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Histological Effect of Ultra Violet (UV) Light on the Brain of Albino Wistar Rats

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A R T I C L E I N F O

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A B S T R A C T

UV light, or ultraviolet light, is a type of radiation that falls between the visible light and X-ray regions of the electromagnetic spectrum. It is a form of electromagnetic radiation emitted by the sun and artificial sources, such as tanning beds and UV lamps. It has a shorter wavelength than visible light, ranging from 100 to 400 nanometers (nm). It is categorized into three types based on their wavelengths: UVA, UVB, and UVC. The aim of this study is to evaluate the histopathological effect of uv light on the brain of adult Wistar albino rats. In all, fifty (50) adult Albino Wistar rats were used for this study. Group A served as the control and the rats were given distilled water. Animals in the test groups (B, C, D and E) were exposed to 30mins, 1hr, 2hrs and 4hrs of UV lights. After the administration, the rats were put under light chloroform anaesthesia and the brain harvested for histological processing. Short-term exposure (30 min daily) did not cause significant changes, whereas prolonged exposure (1 to 4 hours daily) led to pronounced gliosis, neuronal congestion, and axonal alterations. These findings suggest that chronic UV radiation exposure can have detrimental effects on neural integrity, possibly through oxidative stress, DNA damage, and vascular compromise. The long-term implications of these findings may extend beyond structural damage to include cognitive and behavioral deficits, underscoring the need for further investigations

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INTRODUCTION

Ultraviolet (UV) radiation is a form of electromagnetic radiation emitted by both natural and artificial sources. The sun is the primary natural source of UV radiation, while artificial sources include UV lamps, welding arcs, and tanning beds (WHO, 2020). UV radiation is classified into three main types based on wavelength: UVA (320-400 nm), UVB (280-320 nm), and UVC (100-280 nm). While UV radiation plays a critical role in vitamin D synthesis, excessive exposure has been linked to various adverse health effects, including skin cancer, cataracts, and immunosuppression (Yousaf et al., 2021). Despite extensive research on its impact on the skin and eyes, the potential effects of UV radiation on the central nervous system, particularly the brain, remain inadequately explored. The brain, as the control center of the body, is highly susceptible to oxidative stress and inflammation, both of which are potential consequences of excessive UV radiation exposure. Studies have suggested that UV radiation can generate reactive oxygen species (ROS), leading to neuronal damage, apoptosis, and cognitive impairment (Zhang et al., 2019). Oxidative stress plays a crucial role in neurodegenerative diseases such as Alzheimer's and Parkinson's disease, highlighting the need to investigate whether UV radiation exposure can contribute to similar neuropathological changes (Chen et al., 2022). Additionally, disruptions in the blood-brain barrier (BBB) caused by excessive UV exposure may allow harmful substances to infiltrate the brain, exacerbating neuroinflammatory responses (Kwon & Kim, 2021). Experimental models, such as albino Wistar rats, are widely used in neuroscience research due to their genetic similarity to humans and their well-documented physiological responses to environmental stressors. Wistar rats provide a suitable model for studying the effects of UV radiation on brain histopathology, particularly when exposed at varying frequencies (Naderi et al., 2020). Previous research on rodent models has demonstrated that environmental stressors, including radiation, can alter neuronal morphology, induce gliosis, and affect cognitive functions (Adebayo et al., 2021). However, few studies have systematically examined the specific histopathological alterations in the brain following UV exposure at different frequencies, thereby necessitating further investigation.

LITERATURE RIVIEW

The histopathological effects of UV radiation on brain structures, including the hippocampus, cerebral cortex, and cerebellum, are of particular interest due to their roles in memory, cognition, and motor control. Preliminary studies have indicated that prolonged UV exposure may lead to neuroinflammation, astrocyte activation, and neuronal degeneration (Lee et al., 2023). Additionally, UV radiation may disrupt neurotransmitter balance, leading to behavioral and cognitive impairments (Choi et al., 2022). Understanding these potential changes is critical for assessing the long-term neurological risks associated with UV exposure and for formulating preventive strategies. This study aims to investigate the histopathological effects of UV radiation on the brain of adult male albino Wistar rats exposed at varying frequencies. By analyzing neuronal integrity, inflammatory responses, and potential degenerative changes, this research seeks to bridge the existing knowledge gap

and provide insights into the neurological consequences of UV radiation exposure.

METHODOLOGY

Study Area

This study was carried out in the experimental site at the histology laboratory college of medicine, Ambrose Alli University Ekpoma, Edo State. Edo state lies between longitude 06o 04IE and 06o 43IE and latitude 05o 44IN and 07o 34IN with a land mass of17, 450 sq.km located in the south south geopolitical zone of Nigeria with a population of 3.1 million people (World Gazzetter, 2007).

Study Duration

The preliminary studies, animal acclimatization, ingredients procurement, actual animal experiment and evaluation of results, lasted for a period of four weeks. However, the actual administration of substances to the test animals lasted for two (2) weeks.

Ethical Approval

Ethical Approval to utilize the animal intended for this work was sought from the Ethical Board of Ambrose Alli University, Ekpoma, Edo State and the ethical permission was given.

Animal Model and Handling

Fifty (50) Adult Albino Wistar rats having a mean weight of 265g were procured from the animal farm, college of medicine Ambrose Alli University Ekpoma and transferred to the experimental Laboratory at the histology laboratory college of medicine, Ambrose Alli University Ekpoma, Edo State, where they were allowed two (2) weeks of acclimatization. They were kept in wire mesh cages with tripod that separates the animal from its faeces to prevent contamination. During this period of acclimatization, the rats were fed with growers' mash and water provided ad libitum. The animals were maintained and utilized in accordance with the standard guide for the care and use of Laboratory animals.

Grouping of Animal Model

The experimental animals were separated into five groups (A – E). Each group contains ten rats each (n = 10) using 10 big cages to house them. Group A served as the control and groups B - E served as the test groups.

Groups B – E were exposed to UV light at different durations while Group A received only the normal feed (grower's mash) and water with no exposure.

Design of the Study

In all, fifty (50) adult Albino Wistar rats were used for this study. They were divided into five groups of ten rats each. Group A served as the control and the rats were given distilled water. Animals in the test groups (B, C, D and E) were exposed to 30mins, 1hr, 2hrs and 4hrs of UV lights. After the administration, the rats were put under light chloroform anaesthesia and the brain harvested for histological processing.

Substance of Study

Ultraviolet light machine was purchased from an electrical store.

Substance Administration

The rats were weighed before exposure to UV light and before they were sacrificed and similar weight measurements were done weekly to the end of the experiment and the average weight recorded accordingly.

a. Group A (Control) received only normal feed (growers' mash) and distilled water daily for 28 days.
b. Group B was exposed to UV light for 30 minutes daily and given feed and water
c. Group C was exposed to UV light for 1hr daily and given feed and water
d. Group D was exposed to UV light for 2hrs daily and given feed and water
e. Group E was exposed to UV light for 4hrs daily and given feed and water

Sample Collection and Analysis

The weights of the animals were measured before and after the experiment and recorded accordingly. The brain of each rat was obtained at the end of the experiment under chloroform anaesthesia and fixed in 10% formol saline for histological processing.

Histological Processing

The tissues were processed using automatic tissue processor according to the processing schedule used in Irrua Specialist Teaching Hospital, Edo State, Nigeria. The fixed plastic cassette tissues in 10% formalin were automatically processed by passing them through different grades of alcohol as follows:

a. 70% alcohol = 2hrs
b. 70% alcohol = 2hrs
c. 90% alcohol = 2hrs
d. 90% alcohol = 2hrs
e. 90% alcohol = 2hrs
f. Absolute = 2hrs
g. Xylene 1 = 2hrs
h. Xylene II = 2hrs
i. Molten paraffin wax 1 = 2hrs
j. Molten paraffin Wax II = 2hrs

After the last timing, the tissues were removed from their plastic cassettes and placed at the centre of the metallic tissue mould and then filled with molten paraffin wax. They were also left to solidify after which they were now placed in the refrigerator at 5oC for 15 minutes. After the blocks were cool in the refrigerator for the time stated above (15 minutes), the blocks were then removed from the metallic case using a knife and after which the paraffin wax at the side of the blocks were removed.

The blocks were then trimmed at 10microns and cut serially at 5microns on a rotary microtome. The sections were floated in water bath at 55oC and picked up by the use of a clean frosted end slides. The frosted end slides were now placed on the hot plate for 40 minutes for adequate attachment of the sections on the slides after which the sections were de-waxed, hydrated, air dried and stored in a slide box ready for staining process.

Staining Procedure

Sections for general tissue structure were stained by Haematoxylin and Eosin technique.

1. The sections were dewaxed in 3 changes of xylene 5 minutes
2. The sections were hydrated through descending grades of alcohol (absolute, 95%, 80% and 70%).
3. The sections were stained with Cole's haematoxylin 5 minutes
4. The sections were rinsed in running tap-water to remove excess stain
5. The sections were differentiated in 1% acid alcohol 15 seconds
6. The sections were blued in running tap water 10 minutes
7. The sections were counterstained with 1% eosin 2 minutesa
8. Sections were finally rinsed in water, dehydrated in ascending grades of alcohol (70%, 80, 95% and absolute)
9. The sections were cleared in xylene, air-dried and mounted with dibuthylphthalate propylene xylene (DPX).
10. The slides were examined under a light microscope and photomicrographs were taken.

Data Analysis

The mean weight ± SD of the test animals and control before and after the administration of rohypnol were calculated using SPSS (version 27)

RESULT AND DISCUSSION

Table 4.1 show variations in the mean and standard deviation of body weight across different groups of Albino Wistar rats exposed to UV light for different durations. On Day 1, the control group (Group A) recorded a mean weight of 166.7 ± 12.2 g, which was comparable to Group B (166.0 ± 18.5 g). However, Groups C, D, and E exhibited lower mean weights of 142.5 ± 14.1 g, 140.2 ± 12.3 g, and 158.1 ± 14.7 g, respectively. The F-value of 1.682 and p-value of 0.177 indicate no statistically significant difference on this day.

By Day 3, the control group maintained a mean weight of 163.8 ± 12.8 g, while Group B showed a slight increase to 166.7 ± 25.0 g. Conversely, Groups C and D showed reductions to 138.7 ± 16.9 g and 138.2 ± 14.7 g, respectively, whereas Group E had a mean of 154.7 ± 17.8 g. Despite these differences, the p-value of 0.172 suggests no significant variation among the groups.

On Day 7, the control group recorded a higher mean of 170.5 ± 13.3 g, whereas Group B dropped to 142.5 ± 6.6 g. Groups C and D showed further reductions in mean weights to 133.0 ± 18.2 g and 131.7 ± 12.0 g, respectively, while Group E stood at 150.8 ± 19.1 g. The F-value of 3.341 and p-value of 0.020 indicate a statistically significant difference among the groups at this stage.

By Day 14, weight differences became more pronounced. The control group exhibited a mean weight of 175.6 ± 10.3 g, while Group B significantly dropped to 130.3 ± 8.0 g. Groups C and D further declined to 122.6 ± 19.8 g and 120.4 ± 9.4 g, respectively, with Group E maintaining a slightly higher mean of 138.5 ± 18.2 g. The F-value of 6.709 and p-value of 0.000 indicate a highly significant difference among the groups.

On Day 28, the control group maintained a mean weight of 166.5 ± 18.6 g, whereas Group B decreased further to 128.4 ± 11.4 g. Groups C and D displayed the lowest values of 114.5 ± 15.7 g and 109.9 ± 6.3 g, respectively. Meanwhile, Group E had a mean of 129.5 ± 16.3 g. The F-value of 6.437 and p-value of 0.001 confirm a statistically significant difference at this stage, indicating that prolonged UV exposure led to significant weight reductions across the experimental groups.

Table 4.1: Distribution of Mean±S.D of Days of Albino Wistar Rats Administered with UV LIGHT

Days	Control (Group A)	Uv light for 30 mins and feed (Group B)	Uv light for 1 hr and feed (Group C)	Uv light for 2hrs and feed (Group D)	Uv light for 4 hrs and feed (Group E)	F-value	P-value
Day 1	166.7 ± 12.2[a]	166.0 ± 18.5[b]	142.5 ± 14.1	140.2 ± 12.3[ab]	158.1 ± 14.7	1.682	0.177
Day 3	163.8 ± 12.8	166.7 ± 25.0[ab]	138.7 ± 16.9[a]	138.2 ± 14.7[b]	154.7 ± 17.8	1.706	0.172
Day 7	170.5 ± 13.3[abcd]	142.5 ± 6.6[a]	133.0 ± 18.2[b]	131.7 ± 12.0[c]	150.8 ± 19.1	3.341	0.020*
Day 14	175.6 ± 10.3[abcde]	130.3 ± 8.0[a]	122.6 ± 19.8[b]	120.4 ± 9.4[c]	138.5 ± 18.2[d]	6.709	0.000*
Day 28	166.5 ± 18.6[abcde]	128.4 ± 11.4[a]	114.5 ± 15.7[b]	109.9 ± 6.3[c]	129.5 ± 16.3[d]	6.437	0.001*

Asterisk *: Similar Superscript Shows Statistically Significant Difference
Same Superscript Represents a Statistically Significant Occurs

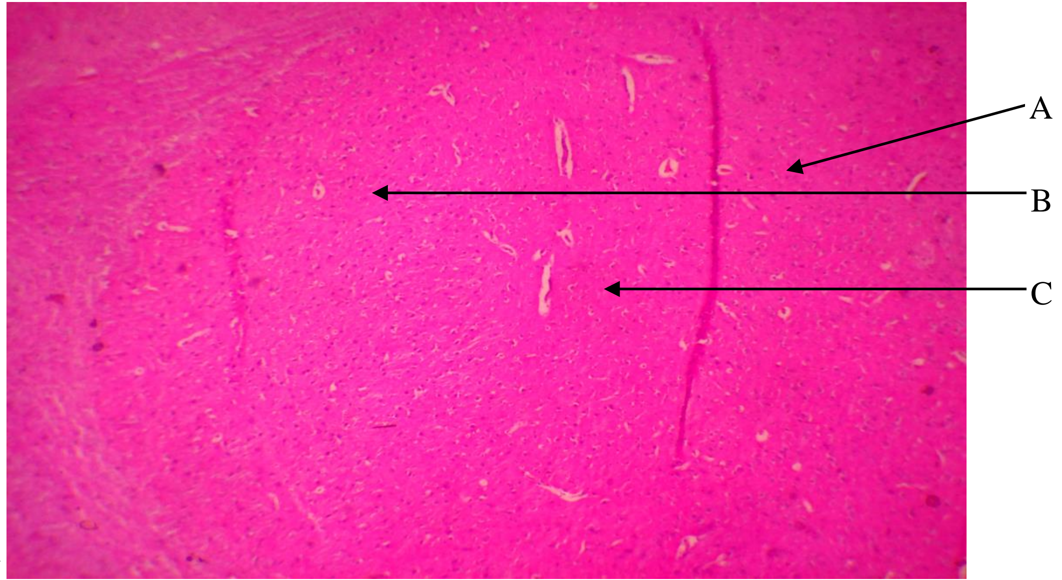

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Fig 1. Plate 1a Shows the Normal Histological Feature of the Brain of the Untreated (Positive Control) Albino Rat with Presence of Microglial Cell (A), Neuron (B) and Capillary (C) of the Brain. Handex100

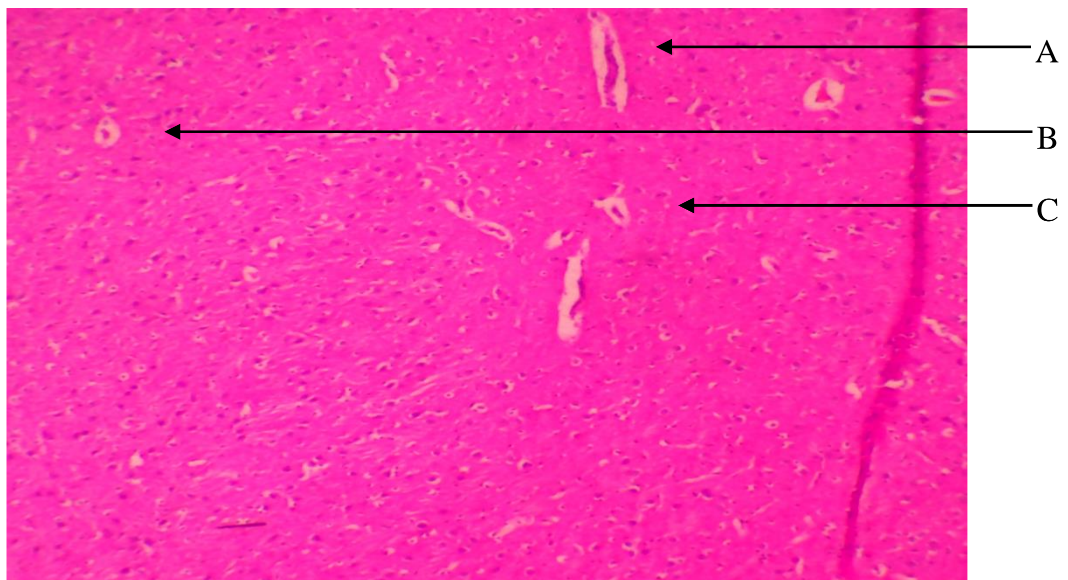

Fig 2. Plate 1b Shows the Normal Histological Feature of the Brain of the Untreated (Positive Control) Albino Rat with Presence of Microglial Cell (C), Neuron (B) and Capillary (A) of the Brain. Handex400

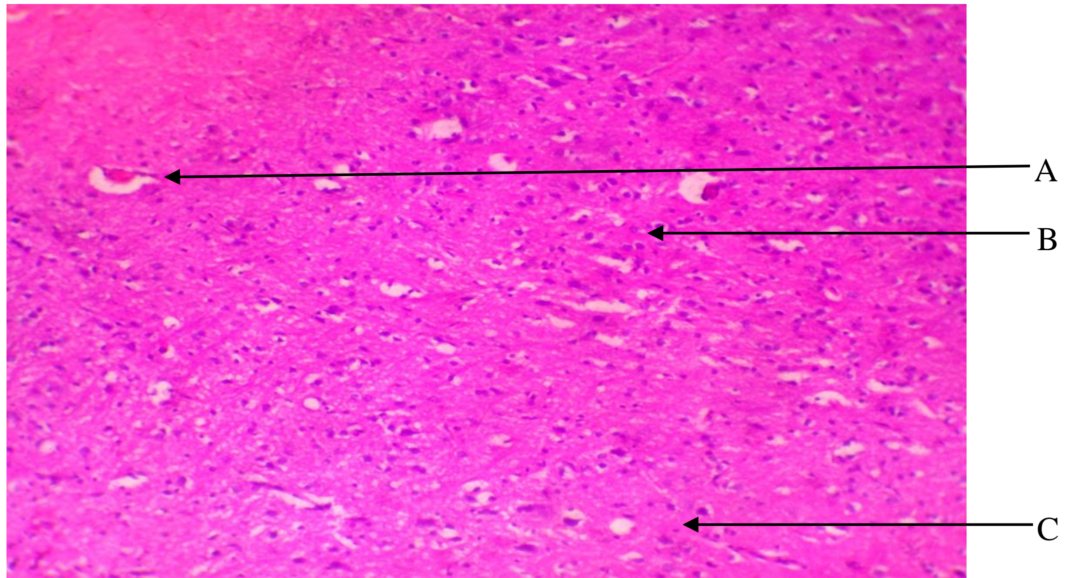

Fig 3. Plate 2a Shows the Histological Feature of the Brain of the Treated (Group B- 30 Min of UV Exposure Daily for 21 Days) Albino Rat with Presence of Neuron (A), Axon (B) and Axon Space (C) of the Brain Which Appear Normal in the Architecture. Handex100

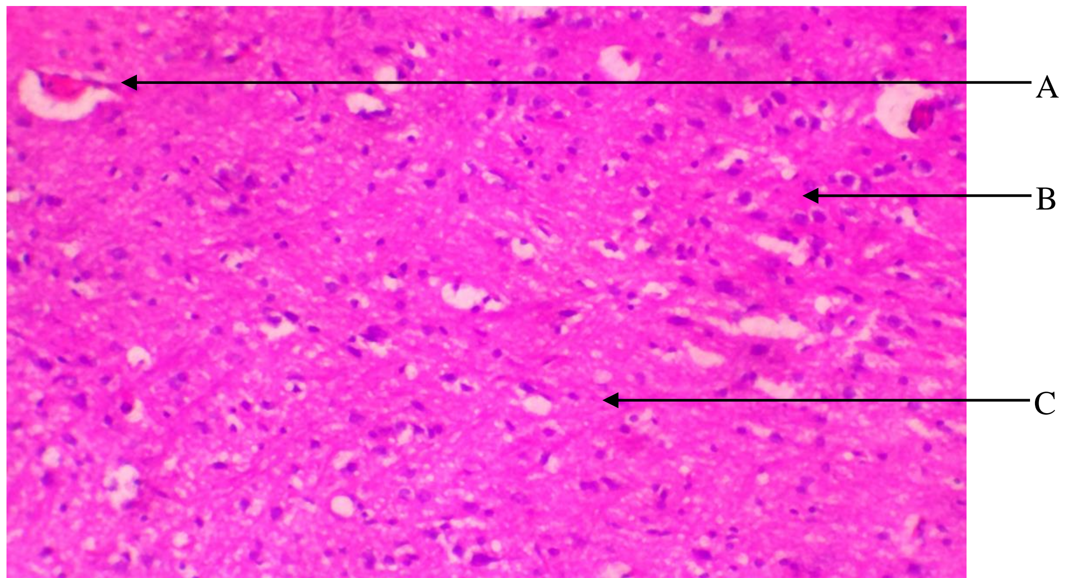

Fig 4. Plate 2b Shows the Histological Feature of the Brain of the Treated (Group B- 30 Min Of UV Exposure Daily for 21 Days) Albino Rat with Presence Of Neuron (A), Axon (B) and Axon Space (C) of the Brain Which Appear Normal in the Architecture. Handex400

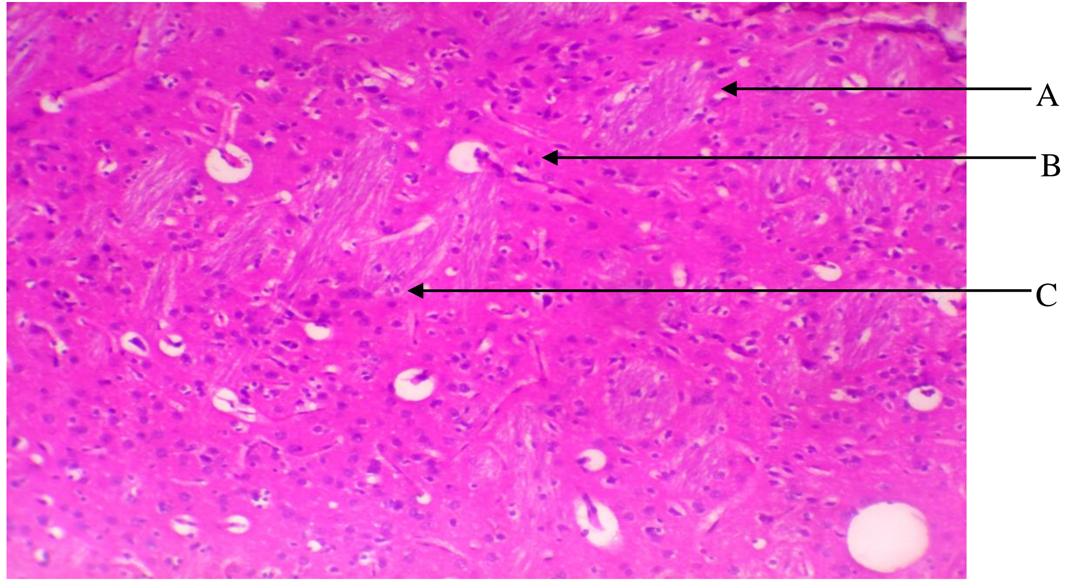

Fig 5. Plate 3a Shows the Histological Feature of the Brain of the Treated (Group C- 1 Hr of UV Exposure Daily for 21 Days) Albino Rat with Presence of Gliosis (A), Neurons (B) and Axon (C) of the Brain. Handex100

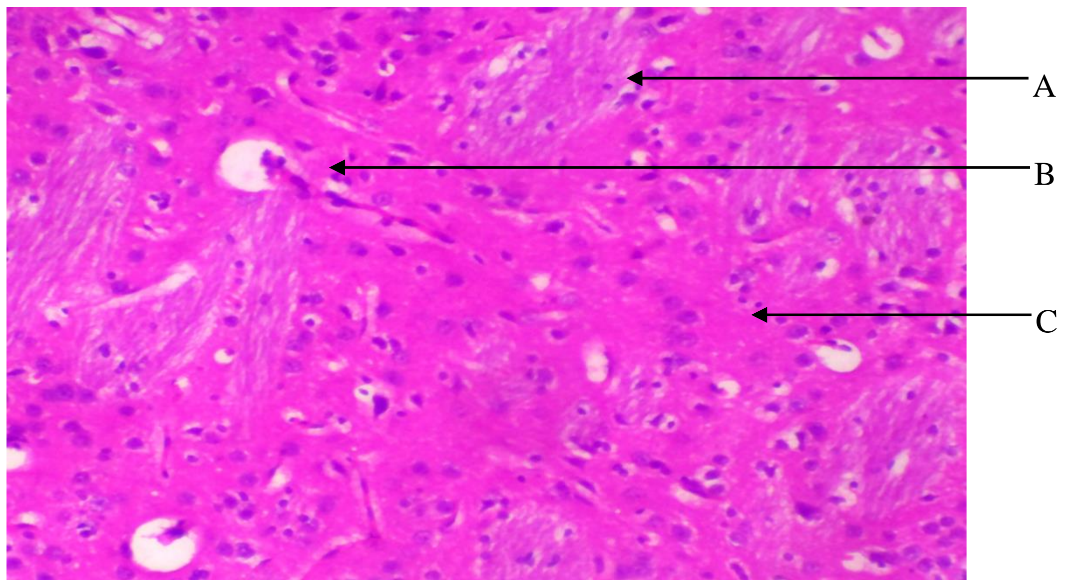

Fig 6. Plate 3b Shows the Histological Feature of the Brain of the Treated (Group C- 1 Hr of UV Exposure Daily for 21 Days) Albino Rat with Presence of Gliosis (A), Neurons (B) and Glia Cells (C) of the Brain. Handex400

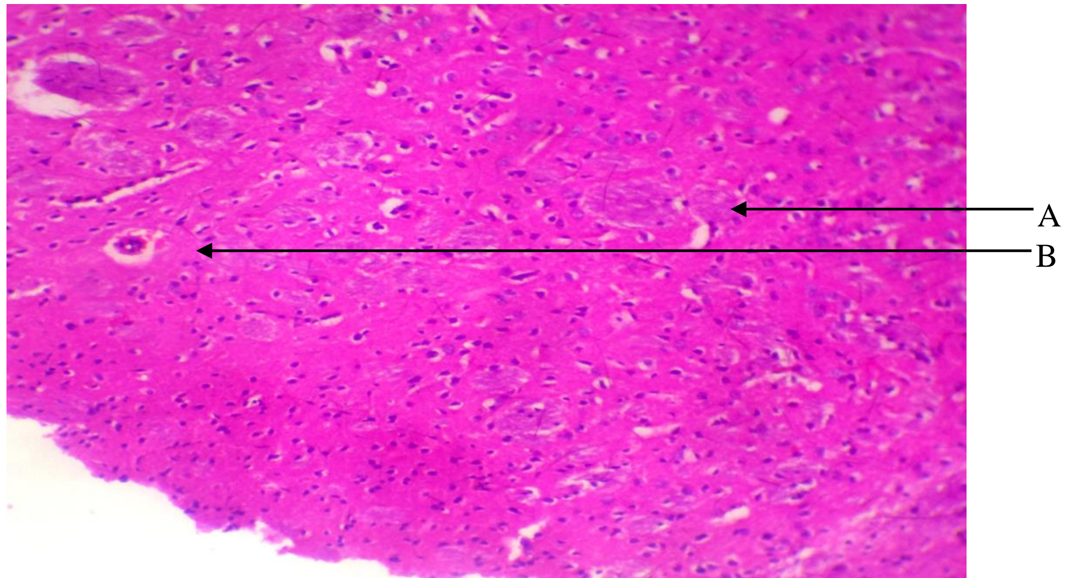

Fig 7. Plate 4a Shows the Histological Feature of the Brain of the Treated (Group D- 2 Hr of UV Exposure Daily for 21 Days) Albino Rat with Presence of Gliosis (A) and Congestion of Neurons (B) of the Brain. Handex100

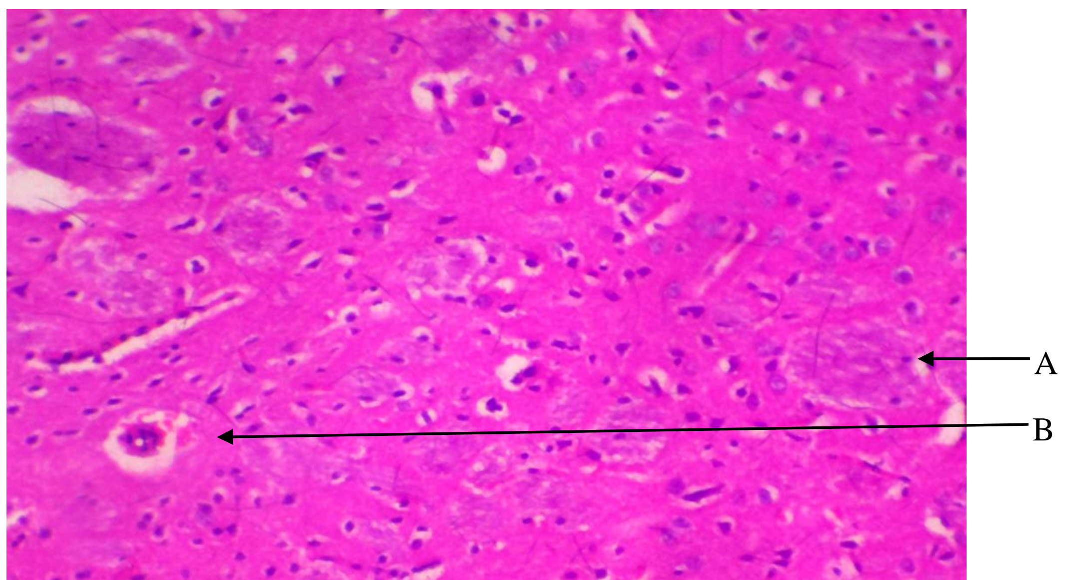

Fig 8. Plate 4b Shows the Histological Feature of the Brain of the Treated (Group D- 2 Hr of UV Exposure Daily for 21 Days) Albino Rat with Presence of Gliosis (A) and Congestion of Neurons (B) of the Brain. Handex400

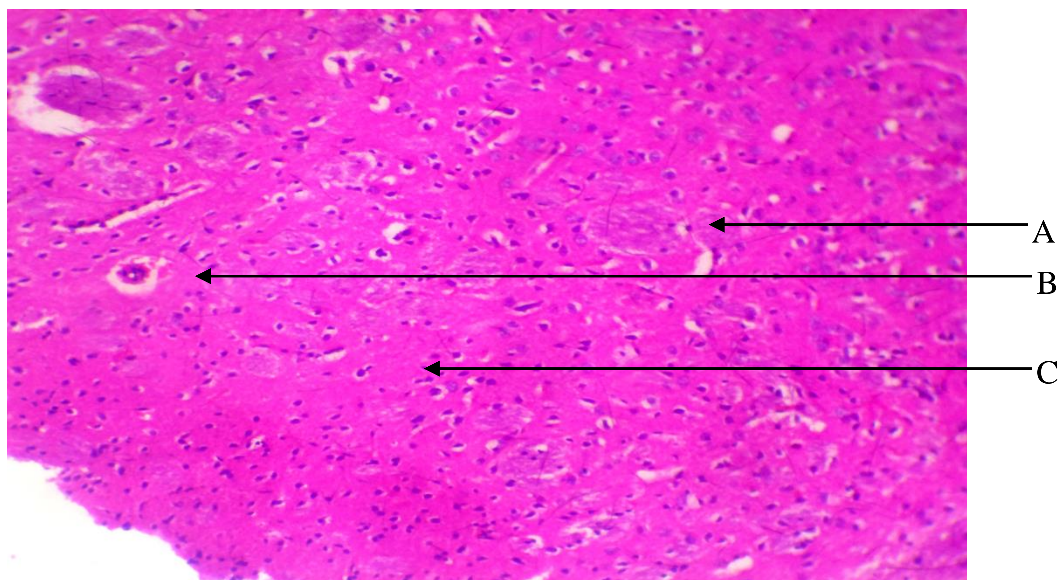

Fig 9. Plate 5a Shows the Histological Feature of the Brain of the Treated (Group E- 4 Hr of UV Exposure Daily for 21 Days) Albino Rat with Presence of Gliosis (A), Enlarged Axon Region with Congested Neurone (B) and Neuron (C) of the Brain. Handex100

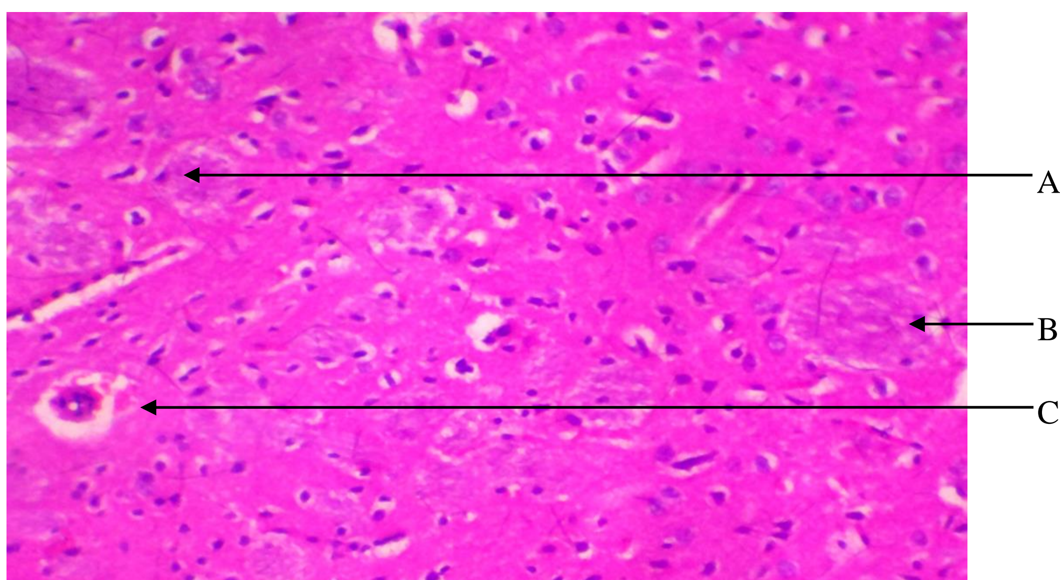

Fig 10. Plate 5b Shows the Histological Feature of the Brain of the Treated (Group E- 4 Hr of UV Exposure Daily for 21 Days) Albino Rat with Presence of Neuron (A), Gliosis (B) and Enlarged Axon Region with Congested Neurone (C) of the Brain. Handex400

The findings from Table 4.1 reveal significant alterations in the body weight of Albino Wistar rats exposed to different durations of UV light while being fed. The control group (Group A) maintained relatively stable weight values across the 28-day period, whereas the experimental groups exhibited varying degrees of weight reduction, with greater UV exposure correlating with a more pronounced decline in body weight. Statistical significance was observed on Days 7, 14, and 28

($p < 0.05$), suggesting that prolonged UV exposure had a considerable impact on the physiological state of the rats.

One possible explanation for these findings is the impact of UV radiation on metabolic processes. UV light has been reported to induce oxidative stress, which may lead to increased metabolic demands and tissue damage (Pérez et al., 2020). Prolonged exposure to UV light can result in the excessive production of reactive oxygen species (ROS), which in turn damages cellular structures, including proteins, lipids, and DNA (Wang et al., 2019). This oxidative damage may contribute to reduced energy efficiency and impaired nutrient absorption, leading to weight loss.

Another potential reason for the observed weight loss is the effect of UV radiation on appetite regulation and feeding behavior. UV exposure has been linked to alterations in the hypothalamic-pituitary-adrenal (HPA) axis, which plays a crucial role in stress response and energy balance (Smith & Kumar, 2021). Chronic exposure to UV radiation can disrupt hormonal homeostasis, leading to decreased appetite and food intake. Studies have demonstrated that excessive UV exposure triggers the release of stress-related hormones such as cortisol, which can suppress appetite and reduce overall caloric intake (Lin et al., 2018).

Additionally, the reduction in weight gain across experimental groups could be attributed to UV-induced immune system suppression. Research has shown that excessive UV exposure can compromise immune function by reducing the production of immune-regulatory cytokines and impairing T-cell activity (He et al., 2017). A weakened immune system may increase susceptibility to infections or other stress-related physiological impairments, which can negatively affect weight maintenance and overall health.

When comparing these findings with previous research, similarities and contrasts emerge. The findings align with the study by He et al. (2017), which demonstrated that prolonged UV exposure leads to metabolic disruptions and increased oxidative stress, resulting in decreased body weight. However, studies by Lin et al. (2018) suggest that moderate UV exposure may have some beneficial effects, such as enhancing vitamin D synthesis and regulating metabolism. This contrast indicates that the duration and intensity of UV exposure play crucial roles in determining its overall impact on physiological health.

The findings from the histological analysis of the brain tissues of albino rats exposed to varying durations of UV radiation demonstrate significant alterations in neuronal architecture, particularly in groups subjected to prolonged exposure. The control groups (Plates 1a and 1b) exhibited normal histological features, including the presence of microglial cells, neurons, and capillaries. In contrast, the experimental groups exposed to UV radiation showed a progressive increase in neural alterations, with longer durations of exposure correlating with greater histopathological changes. These observations strongly indicate that UV exposure, particularly over extended periods, contributes to neuroinflammation and structural disruptions in the brain.

One possible reason for these findings is the oxidative stress induced by UV radiation. UV exposure has been well-documented to generate reactive oxygen species (ROS), leading to oxidative damage and neuroinflammation (Pérez et al.,

2020). The presence of gliosis in Plates 3a, 3b, 4a, 4b, 5a, and 5b suggests a neuroinflammatory response, which aligns with previous studies indicating that prolonged UV exposure can activate glial cells, leading to chronic neuroinflammation (He et al., 2017). Gliosis is a well-known marker of neural distress and is often associated with neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. The activation of glial cells can result in the release of pro-inflammatory cytokines, further exacerbating neuronal damage and dysfunction. This suggests that the brain's response to UV radiation follows similar inflammatory pathways observed in neurodegenerative conditions.

Another plausible explanation is direct DNA damage caused by UV radiation. Studies have shown that excessive UV exposure can lead to DNA strand breaks and neuronal apoptosis, contributing to the observed neural congestion and gliosis (Wang et al., 2019). UV radiation is known to cause the formation of pyrimidine dimers and other mutagenic lesions in DNA, which can result in transcriptional errors and apoptosis in neurons. In severe cases, prolonged UV exposure may compromise the brain's ability to repair DNA damage, leading to cumulative neurodegeneration. DNA damage can also impair synaptic function, which may lead to cognitive deficits if left unchecked. Future studies should investigate whether the histological changes observed in this study translate to functional impairments in memory and cognition.

Additionally, vascular compromise could be a contributing factor. UV radiation has been linked to microvascular damage, which can lead to reduced oxygen and nutrient supply, exacerbating neuronal stress and damage (Smith & Kumar, 2021). The congestion of neurons seen in Plates 4a, 4b, 5a, and 5b could be indicative of a compromised blood-brain barrier (BBB), which may allow harmful substances to infiltrate the brain parenchyma, further aggravating neural distress. The role of cerebrovascular health in UV-induced neurotoxicity warrants further investigation, as disruptions to the BBB have been implicated in a variety of neurological disorders.

Comparing these findings with previous studies, there are both similarities and contrasts. Similar to the study by He et al. (2017), which demonstrated glial cell activation following prolonged UV exposure, this study found significant gliosis in groups subjected to 1 to 4 hours of daily UV exposure. However, a study by Lin et al. (2018) found that UV-induced neuroinflammation was reversible upon cessation of exposure, whereas the extent of gliosis observed in the present study suggests a potentially more persistent impact. The discrepancy may be due to differences in experimental protocols, including UV wavelength, intensity, and the duration of exposure. Additionally, while Pérez et al. (2020) reported neuronal apoptosis as a key consequence of UV-induced oxidative stress, the present study did not directly assess apoptosis but highlighted neuronal congestion, which may be an early indicator of cellular distress before apoptosis occurs.

In another related study, Xu et al. (2022) found that UV exposure resulted in increased microglial activation and synaptic loss in rodent models. These findings align with the results of the present study, suggesting that UV radiation can have both direct and indirect effects on neural integrity. The presence of gliosis,

neuronal congestion, and axonal abnormalities in the present study supports the hypothesis that UV exposure leads to progressive neural damage, potentially predisposing individuals to neurodegenerative conditions over time.

CONCLUSIONS AND RECOMMENDATIONS

This study demonstrates a clear relationship between UV exposure duration and the severity of histopathological alterations in the brain. Short-term exposure (30 min daily) did not cause significant changes, whereas prolonged exposure (1 to 4 hours daily) led to pronounced gliosis, neuronal congestion, and axonal alterations. These findings suggest that chronic UV radiation exposure can have detrimental effects on neural integrity, possibly through oxidative stress, DNA damage, and vascular compromise. The long-term implications of these findings may extend beyond structural damage to include cognitive and behavioral deficits, underscoring the need for further investigations.

Based on the Findings in this Study, the Following are Recommended;

1. Further studies should employ molecular assays to quantify oxidative stress markers and apoptosis-related proteins to confirm the underlying mechanisms of UV-induced neural damage.
2. Protective strategies, such as antioxidant supplementation, should be explored to mitigate UV-induced neuroinflammation and neuronal damage.
3. Behavioral and cognitive assessments should be incorporated in future studies to evaluate the functional implications of these histological changes.
4. Investigations on potential neuroprotective agents, such as melatonin or vitamin E, should be conducted to assess their efficacy in preventing or reversing UV-induced brain damage.
5. Future research should explore the effects of different UV wavelengths and intensities to determine whether specific types of UV radiation are more neurotoxic than others.

FURTHER STUDY

This research still has a delay, so it is necessary to conduct further research related to the topic of Histological Effect of Ultra Violet (UV) Light on the Brain of Albino Wistar Rats to improve this research and add insight for readers

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