total doses received with and without the countermeasure. Temporary relocation, for example, can be suspended when :

$$\frac{d\Delta Y}{dt} = -c_{rel} = -\alpha_0 \beta_0 F_D (\nu+1) E_{res}^\nu \dot{E} \quad (6)$$

where c_{rel} is the cost per person per month of continuing relocation. This can be solved depending on the relationship between the residual dose, E_{res} and the optimum dose rate for return, \dot{E} . Various functional forms and source term characteristics were considered in support of Safety Series No. 109 (IAEA94a). Considering as an example a release of a single nuclide with an effective removal rate constant, λ , the optimum dose rate for return can be evaluated as:

$$IL_{ret} = \left[\frac{c_{rel} \lambda^\nu}{\alpha_0 \beta_0 F_D (\nu+1)} \right]^{\frac{1}{\nu-1}} \quad (7)$$

A parameter uncertainty analysis was carried out (CB93) to evaluate the likely range of values within which a generic intervention level might reasonably lie, and to identify which parameter uncertainties influence most strongly the selection of a generic value.

The results indicated that the range of values derived from the simplistic approach are not drastically different from those obtained by a more sophisticated approach. This analysis and others underpinned the final selection of the values that appear in Safety Series No. 109 (IAEA94a), which also took into account qualitative factors.

3.4 Temporary and permanent relocation

Permanent relocation of a population can also be used as a protective measure where this action can be justified and optimized in accordance with the principles for intervention. Because the penalties associated with this action are of a one-off nature, the intervention level for permanent relocation is expressed in terms of total dose averted rather than avertable doses per month of temporary relocation. In addition to this criterion for permanent relocation based on avertable dose, there is a limit to the period of any temporary relocation that can normally be tolerated. The maximum length of this period is dependent on many social and economic factors. An argument based on economic grounds shows that continuing temporary relocation costs will begin to exceed permanent relocation costs between about one and five years. However social factors would indicate that the period of temporary relocation should be no more than a year or so.

The final guidance of the International Basic Radiation Safety Standards (IAEA94b) is as follows:

The generic optimized intervention levels for initiating and terminating temporary relocation are 30 mSv in a month and 10 mSv in a month, respectively. If the dose accumulated in a month is not expected to fall below this level within a year or two, permanent resettlement with no expectation of return to homes should be considered. Permanent resettlement should also be considered if the lifetime dose is projected to exceed 1 Sv.

5 Conclusions

Over the past decade considerable progress has been made in developing and clarifying internationally recognized principles for decisions on protective measures following nuclear or radiological emergencies, and in providing quantitative guidance for applying these principles. However, experience has shown that, in spite of these efforts, there remain discrepancies in the application of both principles and guidance.

An accident resulting in the dispersion of radioactive material to the environment requires measures to protect the general public against the exposure to ionising radiation from the released and dispersed activity. The effective implementation of these measures will be largely dependent upon the adequacy of emergency response plans. Such plans should specify intervention levels for the various protective actions, and detailed considerations of site specific and accident specific conditions should be taken into account at the planning stage when specifying these levels based on the justification/optimization principles.

In theory, the optimum intervention level for each kind of countermeasure could take a range of numerical values, depending on the exact circumstances following the accident and on social, political and cultural factors that national authorities might need to consider. However, to avoid confusion, there are obvious advantages to have a single internationally accepted value for the appropriate level of protection instead of a range of values as have been recommended earlier by international organisations.

Generically optimized intervention levels based alone on the premises presented in this paper might be used with equal benefit both in developing countries and in more developed countries, even if there are large differences in the absolute cost levels for specific countermeasures between such countries. The reason for this is that the outcome of an optimization normally is a cost ratio, which is much less sensitive to geographical location than the absolute cost values alone all of which are similarly related to the GDP of the country.

Measurable quantities can be applied as surrogates for intervention levels using models that link avertable doses with these quantities. Modelling of the various processes describing the exposure of man to environmental contaminants would include parameters such as type of radionuclides, environmental half-lives, transfer functions as well as location and filtering factors for housing conditions. The models may be of varying complexity but both models and parameter values used to determine avertable doses should be realistic and particular to the circumstances under consideration. Incorporation of pessimism should be avoided by using central values from the parameter ranges. The measurable quantities normally used as so-called operational intervention levels include dose rate and activity concentration in air, in foodstuffs and on ground surfaces. The operational intervention levels will be both accident and site specific as they are derived from dose models that include accident and site specific parameters. Operational quantities should therefore be used carefully.

In conclusion, generic optimized intervention levels and their derived operational quantities based on the principles given in this paper are judged to provide protection that would be justified and reasonably optimized for a wide range of accident situations although they can only be used as guidelines. Any specific optimization would lead to intervention levels that might be either higher or lower than those emerging from a generic optimization.

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