



Hepatoprotective Effect of Tropical Medicine Curcumin and Ascorbic Acid against the Ultraviolet B Irradiated Hyperthyroidism Female Wistar Rat

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Abstract

This study evaluate the protective role of tropical medicine curcumin and ascorbic acid against ultraviolet B (UVB) exposure in female Wistar rats. The Experimental study consisted of 24 rats, divided into four groups, each comprising six animals. Group was named Group first Control, second ultraviolet B (2 hours/day) induced hyperthyroidism group, third ultraviolet B induced hyperthyroidism group, and after 30 min given curcumin (25mg/kg BW) orally, and last fourth ultraviolet B induced hyperthyroidism and after 30 min given ascorbic acid (250mg/kg BW) orally. The duration of each treatment is 15 days straight. The liver marker changes were measured by methods. The histopathological and apoptotic changes in the liver were done by different staining and Caspase 3, and observed the slides under the compound and confocal microscope. UVB radiation damaged the liver cells and showed cellular injury. Curcumin and ascorbic acid protected UVB radiation-induced changes in liver. Little doses of both antioxidants can reduce liver marker responses and histological changes brought on by UVB radiations in Wistar rat's cells. The liver's ability to function was impeded by UVB radiation, which was also seen as liver damage by several stains. Female Wistar rats' livers exhibited protective effects from curcumin and ascorbic acid.

Keywords: Liver; UVB; Histology; Tropical Medicine; Hyperthyroidism; Apoptosis

Abbreviations

UVB: Ultraviolet B; BW: Body Weight

Introduction

Curcumin and ascorbic acid, found in tropical plants, protect Wistar rats' livers from UVB-induced hyperthyroidism. UVB radiation, a harmful energy source, damages cells and DNA by generating free radicals and increasing reactive oxygen species production. Both tropical medicines are crucial in preventing hyperthyroidism in Wistar rats [1]. Hyperthyroidism can affect various organ systems, including the digestive, neurological, cardiovascular, and hepatic systems. The balance between thyroid and liver is regulated by thyroid hormones, which maintain bilirubin metabolism through glucuronyl transferase and ligandin threshold. T3 and T4 are glucuronidase and sulphate in the liver, excreted into bile. Radiation harms cells by increasing superoxide free radicals, damaging

membrane-bound lipids, and increasing lipid peroxidation in liver cells. This can stimulate the apoptotic gene Bcl, causing cell membrane rupture and activating caspases 9 and 3, leading to liver cell apoptosis. Radiation-generated cancers are often seen as late effects [2]. After irradiation, the liver develops the capacity to repair DNA damage and fight free radicals [3-5], demonstrating that the liver responds to radiation and that this appears to control the liver's radiation sensitivity. Furthermore, irradiation promotes DNA repair activities through DNA damage even in the non-proliferating cells, even though hepatocytes in the liver are typically in the interphase of the cell cycle [6]. By triggering inflammatory responses and other after-irradiation processes, the various types of liver cells also contribute to the harmful consequences of radiation [7-9]. Many other potential factors could alter the effects of radiation on the liver. For instance, several distinct environmental conditions are present in space and it has been claimed that prolonged stays

in space may raise the risk of non-alcoholic fatty liver conditions in mouse liver [10]. Stains other than the standard hematoxylin and eosin are required for the examination of liver histopathology to bring out the structure, level of fibrosis, inflammation, and some significant characteristics, such as infectious particles and pigments [11]. The stain shows the disarrangements in the tissues' architecture and a clear image of the infections and diseases related to the tissues like the liver [12]. A set of special stains will be applied to liver tissues diagnosed using H and E, Periodic Schiff reagents, Sudan, and Mercuric bromophenol blue staining to identify discrepancies in diagnosis. Hyperthyroidism affects all physiological systems, while thyrotoxicosis-related liver damage is easily classified as hepatitis.

The goal of the current investigation was to determine whether tropical medicine's curcumin and ascorbic acid defended female rats' livers from the effects of UVB radiation.

Materials and Methods

Chemical requirement

Analytical-grade materials were utilized throughout. We bought ascorbic acid from Sigma-Aldrich Co., USA. The other chemicals utilized came from Central Drug House (P) Ltd in New Delhi, India, while curcumin was purchased from Himedia in India.

UVB Radiations

The Wistar rats were exposed to UVB light (TL 20W/01 UVB Narrowband manufactured in Germany), which emits UVB at a wavelength of 280 nm. For 15 days, there was a two-hour daily irradiation [13].

Ethical statement

The Department of Pharmaceutical Sciences Dr. Harisingh Gour Vishwavidyalaya (A Central University) Sagar (M.P.), India granted permission for the study (Ethical registration No. 379/Go/ReBi/S/01/CPCSEA), and all applicable international and national regulations for the usage and concern of animal research were adhered to.

Experimental design

24 female Wistar rats weighing 130–150g each were bought for this study from the Veterinary Sciences and Animal Husbandry College in Mhow, India. All the animals were kept in plastic cages

and always given a regular laboratory diet of daily food and water available. Rats were housed in a laboratory setting with a standard light and dark cycle, a standard temperature (25 ± 2), and relative humidity levels between 40% and 60%. Experimental rats were randomly assigned to the four groups. The control group was given free access to regular food and water. During fifteen days, the UVB group was exposed to UVB radiation at a dose of 280 nm for two hours each day. The UVB-Curcumin group was administered an oral dose of curcumin (25 mg/kg body weight) for 15 days along with a daily dose of 280 nm UVB radiation. The UVB-Ascorbic acid group was exposed to 280 nm UVB radiation for two hours every day for fifteen days, along with an ingested dose of ascorbic acid (250 mg/kg body weight) [13,14].

Measurement of Body and Liver Weight

Before and after the experiment, BW was measured. Following the experiment, liver weight was measured. With the aid of an electronic balance, both liver weight measures were taken (Sartorius, BP210 S).

Serum Collection

Following general anesthesia, a blood sample was obtained through direct heart puncture, collected in clot-activating tubes, and centrifuged at 2000 rpm for 15 min to separate serum. To estimate the serum's SGOT, SGPT, and ALP analyses, the serum was employed.

Tissue processing

With the use of Zamboni fixative, female Wistar rats were perfused [15,16]. After being removed, the liver was fixed for 48 hours. Fixed tissues were cleansed with xylene before being dehydrated in progressively higher alcohol concentrations and being wrapped in paraffin. After that, 5 μ m pieces were cut out and stained with Sudan black B, PAS, MBB, haematoxylin, and eosin. Use a microscope to examine liver cells at 100x and 400x magnifications.

Estimation of MDA Levels

The MDA test was used to identify lipid peroxidation [17]. MDA is the by-product of lipid peroxidation. 100 microliters of tissue homogenate were treated in 1 ml of 0.5 M Tris-maleate buffer for 30 minutes at 30°C in a water bath (pH 5.9). Tight condensers were used to incubate the mixture in a boiling water bath for 10 minutes after adding 1.5 ml of thiobarbituric acid. The mixture was then

given 3 mL of pyridine: n-butanol (3:1 v/v) to cool. The addition of 1 ml of 1 N NaOH was followed by a 10-minute waiting period. At 548 nm, the absorbance was measured. In nmol MDA/mg protein, the degree of lipid peroxidation was expressed.

Estimation of SOD

SOD activity was assessed using the photochemical inhibition of nitro blue tetrazolium (NBT) chloride. A reaction cocktail consisting of 2.25 mM methionine, 100 mM phosphate buffer (pH 7.8), 2.25 mM NBT, 200 mM L-methionine, 1 M sodium carbonate, and 3 mM EDTA was employed. A reaction cocktail and 50 µl of the tissue sample were added to the test tube. The reaction starts when 60 M riboflavin is added. The absorbance at 560 nm was measured while the reaction cocktail was illuminated in both light and darkness. The units per milligram of protein were used to calculate the enzyme activity. The amount of 50% enzyme that slows down the rate of reaction is known as an enzyme unit [18].

Estimation of GSH

On fresh homogenates, GSH was ascertained right away using the spectrophotometric technique described by Sedlak and Lindsay [19]. Briefly, the supernatant was combined with 1 M Tris-cl, 2 mM 5, 5'-dithiobis-2-nitrobenzoic acid, and 50 mM sodium acetate, and incubated at room temperature for 15 to 30 minutes. The absorbance of the resulting yellow colour was assessed at 412 nm in comparison to a control. At ambient temperature, biochemical assays were made using a spectrophotometer.

Haematoxylin and Eosin staining

One common staining technique used in histology is the haematoxylin and eosin stain. It is the most widely used stain in medical diagnosis. Haematoxylin stains blue to nuclei, and eosin stains pink to the cytoplasm. The tissue fix slide was taken and processed in alcohol series, and haematoxylin and eosin stain after staining rinsed the slide in xylene and then mounted with DPX [16].

Periodic acid Schiff (PAS)

A staining technique called periodic acid-Schiff is used to identify polysaccharides like glycogen and mucus components including glycoproteins, glycolipids, and mucins in tissues. The tissue fixes

slide was taken and processed in alcohol series and then stained with PAS stain then after staining rinse the slide was in xylene and then mounted with DPX.

Mercuric bromophenol blue (MBB)

Mercuric ions of the bromophenol blue solution react with acidic, sulfhydryl, and aromatic residues of the protein to give blue color. The tissue fix slide was taken and processed in alcohol series and then stained with Mercuric bromophenol blue stain; then after staining, rinse the slide in xylene and then mount with DPX.

Sudan black B staining

Sudan stain employs frozen tissue sections that have been paraffinized or fixed with formalin solution. The Sudan dye group's Sudan Black B dye is the most popular dye. The phospholipids, lipoproteins, and triglycerides present in the staining specimen are stained by Sudan Black B, a slightly basic dye that reacts with the acidic groups in lipid molecules. The tissue fix slide was taken and processed in alcohol series and then stained with Sudan black B stain. Then after staining, rinse the slide in xylene and then mounted it with DPX.

Immuno-fluorescence analysis

Caspase-3 immunofluorescence at a dilution of 1:50 on liver sections that were 5 m thick and mounted on glass slides. Sections were stained with FITC (1:100) and DAPI and then seen under a confocal microscope (Nikon ECLIPSE Ti-E) [16].

Analysis of image

Using Image J software, the integrated optical density (IOD) of Caspase-3 immunoreactivity was assessed in liver sections. For each antigen, average IOD values were calculated as arbitrary unit (a. u.) thresholds.

Analysis of statistics

The data are shown as Mean S.E. to ascertain the statistical distinction between several experimental groups, a one-way analysis of variance test (ANOVA) was conducted, followed by the Dunnett test. Significant differences from the control group are indicated by the following symbols: *p < 0.05, **p < 0.01, ***p < 0.01, # significant differences from the exposed group.

Results and Discussion

Body weight (BW) and Liver Weight

The BW of the UVB-treated female Wistar rats is considerably lower as compared to the control group. In comparison to UVB, BW is dramatically increased by the Cur and AA treatments. There are also notable differences when compared to the control group. In female Wistar rats, UVB treatment significantly reduced liver weight, but antioxidant delivery slightly increased liver weight in both groups (Table 1).

GROUPS	Body weight (gm)	Liver weight (gm)
CONTROL	117.8 ± 1.24	2.9 ± 0.30
UVB	87.6 ± 1.21**	3.4 ± 0.66
UVB+Cur	95.1 ± 2.82**	3.7 ± 0.46
UVB+AA	98.6 ± 2.67**	2.3 ± 0.13

Table 1: Effect of ultraviolet B radiation and both antioxidants administration on (a) body weight, (b) liver weight. Values are presented as mean ± SE. Significant difference from control group (*p < 0.05, **p < 0.01).

Biochemical parameters

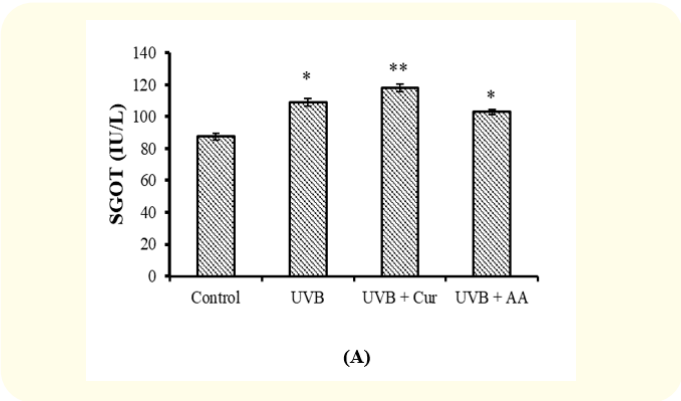
The MDA levels in the liver of the female Wistar rat changed in response to the stress of UVB treatment. The UVB-treated group's liver MDA concentration was noticeably higher than that of the control group. The MDA concentration was significantly lower in the UVB + Cur group compared to the control group. According to the Wistar rat liver's enzymatic antioxidant state, UVB treatment significantly reduces SOD-specific activity in the liver (P < 0.05) when compared to control groups. When compared to the UVB-treated group, SOD activity rises considerably in the UVB + Cur group and falls by P < 0.05 in the UVB + AA group. When compared to control groups, GSH activity in the liver diminishes in UVB-treated groups. When compared to the control and UVB-treated groups, GSH activity considerably rises in the UVB + Cur and UVB + AA groups (Table 2).

Groups	Control	UVB	UVB + Cur	UVB + AA
Variables				
LPO	7.179 ± 0.78	7.881 ± 0.43	7.184 ± 0.53	6.184 ± 0.20
SOD	301.7 ± 9.79	250.5 ± 1.54*	268.2 ± 22.15	211.5 ± 22.13*
GSH	2.95 ± 0.98	2.76 ± 0.25	3.25 ± 0.48	3.38 ± 0.78

Table 2: Effect of UVB radiations, Curcumin and Ascorbic acid on different biochemical parameters. Values are presented as mean ± SE. Significant difference from control group (*p < 0.05, **p < 0.01).

Liver markers Serum Glutamate Oxaloacetate Transaminase (SGOT), Serum Glutamate Pyruvate Transaminase (SGPT), and Alkaline phosphatase (ALP)

SGOT and SGPT are the two most important indicators of liver impairment. The level of these enzymes typically rises in response to moderate hepatotoxic damage. Hepatocyte destruction and reduced liver function are both confirmed by the significantly higher levels of GOT (*p < 0.05) and GPT (**p < 0.01) in the serum of UVB-induced hyperthyroidism rats. Curcumin and ascorbic acid treatment provided significant hepatoprotection by restoring SGPT and SGOT levels to control levels in UVB-induced hyperthyroidism rats. Serum ALP activity is used primarily as an indicator of hepatic disease. Serum ALP activity was higher (**p < 0.01) in UVB-induced hyperthyroidism rats compared to controls. When curcumin and ascorbic acid were administered, the serum ALP was lower in the UVB + Cur and UVB+AA groups than in the UVB-induced hyperthyroidism group (Figure 1).



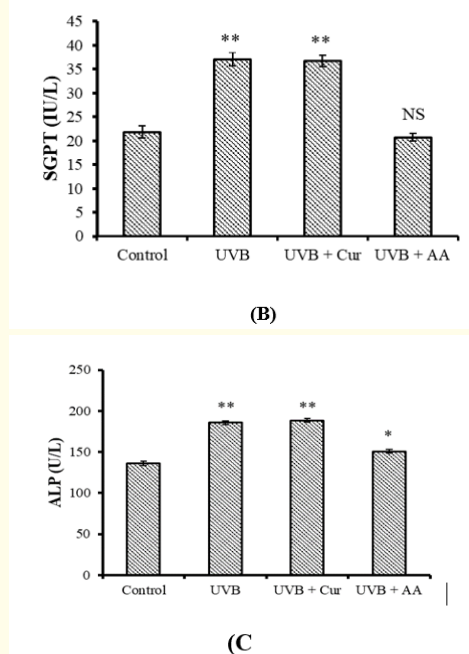


Figure 1: Effect of UVB-induced hyperthyroidism and the role of Curcumin and Ascorbic acid administration on (A) serum SGOT, (B) SGPT, and (C) ALP. Values are presented as mean $\pm$ SE. Significant difference from the control group (* $p < 0.05$, ** $p < 0.01$).

Histopathological observations

A liver histopathological observation of rats stained with H & E, PAS, Mercuric bromophenol blue (MBB), and Sudan black B in the liver (Figure 2 to 5). Hepatocyte degeneration was visible in microscopic parts of the liver of UVB-exposed rats, advancing from stages of temporary to permanent cellular destruction. Cellular enlargement, together with granular and vascular alterations of the hepatocytes, were more apparent in the periportal region than in the centrilobular zone, and these changes were reversible. Cells that have sustained irreversible damage and exhibit coagulative necrosis, as seen by karyorrhexis, pyknosis, karyolysis, and nucleosis, as well as cytoplasmolysis, individualization, and hepatocyte disintegration. However, there was also a lack of globular structure in some focus areas because of more pronounced coagulative necrosis. The groups receiving ascorbic acid and curcumin demonstrated a beneficial effect on UVB-induced liver hyperthyroid damage. (Figure 2). The control rat's liver was examined and

demonstrated a robust PAS-positive reaction, which denoted the presence of significant volumes of polysaccharides in the form of glycogen accumulation. Bright pink granular material was found in the cytoplasm, which was used to demonstrate the reaction product. Due to the fixative's effects on the tissue, most of the PAS-positive results were moved laterally, toward one side of the cell. The responses in the nucleus were PAS-negative. When compared to the hepatocytes from the control group, the liver of UVB-induced hyperthyroidism-induced rats showed a severe depletion of carbohydrate contents. Only a small number of cells showed mild to moderate reactivity. In contrast to the UVB-induced hyperthyroidism group, most liver cells examined in the liver section of the UVB-induced hyperthyroidism rats with curcumin and ascorbic acid revealed a practically normal distribution of polysaccharides (Figure 3). The mercuric bromophenol blue technique revealed the presence of several soluble proteins in the liver cells of the control rat as blue granules against a light-blue ground cytoplasm. The cytoplasm was covered in a smattering of protein granules. Intensely stained plasma membranes limited the cells. When compared to the hepatocytes from the control group, the hepatocytes of the UVB-induced hyperthyroidism rats revealed a marked reduction in the amount of protein. Only a few cells showed a minimal response. The co-administration of curcumin and ascorbic acid roughly restored the protein levels and overall appearance of the liver cells. The cytoplasm of several cells was filled with a lot of protein granules that were well labeled. The nuclear and plasma membrane staining affinities, as well as the chromatin components, are higher than in the UVB-induced hyperthyroidism group (Figure 4). A moderate amount of lipids was present in the form of Sudan Black B-positive materials, according to the histochemical analysis of the rat liver from the control group. Rat livers from the UVB-induced hyperthyroidism group displayed an increase in lipid content in the form of lipid droplets with a pronounced black color inside the hepatocytes. In contrast to the control group, the vacuoles appeared to contain lipids that were positively labeled. Rat livers administered concurrently with curcumin and ascorbic acid in the UVB+Cur and UVB+AA groups displayed considerable cytoplasm staining but no nuclear staining. Comparatively to the UVB group, Sudan's black-stained sections displayed a moderate buildup of lipid droplets (Figure 5).

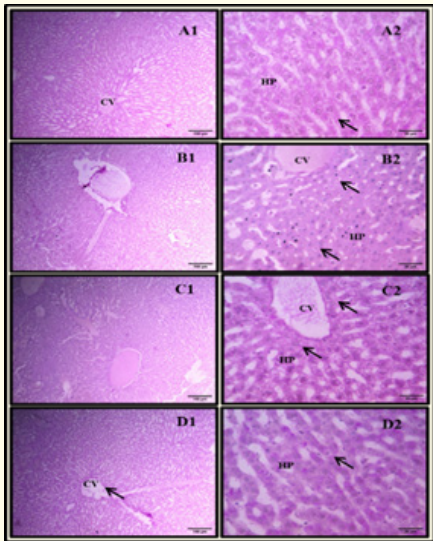


Figure 2: Representative photomicrographs of liver tissues for the control (A), UVB-induced hyperthyroidism group (B), UVB+Cur (C), and UVB+AA (D) groups (Magnification 100X and 400X, 1 and 2 respectively). CV indicate central vein and HP indicate Hepatocytes.

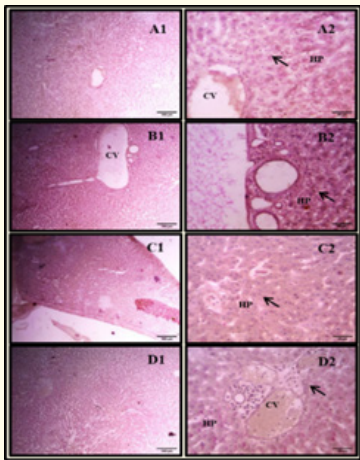


Figure 3: Representative photomicrographs of liver tissues of PAS staining. The control (A), UVB-induced hyperthyroidism group (B), UVB+Cur (C), and UVB+AA (D) groups and arrows indicate the carbohydrate concentration (Magnification 100X and 400X, 1 and 2 respectively). CV indicate central vein and HP indicate Hepatocytes.

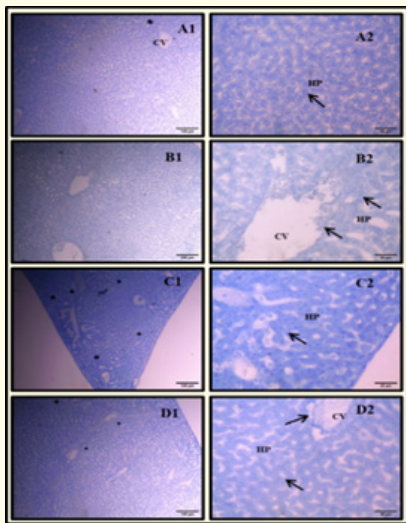


Figure 4: Representative photomicrographs of liver tissues of Mercuric bromophenol blue staining. The control (A), UVB-induced hyperthyroidism group (B), UVB+Cur (C), and UVB+AA (D) groups and arrows indicate the protein concentration (Magnification 100X and 400X, 1 and 2 respectively). CV indicate central vein and HP indicate Hepatocytes.

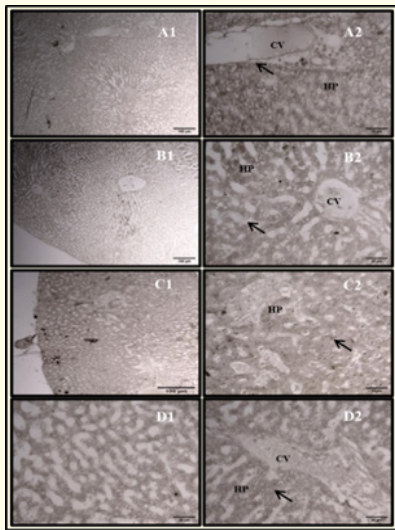


Figure 5: Representative photomicrographs of liver tissues of Sudan black B staining. The control (A), UVB-induced hyperthyroidism group (B), UVB+Cur (C), and UVB+AA (D) groups and arrows indicate the lipid concentration (Magnification 100X and 400X, 1 and 2 respectively). CV indicate central vein and HP indicate Hepatocytes.

Immunoreactivity of caspase 3 in the liver

The immune expression of caspase-3 differed in all groups. In the UVB-induced hyperthyroidism group (B1&B2), UVB radiation enhanced the expression of caspase-3 in rat liver, represent-

ing maximum apoptosis with a higher integrated optical density of caspase-3. Curcumin and ascorbic acid reduce caspase-3 levels to combat the effects of apoptosis in the UVB + Cur group and UVB+AA (C1 and C2). (Figure 6).

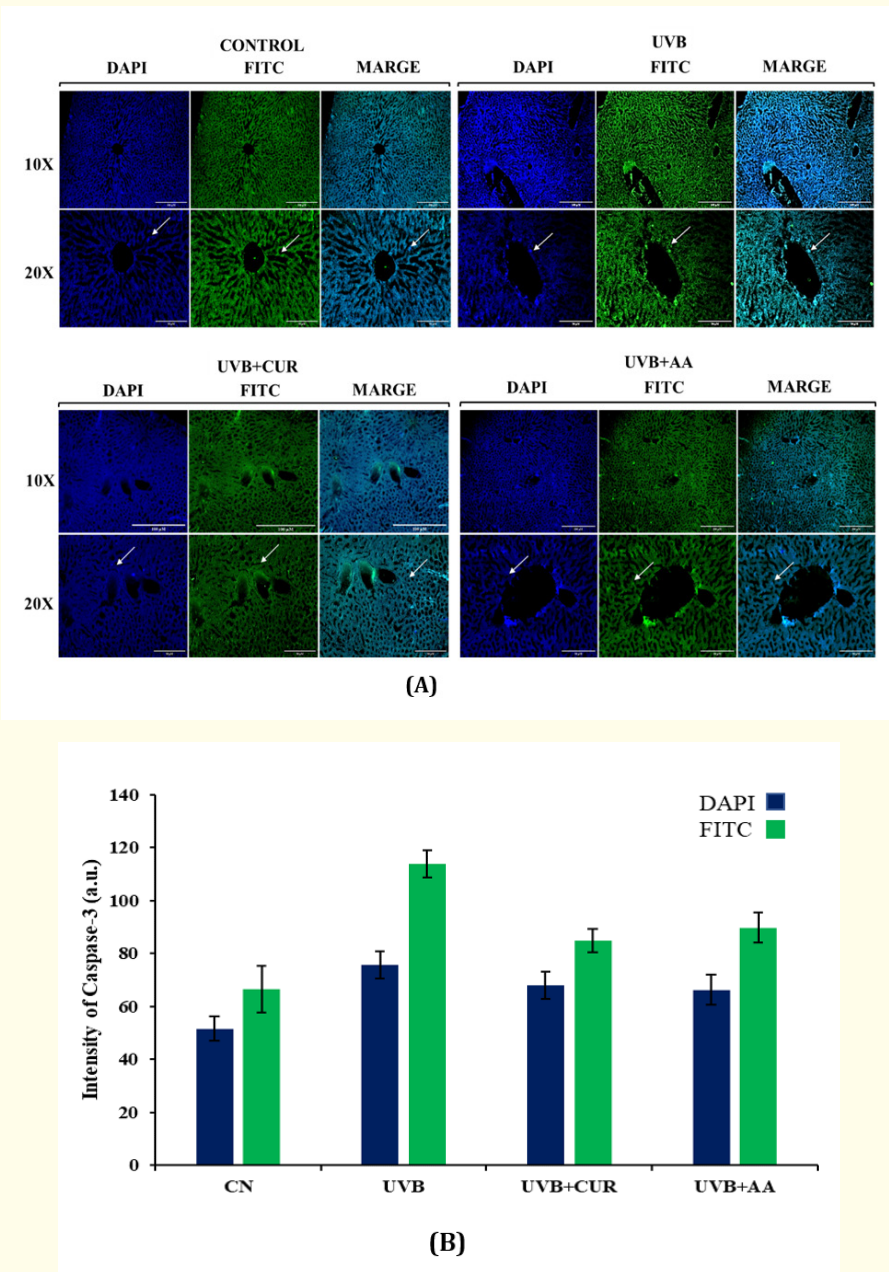


Figure 6: Immuno-fluorescence expression of caspase 3 in UVB-induced liver toxicity. IHC was performed, and images were scanned. (A) Images showed expression of Caspase-3 in the liver and images were scanned at low and high magnification. (10X and 20X). (B) intensity profile showed the expression of Caspase 3-FITC and DAPI.

Discussion

Studies on the effects of radiation have been conducted on a variety of animal species. However, there are data on the immediate effects of UV light and little information on UVB toxicity *in vivo*. There is very little data on how UVB radiation affects “non-skin” tissues, except for studies that looked at photoprotection. Due to these factors, we assessed and studied the impact of a single UVB exposure on the histology and function-related parameters in the liver of female Wistar rats just 15 days after exposure, as well as the anti-toxin properties of curcumin and ascorbic acid on UVB-induced hyperthyroidism. The mode of action of UVB radiation can be anticipated to have two possible outcomes: either it will produce ROS, which causes oxidative stress, or it will collect in cell membranes and disrupt membrane structure because of its capacity to produce free radicals, which cause tissue damage [20].

Previous investigations that ultraviolet B radiation lost body and thyroid weight and increased the levels of thyroid hormones and decreased the level of TSH. This investigation showed UVB radiation-induced hyperthyroidism [1,21]. The Wistar rat liver was exposed to additional UVB irradiation, which resulted in tissue damage as seen by elevated MDA levels and decreased SOD and GSH levels [22]. A single UVB treatment increased TBARS concentration and GSH level (200mJ/cm², 24 hours), as well as a decrease in SOD activity. It found, in line with the data, that guinea pig erythrocytes treated with a single dose of 900mJ/cm² UVB had significantly higher GSH levels and SOD activity after 24 hours [23]. However, the TBARS level initially decreased a little (over 24 hours) before rising (48h). In irradiated hairless rats, Mulero et al. discovered no alterations in the amount of erythrocyte GSH [24]. UVB-induced hyperthyroidism-induced toxicity in the liver. In the present study, SGOT and SGPT are the two most important indicators of liver impairment. The level of these enzymes typically rises in response to moderate hepatotoxic damage. Hepatocyte destruction and reduced liver function are both confirmed by the significantly higher levels of SGOT (**p* < 0.05) and SGPT (***p* < 0.01) of UVB-induced hyperthyroidism rats. Curcumin and ascorbic acid treatment provided significant hepatoprotection by restoring SGPT and SGOT levels to control levels in UVB-induced hyperthyroidism rats. Serum ALP activity is used primarily as an indicator of hepatic disease. Increased (***p* < 0.01) level of serum ALP activity in UVB-induced hyperthyroidism rats as compared to the control group. Similarly, radiation raised the levels of GOT, GPT, and ALP

in serum [25]. The levels of the enzymatic antioxidants SOD, CAT, GSH, and LPO were increased, according to a previous study. Oxidative stress is regarded as a primary pathogenic mechanism since it contributes to the beginning and development of liver injury [26]. Oxidative stress occurs when ROS generation exceeds the capacity of antioxidative defense systems to safeguard the target cell [27]. It has been associated with the onset and progression of a variety of illness states, including inflammation, photoaging, and skin cancer. A wide variety of environmental influences quickly affect the liver because it is a metabolically active organ. Ionizing radiation is one such environmental factor, and living beings exposed to it at relatively large doses can suffer serious harm or perhaps quickly expire because of its acute effects [28].

In the present study, UVB-induced hyperthyroidism injured the hepatic cells. However, there was a lack of globular architecture in some focus areas because of more pronounced coagulative necrosis. When compared to the hepatocytes from the control group, the liver of UVB-induced hyperthyroidism-induced rats showed a severe depletion of carbohydrate contents. When compared to the hepatocytes from the control group, the hepatocytes of the UVB-induced hyperthyroidism rats revealed a marked reduction in the amount of protein. Rat livers from the UVB-induced hyperthyroidism group displayed an increase in lipid content in the form of lipid droplets with a pronounced black color inside the hepatocytes. In contrast to the control group, the vacuoles appeared to contain lipids that were positively labeled. In another investigation, the liver's lobular architecture was found to be intact, except for a few cases in which mildly reversible degenerative alterations in the hepatocytes and leukocyte infiltration in the portal region were noted. The livers of irradiated rats, on the other hand, displayed hydropic degeneration, necrosis in sizable clusters of hepatocytes within the lobule, dilated central veins, and micro-vesicular steatosis [29]. The liver is subject to a wide range of effects from radiation. The medical world is concerned about fibrosis since it is one of the acute side consequences of high-dose irradiation, such as with cancer radiotherapies. Following radiation exposure, cancer is a significant risk as well [30]. With both radiations, the reactivation of Hepatitis B is a well-known event [31]. Where Curcumin exhibits a shielding effect against radiation exposure [32]. The reduction in DNA-repairing protein expression that happens when cells are exposed to UVA and UVB radiation could possibly be explained by ascorbic acid. It is generally known that UV radiation exposure harms nucleic ac-

ids either directly or indirectly by oxidizing DNA under the control of ROS [33]. In the current investigation, the liver sections of Wistar rats were examined for histological changes. These included clogged blood vessels, inflammatory cell infiltration, a clogged portal vein, and more degraded hepatocytes that had undergone fatty change and fibrosis [34]. This study demonstrates the changed liver tissue structure together with increased marker enzymes and lipid peroxidation. Contrarily, curcumin has antioxidant capabilities that have substantial ROS scavenging effects and protect the hepatic tissue from UVB radiation. They also have a protective impact against oxidative damage (Piantanida *et al.*, 2020).

Conclusion

The liver damage caused by UVB radiation in female Wistar rats is interpreted in this study. Additionally, pay attention to the antioxidant curcumin and ascorbic acid's medicinal effects. The histopathology and functionality of liver tissue are changed by UVB radiation. A Variety of stains demonstrates the significant variations in the histology between the several treated groups, and the liver function test demonstrates the rise of the marker enzymes and the degree of liver injury. The current study is the first, as far as the authors are aware, to suggest that curcumin plus ascorbic acid may help treat liver structure and function to support the healing of UVB-induced damage. Ascorbic acid and curcumin may be created and used in the prevention of liver damage as a result of the current study's findings.

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Conflict of Interest

There are no competing interests.

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