

Remediating and Modulatory Potential of Bark of *Ficus platyphylla* on NMU-Induced Breast Cancer in *Mus musculus* (Albino mice)

Oloyede, M. Adeola^{1*}, Aina, O. Matthew¹, Lawal, B. Oluwatoyin¹, Ottu, Bodunrin¹ and Agbonifo, O. Worship¹

¹Department of Cell Biology and Genetics, University of Lagos, Akoka, Lagos-Nigeria.

*Correspondence: moloyede@unilag.edu.ng; +2348022215162

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Abstract

Ficus platyphylla (Fig tree) is one of many medicinal plants used in Nigeria in managing various medical conditions. This study evaluated the protective effects of *Ficus platyphylla* on Nitrosomethylurea (NMU)-induced breast carcinogenesis. A total number of 25 female mice (19g -25g) were divided into five groups. They were pre-treated with NMU, then orally administered 1600, 800 and 400 mg kg⁻¹ of extract respectively for 10 weeks, others received cisplatin (reference drug) and normal saline (control). Upon expiration, mice were euthanized via jugular puncture. Breast tissue was harvested for biochemical, antioxidant, histologic and DNA analysis. There was insignificant body weight increase. Biochemical analysis showed significant increase in ALP for cisplatin (p<0.05), urea for 800 mg kg⁻¹ (p<0.05) and 400 mg kg⁻¹ (p<0.01). Total protein significantly reduced (p<0.01) in 1600 mg kg⁻¹. GSH significantly increased (p<0.01) at 400 and 800 mg kg⁻¹ doses, and significantly decreased (p<0.01) in MDA at 400 mg kg⁻¹. Breast histoarchitecture was conserved for treatments. DNA purity and concentration increased significantly in treatments. AXIN1 gene electrophoresis displayed clear bands for treatments. The increased GSH and decreased MDA suggest that extract counteracted oxidative stress associated with NMU. Reduced urea could imply potentially decreased metabolic burden from NMU toxicity. Conserved breast histoarchitecture suggests extract's protective effects. Enhanced DNA concentration, purity, and clear AXIN1 gene bands, may pontificate that extract supports DNA repair mechanisms to mitigate carcinogenic damage. This study, therefore infer that the extract offers protection against NMU-induced carcinogenesis, possibly elicited by flavonoids, steroids, alkaloids, triterpenoids, phenols, terpenoids, and coumarins in extract.

Keywords: AXIN1, Breast Cancer, DNA, *Ficus platyphylla*, Toxicity

1.0 Introduction

Ficus platyphylla, commonly known as broad leaf fig, is a deciduous plant belonging to the Moraceae family, predominantly found in Northern Nigeria, where it is locally referred to as "Gamji" [1]. Historically, Nigerian traditional medicine has utilized preparations of this plant to treat depression, pain, ulcer, epilepsy, psychosis, and inflammation [2]. Many rural communities in Nigeria have highly commended the medicinal properties of the plant [2]. Various components of the plant, including its leaves, stem, bark, and root, are utilized for these therapeutic effects [1]. In some parts of Nigeria, a brew made by

combining the stem bark, seeds, and leaves of *Ficus platyphylla* is consumed as tea. This is believed to enhance fertility [3]. Additionally, the stem bark is also utilized locally to treat malaria and tuberculosis [4].

The therapeutic effects of *Ficus platyphylla* in traditional medicine are largely due to its significant concentration of phytochemicals [5]. Phytochemicals are biologically active constituents that are found naturally in plants, exhibiting variations in their occurrence and secretion across different plant species [6]. They play a crucial role in human health through a range of effective mechanisms, such as

possessing antioxidant properties, enhancing the activity of detoxifying enzymes, impacting DNA metabolism, and facilitating DNA repair, among others [6]. Hassan *et al.* [1] reported after a qualitative screening that *F. platyphylla* contains phytochemicals such as tannins, alkaloids, saponins, cardiac glycosides, flavonoids, steroids, and phenols. Furthermore, several additional phytochemicals have been discovered in various *F. platyphylla* extracts using sophisticated analytical techniques like gas chromatography-mass spectrometry (GC-MS) and liquid chromatography-mass spectrometry (LC-MS) [1]. N-hexadecenoic acid is one such phytochemical that has been shown to have antifungal, antioxidant, and antibacterial qualities in the past [1]. 9, 12-Octadecadienoic acid was also identified and it has been discovered to have anti-inflammatory and antibacterial qualities [7]. Other compounds detected were squalene, cis-vaccenic acid, beta-sitosterol, and lanosterol, among several others [1]. Squalene possesses anti-inflammatory, antioxidant, and anticancer properties [1]. Cis-vaccenic acid is an omega-7 fatty acid that has been seen to improve insulin sensitivity and decrease low-density lipoprotein cholesterol (LDL-C) [1]. Also, beta-sitosterol has anti-inflammatory properties and inhibits cholesterol uptake into the bloodstream [1]. This study aimed to investigate the potential ameliorating effects of *Ficus platyphylla* on N-Methyl-N-Nitrosourea (NMU) induced breast carcinogenesis in *Mus musculus*.

2.0 Materials and Methods

2.1 Ethical Approval

The ethical approval was issued by Mr Adebisi of College of Medicine University of Lagos with reference no CMUL/HREC/0887/19.

2.2 Plant Collection and Identification

Bark of *Ficus platyphylla* were collected in the month of March, 2022, from traditional merchants in Bariga Market, Lagos. The leaves were identified and authenticated at the department of Botany, University of Lagos, with voucher number LUT 8746 of the plants deposited in the herbarium.

2.3 Plant Preparation and Extraction

The barks of *Ficus platyphylla* were cut in bits and air dried, plant samples were then dehydrated at 35°C in an oven; for total removal of any present water molecules. The dehydrated bark of *Ficus platyphylla* was granulated to a fine powdered form. The bark extracts were obtained by using crude extraction protocol, using a ratio of 1:3 solutes to solvent. The preparation was soaked for 72 hours after which it was filtered using a funnel and cotton wool. The filtrate was subjected to heating at 40±1°C using a regulated hot plate. Extract derived was weighed and reconstituted for bioactivity.

2.4 Experimental Animals

Twenty-five female albino mice obtained from the National Agency for Food and Drug Administration and Control, Lagos, Nigeria (NAFDAC) of average weight of 19-28g were used for the study and were acclimatized for two weeks in animal house of the Department of Cell Biology and Genetics, University of Lagos. The mice were thereafter divided into five groups of five mice each for the experiment.

2.5 Tumour Induction and Treatment

NMU (50mg kg⁻¹) was prepared for administration to the mice. After acclimatization of the mice, the NMU was administered intraperitoneally in the ventral midline of the animal (between the third and fourth pair of the mammary gland) as described by Rajmani *et al.* [8]. Mice were allowed to stay 7 days before the commencement of the treatment. The twenty-five albino mice were weighed and grouped into five groups of five animals each. Three groups (A, B, C) received treatments while two (D, E) served as control (positive and negative). All the mice were induced with the prepared NMU, the positive control group was treated with 3mg kg⁻¹ Cisplatin. Negative control group was administered normal saline intra-peritoneally, same time and duration as positive control. The treatment groups A, B, C were orally administered 400mg kg⁻¹, 800mg kg⁻¹ and 1600mg kg⁻¹ respectively for 10 weeks. All mice were weighed weekly and inspected for any irregularities. The mice were sacrificed via jugular puncture at the expiration of treatments.

2.6 Histologic Preparation

According to Oloyede [9], the breast tissue was collected from each animal and fixed in 10% formaldehyde. The tissue biopsies were dehydrated and embedded in paraffin, cut into 4-5µm sections with rotary microtome (LEICA RM 2235), and stained with haematoxylin-eosin for photo microscopic examination. Photographs of the prepared slides were taken with a camera attached to the compound light microscope.

2.7 DNA Extraction

The DNA extraction was done according to García-Alegría *et al.* [10] with slight modification, using DNA miniprep plus kit from Zymo research group purchased from Inqaba Biotec West Africa LTD. DNA extraction kit protocol was used. Breast tissue of about 25mg was put in an Eppendorf tube, 95µl of water, 95µl of solid tissue buffer and 10µl of Proteinase K was added to the tube. It was mixed thoroughly and incubated at 55°C for 2 hours till tissue solubilized. The solution was mixed thoroughly after incubation and supernatant carefully poured in another tube. The undissolved tissues were discarded. 400µl of Genomic binding buffer was added to the supernatant and mixed thoroughly. The mixture was then transferred to a Zymo-spin HC-XLR column in a collection tube and centrifuged ($\geq 12000g$) for 1 minute. The collection tube and the flow through was discarded. 400µl of DNA pre wash buffer was then added to the column in a new collection tube and centrifuged for 1 minute. Collection tube was discarded again. 700µl was then added to the DNA wash buffer and centrifuged for 1 minute, flow through and collection tube was then discarded. Previous step was repeated only that 200µl was added. Lastly 100µl of DNA elution buffer was added to the spin column and placed in a clean micro-centrifuge tube. Content was incubated at room temperature for 5 minutes and centrifuged at top speed for 30 seconds to elute the DNA. The eluted DNA was immediately used and stored at $\leq -20^{\circ}\text{C}$ for future use.

2.8 Primer

The primer sequence was adapted from the work of Vuttariello *et al.*, [11]. The primer was

designed for AXIN 1 and mutation in breast cancer. The sequence of the forward and backward primer was 5'-GGCGTAGGTTTTAGAGATGC-3' and 5'-AAACGAACTCGCTCCCTT-3' respectively.

2.9. Concentration and Purity Determination

A quantitative spectrophotometric assay of extracted DNA was performed with reference to García-Alegría *et al.* [10], using a Nano-Drop lite spectrophotometer. Absorbance was measured at wavelengths of 260 and 280 (A_{260} and A_{280} , respectively) nm. The absorbance quotient (OD_{260}/OD_{280}) provided an estimate of DNA purity. An absorbance quotient value of $1.8 \leq 2.0$ was considered to be good; purified DNA. A ratio of < 1.8 was indicative of protein contamination; whereas a ratio of > 2.0 indicated RNA contamination.

2.9.1 Polymerase Chain Reaction (PCR)

Polymerase chain reaction, according to Kadri, [12] was carried out to amplify the AXIN 1 gene of the DNA. The PCR reaction was carried out using the Solis Biodyne 5X HOT FIREPol Blend Master mix. PCR was performed in 20 µl of a reaction mixture containing 4 µl of Master Mix, 0.6 µl of each primer, 12.8 µl of sterile distilled water and 2 µl of extracted DNA. Thermal cycling was conducted in a PTC 100 Peltier thermal cycler (MJ Research) for an initial denaturation of 95 °C for 10 min followed by 40 amplification cycles; Denaturation at 95 °C for 30secs; Annealing at 60 °C for 30 secs and 30 s at 72 °C. This was followed by a final extension step of 10 min at 72 °C

2.9.2 Electrophoretic Analysis

The amplification product was subjected to electrophoresis (Bioad) in a 2.0% agarose gel in 0.5% Tris-borate-EDTA buffer at 70v for 1h and stained with ethidium bromide and it was then visualized under ultra-violet trans-illuminator and the expected band were observed using a polaroid camera Lee *et al.* [13].

2.9.3 Statistical Analysis

Data obtained from the study were expressed as mean $\pm$ standard error of the mean (mean $\pm$ SEM). Analysis of data was carried out using

student t-test. Difference among the treatment groups were analyzed using one-way analysis of variance. Values of $p < 0.05$ were considered significant.

3.0 Results

Table 1 shows variation in the body weight of mice during the duration of extract administration. Though not significantly different when compared to the negative control. There was insignificant increase in the body weight of the treated mice compared to the control.

Table 1: Weight of the mice during treatment with *F. Platyphylla* for 10 weeks

Week	Control	Cisplatin	400mg kg ⁻¹	800 mg kg ⁻¹	1600 mg kg ⁻¹
1	20.38±0.37	21.22±0.32	22.42±0.09	23.60±0.22	25.92±0.69
2	20.72±0.58	23.04±0.32	24.44±0.48	25.90±0.88	25.80±1.12
3	21.57±0.77	24.45±0.29	25.42±1.72	25.90±0.85	26.40±1.31
4	22.72±0.57	25.40±0.40	27.12±1.58	26.76±0.81	28.12±1.26
5	23.65±0.75	26.45±0.35	27.26±0.96	27.66±0.63	28.40±0.45
6	23.87±0.79	26.62±0.34	27.46±0.94	27.80±0.62	28.56±0.42
7	24.05±0.80	23.95±1.35	24.40±0.98	24.96±1.62	25.46±0.66
8	24.15±0.73	23.63±1.25	26.83±0.82	27.65±2.45	27.10±2.30
9	24.35±0.74	23.05±1.55	27.13±0.86	27.80±2.60	27.30±2.30
10	24.82±0.84	23.70±1.40	27.56±0.78	28.25±2.55	27.90±2.30

Data presented as Mean ± SEM (N=5) (a=P<0.05, b=P<0.01, c=P<0.001)

3.1 Biochemical result of the Breast tissue

A comparison of the different biochemical parameters in breast tissue of the mice in this study is shown in Table 2. Mice treated with 800 mg kg⁻¹ of the plant extracts showed a significant increase (P<0.05) in the Alanine Transaminase values when compared with the control group. In the Alkaline phosphatase values observed in all mice, only mice treated with cisplatin showed a significant decrease (P<0.05) when compared

with the control group. There was also a significant decrease in the group of mice treated 800 mg kg⁻¹ (P<0.05), 400 mg kg⁻¹ (P<0.01) of the plant extract and mice treated with cisplatin (P<0.01), when compared with the control group in the Urea value seen. Only mice treated cisplatin showed a significant decrease (P<0.01) when compared with the control group in the creatinine values seen.

Table 2: Breast tissue biochemical analytes expression between treatments and control

Treatment	AST	ALT	ALP	UREA	Albumin	Creatinine
-ve Control	3.40±0.92	14.63±0.66	96.44±4.50	71.09±0.60	2.13±0.55	0.92±0.07
1600 mg kg ⁻¹	3.78±0.07	39.90±20.59	63.90±3.46	36.84±32.46	1.98±0.15	0.65±0.19
800 mg kg ⁻¹	21.90±8.70	93.26±5.06 ^a	66.45±4.08	3.42±0.47 ^a	1.91±0.02	1.01±0.01
400 mg kg ⁻¹	13.53±1.83	92.89±17.84	71.79±1.99	3.03±0.42 ^b	1.55±0.22	0.88±0.07
Cisplatin	8.88±0.13	89.81±5.32	72.26±5.88 ^a	3.29±0.09 ^b	2.01±0.09	0.75±0.06 ^a

Values are mean ± Standard error of the mean (SEM). Value a= <0.05, b= <0.01, c=0.001.

Table 3; shows the results obtained from the evaluation of selected antioxidant biomarkers of mammary gland of experimental mice. There is significant decrease in total protein values at 1600 mg kg⁻¹ concentration as well as in the Cisplatin group (p<0.01) when compared to the

control group. Catalase showed significant decrease in the values obtained at 1600 mg kg⁻¹ and 400 mg kg⁻¹ (p<0.01), but not at 800 mg kg⁻¹. Superoxide dismutase showed no significance in any of the treatment groups, but showed significance increase in Cisplatin group (p<0.01).

Malondialdehyde showed significant decrease at the 400 mg kg⁻¹ group (p<0.05), while Glutathione showed significant increase at both 400 mg kg⁻¹ and 800 mg kg⁻¹ (p<0.01) concentrations

Table 3: Antioxidants profile of mammary gland of experimental mice

Treatment	Total protein	Catalase	SOD	Malondialdehyde	Glutathione
1600 mg kg ⁻¹	8.46±2.77 ^b	5.55±0.11 ^b	70.34±3.88	5.86±0.47	0.25±0.09
800 mg kg ⁻¹	18.05±3.48	3.12±0.34	41.37±12.35	3.97±1.82	0.22±0.20 ^b
400 mg kg ⁻¹	6.69±0.37	6.06±0.17 ^b	77.67±60.05	4.039±1.48 ^a	0.21±0.86 ^b
Cisplatin	16.04±4.57 ^b	3.28±0.18	139.70±5.20 ^b	9.96±3.63	0.09±0.02
Control	10.19±0.30	10.05±3.25	118.20±9.60	5.39±0.54	0.13±0.08

Values are mean ± Standard error of the mean (SEM). Value a= <0.05, b= <0.01, c=0.001.

3.2 Breast histology of experimental Mice

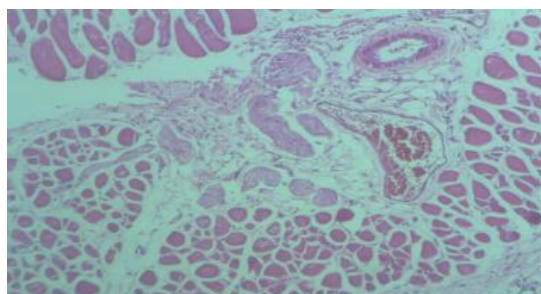


Plate 1A; 1600mg kg⁻¹ X40

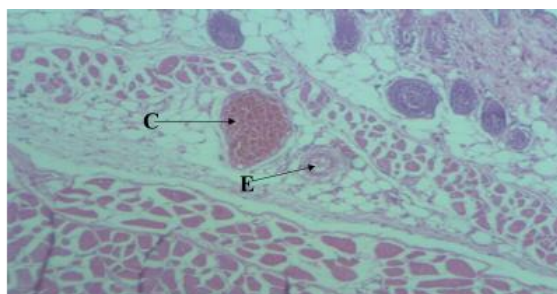


Plate 1B; 800 mg kg⁻¹ X40

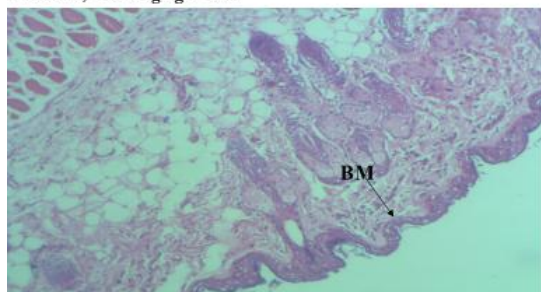


Plate 1C; 400mg kg⁻¹ X40

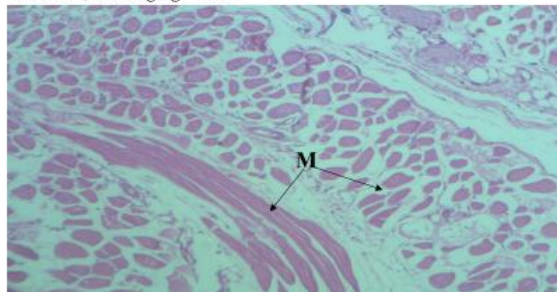


Plate 1D; Cisplatin X40

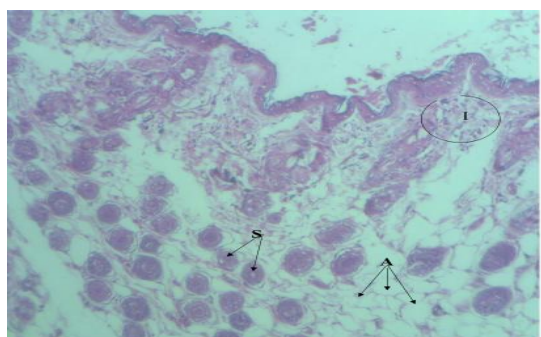


Plate 1E; Control X40

Section of the breast in all groups shows wavy orthokeratinised stratified squamous epithelium overlying loose collagenous stroma within which are skin adnexal structures. Plate 1A, B, C and D, revealed normal histology with C-Capillaries, E- Excretory duct, BM-Basal Membrane, and M-Muscle fibre. Plate 1E (negative control) showed I-Invasive breast carcinoma with abnormal nuclear pleomorphism, an invasion into normal breast stroma, S-Sebaceous gland and A-Adipocytes. H&E staining.

3.3 Purity and Concentration of DNA

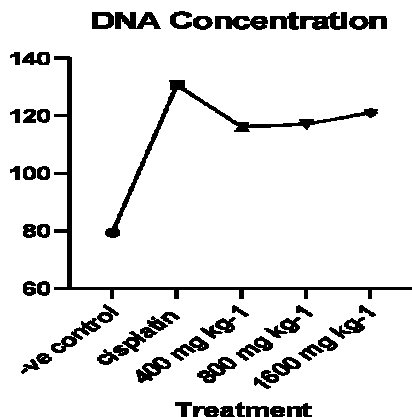


Fig 1A

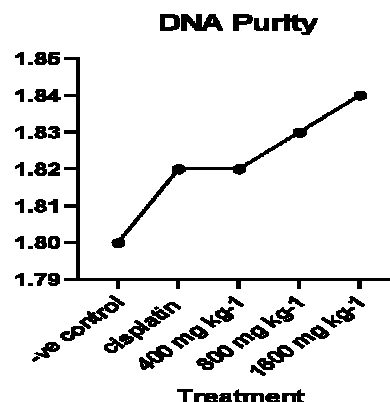


Fig. 1B

Figure 1a and b shows the purity and the different concentration of DNA extracted from breast tissue of mice in each group. In DNA concentration, *Ficus platyphylla* compared favourably with the reference drug (cisplatin) and was higher in treatments than control (1A). In figure 1B a significant ($p < 0.05$) dose-dependent increase in DNA purity was observed and well established in treatment than both the reference drug (cisplatin) and control.

3.4 Electrophoresis Image of PCR Amplification

Gel Electrophoresis image of the PCR amplified AXIN1 gene of the experimental female albino mice is shown in Plate 1. Clear bands and smear were observed in all the mice groups.

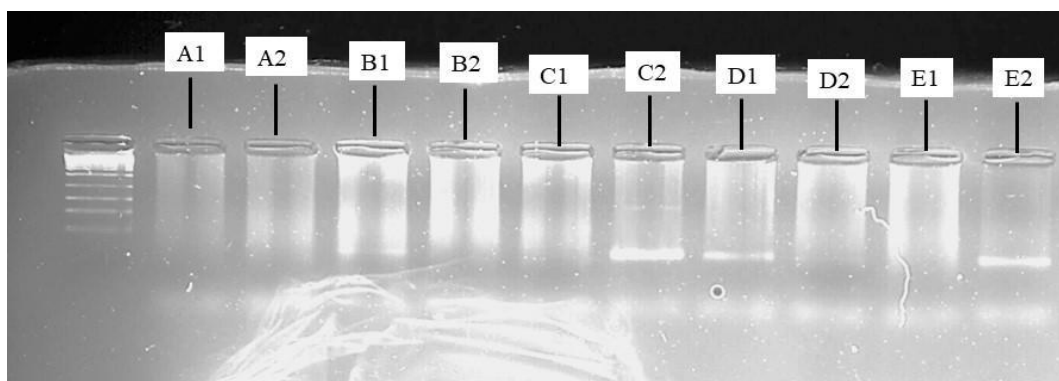


PLATE 2: DNA gel electrophoretic band pattern of PCR amplified AXIN1 gene of the experimental female albino mice.

A: Animals treated with 1600 mg kg⁻¹ of *Ficus platyphylla* after being injected with NMU.

B: Animals treated with 800 mg kg⁻¹ of *Ficus platyphylla* after being injected with NMU.

C: Animals treated with 400 mg kg⁻¹ of *Ficus platyphylla* after being injected with NMU.

D: Animals treated with Cisplatin after being injected with NMU.

E: Animal had access to feed and water only after injection of NMU

4.0 Discussion

In exploring the multifaceted benefits of *Ficus platyphylla* in mitigating the effects of carcinogenesis and toxicity, this study delved into various aspects of its therapeutic potential.

The effectiveness of an extract can be observed through changes in body weight, which may demonstrate its ability to counteract toxic effects [14]. The insignificant increase in the body weight of rats treated with *Ficus platyphylla* suggests that it may have a non-harmful physiological impact on body weight in the context of carcinogenesis [14]. This indicates that the extract could possess properties that help to stabilize or maintain body weight, even in the presence of potentially harmful stressors [14]. This aspect of the potential of *Ficus platyphylla* highlights its ability to provide protection without causing negative physiological effects [15].

Antioxidants are crucial in the body for minimizing oxidative processes and the harmful effects of reactive oxygen species (ROS) [16]. They achieve this by neutralizing free radicals and donating electrons to stabilize them without jeopardizing their stability, thus preventing ROS from causing cellular damage [16]. The significant increase in GSH levels at both 400 and 800 mg kg⁻¹ dosages implies that *Ficus platyphylla* has antioxidative properties that could help in counteracting the oxidative stress associated with induced toxicity and cancer [17]. GSH is an essential intracellular antioxidant that is composed of thiol and is effective in reducing H₂O₂, hydroperoxide (ROOH), and xenobiotic toxicity [18].

The antioxidant effects of *Ficus* have been described in a study by Hassan *et al.* [1] and are primarily attributed to its high content of polyphenolic compounds such as flavonoids, saponins, and tannins. By neutralizing free radicals and reducing oxidative stress, these antioxidants help prevent DNA damage and cellular mutations that can cause the progression of cancer [1]. The observed reduction in MDA levels of animals treated with 400 mg kg⁻¹ dosage of the *Ficus* extract further confirms the antioxidant properties of the treatment, and its ability to decrease lipid peroxidation, a process linked to tumorigenesis, highlighting its potential in cancer prevention and treatment strategies

[18]. By decreasing lipid peroxidation, *Ficus platyphylla* may help alleviate oxidative damage associated with cancer progression [18], also, the presence of Squalene and 9,12-octadecadienoic acid (Z, Z) methyl ester may have reinforced the antioxidant activities [1]. The results align with recent studies by Oraebosi, [19] that have demonstrated the antioxidant properties of *Ficus platyphylla*.

The therapeutic potential of *Ficus platyphylla* extends to its impact on kidney function [19]. Urea, a byproduct of amino acid metabolism in the liver, is filtered by the glomerulus, reabsorbed by the renal tubules, and ultimately excreted in urine [20]. This biomarker is essential for diagnosing and assessing kidney injury because it sheds light on the capacity of the kidney to cleanse and expel waste from the body [17]. The significant decrease in urea levels in mice treated with 800 mg kg⁻¹ and 400 mg kg⁻¹ of *Ficus platyphylla* extract suggests that at those doses, the extract might be reducing the metabolic burden of the effects of NMU toxicity, and improving kidney function [20]. Additionally, the decrease in total protein levels observed at a concentration of 1600 mg kg⁻¹ of *Ficus platyphylla* extract treatment may indicate a decrease in protein synthesis [21]. This reduction in protein levels might suggest that *Ficus platyphylla* helps mitigate the damage or stress caused by NMU on protein synthesis or turnover in the body [21].

Cancer diagnosis and monitoring increasingly rely on biochemical markers, with enzymes like Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), and Alkaline phosphatase (ALP) playing a vital role in this process [22]. AST is a pyridoxal phosphate-dependent enzyme involved in amino acid metabolism, catalyzing the reversible transfer of an α -amino group between aspartate and glutamate [17]. The lack of significant changes in AST levels across all treatment doses may suggest that *Ficus platyphylla* extract maintain normal enzyme function, suggesting its non-toxic nature in tissues where AST is prevalent [23]. Furthermore, the integrity of liver cell plasma membranes is evaluated using ALP [17]. The lack of significant changes in ALP levels for the groups treated with the plant extract could indicate the potential of *Ficus platyphylla* as a safe therapeutic agent in managing NMU-induced

carcinogenesis, without adversely affecting liver metabolism [24].

Furthermore, the histopathological studies reinforce the efficacy of *Ficus platyphylla* in combating the carcinogenic effects of NMU, as tissue biopsy can be a valuable diagnostic tool in this regard [4]. The normal histology displayed in the breast tissue sections from the groups treated with *Ficus platyphylla* indicates that the extract, administered in varying dosages, effectively mitigates the carcinogenic effects of NMU [25]. This is evidenced by the preservation of normal histology in breast tissue sections of the treated groups [25]. The presence of typical tissue structures such as excretory ducts (E), capillaries (C), the basal membrane (BM), and muscle fibers (M) in these groups, as observed in normal breast tissue samples of rats in a study by Feng *et al.*[251], indicates a significant protective or remedial action of *Ficus platyphylla* against NMU-induced carcinogenesis.

In assessing molecular pathways in carcinogenesis, Axis Inhibition Protein 1 (AXIN1), a crucial component of the Wnt signaling pathway, emerges as a key player [22]. AXIN1 plays a vital role in various cellular processes, such as proliferation, differentiation, and apoptosis [27]. The disruption of Wnt signaling is a common characteristic of several types of cancer, emphasizing the significance of its components, including AXIN1, in cancer development [27]. AXIN1 primarily functions as a tumor suppressor, and genetic mutations or alterations in the AXIN1 gene can impair its regulatory mechanism, leading to uncontrolled activation of Wnt signaling and promoting the development of cancerous cells [27]. The observation of clear bands in gel electrophoresis for the groups treated with *Ficus platyphylla* is indicative of successful amplification of the AXIN1 gene [13]. This finding implies that *Ficus platyphylla* administration plays a substantial role in maintaining the normal expression levels of the AXIN1 gene, even in the face of NMU-induced carcinogenesis [29]. This highlights the potential protective role of *Ficus platyphylla* against the genetic disruptions commonly associated with the carcinogenic effects of NMU.

To effectively evaluate the genotoxic effects of carcinogens, such as NMU, and the impact of treatments, it is necessary to assess the level of purity and concentration in DNA samples [10].

This is vital for evaluating DNA damage, including the formation of DNA adducts and mutations associated with tumorigenesis [31]. Moreover, it is important to assess the efficacy of cancer-prevention therapies based on decreased or absent DNA damage [31]. The increased DNA concentration in the treatment groups compared to the control group suggests a significant protective or restorative effect of *Ficus platyphylla* on cellular DNA integrity against carcinogenic damage induced by NMU [31]. This implies that the extract may exert modulatory effects on the carcinogenesis process, possibly by improving DNA repair mechanisms or interfering with the tumor development pathway, preventing further DNA damage [31]. Furthermore, the increase in DNA purity in groups treated with *Ficus platyphylla*, in comparison to the control group, suggests that the extract may have a more favorable mechanism of action that is linked to fewer side effects or increased effectiveness in maintaining or restoring DNA integrity[13]. Notably, the dose-dependent increase in DNA purity indicates that the mitigative effects of the extract on DNA are directly correlated with its dosage, emphasizing the significance of dosage in attaining intended therapeutic outcomes [29].

4.0 Conclusion

The therapeutic efficacy of *Ficus platyphylla* in mitigating oxidative stress, improving kidney function, preserving normal tissue histology, maintaining the AXIN1 gene to preserve crucial molecular pathways, improving DNA repair mechanisms, and safeguarding DNA integrity positions it as a promising candidate for cancer therapy. Future research to further elucidate the mechanisms behind these effects will potentially pave the way for new therapeutic strategies in the treatment of cancer.

5.0 Acknowledgement

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6.0 Conflict of Interest

We hereby declare that there is no conflict of interest whatsoever.

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