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Research Article

Ameliorative effect of curcumin and ascorbic acid against ultraviolet B radiation-induced thyroid toxicity in female Wistar rats: A haematological and biochemical study

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Abstract
The present study aimed to investigate the ameliorative effect of curcumin and ascorbic acid against ultraviolet (UVB)-induced thyroid toxicity and to study the haemtological and biochemical parameters. Twenty-four female Wistar rats, aged 3-4 months and weighing 130-150 g, were used, and the rats were divided into four Groups (Groups I to IV). Group I received standard food and water ad libitum and was treated as a control; Group II received a dose of 280 nm of UVB radiation for 2 hrs/day. Group Ⅲ received 280 nm UVB radiation for 2 hrs/day and received curcumin 25 mg/kg body weight given orally. Group IV received 280 nm of UVB radiation for 2 hrs/day and received ascorbic acid 250 mg/kg body weight given orally. All the treatments were consequently performed for 15 days. The results showed that haemtological parameters such as haemoglobin (Hb) ($p<0.05$), red blood cells (RBCs), white blood cells (WBCs), MCV, MCH, and MCHC decreased significantly. Biochemical parameters included lipid peroxidation (LPO) ($p<0.05$), H_2O_2 ($p<0.01$), nitric oxide (NO), superoxide dismutase (SOD) ($p< 0.01$), catalase ($p<0.01$), glutathione-S-transferase (GST) ($p<0.01$), and glutathione reductase. NO increased, and glutathione (GSH) ($p<0.01$) decreased significantly. However, cotreatment with curcumin and ascorbic acid significantly increased the haemtological parameters. In addition, oxidative parameters such as LPO ($p<0.01$), SOD ($p< 0.01$), CAT ($p<0.01$), GST ($p<0.01$), and NO ($p<0.01$) significantly increased, and GSH ($p<0.01$) significantly decreased upon cotreatment with curcumin and ascorbic acid. The results indicated the ameliorative effect of curcumin and ascorbic acid against UVB-induced thyroid toxicity in female Wistar rats.

Keywords: Ascorbic acid, Curcumin, Hematology, Oxidative stress, UVB radiation

INTRODUCTION

UVB radiation generates free radicals and causes oxidative stress affecting the endocrine system and its physiological processes, such as thyroid hormone action and the ovarian cycle (Rai *et al.,* 2018, and Rai *et al.,* 2020). Thyroid hormones have played a biological role in animals; they regulate haematopoiesis in the bone marrow (Golde *et al.,* 1977). It is known that haematological imbalance and thyroid dysfunction are linked together. Fein reported that Graves' disease correlated with anemia (Fein *et al.,* 1975). Previously, hypothyroidism was shown to cause positive forms of anemia and hyperproliferation of immature thyroid progenitors. The anemia is commonly hypochromic anemia of slight severity (JP and Srikrishna, 2012). Erythrocytosis is relatively not unusual, and anemia is not regularly determined in sufferers with hyperthyroidism (Iddah *et al.,* 2013). Previously, it was found that when the euthyroid condition earns returns to normal, all haematological parameters (Lima *et al.,* 2006).

In the hypothyroid, patients had decreased overall leucocyte matter, neutropenia, and thrombocytes (Axelrod *et al.,* 1951). Moreover, reduced general leucocyte counts were observed in hyperthyroid sufferers. There is a relatively lower variety of neutrophils and a boom in the range of eosinophils and mononuclear cells (MNCs). Nonetheless, hyperplasia of all myeloid mobile traces occurs in hyperthyroidism (Chen *et al.,* 2006).

Ultraviolet B (UVB) radiation induces various effects on all life forms on Earth, causing damage at both the physiological and molecular levels (Neugart *and* Schreiner, 2018). The intensity of UVB radiation reaching the Earth's surface is dependent on the ozone concentration in the stratosphere. Biological responses to changes in UVB radiation may have harmful effects on animals and humans. The impact of UVB radiation on humans and the environment has been reviewed regularly by the United Nations (Andrady *et al*., 2004 and United Nations Environmental Programme, 2004, 2017).

The hematopoietic system is one of the most radiosensitive systems. Its harm leads to the development of the hematopoietic syndrome and consequences in death. Survival after irradiation outcomes inside the restoration of several target structures along with the bone marrow, gastrointestinal tract, pores and skin, and hemostatic system (Raukar *and* Simpraga*,* 2005). Haematological parameter analysis of various illnesses, each metabolic and infectious (Romdhane *et al.,* 2000 and Chattopadhyay *et al.,* 2003).

Hypothyroidism and hyperthyroidism can each be associated with oxidative stress. Furthermore, thyroid hormone-triggered oxidative damage might be an issue responsible for the development of heart failure, as cautioned by the advantage of T3 management on antioxidant structures in rat hearts after pharmacological-prompted hypothyroidism. (Mahobiya, 2020 and Mancini *et al.,* 2013).

This oxidative pressure has been implicated in a selection of pathological situations collectively with cancer, irritation, diabetes mellitus, thyroid damage, aging, and so forth. Previous research found that every one of the stressors always causes growth stress in all tissues, as evident from the markers of oxidative stress, which incorporate lipid peroxidation (LPO), hydrogen peroxide (H2O2), nitric oxide (NO), catalase (CAT), superoxide dismutase (SOD), glutathione (GSH), and glutathione-s-transferase GST in numerous tissues, such as blood, muscle tissue, thyroid, and liver. Additionally, chemical pressure produces the most oxidative stress, and changes in oxidative stress markers in the blood are parallel with adjustments in blood and thyroid (Rani and Yadav*,* 2015). In this view, the present study investigated the ameliorative effect of curcumin and ascorbic acid against UVB-induced thyroid toxicity in female Wistar rats.

MATERIALS AND METHODS

Experimental design and treatment

Female Wistar rats weighing 130-150 gm were purchased from the College of Veterinary Sciences and Animal Husbandry Mhow, India. Animals were acclimatized to the animal housing with an ambient temperature of ~20-25°C, relative humidity of 50±5%, and a 12 h light:12 h dark cycle. Ethical approval was obtained from the Department of Pharmaceutical Sciences Dr. Harisingh Gour Vishwavidyalaya (Registration 379/Go/ReBi/S/01/CPCSEA), Sagar (M.P.). The care and use of laboratory animals followed international guidelines. All animals (n=24) were housed in plastic cages and given standard food and water ad libitum. The experimental animals were divided into four Groups, Group I to Group IV, and administered different doses as follows:

Group I (Control) - Received standard food and water ad libitum,

Group II (UVB exposed)- Received a dose of 280 nm of UVB radiation for two hours/day for 15 days.

Group III (UVB exposed+Curcumin)- Received a dose of curcumin (25 mg/kg body weight) orally for 15 days**.**

Group IV (UVB exposed+Ascorbic acid)- Received a dose of ascorbic acid (250 mg/kg body weight) orally for 15 days.

Collection of blood samples

At the end of exposure, each Group of animals was sensitized with chloroform, and then blood samples were collected by cardiac puncture and used for different blood parameter measurements.

Tissue homogenization

For biochemical estimation of the thyroid, tissues were homogenized in 0.02 M Tris HCl pH 7.4. After making a 10% homogenate of each tissue, the samples were centrifuged at 1000 g for 30 minutes at 4°C. The supernatant was collected and stored at -20°C for further analysis.

Determination of haematological parameters

The haematological study followed Dacie and Lewis*.,* 1991 and Osman *et al.,* 2010.

Haemoglobin (Hb)

Hb (g/dl) was estimated by following Sahli's acid haematin method. Twenty microliter of blood was poured into a glass tube containing HCl, and the acid haematin solution was mixed thoroughly with a glass rod and allowed to stand for 10 min. After 10 min, the solution of acid haematin was diluted by adding distilled water with continuous stirring. The addition of water was carried out until the colour of the solution matched perfectly with the standard.

Reading became in grams.

Red blood cells

The red cell pipette was filled exactly up to 0.5 marks with blood and filled by fluid to 101 mark and mix solution. After mixing, a few drops were placed into the

chamber and allowed to stand for a few minutes. The RBC (million/cmm) counts in the central double-ruled squares of Neubauer's chamber with the help of the microscope.

White Blood Cells

The WBC pipette filled up to 0.5 marks with blood and diluted by fluid to mark 11. After thoroughly mixing, a few drops of the contents were placed into the Neubauer chamber. The chamber was left undisturbed for a few minutes. The WBC (million/cmm) count was performed in the single ruled four corner squares of the Neubauer chamber.

The mean cell haemoglobin concentration (MCHC), mean cell haemoglobin (MCH), and mean cell volume (MCV) were calculated using the following formulas (Osman *et al.,* 2010).

MCHC (%) = Hb/Ht×100 Eq.

MCH (pg)= Hb/RBC×10 Eq.

MCV (μ^3) = Ht/RBC×10 Eq.

Biochemical parameters

Measurement of LPO

The MDA (malondialdehyde) assay was determined by thiobarbituric acid assay and using the method of Placer (Placer *et al.,* 1966). The absorbance from the spectrophotometer at 548 nm was recorded, and the results were expressed as nmol MDA/mg protein.

Measurement of hydrogen peroxide (H_2O_2)

Hydrogen peroxide was estimated by following the method of Sinha (Sinha, 1972), and the absorbance was measured at 570 nm. The H_2O_2 concentration was expressed in µM/mg protein.

Measurement of Nitric Oxide (NO)

Nitric oxide was estimated by following the method of Griess (Griess *et al.,* 1879), and the absorbance was taken at 550 nm. The concentration of nitric oxide was calculated against a standard curve plot of sodium nitrate, and the values were expressed as µM/mg protein.

Catalase activity assay

The activity of catalase was determined as per the method of Bergmeyer et al. *(*1983). The rate of H_2O_2 decomposition for 180 sec at 240 nm was recorded and expressed as µmol/min/mg protein.

Measurement of superoxide dismutase (SOD)

In the activity of SOD, the photochemical inhibition of nitro blue tetrazolium was measured (Beauchamp and Fridovich*,*1971). The absorbance was measured at 560 nm, and the results are expressed in units/mg protein. One unit of the enzyme is a 50% enzyme inhibiting reaction rate.

Measurement of GSH

The levels of glutathione (GSH) in the tissue homogenates were determined spectrophotometrically by using the method of Sedlak and Lindsay (Sedlak and Lindsay 1968). The absorbance was read against the blank at 412 nm, and the results were expressed as µmol/mg protein.

Measurement of glutathione S-transferase (GST)

The activity of glutathione-S-transferase (GST) was assessed spectrophotometrically as per the method of (Seyyedi *et al.,* 2005). The absorbance was measured at 340 nm for 5 min, and the results were expressed as µmol/min/mg protein.

Measurement of glutathione reductase assay (GR)

The activity of glutathione reductase was measured by Carlberg and Mannervic (1975). The reaction was started by the addition of tissue supernatant, and NADPH (nicotinamide adenine dinucleotide phosphate hydrogen) was recorded as a decrease in absorbance at 340 nm for 10 min. Nonspecific oxidation of NADPH was calibrated by the absence of GSSG. The unit of the enzyme activity was defined as µmole NADPH/min, and the activity of the enzyme was expressed as units/mg protein.

The protein concentration in the supernatants was determined by following the method described by Lowry (Lowry *et al.,* 1951).

Data analysis

All statistical data analyses were performed using one-way ANOVA (analysis of variance), and the data are expressed as the mean ± SE (standard error). Dunnett's test was applied for comparisons between the control and exposed Groups. The level of significance at $p < 0.05$ was considered significant.

RESULTS AND DISCUSSION

Haematological study

Haematological parameters manifested alterations after UVB-induced thyroid toxicity and curcumin and ascorbic acid treatment in Wistar rats. All blood parameters are listed in Table 1.

The RBC count decreased significantly in Group II (UVB exposed) (6.25±0.31) compared to Group I (control) (7.535±0.29) and increased significantly in the coadministrative Groups (Group III and IV) (6.79±0.19 and 7.40±0.11) compared to Group II (UVB exposed). In comparison, MCV increased (**$p<0.01$) significantly in Group II (57.42±0.38) compared to Group I (control) (53.27±0.14) and decreased significantly in the coadministrative Groups (Group III and IV) (53.80±0.30 and 53.77±0.37) compared to Group II (UVB Exposed).

These variations were remarkably ameliorated with antioxidants in all the parameters of Groups III and IV.

The Hb% (12.075±0.51) and MCH (17.55±0.07) decreased and MCHC (36.32±0.17) increased significantly in Group II (UVB exposed) compared to Group I (control), and Hb% (**p<0.01) and MCH increased (**p<0.01) and MCHC decreased (**p<0.01) significantly in the coadministrative Groups (Group III and IV) compared to Group II (UVB exposed).

The total leukocyte count (7870±50.33) and neutrophil percentage (12.75±1.19) significantly decreased (**p<0.01), and the lymphocyte percentage (80.25±2.95) significantly increased (**p<0.01) in Group II (UVB exposure). The alteration in total and differential WBC counts alters the immune response. Coadministration of curcumin and ascorbic acid (Groups III and IV) significantly increased the total WBC counts and neutrophil percentage (12.75±1.19) (**p<0.01), and the lymphocyte percentage (80.25±2.95) significantly decreased (**p<0.01).

The platelet count (6.99±0.30) decreased (**p<0.01) significantly in Group II (UVB exposed) compared to Group I (control) (8.22±0.12) and increased significantly in Groups III and IV compared to Group II (UVB exposed).

Biochemical study

Biochemical parameters were altered after UVB exposure and curcumin and ascorbic acid treatment in Wistar rats. All biochemical parameters are represented in Table 2.

The MDA concentration of the thyroid gland significantly increased (**p<0.01) in Group II (UVB exposed) (61.09±1.10) compared to Group I (control) and significantly decreased (**p<0.01) in the curcumin and ascorbic acid coadministrative Groups (54.38±0.76 and 50.99±0.58) (Groups III and IV) compared to Group II (UVB exposed). The H_2O_2 concentration of the thyroid gland significantly increased (**p<0.01) in Group II (UVB exposed) (0.37±0.014) compared to Group I (control) and significantly decreased (**p<0.01) in the curcumin and ascorbic acid coadministrative Groups (0.19±0.011 and 0.11±0.013) (Group III and IV) compared to Group II (UVB exposed). The NO activity significantly increased in Group II (UVB exposed) (1.14±0.072) (**p<0.01) compared to Group I (control) and significantly decreased in the coadministrative Groups (UVB+Cur and UVB+AA) (1.07±0.054 and 1.03±0.072) (Group III and IV) (**p<0.01) compared to Group II (UVB exposed).

The catalase activity significantly increased in Group II (UVB exposed) (16.0±0.63) (**p<0.01) compared to Group I (control) and significantly decreased in the curcumin and ascorbic acid coadministrative Groups (10.30±0.250 and 13.57±0.257) (Group III and IV) (**p<0.01) compared to Group I (control).

The SOD activity increased significantly (**p<0.01) in Group II (UVB Exexposed) (328.7±10.4) compared to Group I (control) and significantly decreased (**p<0.01) in the curcumin and ascorbic acid coadministrative Groups (UVB+Cur and UVB+AA) (284.9±7.0 and 301.5±5.2) (Group III and IV) compared to Group I (control).

The GSH activity significantly decreased in Group II (UVB exposed) (1.31±0.13) (***p<0.001) compared to Group I (control) and significantly increased in the curcumin and ascorbic acid coadministrative Groups (UVB+Cur and UVB+AA) (3.24±0.14 and 3.85±0.10) (Group III and IV) (**p<0.01) compared to Group II (UVB exposed).

The GST activity significantly increased in Group II (UVB exposed) (113.26±1.12) (**p<0.01) compared to Group I (control) and significantly decreased in the coadministrative Groups (UVB+Cur and UVB+AA) (**p<0.01) compared to Group II (UVB exposed).

GR activity significantly increased in Group II (UVB exposed) (27.26±0.50) (**p<0.01) compared to Group I (control) and significantly decreased in the coadministrative Groups (UVB+Cur and UVB+AA) (19.15±0.94 and 26.68±1.63) (Group III and IV) compared to Group II (UVB exposed).

The results of the study indicated that thyroid toxicity induced by 15 days of UVB exposure in Wistar rats changed the haematological parameters, but it did not cause anemia. Many studies have reported anemia and the abnormalities of blood profiles in patients with hyperthyroidism, suggesting both direct and indirect effects of thyroid hormones (Ahmed. In addition, Mohammed 2020; Maggio *et al.,* 2015). The existence of micro-cytic anemia is reported in hyperthyroid patients (Miyamoto H *et al.,* 2018). Some studies found that the decreased Hb% and MCV in hyperthyroid patients returned to normal after treatment. (Zahediasl *et al.,* 2010). In previous studies, it was reported that UVB radiation increased the levels of T3 and T4, decreased TSH levels, and caused hyperthyroidism (Rai *and* Mahobiya*,* 2020). In the present study, UVB-induced thyroid toxicity altered blood and biochemical parameters, while ascorbic acid increased Hb and RBC levels. Abojassim et al (2015) reported that haemoglobin concentration was also increased due to the role of vitamin C, which increases absorption of iron from the digestive system and acts on the reduction of bivalent copper ion (Cu++) into monovalent copper ion (Cu+) and thus acts as a coenzyme to increase haemoglobin and the increased in total RBC count.

In the present study, UVB radiation decreased the WBC count (7870±50.33), and ascorbic acid (8867.5±70.13) (**p<0.01) and curcumin (7935±115.71) (**p<0.01) significantly elevated the WBC count

Table 1. Effect of UVB radiation and the role of curcumin and ascorbic acid administration on blood parameters of female Wistar rats

Groups	RBCs	WBCs	Hb%	MCV	MCH	MCHC	Neutrophil	Lymphocytes	Platelets
Group I (Control)	7.535±0.29	8825±55.27	14.05±0.68	53.27±0.14	18.17±0.05	34.37±0.12	32.5±1.37	66.5±1.91	8.22±0.12
Group II (UVB exposed)	6.25±0.31**	7870±50.33 **	12.075±0.51**	57.42±0.38**	17.55±0.07**	36.32±0.17**	12.75±1.19**	80.25±2.95**	6.99±0.30**
Group III (UVB+Cur)	6.79±0.19**	7935±115.71**	12.75±0.27**	53.8±0.30**	18.225±0.02**	34.02±0.15**	34.32±0.72**	66.00±3.85**	7.13±0.18**
Group IV (UVB+AA)	7.40±0.11	8867.5±70.13	13.0±0.11**	53.775±0.37**	17.875±0.05**	33.75±0.19**	21.75±1.51**	69.25±2.76**	8.83±0.05**

(Data are expressed as the mean±SE. *p<0.05, **p<0.01, ***p<0.001 (RBCs - red blood cells, WBCs- white blood cells, Hb- haemoglobin MCV- mean cell volume, MCH- (mean cell haemoglobin), and MCHC- mean cell haemoglobin concentration, SE- standard error);RBC-Red blood cells; WBC- White blood cells; Hb-Haemoglobin ; MCV-Mean cell volume; MCH-Mean cell haemoglobin; MCHC-Mean cell haemoglobin concentration; Cur-Curcumin; AA-Ascorbic acid

Table 2. Effect of UVB radiation and the role of curcumin and ascorbic acid administration on the thyroid gland of female Wistar rats

Groups	LPO	H_2O_2	NO	SOD	CAT	GSH	GST	GR
Group I (Control)	51.33±2.77	0.15±0.017	0.99±0.075	302.4±7.5	12.36±0.46	4.28±0.21	95.88±1.25	16.71±0.91
Group II (UVB Exposed)	61.09±1.10 ***	0.37±0.014**	1.14±0.072**	328.7±10.4**	16.0±0.63**	1.31±0.13**	113.26±1.12**	27.26±0.50**
Group III (UVB+Cur)	54.38±0.76***	0.19±0.011**	1.07±0.054**	284.9±7.0**	10.30±0.250**	3.24±0.14**	91.71±0.54**	19.15±0.94*
Group IV (UVB+AA)	50.99±0.58***	0.11±0.013**	1.03±0.072**	301.5±5.2**	13.57±0.257**	3.85±0.10**	101.11±3.1**	26.68±1.63**

Data are expressed as the mean±SE. *p<0.05, **p<0.01, ***p<0.001;LPO- lipid peroxidation; H_2O_2- hydrogen peroxide; NO- nitric oxide; SOD- superoxide dismutase; CAT- catalase; GSH- glutathione; GST- glutathione S-transferase; GR- glutathione reductase; Cur-Curcumin; AA-Ascorbic acid; SE- standard error

(**p<0.01). In other studies, gamma irradiation has been reported to reduce the total counts of WBCs, lymphocytes, monocytes, neutrophils, basophils, and eosinophils (Mishima *et al.,* 2004). The results are consistent with previous findings that irradiation decreased leukocytes and reduced lymphocyte, neutrophil, and monocyte counts. It has been reported that gamma irradiation causes reduced Hb% and RBC counts in mice attributed to the impairment of cell division, removal of blood-forming organs, alimentary tract injury (Verma *et al.,* 2011), depletion of factors needed for erythroblast differentiation, reticulocyte release from the bone marrow and the loss of cells from the circulation by hemorrhage or leakage through capillary walls and/ or the direct destruction of mature circulating cells (Abdelhalim *et al.,* 2015).

The effects of hyperthyroidism on the red cell profile have been less studied. Most patients with hyperthyroidism are not anemic. The microscopic appearances of the red cells are unremarkable (Kerr *et al.,* 2008), and platelet changes in patients with hyperthyroidism have been regarded as an autoimmune phenomenon. Indeed, platelet-associated immunoglobulin is increased in many patients with Graves' disease (Provan and Semple, 2019). The increased MDA levels in the irradiated Group. In addition, curcumin and ascorbic acid may have curative effects. An increase in MDA concentration in the thyroid gland is a good indicator of lipid peroxidation (Joshi B, *et al.,* 2018). Research reported that whole-body exposure of rats to high-energy radiation from Co60 causes tissue damage in several organs as assessed by increased lipid peroxidation at 2, 12, and 72 h after irradiation (Makhlouf and Makhlouf*,* 2012). Thus, radiation-induced damage might result in adverse health effects within hours to weeks, and delayed effects may be observable many months after exposure.

Additionally, there is evidence of an increase in the activity of antioxidant enzymes such as SOD and GSH-Px and a decrease in MDA after precautions (Suntres ZE, *et al.,* 2011). In our results, UVB exposure-induced thyroid toxicity increased nitric oxide (NO) activity, and administration of curcumin decreased NO activity compared to UVB-induced hyperthyroidism. Nicola *et al. (*2015) and de mar Montesinos *et al.* 2016) reported that nitric oxide (NO) is a negative regulator of TPO and NIS involved in various physiological and pathological processes. Radiation stimulates inducible nitric oxide synthase (NOS) activity in cells, thereby generating large amounts of nitric oxide (Mikkelsen and Wardman, 2003). In the present results, there was an increase in nitric oxide (1.14±0.072) in Group II (UVB Exposed). Iodine simulates the pathogenesis of hyperthyroidism and hypothyroidism when NO is involved (Craps *et al.,* 2015 and Rong *et al.,* 2018). The increased oxygen consumption and enhanced ROS formation without a proportional growth inside the enzymatic activity except for GST activity showed less nonenzymatic antioxidants (Araujo *et al.,* 2011). Similarly, the present study found increased NO and GR activity in Group II (UVB Exexposed) animals. High activities of reduced glutathione and GR in the thyroid gland suggest that the system of glutathione metabolism regulates the antioxidant grade of the thyroid gland (Nadolnik *and* Valentyukevich, 2007).

Conclusion

It was concluded that the present study is pioneering in its field, which for the first time established the effect of UVB radiation exposure on the thyroid of female Wistar rats, and antioxidants protect against the oxidative stress in the thyroid gland. UVB exposure induced a significant disturbance in haemoglobin, RBC, WBC, MCV, MCH, MCHC, platelets, and an imbalance in thyroid hormones. UVB radiation affects blood parameters and results in abnormalities. Both curcumin and ascorbic acid protected thyroid hormones in addition to maintaining blood parameters. UVB radiation disturbs LPO, H2O2, NO, SOD, catalase, GSH, GST, and GR, and curcumin and ascorbic acid cure these enzymatic and nonenzymatic antioxidants.

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Conflict of interest

The authors declare that they have no conflict of interest.

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