

MULTIPLE PESTICIDES EXPOSURE OF GREENHOUSE WORKERS AND THYROID PARAMETERS

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ABSTRACT

Objective: The persistence of high prevalence of thyroid pathology (goiter, nodules) in Romanian population, despite the correction of iodine deficiency, determined us to evaluate the impact of factors different than iodine intake on the thyroid gland as environmental endocrine disruptors. We studied the potential correlation between pesticide exposure and parameters of the function, autoimmunity and morphology of the thyroid in a group of greenhouse workers (GHWs) exposed to multiple uncontrolled pesticides across agricultural seasons.

Materials and methods: A group of 108 GHWs, 18–78 year olds, with normal iodine intake, from a plain village, exposed to multiple pesticides was enrolled voluntarily in this study. Thyroid echography, thyroid parameters [thyroid stimulating hormone (TSH), free T4 (FT4), and antibody to thyroid peroxidase (ATPO)], some of the used pesticides (chlorpyrifos, trichloropyridinol (TCP), carbofurane, cypermethrin, dimethoate) and cholinesterase activity were determined in biological samples (urine, blood) collected across two agricultural seasons.

Results: The median urinary iodine concentration in children from this village was 135.20 µg/L, while median TSH and median FT4 was 1.72 µUI/mL and 16.68 pmol/L, respectively. Hypothyroidism (TSH > 4.2 µUI/mL) was present in 12.4% of patients, median TSH value was 7.26 µUI/mL. In four of them post-thyroidectomy hypothyroidism was present. Elevated titres of anti-TPO antibodies were found in 22 (20.37%). Echographic pattern of thyroiditis was present in 16.49% of the patients, and thyroid nodules were detected by ultrasonography in 38.13%. There were two subjects with Graves' disease.

The detectable level of TCP was bigger in the first season (range 120–190 µg/L) than in the second (range 0.7–1.7 µg/L). The range of seasonal concentration of urinary carbofurane was 0.004–0.25 µg/mL. Cypermethrin was detected in a small number of subjects (range: 12.5–13.3 µg/g creatinine). Dimethoate was undetectable.

Conclusions: The distribution of thyroid disorders in study group living in an area with normal iodine intake did not differ from known epidemiological studies. The most frequently encountered pesticide was chlorpyrifos. Some samples presented several pesticides simultaneously.

Keywords: Endocrine disrupter, pesticides exposure, thyroid autoimmunity, thyroid function.

1 INTRODUCTION

The effect of pollutant substances on ecosystem in general and on human health especially has increased evidence [1, 2]. The polluted environment has a permanently impact on thyroid gland due to the gland's dependence on essential elements and because of contamination by different pollutant substances from environment and food [3].

In Romania, endemic goiter was reported in two-thirds of the population. The prophylactic program introduced in 1956, reinforced many times, was partially efficient. Mandatory universal salt iodization for house and baking industry normalized iodine uptake (generalized epidemiologic inquires in schoolchildren, 2005–2008, showed a median urinary iodine concentration (UIC) of 135 µg/L [4–6], a value considered normal for iodine intake in a population [7]).

A small study conducted by our group in 2000 on 'Nitrates and organochlorine pesticide elimination in subjects with normal and borderline iodine intake and their effects on thyroid parameters' hypothesized the possible interference of pollutant substances with thyroid and was received with great interest in the European symposium 'Thyroid and Pollution' and determined us to continue the population studies regarding this topic [8].

The persistence of an important thyroid pathology despite the iodine implementation program in progress, and the observation of a high incidence of goiter in areas with normal iodine intake (revealed through discordance between goiter incidence and urinary iodine values) determined us to suspect that there are certain goitrogenic substances that interfere with thyroid metabolism and determine the persistence of thyroid pathology.

In the context of a project developed in 2006 entitled 'Promotion of Romanian participation in European programs in research field of endocrine disruptor effects on thyroid physiopathology' [9], we had the opportunity to invite in a workshop three hygiene institutes from our country and Institute of Food Chemistry that brought us information about the presence of pesticides in different vegetables, which often are above the permissible maximum limit. The analyzed food came from different areas of the country. For this reason, we chose the population group of agriculture workers from greenhouses in the vicinity of Bucharest. These farmers are in fact private farmers and they work without being instructed and use commercial pesticides, insecticides and fungicides, some of them from uncontrolled sources.

The aim of our study was to evaluate the impact of other factors than iodine intake on the thyroid gland as environmental endocrine disruptors in a group of greenhouse workers (GHWs) exposed to multiple uncontrolled pesticides across agricultural season. The potential correlations between pesticide exposure and parameters of thyroid function, autoimmunity and morphology were analyzed.

2 STUDY SITE AND POPULATION

The GHWs were from a village near Bucharest (30 km) in a plain area. Because these farmers own an average field size of 2000–3000 m², the village farms are small. As we observed, the private individual GHW cultivate different kinds of vegetables, and do not wear the personal protection equipment at work. The used pesticides are inappropriately handled, and there are not special instructions on the pesticide toxicity. A number of 108 GHWs (18–78 years old; 19 males and 89 females) were enrolled voluntary into this study.

3 MATERIALS AND METHODS

Subjects were clinically examined by an endocrinologist and the data were noted in an observation chart designed especially for the study. Morphology of the thyroid gland was assessed by thyroid ultrasonography with an Aloca ultrasonograph having a 7.5-MHz probe.

Detailed information about agricultural activities, pesticide application was obtained. In different months of agricultural season we performed toxicological inquires on the used pesticides, and the obtained data were recorded. Most of the farmers enrolled in the study were exposed to pesticides for more than 20 years. The different types of used pesticides in the agricultural seasons 2010 and 2011 are presented in Table 1.

Depending on the availability of workers, we had to organize clinical endocrinologic evaluations and collection of samples (blood and urine) on the last Sunday of the month, three times in 2010 and four times in 2011. Aliquots of samples were stored at –80°C. The samples were divided between the laboratory of National Institute of Endocrinology for thyroid parameters analysis and three partners involved in pesticides determination.

Table 1: Pesticides used in agricultural season 2010 and 2011.

Commercial name	Basic substance
SINORATOX 5G	5% Dimethoate
VICTENON 50WP	50% Benzultap
TOPSIN AL 70 PU	70% Methyl tiophanate
SHAVIT 72WDG	2% Triadimenal + 70% folpet
ACTARA 25 WG	25% Thiametoxan
LIDER 70 WG	70% Imidacloprid
NOVOZIR MN80	Mancozeb 80%
CAPTADIN	Captan – 50% ethane thiol or ethyl mercaptan
CHAMPION	50% Copper hydroxide
SUMILEX	50% Procimidon (dicarboximide group)
OMITE	57% Propargit
CYPERMETHRIN	Pyrethroid
DECIS	Pyrethroid (50% deltametrin)
FASTAC	α - Cypermethrin – pyrethroid
LASER	Cyfluthrin
LANATE	90% Methomyl – carbamate
NUTRILIVE	Pyrethroid
ATONIK	Sodium 5-nitroguaiacolate, sodium <i>o</i> -nitrophenolate and sodium <i>p</i> -nitrophenolate
CRONPAX	Natural product
NUTRISOL	Peat, humus, whitewood bark
REALDAN	Organophosphoric insecticide (chlorpyrifos)
DITHANE NEOTEC 75W	80% Mancozeb – carbamate
POLITRIN	Cypermethrin
ANTRACOL	70% Propineb
ROVRAL	Iprodion
STOMP	Pendimetalin
RANDUP	Glyphosphate
FOLPAN	80% Folpet
BAVISTAN	methyl <i>N</i> -(1H-benzimidazol-2-yl) carbamate
CONFIDOR	Imidacloprid si deltametrin
OPTIMOL	4% Methaldehyde
RIDOMIL	Metalaxil-m and mancozeb
FOLIAR	Aminopoliethilenurea
LIDER	70% Imidacloprid
AGROTHRIN 10 EC	10% Cypermethrin
EMERITE	240 g/L Imidacloprid 32 g/L Deltametrin

(Continued)

Table 1 (cont.)

DRAGO 76 WP	6% Cimoxanil 70% Mancozeb
MELODY compact	84 g/kg Iprovalicarb 406% copper oxychloride
MESUROL	2% Methiocarb
Alfametrin 10 CE	100 g/L α -Cypermethrin
SILWET L – 77	84% Heptametiltrisiloxat modified with polialchilenoxid
Ferbanat L	
BIONAT plus	6.9% Nitrogen, 0.003% phosphorus (P ₂ O ₃), 0.76%, potassium (K ₂ O), 0.47% magnesium (Mg) 1.3% sulfur (S), 0.0005% calcium
MEGASOL	
AECTRA	20% Total nitrogen (N), 20% available phosphorus, 20% soluble potassium

The thyroid parameters [thyroid stimulating hormone (TSH); free T4 (FT4) and autoimmunity marker (antibody to thyroid peroxidase (ATPO))] were assessed by electrochemiluminometric immunoassay using Elecsys autoanalyzer (Roche Diagnostics). The reference ranges were 0.27–4.2 μ IU/mL for TSH, 12–22 pmol/L for FT4, and <34 IU/mL for ATPO. The UIC in a group of children from Adunații-Copăceni village was measured in the Department of Isotopic Diagnostics, Sosnowiec, Poland, using Sandell and Kolthoff method (cerium–arsenite method).

From the multiple pesticides to which farmers were exposed we choose to analyze those compounds with possible endocrine disrupting effects on thyroid gland as chlorpyrifos, its metabolite trichloropyridinol (TCP), dimethoate, cypermethrin and carbofuran (Table 1) [10].

In the first stage of experiment, we analyzed in the urine samples biological indicators for the exposure to the toxic chemicals – thioethers [11–14]. Urinary thioethers were spectrophotometrically evaluated when the SH groups obtained by alkaline hydrolysis of the thioether bonds react with the Ellman's reagent [5,5'-dithiobis-(2-nitrobenzoic acid)] and a light yellow compound is formed.

The blood cholinesterase activity was also determined to monitor the exposure to organophosphorus and carbamates pesticides [15–20]. The activity of cholinesterase was determined in the samples of sera using as substrate the butyrylthiocholine, which is hydrolyzed by the enzyme into butyrate and thiocholine. The later reduces the hexacyanoferrate(III) to hexacyanoferrite(II), and the absorbance was recorded at 405 nm. The decrease of the absorbance in the time unit is proportional with the cholinesterase activity in the sample.

Chlorpyrifos is the major representative of the organophosphorus insecticides that are widely used in agriculture and even with all restrictions of their use, they still account for 50% of all pesticide applications worldwide. These compounds receive an increasing consideration as potential endocrine disrupters [21–24]. Chlorpyrifos is rapidly metabolized and excreted in the urine. It does not accumulate appreciably in the human body. For biomonitoring the exposure, their urine metabolites have been typically measured. Urine samples were analyzed for the primary metabolite of chlorpyrifos, 3,5,6-trichloro-2-pyridinol (TCP). This metabolite of chlorpyrifos was determined in urine using gas-chromatography with mass spectrometric detector (gas chromatography-mass spectrometry or GC-MS). Briefly, urine samples (1 mL) were hydrolyzed with 100 μ L concentrated HCl at 80°C for

2 hours, in order to break double bonds. The samples were then cooled at room temperature by adding 0.5 mL 20% NaCl and 1 mL of toluene, stirred and centrifuged at 3400 rpm for 10 minutes. The collected extracts (approximately 750 μ L) were concentrated to 120 μ L under nitrogen stream and analyzed for the presence of TCP by GC-MS equipped with a capillary column DB-5 (30 m, 0.25 mm I.D., film thickness 0.25 μ m), carrier gas He at 1 mL/min, for m/z ratios: 161(35Cl-TCP) and 163(37Cl-TCP). The response of the detector was linear in the range 0.002–0.455 μ g/mL, based on the equation: $y = 56.119x + 0.0024$, with a correlation coefficient $r^2 = 1.0000$. The recovery of the characterized method was calculated on three levels of the TCP concentrations for a sample with an initial content of 4.97 ng/mL. It was found that for the first level of enrichment with 1 ng/mL, the recovery value ($n = 4$) is 87% (s.d.% = 2.65); for the second level of enrichment with 10 ng/mL, the recovery value ($n = 4$) is 93.2% (s.d.% = 0.95); for the third level of enrichment with 110 ng/mL, the recovery value ($n = 4$) is 86.03% (s.d.% = 0.99). The limit of detection (LOD) and the limit of quantification (LOQ) were calculated as the concentration of TCP with signal-to-noise (S/N) ratios of about 3/1 and 10/1, respectively and have the following values: LOD = 0.001 μ g/mL and LOQ = 0.005 μ g/mL.

From the group of carbamate pesticides, carbofuran (2,3-dihydro,2,2-dimethyl-7-benzofuranyl carbamate) was analyzed in the biological samples by its reaction with diazotized *p*-aminoacetophenone under alkaline condition, when the orange dye formed was measured at 460 nm [25, 26].

Cypermethrin is a synthesis pyrethroid, most frequently used in gardens and homes; it does not accumulate in the human body and is rapidly metabolized being excreted in the urine [27–30]. Pyrethroids are rapidly metabolized through esterase and their urine metabolites have a half-time of approximately 6 hours. We aimed to determine the unmetabolized cypermethrin in the urine of farmers professionally exposed to this compound. Cypermethrin in biological samples was analyzed by its alkaline hydrolysis to cyanide ion, which further reacts with potassium iodide and leucocrystal violet. The absorption at the highest level of the formed crystal violet dye was measured at 595 nm in acidic medium.

Dimethoate determination was based on the pesticide extraction in dichloromethane/methylene chloride (a nonpolar solvent), followed by the extract purification (if applicable), using separation on a capillary column with either nonpolar or medium polar reagents and detection with an Nitrogen-Phosphorus detector. The data were then confirmed by mass spectrometry [31].

Statistical analysis was performed using SPSS 10.0 software.

4 RESULTS

The evaluation of iodine uptake in Adunații-Copăceni was assessed in a group of 104 schoolchildren from this village. The median UIC for this group was 135.20 μ g/L, showing a normal iodine intake for the studied area (Table 2).

Table 2: Urinary iodine concentration in schoolchildren from Adunații-Copăceni.

Urinary iodine concentration (μ g/L)	
No of subjects	104
Mean	134.04
Median	135.20
Standard deviation	59.28
Percentile 10	52.76
Percentile 50	135.20
Percentile 97	230.74

In this study, 108 GHWs (19 males and 89 females) between 18 and 78 years of age were enrolled. Median TSH and FT4 values in studied subjects and controls are presented in Table 3.

Median TSH was 1.72 $\mu\text{IU/mL}$ with the range being 0.051–14.97 $\mu\text{IU/mL}$. Median TSH in the control group was 1.28 $\mu\text{IU/mL}$ and the range was 0.47–4.14 $\mu\text{IU/mL}$.

Median FT4 was 16.68 pmol/L with a range of 9.49–22.18 pmol/L. Median FT4 in the control group was 16.84 pmol/L with a range of 12.79–21.2 pmol/L.

Regarding the distribution of thyroid disorders in this group with normal iodine uptake [32], 13 female patients (12.4%) have hypothyroidism (TSH > 4.2 $\mu\text{UI/mL}$), with a median TSH value of 7.26 $\mu\text{UI/mL}$. In four of these patients, a post-thyroidectomy hypothyroidism was present (Table 4). Age of the patients with hypothyroidism was between 19 and 68 years, with 15.38% of them being under the age of 30. ATPO were positive for nine of the subjects with hypothyroidism.

In studied group, elevated titres of thyroid peroxidase antibodies were found in 22 females (20.37%), with 22.73% under the age of 30; it is known that ATPO increases with age.

Thyroid nodules were detected by ultrasonography in 38.13% of the subjects. The prevalence of single thyroid nodule was 17.52% and of multinodular goiter was 20.61%.

Echographic pattern suggesting chronic thyroiditis was found in 16.49% of the subjects (Fig. 1).

In the biological samples of GHWs, we measured some of the pesticides used in agricultural seasons 2010–2011 [33, 34]. Given that the most organophosphorus and many carbamate pesticides cause the inhibition of acetylcholinesterase, we considered the determination of this parameter in the biological samples [35]. The serum cholinesterase values were all in normal

Table 3: Thyroid parameters in greenhouse workers.

	TSH		Free T4	
	$\mu\text{IU/mL}$		pmol/L	
	Subjects	Controls	Subjects	Controls
No	108	27	108	28
Mean	2.58	1.53	16.13	17.64
SD	2.71	0.86	3.32	2.93
Range	0.05–14.97	0.47–4.14	9.49–22.18	12.79–21.2
Median	1.72	1.28	16.68	16.84

Table 4: Thyroid parameters in subjects with thyroidectomy.

Sex	Age (years old)	TSH under Euthyrox ($\mu\text{UI/mL}$)	ATPO (UI/mL)	Thyroidectomy
F	53	7.17	Positive	Hashimoto thyroiditis
F	49	10.13	Positive	Graves' disease
F	41	12.76	Positive	Hashimoto thyroiditis
F	37	1.4	Negative	Toxic adenoma

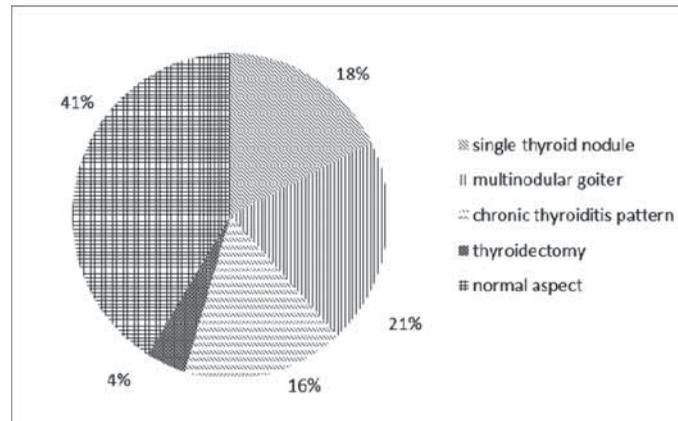


Figure 1: Echographic pattern in greenhouse workers.

Table 5: Seasonal variation of serum cholinesterase activity.

	Cholinesterase activity (U/L)					
	Control group	GHWs				
		April 2010	August 2010	April 2011	May 2011	August 2011
No	18	15	25	12	14	12
Mean	8444	7976	6944	5345	6320	6355
SD	1056	1758	1337	553	1483	2226
Range	6760–11014	5177–11295	4844–9394	4407–6229	4354–8343	4695–11010
Median	8246	7829	6851	5347	6259	5521

ranges, but the seasonal mean values for the GHWs were lower compared with that of the control group (Table 5).

The lowest value was obtained in April 2011 suggesting that in Spring 2011 there were used many more products based on organophosphoric and carbamic compounds.

Chlorpyrifos was found in the urine samples analyzed confirming the exposure of our subjects to this group of substances. Samples were collected in two seasons. There were collections two times in the first season and four times in the second one. In different time-season exposures in 2010, TCP (the metabolite of chlorpyrifos) was detectable in proportion of 64.70% (mean: 160 µg/L, range: 120–190 µg/L) and of 100% (mean: 150 µg/L, range 120–180 µg/L), respectively (Table 6).

In all the collections of 2011, TCP was found in lower proportion 21.43%, 31.25%, 21.43% and 12.5%, respectively (Table 7) and lower concentration (range: 0.7–1.7 µg/L).

While interpreting results, we must take into account that determinations were made from urine samples collected during the day [36–38].

Table 6: TCP values in urine samples collected in 2010.

	TCP ($\mu\text{g/L}$)	
	June	August
No	17	25
Mean	160	150
SD	18	20
Range	120–190	120–180
Median	170	150

Table 7: TCP values in urine samples collected in 2011.

	TCP ($\mu\text{g/L}$)			
	April	May	June	August
No	14	16	14	8
Mean	1.1	1.18	1	1.4
SD	0.5	0.2	0.3	–
Range	0.7–1.7	0.8–1.4	0.7–1.3	–
Median	0.9	1.2	1	–

Table 8: Urinary concentration of carbofurane in urine samples collected in 2010.

	Carbofurane ($\mu\text{g/mL}$)	
	June	August
No	14	21
Mean	0.089	0.044
SD	0.071	0.020
Range	0.02–0.025	0.009–0.08
Median	0.06	0.044
Percentage of positive samples	71.43%	71.43%

The presence of carbofurane in the urine samples of the studied subjects varied in different months between 33.33% and 71.43%. Seasonal mean concentration of urinary carbofurane varied between 0.024 and 0.089 $\mu\text{g/mL}$ (range: 0.004–0.25 $\mu\text{g/mL}$) (Tables 8 and 9), while mean values of serum samples varied between 0.125 and 0.24 $\mu\text{g/mL}$ (range: 0.04–0.52 $\mu\text{g/mL}$) (Table 10).

Urinary concentrations are smaller than serum concentrations due to the fact that carbamic compounds can be metabolized through different pathways.

Table 9: Urinary concentration of carbofurane in the urine samples collected in 2011.

	Carbofurane			
	(µg/L)			
	April	May	June	July
No	14	18	18	17
Mean	0.025	0.034	0.061	0.024
SD	0.025	0.027	0.027	0.022
Range	0.006–0.08	0.004–0.1	0.035–0.11	0.004–0.06
Median	0.9	1.2	1	–
Percentage of positive samples	0.7–1.7	0.8–1.4	0.7–1.3	–

Table 10: Serum concentration of carbofurane in samples collected in 2011.

	Carbofurane – Serum concentration	
	(µg/mL)	
	April	May
No of greenhouse workers investigated	12	14
Mean	0.125	0.24
Range	0.04–0.23	0.020
Percentage of positive samples	50%	85.7%

In the control group (24 adults), carbofurane concentrations were under the detection limit. In addition, in the urine samples collected from a group of 22 children, we did not find the presence of carbofurane.

Cypermethrin concentration in the majority of samples was under the quantification limit in samples from 2010. Only in two samples we found cypermethrin concentrations of 12.5 µg/g creatinine and of 13.3 µg/g creatinine. In samples from 2011, we detected cypermethrin in the urine of a small number of exposed subjects. This can be explained by the fact that pyrethroids are rapidly metabolized through esterase and urine metabolites have a half-time of approximately 6 hours.

Dimethoate concentration was determined only in 2010 and all samples had values under the quantification limit.

Because of temporal exposure of workers to these substances, we evaluated the rate of exposure and the effects of exposure on thyroid pathology with reluctance.

5 DISCUSSIONS

Toxicological inquiry of our group showed that GHWs used a wide range of substances for the cultivation and growing of vegetables. A number of these substances were found in the EPA list from 2009 [10] as having possible endocrine disruptor effect.

There is increasing experimental evidence that a wide range of chemicals can interfere with thyroid [39–42].

Organophosphorus pesticides, e.g. chlorpyrifos, are widely used in agriculture (despite increasing restrictions in industrialized countries) and are receiving increasing consideration as potential endocrine disruptors [43].

The thyroid may be a sensitive target to chlorpyrifos.

Studies with chlorpyrifos (at dose levels without effect on brain acetylcholinesterase) showed that experimental exposure to chlorpyrifos induces histologic alterations on thyroid – necrotic follicular epithelial cells exfoliated into the lumen – in adult F1 mice and alteration of thyroid hormone levels in Cd1 mice [44].

In addition, Jeong *et al.* [45] found decreased T4 levels and histopathological damage corresponding to vacuolation or necrosis of thyroid follicular cells in F1 male rats exposed during pregnancy and for 13 weeks after birth.

Organophosphorus pesticides enter into the human body through inhalation of air dust, dermal contact or ingestion of food and water. Generally, they do not accumulate in the tissues, as they are metabolized rapidly and the metabolites are excreted in urine. Urine is the preferable matrix for pesticides measurement.

There is a temporal variability of a given chemical in multiple samples collected.

Literature data on TCP level showed in a US general population values in the range of 1.5–1.87 $\mu\text{g/g}$ creatinine [45] (NHANES 2000–2001). In the other two studies in the United States [46, 47], the level of TCP was 10.5 (geometric mean) and 12–49 $\mu\text{g/g}$ creatinine, respectively.

More recently, a control group in an Egyptian study showed a TCP value of 6.25 $\mu\text{g/g}$ creatinine [23].

In our study group, chlorpyrifos and TCP were determined in urine samples. There is considerable variability between the TCP concentrations in the urine samples of the GHWs during the two agricultural years. In 2010, TCP was detectable in a proportion of 64.70%–100% showing high values (120–190 $\mu\text{g/L}$). In seasons of 2011, we registered lower proportions in which TCP was determined (12.5%–31.25%). The TCP values were lower as well during the same period (0.7–1.7 $\mu\text{g/L}$).

The demonstration of an increased level of chlorpyrifos metabolite promotes convincing evidence that GHWs have substantial exposure during organophosphate pesticides application.

Carbofuran is another pesticide with effects on thyroid function and morphology; it was measured in urine samples in a proportion of 50%–71.43%, showing the exposure to another pesticide compound.

We detected the pyrethroid cypermethrin in the urine samples of a small number of exposed subjects.

A possible explanation of this fact could be that collection of samples was made on Sunday and the farmers handled the pesticides during the week on Tuesday and Wednesday. In the literature, it is mentioned that pyrethroids are rapidly metabolized through esterase and urine metabolites and have a half-time of approximately 6 hours depending on the isomer; it is necessary to collect the urine samples immediately after exposure, but we did not get the opportunity to collect the samples in this way.

We have to mention that some samples presented several pesticides simultaneously.

The iodine availability is within normal recommended values for the studied GHWs who are living in a plain area. The median UIC in schoolchildren from this village was 135.20 $\mu\text{g/L}$. Regarding the distribution of thyroid disorders in the studied group, we registered TSH values higher than 4.2 $\mu\text{IU/mL}$ in nine subjects and other four subjects presented hypothyroidism after thyroidectomy.

The presence of high level of ATPO was detected in 20.37% of subjects from the studied group, with 22.73% under the age of 30 years; it is known that having ATPO increases with age. The echographic pattern suggestive of Hashimoto thyroiditis was observed in 16.49% of the subjects. The causes of hypothyroidism in the studied group were chronic autoimmune thyroiditis and thyroidectomy. Thyroid nodules were detected by ultrasonography in 38.13%. The prevalence of single thyroid nodule was 17.52% and of multinodular goiter 20.61%.

6 CONCLUSIONS

The distribution of thyroid disorders in the study group living in an area with normal iodine intake did not differ from known epidemiological studies. The most frequently encountered pesticide (70%–90%) was chlorpyrifos with concentrations significantly higher than the known concentrations for exposure background. There is increasing experimental evidence that the organophosphorus and carbamate pesticides interfere with thyroid (histopathological damage and functional involvement). It is necessary to extend these data.

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