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## Lipid peroxidation and superoxide dismutase levels variation in hypothyroidism patients

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### ABSTRACT

The study aims to find the antioxidant status in hypothyroidism. Hypothyroidism is otherwise known as an underactive thyroid. It takes place when the thyroid gland does not produce enough thyroid hormone for the body need. 40 subjects were divided into two groups of the normal healthy individual (20 in numbers) as Group I and hypothyroidism patients (20 in numbers) as Group II from the dental outpatient department of Saveetha Dental College and Hospitals. Blood samples were collected from the participants, and it was distributed in a plain collection tube and centrifuged in 2500 rpm for 10 minutes. The Serum was separated and analysed for Malanaldehyde and Superoxide dismutase by TBARS method and Pyrogallol Autoxidation method using ERBA CHEM 5 plus analyser. There is a significant increase in (MDA) lipid peroxidation ( $p < 0.005$ ) as well as there is a significant decrease in (SOD) superoxide dismutase ( $p < 0.005$ ) in Hypothyroidism patients. Our study revealed that the women with PCOS have an imbalance in their antioxidant status. Increased oxidative stress and decreased antioxidant capacity may contribute to the increased risk of CVD in women with Hypothyroidism. Further research on oxidative stress in hypothyroidism is needed.



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### INTRODUCTION

Hypothyroidism occurs when the thyroid gland lacks enough thyroid hormones to meet the needs of the body. T<sub>3</sub> and T<sub>4</sub> are the thyroid hormone produced by the thyroid gland. These two hormones control the metabolism of the body (Prakash A *et al.*, 2006). This is a very common thyroid disorder. It occurs most often in women and the age changes and may be a genetic cause. Hypothyroidism has an adverse effect on pregnant women and growing

foetus (Abalovich M *et al.*, 2007). If the thyroid gland is not developed correctly, hypothyroidism can also be from birth. Hashimoto disease is one of the most common causes of hypothyroidism in adults. This is where your immune system is in a position to attack your thyroid, which can cause it to be damaged and not enough hormones. Radiation iodine or surgery can cause hypothyroidism on the thyroid gland (which is used to treat other types of thyroid disorders). The problem with the Pituitary is another rare cause. Medicinal and congenital hypothyroidism and thyroiditis are risk factors that affect hypothyroidism (Prakash A *et al.*, 2006, Abalovich M *et al.*, 2007). Depression of metabolism due to hypothyroidism has been reported to be decreased in oxidant production and thus protects the tissues against oxidant damage (M. J. Coria *et al.*, 2009).

The antioxidant is the low concentration of proteins, carbohydrates, lipids and oxidation of DNA. Antioxidants neutralise the effects of ROS, and this helps in preventing diseases. Antioxidants are natural or synthetic. Natural antioxidants can be taken

directly through food as it contains fruits, vegetables and spices. Some synthetic antioxidants, such as BHT and BHA, also prevent oxidation. However, these synthetic antioxidants have now been reported to be hazardous to humans, and the search for poisonous antioxidants (Vinita Sindhia *et al.*, 2013).

The oxidation stress results are the antibody between the antioxidant protection systems and the production rate of the reactive oxygen species (ROS). This leads to lipid peroxidation and oxidative DNA loss but can interfere with physiology and intracellular signal transduction. The resulting change in intracellular redox status leads to the activation of protein kinase, for example, tyrosine kinase, protein kinase c, and the Mario-activated protein kinase cascade leading to modified cellular functions.

Oxidative stress compounds hypothyroidism. Hypothyroidism is a state that increases the oxidative stress. In this study, the biomarker is high in MDA level therapy-innocent primary hypothyroid patients. After treatment with L-thyroxine, the pressure marker is reduced to a considerable degree. MDA can also be used as a useful biomarker to measure and monitor oxidative stress. The role of antichrist in the form of selenium is incomplete (Sumit Kumar Chakrabarti *et al.*, 2016). In our research, we find antioxidant status in hypothyroidism

## MATERIAL AND METHODS

40 subjects were selected from the outpatient department of Saveetha Dental College and Hospitals. They were divided into two groups.

Group I (Control group) – Normal healthy individuals – 20 in numbers

Group II (Study group) – Hypothyroidism patients – 20 in numbers

### Inclusion Criteria

1. A normal healthy individual with normal BMI (19.9-249)
2. Known Hypothyroidism Patients

### Exclusion Criteria

1. Subjects with systemic diseases like Diabetes Mellitus, CVD, Hypertension and other endocrine disorders.
2. Immunocompromised persons

### Sample collection and procedure

5 ml of venous blood was collected from the participants and blood was distributed in the plain collection tube and centrifuged in 2500 rpm for 10 minutes. The Serum was separated and analysed

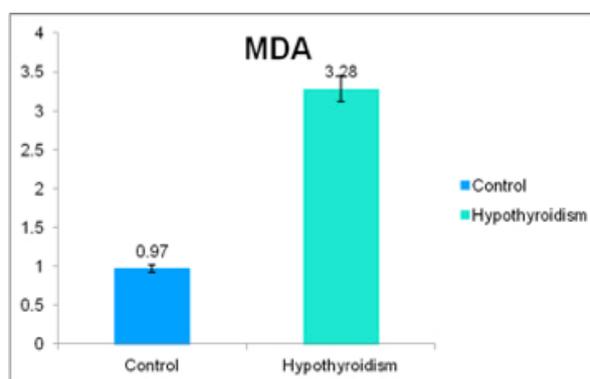
for Melanaldehyde and Superoxide dismutase by TBARS method and Pyrogallol Autoxidation method using ERBA CHEM 5 plus analyser.

## RESULTS AND DISCUSSION

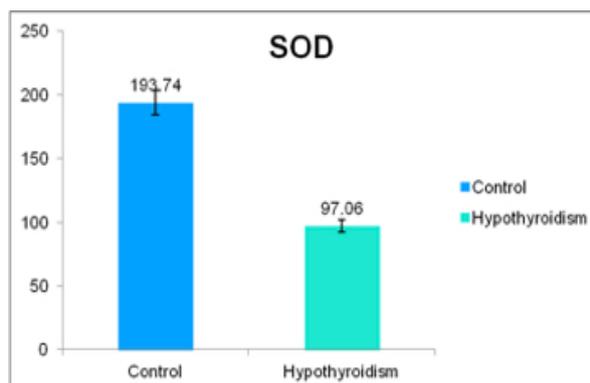
They postulated the role of excess TSH to produce oxidative stress directly. Hypothyroidism is associated with increased oxidative stress response. Treatment with L-thyroxine is effective in bringing a reduction in the level of stress markers (Sumit Kumar Chakrabarti *et al.*, 2016).

**Table 1: Mean, SD and p value of Control and Study groups**

Parameters	Control	Hypothyroidism	p value
MDA(nmol/ml)	0.97 ± 0.57	3.28 ± 1.01	<0.005
SOD(U/ml)	193.74 ± 27.34	97.06 ± 21.65	<0.005



**Figure 1: comparison of MDA level in a normal healthy patient with the known hypothyroidism patient**



**Figure 2: Comparison of SOD level in a normal healthy patient with the known hypothyroidism patient**

Hypothyroid patients present higher lipoperoxide (LPx) levels, a significant higher LDL content in the lipid peroxides and higher oxidation rate; they also exhibit an elevation in  $\beta$ -carotene levels with higher LDL oxidation. Finally oleic to linoleic acid ratio, which is inversely proportional to oxidative stress, is lower in hypothyroidism (Antonio

Mancini *et al.*, 2013). The treatment with thyroid hormones decreased MDA levels and increased PON-1 activity, even though values similar to those observed in controls were not reached. They hypothesised that in patients with hypothyroidism the pro-oxidant environment could play a role in the development of atherosclerosis. Elevated MDA levels were also shown in subclinical. Hypothyroidism causes immunosuppression that may lead to oxidative stress. According to Kale, Thyroid hormones accelerate cellular reactions and increase oxidative metabolism. By stimulating enzymes that control active transport pumps, demand for cellular oxygen increases, and as ATP production goes up, heat is produced. It can eventually lead to many other complications. Antioxidant therapy and antioxidant diet should be advised along with thyroid hormone replacement therapy to diminish further complications (Kale M *et al.*, 2006).

On the other hand, hyperthyroidism is characterised by an increasing cellular metabolic rate, and thus an increased amount of free radicals and an increase in peroxides levels (Nabila A *et al.*, 2016). Hypothyroidism always tends to decrease lipid peroxide level, and low level of thyroid hormone always diminishes SOD and GPX activity (Pereira B *et al.*, 1994). Free radical-scavenging enzymes such as SOD and CAT acts as the first line of cellular defence against the oxidative injuries, decomposing  $O_2^-$  and  $H_2O_2$  before it get interacted to form a more reactive hydroxyl radical (OH). These enzymes protect the red cells against  $O_2^-$  and  $H_2O_2$  mediated by lipid peroxidation (S. Senthil *et al.*, 2004). Hypothyroidism drops to change the complete dismutation capacity of the mitochondrial fractions of the heart but decreases significantly when challenged by  $T_3$  (S. Chattopadhyay *et al.*, 2003). The reason being, the introduction of hypothyroidism into the Oxidative stress model because it reduces the generation of free radicals and hence reduced pre-existing of Oxidative stress. All the reports strengthened the previous notion that hypothyroidism that associated with decreased Oxidative stress (Nivedita Nanda *et al.*, 2016). Hypothyroidism does not control the free-radical-to induce the oxidative damage, and that hypothyroidism may not present some protection against lipid peroxidation. Thus, enhances the lipid peroxidation may play a role in the free-radical-induced oxidative damage of some tissues in hypothyroidism. These may show that there is an essential relation between hypothyroidism and lipid peroxidation. The hypothyroidism is associated with increased susceptibility to lipid peroxidation compared to that in the euthyroid state (Sawant *et al.*, 2003). Hypothyroidism has been associated with glucose and insulin metabolism disorders that affect insulin secretion in response to glucose, hyperinsulinemia,

altered peripheral glucose disposal, and insulin resistance (Aniruddh Menon *et al.*, 2016). TSH production is regulated by the transmitters and hormones which regulate the body weight and satiation like neuropeptide Y,  $\alpha$  melanocytes stimulate hormone, and the agouti-related peptide innervates hypophysiotropic TRH neuron (Faazila Fathima *et al.*, 2016). Obesity affects thyroid function via many mechanisms including increasing levels of leptin hormone which subsequently affects TSH production (Maureen Jepakorir Cheserek *et al.*, 2015). The level of serum in the oxidant-antioxidant system as well as pre and post treatment showed that there is an increase in MDA level in hypothyroidism patient before the treatment and decreased after treatment but yet it remains higher than the control group (Kadayam G Gomathi *et al.*, 2012). Improper function of thyroid brings about pathological changes in various organs of the body. This was found from the in vivo and in vitro studies that how the thyroid hormones have a substantial impact on oxidative stress (Erdamar H *et al.*, 2008). The thyroid hormone-enhances the ROS generation in liver and damages the polyunsaturated fatty acids, proteins, and DNA occurs (Claudio Tomella *et al.*, 2014). Enzymatic antioxidants like SOD, CAT, GSH, and GSH-PX, which are considered to serve as protective responses for the elimination of reactive free radicals (Lingfa KONG *et al.*, 2015).

## CONCLUSION

Our study revealed that there is an imbalance of antioxidant status in women with PCOS. Increased oxidative stress and decreased antioxidant capacity may contribute to the increased risk of CVD in women with Hypothyroidism. Further research on oxidative stress in hypothyroidism is needed.

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