



## Original Research Article

## Evaluation of thyroid profile in active and passive smokers

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## ABSTRACT

Tobacco has multiple effects on the hypothalamic-pituitary-thyroid axis and the functioning of the thyroid gland. The adverse mechanisms of smoke exposure include alteration of thyroid hormone synthesis, binding, transport, storage and clearance, thereby resulting in changes in the circulating hormone concentrations. This study was designed to analyze the effect of smoking on thyroid profile, measuring serum TSH, total T3 and total T4 levels in active and passive smokers. The results revealed that both active and passive smokers had highly significant decreased serum total T3 and T4 levels along with substantially increased serum TSH levels in comparison to non-smokers ( $p < 0.001$ ). However, the variation of hormone status between the both case groups was less statistically significant ( $p < 0.05$ ), indicating the deleterious nature of tobacco smoke on the activity of thyroid, irrespective of its nature and origin.

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## 1. Introduction

Cigarettes are considered as the commonest source of toxic chemical exposure and chemically mediated illness in humans. The tobacco epidemic is one of the biggest public health threats the world has ever faced, killing more than 8 million people a year. More than 7 million of those deaths are the result of direct tobacco use, while around 1.2 million are the result of non-smokers being exposed to second hand smoke (SHS).<sup>1</sup> The International Agency for Research on Cancer classifies cigarette tobacco smoke as a human carcinogen.<sup>2</sup>

Active and passive smoking can result in smoke induced interference with thyroid hormone homeostasis.<sup>3</sup> The effect of cigarette smoke on thyroid is believed to be mostly due to a compound, thiocyanate, a derivative of hydrogen cyanide with a half life of  $> 6$  days.<sup>4,5</sup> It has been studied extensively as a potential goitrogen.<sup>6</sup> It inhibits iodide transport and organification, and in the presence of iodine deficiency, can cause goiter.<sup>7</sup> A burning cigarette emits both mainstream and sidestream smoke. Second Hand Smoke

(SHS), to which to which passive smokers are exposed to, comprises of both mainstream (11%) and sidestream smoke (85%) along with other contaminants.<sup>8</sup> Thus passive smokers are exposed to a different spectrum of toxicants than active smokers.

The present study was undertaken to evaluate the effect of exposure of different types of smoking on the thyroid status of the study group. This variation of hormone profile could be taken into account while considering management for such individuals

## 2. Materials and Methods

Apparently healthy non smokers and smokers (active and passive) with no history of thyroid disorders, in the age group of 18-50 years visiting the OPD of Pradyumna Bal Memorial Hospital, Kalinga Institute of Medical Sciences, Bhubaneswar were selected as controls and cases respectively. The cases were categorized into active and passive smokers based on the declaration of the participants through a questionnaire based on tobacco smoke exposure profiles. 50 non smokers, 50 active and 50 passive smokers were included in the study group.

Inclusion criteria of the participants were as follows:

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1. Active smokers: Participant s who have been smoking for the past 6 months
2. Passive smokers: Participant s who have been regularly exposed to SHS within the previous 6 months
3. Controls/non -smokers: Participants who have not been exposed to SHS within the previous 6 months.

Known cases of thyroid disorders or with any past history of thyroid dysfunction, patients on anti-thyroid drugs or other medications like corticosteroids and beta blockers, smoking for less than 6 months, conditions that alter thyroid profile like related endocrinological disorders or suffering from major illness, infections or immunological disorders were excluded from the study.

Fasting blood samples were collected from all subjects (cases and controls), centrifuged and serum was used to estimate thyroid profile: Total T3 (Triiodothyronine), Total T4 (Thyroxine), and TSH (Thyroid Stimulating Hormone). These parameters were estimated by Electrochemiluminescence immunoassay (ECLIA) by Roche Cobase 411 immunoassay analyzer by sandwich principle.<sup>9</sup> Analysis was completed within 24 hours of collection. The data was statistically analyzed using STATA software.

### 3. Results

The baseline parameters like age, gender and BMI were not significant predictors in the study group (Table 1).

**Table 1:** Baseline characteristics of study participants

Parameter	Analysis variable	%
Age	20 - 35	28
	35 – 50	72
Gender	Male	76
	Female	24
BMI	<25	23
	25 – 30	60
	>30	17

Out of the 50 non- smokers, 78% of the participants had normal thyroid profile while 12% & 10% were hypothyroid and hyperthyroid respectively. Among the active smokers, 64% of the participants had hypothyroid, 18 % had hyperthyroid & 18% were euthyroid while in the passive smokers group, 52% of the participants had hypothyroid, 20% had hyperthyroid & 28% were euthyroid. (Table 2)

**Table 2:** Percentage of participants having hypothyroid, hyperthyroid and euthyroid states

Groups	Hypothyroid (%)	Hyperthyroid (%)	Euthyroid (%)
Non-smokers	12	10	78
Active smokers	64	18	18
Passive smokers	52	20	28

Thyroid profile in the study group as depicted in Table 3, revealed a highly significant variation (decrease in serum T3 And T4 and increase in Serum TSH) of  $p < 0.001$ , in both active and passive smokers in comparison to non- smokers.

**Table 3:** Thyroid Profile in the study groups

Parameter	Non - smokers	Active smokers	Passive smokers
Serum Total T3 (ng/ml)	1.55 ± 0.29	0.75 ± 0.36**	0.9 ± 0.48**
Serum Total T4 (µg/dl)	5.91 ± 1.02	3.26 ± 0.83**	3.76 ± 1.38**
Serum TSH (µIU/ml)	3.03 ± 0.73	7.12 ± 2.18**	5.92 ± 2.52**

\*\*Indicates  $p < 0.001$  – highly significant difference as compared to controls

Table 4: illustrates a fall in serum T3 and T4 and a rise in serum TSH in active smokers in comparison to passive smokers which was however less significant ( $p < 0.05$ ), indicating alteration in thyroid status in both the case groups due to smoking irrespective of their different modes exposure.

**Table 4:** Comparison of thyroid status between the Cases

Parameter	Active smokers	Passive smokers
Serum Total T3 (ng/ml)	0.75 ± 0.36	0.9 ± 0.48*
Serum Total T4 (µg/dl)	3.26 ± 0.83	3.76 ± 1.38*
Serum TSH (µIU/ml)	7.12 ± 2.18	5.92 ± 2.52*

\*Indicates  $p < 0.05$  - significant difference between the case groups

### 4. Discussion

Tobacco smoke modifies almost all functions of the thyroid gland. The injurious effect of smoking becomes apparent when thyroid function is compromised,<sup>10</sup> contributing towards hypothyroidism.<sup>11</sup> It might have a dual mode of action on the thyroid gland, one of direct suppression by thiocyanate, and other by indirect activation through the Hypothalamus –pituitary axis. Thiocyanate inhibits iodine uptake and inhibits hormone synthesis as it competes with iodide in the organification process.<sup>7,12</sup> Other components of smoke like 2,3 hydroxypyridine, also interfere with thyroid function by inhibiting deiodination by reducing iodothyronine deiodinase activity.<sup>13</sup>

Numerous studies have reported variable results with a decrease, increase or no effect of smoking on peripheral thyroid hormones.<sup>14,15</sup> T4 and reverse T3 (rT3) have been found to be increased along with normal T3 levels in some publications,<sup>16</sup> where others have reported a rise in T3 without any accompanying elevation in T4.<sup>17</sup> The effect of thyroid smoke on thyroid function is related to higher levels of thyroxine binding globulin among smokers compared to

non smokers as well as higher levels of thyrotoxins in tobacco smoke in heavy smokers compared to light and moderate smokers.<sup>18</sup>

The present study illustrated low serum T3 and T4 levels and significantly high TSH values in both active and passive smokers which corroborated with the findings of Nystrom et al<sup>11</sup> and S Fukata et al<sup>19</sup> respectively. Nystrom demonstrated significantly low values of serum T3 in comparison to non-smokers while Fukata indicated a relationship between smoking and hypothyroidism attributing this phenomenon to increased serum thiocyanate which is contributed to smoking.

To conclude this study provides additional valuable information on how cigarette smoking affects thyroid functions in and will contribute to the existing knowledge of its detrimental effects. Thus it may be instrumental in early detection and management of thyroid disease in smokers. An increase in awareness among participants regarding their individual thyroid status may act as a deterrent for smoking and thus motivate them in improving their lifestyle habits.

## 5. Source of funding

None.

## 6. Conflict of interest

None.

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