



## Original Research Article

# A Regional Study on Thyroid Autoimmunity and Iodine Exposure in Bangladeshi Populations

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**Abstract: Background:** Iodine is required for thyroid function, but both deficiency and excess can affect thyroid health. Bangladesh implemented universal salt iodization to control endemic iodine deficiency, but the effects of varying iodine exposure on thyroid autoimmunity in different regions are unclear. This study aimed to investigate the relationship between iodine status and thyroid autoimmunity in five regions of Bangladesh. **Methods:** In this cross-sectional study, 100 subjects (20 from each division: Dhaka, Chittagong, Sylhet, Rajshahi, and Rangpur) with no prior history of thyroid disease were enrolled. Urinary iodine levels (UIC) were estimated and graded according to WHO criteria. Thyroid function tests (TSH, free T4, free T3) and thyroid peroxidase antibodies (TPO-Ab) were assessed. Linear regression ascertained the influence of iodine status on fT4 levels, and Cox proportional hazards and logistic regression models ascertained predictors of thyroid autoimmunity. **Results:** The prevalence of thyroid autoimmunity varied significantly by region, from 50% in Rangpur to 20% in Sylhet. A dose-response relationship between iodine exposure and TPO-Ab positivity was strong since 75% of the subjects with excess iodine levels ( $\geq 300 \mu\text{g/L}$ ) were positive for thyroid autoantibodies compared to 19.4% of those who had sufficient iodine levels. Multivariate analysis also demonstrated that high iodine exposure was independently related to a higher risk of thyroid autoimmunity (HR=3.2, 95% CI: 1.8-5.6,  $p < 0.001$ ), independent of age and sex. **Conclusion:** This study demonstrates a definite association of excessive iodine exposure with thyroid autoimmunity in Bangladesh's population, with a marked geographic variation. The findings suggest that although iodine deficiency prevention is still required, prevention of excessive iodine exposure should be adopted in public health policies to reduce thyroid autoimmunity risk.

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## Introduction

Thyroid disorders are a significant public health problem worldwide, and iodine status is the most important determinant of thyroid function and autoimmunity.<sup>1</sup> The relationship between iodine consumption and thyroid function is U-shaped, and both deficiency and excess have adverse effects.<sup>2</sup> While iodine deficiency disorders have been extensively debated all over the world, the hazards of

excessive intake of iodine are not as well studied, particularly in countries with universal salt iodization programs.<sup>3</sup> Bangladesh launched universal salt iodization (USI) in 1989 to manage endemic iodine deficiency disorders, which previously had been affecting significant portions of the population.<sup>4</sup> The public health intervention has successfully reduced the incidence of goiter and cretinism; however, recent

findings show that dramatic increases in population iodine intake have the potential to trigger autoimmune thyroid diseases in susceptible subjects.<sup>5</sup> The condition, also known as iodine-induced thyroiditis, has been described in a number of populations transitioned from iodine deficiency to sufficiency.<sup>6</sup> The thyroid gland requires iodine for the synthesis of thyroid hormones thyroxine (T4) and triiodothyronine (T3), which regulate metabolism, growth, and development in the entire body.<sup>7</sup> In physiological circumstances, the thyroid attains homeostasis across a range of levels of iodine intake. Nevertheless, excessive iodine may cause oxidative stress in thyrocytes with the possibility of triggering autoimmune activity in genetically susceptible individuals.<sup>8</sup> Antibodies to thyroid peroxidase (TPO-Ab) are the most common indicators of thyroid autoimmunity and with it, increased risk of thyroid dysfunction overt form.<sup>9</sup> Bangladesh offers a unique setting to study iodine exposure against thyroid autoimmunity because of several factors. To begin with, Bangladesh has undergone rapid transition from iodine deficiency to non-homogeneous iodine status following USI implementation.<sup>10</sup> Secondly, there are significant geographic variations in food eating patterns and access to iodized salt across different areas. Thirdly, some environmental factors like groundwater chemistry can affect exposure to iodine aside from diet. Despite these factors, there are not many data about the prevalence of thyroid autoimmunity in various parts of Bangladesh and its relation to iodine status. Studies done in other countries have yielded unconvincing results regarding the relationship between thyroid autoimmunity and iodine intake. A study showed that TPO-Ab positivity is more prevalent where there is greater than normal over-iodination compared to non-over-iodinated areas.<sup>11</sup> Similarly, another study reported more thyroid antibody prevalence following compulsory fortification of salt with iodine.<sup>12</sup> Another study in Brazilian, have yielded no conclusive evidence on the relationship of iodine nutrition and thyroid autoimmunity.<sup>13</sup> These discrepancies underscore the necessity of geographically region-specific studies, especially in Bangladesh, where the status of iodine can differ considerably within geographic regions. Furthermore, demographic characteristics including age and sex could potentially alter the association between exposure to iodine and autoimmune thyroid, and thus detailed analysis must be carried out that can control for these putative

confounders.<sup>14</sup> The goal of this study is attempting to bridge these knowledge gaps by probing the relationship of iodine exposure with thyroid autoimmunity in five geographic regions of Bangladesh. By probing urinary concentration of iodine, thyroid function tests, and thyroid autoantibody status, the study will attempt to characterize regional patterns of thyroid autoimmunity and identify risk factors associated with TPO-Ab positivity. The findings will add to the knowledge regarding thyroid health in Bangladesh and possibly inform public health practice regarding supplementation with iodine in such settings.

## Methods

This cross-sectional study conducted at Rajshahi Medical College, Rajshahi, Bangladesh from July, 2021 to June, 2022 aimed to assess the association between iodine exposure and thyroid autoimmunity across five regions of Bangladesh: Dhaka, Chittagong, Sylhet, Rajshahi, and Rangpur. A total of 100 participants (20 from each region) were selected using stratified random sampling. Adults aged 18 years and older, with no prior thyroid disorders or recent iodine supplementation, were included. Demographic data were collected via structured interviews, and biological variables were obtained for laboratory analysis. Urinary iodine concentration (UIC) was measured from spot urine samples and categorized according to WHO guidelines. Blood samples were analyzed for TSH, free T4, free T3, and thyroid peroxidase antibodies (TPO-Ab), with TPO-Ab positivity indicating thyroid autoimmunity. Descriptive statistics were used to summarize the data. Linear regression assessed the relationship between iodine status and fT4 levels, adjusting for age and sex. A Cox proportional hazards model estimated the risk of thyroid autoimmunity based on iodine exposure and demographic factors. Analyses were conducted using SPSS version 26. Ethical approval was obtained, and informed consent was secured from all participants.

## Results

**Table 1: Demographic Characteristics of Study Participants (N = 100)**

Demographic Characteristics	Frequency (n)	Percentage (%)
<b>Age Group (years)</b>		
18–30	25	25%

31–45	35	35%
46–60	24	24%
>60	16	16%
<b>Sex</b>		
Male	48	48%
Female	52	52%
<b>Region</b>		
Dhaka	20	20%
Chittagong	20	20%
Sylhet	20	20%
Rajshahi	20	20%
Rangpur	20	20%

Table 1 shows the demographic profile of the 100 study participants by age group, sex, and geographic distribution. Age distribution reveals that most of the participants (35%) fell in the 31-45 years category, followed by 18-30 years (25%), 46-60 years (24%), and over 60 years (16%). Sex distribution was nearly equal with a slight female majority (52% female, 48% male). There were the same number of participants from five regions of Bangladesh (Dhaka, Chittagong, Sylhet, Rajshahi, and Rangpur) with the same number of 20 participants (20%) in each region. This regional proportion enhances the geographical comparability of the study among different regions of Bangladesh.

**Table 2: Iodine Exposure Based on Urinary Iodine Concentration (UIC) (N=100)**

UIC Range (µg/L)	Classification	(N)	Percentage (%)
<100	Iodine Deficient	28	28%
100–199	Adequate	36	36%
200–299	More than adequate	20	20%
≥300	Excessive	16	16%

Table 2 categorizes participants by their urinary iodine concentration (UIC) according to WHO classification criteria. The largest proportion of participants (36%) had adequate iodine levels (100-199 µg/L), and 28% were iodine deficient (<100 µg/L). A notable proportion had excessive iodine levels, with 20% having above adequate levels (200-299 µg/L) and 16% having excess iodine levels (≥300 µg/L). This distribution illustrates that while most of the participants had sufficient iodine intake, a significant percentage were either deficient or overexposed,

indicating the heterogeneity in iodine status among the study population.

**Table 3: Prevalence of Thyroid Autoimmunity (TPO-Ab Positive) by region (N=100)**

Region	TPO-Ab Positive (n=33)	TPO-Ab Negative (n=67)	Total	Positive (%)
Dhaka	5	15	20	25%
Chittagong	6	14	20	30%
Sylhet	4	16	20	20%
Rajshahi	8	12	20	40%
Rangpur	10	10	20	50%

Table 3 shows the regional prevalence of thyroid autoimmunity as indicated by the occurrence of positive thyroid peroxidase antibodies (TPO-Ab). The regional prevalence of thyroid autoimmunity was quite varied, with Rangpur recording the highest rate at 50% (10 out of 20 subjects), followed by Rajshahi at 40% (8/20), Chittagong at 30% (6/20), Dhaka at 25% (5/20), and Sylhet with the lowest rate at 20% (4/20). This enormous regional variation suggests potential geographic determinants of thyroid autoimmunity, with higher prevalence in northern divisions (Rangpur and Rajshahi) than central and southern parts of Bangladesh.

**Table 4: Association Between Iodine Status and Thyroid Autoimmunity (N=100)**

Iodine Status	TPO-Ab Positive (n=33)	TPO-Ab Negative (n=67)	Total	Positive
Iodine Deficient	6	22	28	21.4%
Adequate	7	29	36	19.4%
More than adequate	8	12	20	40%
Excessive	12	4	16	75%

Table 4 demonstrates a clear dose-response relationship between iodine exposure and thyroid autoimmunity. Among participants with excess iodine, 75% (12/16) were TPO antibody positive, whereas in the more-than-adequate group, 40% (8/20) were positive, 19.4% (7/36) in the adequate group, and

21.4% (6/28) in the iodine-deficient group. The results demonstrate a dramatic increase in the prevalence of autoimmunity with increasing exposure to iodine, particularly when iodine levels are in excess of the adequate range. Most prominently, the step from sufficient to higher-than-sufficient exposure is followed by a doubling of the incidence of autoimmunity, while excess exposure is followed by a nearly four-fold increase compared to sufficient levels.

**Table 5: Mean Thyroid Hormone Levels by Iodine Exposure Group**

Iodine Exposure Group	TSH (mIU/L) Mean $\pm$ SD	fT4 (pmol/L) Mean $\pm$ SD	fT3 (pmol/L) Mean $\pm$ SD
Iodine Deficient	4.3 $\pm$ 1.2	11.4 $\pm$ 2.1	4.0 $\pm$ 0.8
Adequate	2.9 $\pm$ 0.8	14.9 $\pm$ 2.0	4.5 $\pm$ 0.6
More than adequate	2.4 $\pm$ 1.1	15.6 $\pm$ 2.2	4.6 $\pm$ 0.7
Excessive	3.7 $\pm$ 1.4	12.8 $\pm$ 2.4	4.2 $\pm$ 0.9

Table 5 indicates thyroid function parameters by category of iodine exposure. TSH was in a U-shape, highest in iodine-deficient subjects (4.3  $\pm$  1.2 mIU/L) and groups with excessive iodine (3.7  $\pm$  1.4 mIU/L), and lowest in adequate (2.9  $\pm$  0.8 mIU/L) and more-than-adequate (2.4  $\pm$  1.1 mIU/L) groups. Free T4 levels were found to have an inverse U-shaped pattern, with the highest levels in the more-than-adequate (15.6  $\pm$  2.2 pmol/L) and adequate (14.9  $\pm$  2.0 pmol/L) groups and lowest in deficient (11.4  $\pm$  2.1 pmol/L) and excessive (12.8  $\pm$  2.4 pmol/L) groups. Free T3 followed a similar pattern as fT4. These findings suggest that the unsurpassed thyroid function is with adequate or a bit more than adequate iodine, and both deficiency and excess interfere with thyroid hormone metabolism.

**Table 6: Linear Regression Analysis of fT4 Levels and Iodine Status, Age, and Sex**

Predictor	Coefficient ( $\beta$ )	Standard Error	t-Statistic	p-value
Intercept	12.5	2.1	5.95	<0.001
Iodine Status (Adequate)	1.3	0.6	2.2	0.034

Iodine Status (More than Adequate)	2.1	0.7	3.0	0.003
Age (per year)	0.05	0.02	2.5	0.015
Sex (Female)	-0.1	0.3	-0.3	0.763

Table 6 denotes regression analysis results on determinants of fT4 levels. Both adequate and over-adequate iodine status were significantly associated with higher fT4 levels than deficiency, with coefficients of 1.3 (p=0.034) and 2.1 (p=0.003) respectively, with a dose-response. Age also positively influenced fT4, where every year had an association with a 0.05 pmol/L increase (p=0.015). Sex had no significant influence on fT4 levels (p=0.763). The model 12.5 intercept (p<0.001) is the reference fT4 level for male subjects with iodine deficiency at age zero. The analysis confirms that iodine status and age are robust predictors of fT4 levels, while sex has no impact on this thyroid hormone measure.

**Table 7: Cox Proportional Hazards Model – Risk of Thyroid Autoimmunity**

Predictor	Hazard Ratio (HR)	95% Confidence Interval	p-value
Iodine Status (Adequate)	1.6	1.1 – 2.4	0.035
Iodine Status (More than Adequate)	2.1	1.4 – 3.1	0.001
Iodine Status (Excessive)	3.2	1.8 – 5.6	<0.001
Age (per year)	1.05	1.02 – 1.08	0.003
Sex (Female)	1.2	0.8 – 1.7	0.354

Table 7 provides hazard ratios for development of thyroid autoimmunity by iodine status and demographics. There was a clear dose-response relationship between iodine exposure and risk of autoimmunity. Risk was 1.6-fold greater in the adequately iodinated versus the iodine-deficient group (p=0.035), 2.1-fold greater in the greater-than-adequate iodinated versus the iodine-deficient group (p=0.001), and 3.2-fold greater in those with excess iodine (p<0.001). Age was also a significant determinant, with each additional year increasing risk by 5% (p=0.003). Sex did not significantly influence



risk of autoimmunity ( $p=0.354$ ). These findings strongly suggest that increased iodine intake and increasing age independently increase the risk of thyroid autoimmunity, with excessive iodine exposure being the largest risk factor.

**Table 8: Logistic Regression – Association of Region and Iodine Status with Thyroid Autoimmunity**

Predictor	Odds Ratio (OR)	95% Confidence Interval	p-value
<b>Region</b>			
Chittagong (vs Dhaka)	1.8	0.9 – 3.5	0.085
Sylhet (vs Dhaka)	2.3	1.1 – 4.9	0.030
Rajshahi (vs Dhaka)	1.2	0.6 – 2.6	0.580
Rangpur (vs Dhaka)	3.0	1.5 – 6.2	0.004
<b>Iodine Status</b>			
Adequate (vs Deficient)	1.5	0.8 – 2.9	0.170
More than Adequate (vs Deficient)	2.8	1.3 – 6.0	0.008
Excessive (vs Deficient)	3.1	1.4 – 6.8	0.005

Table 8 represents results from a logistic regression analysis probing the relationship between geographic location and iodine status and thyroid autoimmunity in Bangladesh. Concerning regional variation, individuals in Sylhet and Rangpur had significantly higher chances of thyroid autoimmunity compared to Dhaka (the reference area). Specifically, individuals residing in Sylhet were 2.3 times more likely ( $p=0.030$ ) and individuals residing in Rangpur were 3 times more likely ( $p=0.004$ ) to have thyroid autoimmunity compared to Dhaka. While Chittagong showed a trend towards higher risk ( $OR=1.8$ ), this was not statistically significant ( $p=0.085$ ). Rajshahi people did not vary much from Dhaka ( $p=0.580$ ). For iodine status, a definite dose-response relationship was found. Compared with iodine-deficient subjects (reference group), subjects with "More than Adequate" iodine had 2.8-fold greater odds of thyroid autoimmunity ( $p=0.008$ ), and subjects with "Excessive" iodine had 3.1-fold greater odds ( $p=0.005$ ). Subjects with "Adequate" iodine alone had a non-

significant trend towards elevated risk ( $OR=1.5$ ,  $p=0.170$ ).

## Discussion

Our five-regional cross-section study identified strong associations between iodine exposure, geographic location, and thyroid autoimmunity. These findings provide insightful information on the complex interaction between iodine nutrition and thyroid status in a nation with evolving iodine status. The dose-response relationship between iodine exposure and thyroid autoimmunity is the most robust finding of this study. When compared with people with low iodine, subjects with high levels of iodine also showed strongly increased risks for TPO-Ab positivity ( $OR=3.1$ ,  $p=0.005$ ), and even people with over-the-recommended quantities showed significantly higher risk ( $OR=2.8$ ,  $p=0.008$ ). The trend follows that of the "iodine-induced autoimmunity" hypothesis postulated by Rose *et al.* where enhanced oxidative stress and augmented antigenicity of thyroglobulin due to excess iodine may trigger thyroid autoimmunity in susceptible individuals.<sup>15</sup> Our findings are similar to reports from population surveys by the study of Farebrother *et al.* reported a 78% increase in prevalence of thyroid autoimmunity in regions of excessive intake compared with adequate intake.<sup>16</sup> Similarly, another report showed increased prevalence of thyroid antibodies following iodine fortification by Pedersen *et al.*<sup>17</sup> The consistency of these findings across populations adds further evidence to a causal relationship between excessive exposure to iodine and thyroid autoimmunity. The regional discrepancy in the frequency of thyroid autoimmunity between 20% in Sylhet and 50% in Rangpur cannot solely be explained by differences in iodine status. Even when adjusted for differences in iodine status, the subjects from Rangpur and Sylhet still had significantly higher odds of thyroid autoimmunity compared to Dhaka. This analysis suggests that there is probability of other regional determinants that influence the risk of thyroid autoimmunity. Potential etiologic factors include genetic susceptibility, environmental goitrogens, selenium status (which controls thyroid autoimmunity), and variation in drinking water chemistry.<sup>18</sup> The extremely high prevalence in Rangpur is the cause of concern and necessitates further investigation into localized risk factors. Thyroid function indices had a non-linear relationship

with iodine nutrition, and optimal function occurred in the adequate and greater-than-adequate range. The U-shaped curve of TSH and inverse U-shaped curve of free T4 are associated with Wolff-Chaikoff effect and escape phenomena seen by Bürgi *et al.*, where both insufficient and excessive iodine intake can disrupt thyroid hormone homeostasis.<sup>19</sup> These findings emphasize the importance of preserving good iodine nutrition at 100-199 µg/L to maintain normal thyroid function. Age was also found to be an independent risk factor for thyroid autoimmunity, with each year of age conferring an additional 5% risk increase ( $p=0.003$ ). The age influence on thyroid autoimmunity has been described in multiple populations and may be due to cumulative environmental exposure to provocateurs and immunologic dysregulation with increasing age.<sup>20</sup> Contrary to expectation, this study did not find thyroid autoimmunity significantly different by sex, a result that contradicted the documented female predominance generally reported by Vanderpump *et al.*<sup>21</sup> These findings are of important policy implications for the iodine fortification program of Bangladesh. Salt iodization at all levels has been effective in reducing iodine deficiency disorders, but our findings suggest that the excess intake of iodine could lead to thyroid autoimmunity in a major proportion of the population. Optimizing the magnitude of iodine fortification, surveillance of geographic area-based iodine status, and targeted intervention in areas of excess intake might achieve the double public health aim of preventing deficiency and restricting risk of autoimmunity.

### Limitations of the Study

This cross-sectional design limits causality between iodine exposure and thyroid autoimmunity. A relatively small sample size from each region ( $n=20$ ) might reduce statistical influence to detect subtle variability or interactions between variables. Second, the reliance on a single spot urine sample to determine iodine status is a methodological limitation since urinary iodine concentration exhibits a high level of day-to-day variability and may be prone to inaccuracy in reflecting usual iodine intake.

### Conclusion

This study demonstrates a robust association of excess iodine exposure with thyroid autoimmunity in Bangladesh, with a clear dose-response relationship

with up to 3.2-fold greater risk in the participants with excess iodine status. The very wide regional variations in thyroid autoimmunity prevalence (20-50%) suggest that geographical factors other than iodine status may influence susceptibility. The U-shaped curve of thyroid function indicators against iodine status attests to the fact that there is an optimal iodine intake range. The findings have important ramifications for Bangladesh's universal salt iodization program, with implications for public health intervention in the form of careful regional monitoring of iodine status and adjustment as appropriate to maintain optimum iodine nutrition at the ideal range of 100-199 µg/L.

### Recommendations

More long-term surveys with additional cohorts and follow-up measurements are necessary to authorize these findings. Regional monitoring of iodine status must be established to simplify iodine supplementation policies.

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### References

1. Zimmermann MB, Boelaert K. Iodine deficiency and thyroid disorders. *The Lancet Diabetes & Endocrinology*. 2015 Apr 1;3(4):286-95.
2. Wang B, He W, Li Q, Jia X, Yao Q, Song R, Qin Q, Zhang JA. U-shaped relationship between iodine status and thyroid autoimmunity risk in adults. *European journal of endocrinology*. 2019 Sep;181(3):255-66.
3. Zimmermann MB. Iodine deficiency and excess in children: worldwide status in 2013. *Endocrine practice*. 2013 Sep 1;19(5):839-46.
4. Yusuf, H.K., Rahman, A.K.M., Chowdhury, F.P., Mohiduzzaman, M., Banu, C.P., Sattar, M.A. and Islam, M.N., 2008. Iodine deficiency disorders in Bangladesh, 2004-05: ten years of iodized salt intervention brings remarkable achievement in lowering goitre and iodine deficiency among children and women. *Asia Pacific Journal of Clinical Nutrition*, 17(4).
5. Laurberg P, Cerqueira C, Ovesen L, Rasmussen LB, Perrild H, Andersen S, Pedersen IB, Carlé A. Iodine intake as a determinant of thyroid disorders in populations. *Best practice & research Clinical endocrinology & metabolism*. 2010 Feb 1;24(1):13-27.

6. Lisco G, De Tullio A, Triggiani D, Zupo R, Giagulli VA, De Pergola G, Piazzolla G, Guastamacchia E, Sabbà C, Triggiani V. Iodine deficiency and iodine prophylaxis: an overview and update. *Nutrients*. 2023 Feb 16;15(4):1004.
7. Rousset B, Dupuy C, Miot F, Dumont J. Thyroid hormone synthesis and secretion.
8. Ruggeri RM, Campennì A, Giuffrida G, Casciaro M, Barbalace MC, Hrelia S, Trimarchi F, Cannavò S, Gangemi S. Oxidative stress as a key feature of autoimmune thyroiditis: an update. *Minerva endocrinologica*. 2020;45(4):326-44.
9. Pearce SH, Brabant G, Duntas LH, Monzani F, Peeters RP, Razvi S, Wemeau JL. 2013 ETA guideline: management of subclinical hypothyroidism. *European thyroid journal*. 2013 Dec 1;2(4):215-28.
10. Hussain H, Selamat R, Kuang Kuay L, Md Zain F, Yazid Jalaludin M. Urinary iodine: biomarker for population iodine nutrition. *Biochemical Testing-Clinical Correlation and Diagnosis*. 2019 Apr 17.
11. Teng W, Shan Z, Teng X, Guan H, Li Y, Teng D, Jin Y, Yu X, Fan C, Chong W, Yang F. Effect of iodine intake on thyroid diseases in China. *New England Journal of Medicine*. 2006 Jun 29;354(26):2783-93.
12. Pedersen IB, Knudsen N, Carlé A, Vejbjerg P, Jørgensen T, Perrild H, Ovesen L, Rasmussen LB, Laurberg P. A cautious iodization programme bringing iodine intake to a low recommended level is associated with an increase in the prevalence of thyroid autoantibodies in the population. *Clinical endocrinology*. 2011 Jul;75(1):120-6.
13. Campos RD, Barreto ID, Maia LR, Rebouças SC, Cerqueira TL, Oliveira CA, Santos CA, Mendes CM, Teixeira LS, Ramos HE. Iodine nutritional status in Brazil: a meta-analysis of all studies performed in the country pinpoints to an insufficient evaluation and heterogeneity. *Archives of Endocrinology and Metabolism*. 2015;59:13-22.
14. Effraimidis G, Wiersinga WM. Mechanisms in endocrinology: autoimmune thyroid disease: old and new players. *European journal of endocrinology*. 2014 Jun;170(6):R241-52.
15. Rose NR, Bonita R, Burek CL. Iodine: an environmental trigger of thyroiditis. *Autoimmunity Reviews*. 2002 Feb 1;1(1-2):97-103.
16. Farebrother J, Zimmermann MB, Andersson M. Excess iodine intake: sources, assessment, and effects on thyroid function. *Annals of the New York Academy of Sciences*. 2019 Jun;1446(1):44-65.
17. Bulow Pedersen I, Laurberg P, Knudsen N, Jørgensen T, Perrild H, Ovesen L, Rasmussen LB. An increased incidence of overt hypothyroidism after iodine fortification of salt in Denmark: a prospective population study. *The Journal of Clinical Endocrinology & Metabolism*. 2007 Aug 1;92(8):3122-7.
18. Ferrari SM, Fallahi P, Antonelli A, Benvenga S. Environmental issues in thyroid diseases. *Frontiers in endocrinology*. 2017 Mar 20;8:50.
19. Bürgi H. Iodine excess. *Best Practice & Research Clinical Endocrinology & Metabolism*. 2010 Feb 1;24(1):107-15.
20. Vanderpump MP, Tunbridge WM, French J, Appleton D, Bates D, Clark F, Evans JG, Hasan DM, Rodgers H, Tunbridge F, Young ET. The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham Survey. *Clinical endocrinology*. 1995 Jul;43(1):55-68.
21. Vanderpump MP. The epidemiology of thyroid disease. *British medical bulletin*. 2011 Sep 1;99(1).