IJPSR (2024), Volume 15, Issue 10 (Research Article)

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 22 April 2024; received in revised form, 25 May 2024; accepted, 09 July 2024; published 01 October 2024

ANTIOXIDANT AND ANTITUMOR EFFECTS OF MORINGA OLEIFERA LAM AGAINST EHRLICH'S ASCITES CARCINOMA BEARING MICE

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Keywords:

Moringa oleifera, Glutathione, Ehrlich's ascites carcinoma, Superoxide dismutase

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ABSTRACT: Objectives: *Moringa oleifera* has enormous attention as the 'natural nutrition of the tropics and an essential food commodity. Antioxidants important in deactivating free radicals having damaging role on biological cells. Polyphenolic compounds are the most prevalent antioxidants seen in plants. Present study aims to evaluate the antioxidant and antitumor effect of ethanolic leaf extract of *Moringa oleifera* (ELMO) against Ehrlich's ascites carcinoma bearing Swiss albino mice. **Materials and Methods:** 500 mg/kg body weight of ELMO was administered once in a day about 15 days after 24hrs of tumour inoculation. In mice treated with ELMO we observed decreased body weight, abdominal circumference and tumour volume, when compared to Ehrlich's ascites carcinoma control mice. We also observed in mice Treatment with ELMO decreased levels of lipid peroxidation (LPO) and increased levels of (GSH), and superoxide dismutase (SOD). **Results:** Mice treated with 500mg/kg body weight of ELMO have shown a significant decrease in both the protective and curative group of body weight, tumour volume, and abdominal circumference compared to the Ehrlich's ascites carcinoma tumour group. The levels of lipid peroxidation, GSH, and superoxide dismutase also altered. **Discussion and Conclusion:** The present study demonstrates that the ethanolic leaves extract of *Moringa oleifera* increased the life span of Ehrlich's ascites carcinoma tumour-bearing mice and decreased lipid peroxidation. These parameters suggest that the ethanolic leaves extract of *Moringa oleifera* exhibits potential antioxidant and antitumor effects.

INTRODUCTION: Free radicals are fundamental to any biochemical process and contain a pivotal part of life processes. Production of reactive oxygen species such as hydroxyl radical (OH), superoxide (O2−.), hydrogen peroxide (H_2O_2), and singlet oxygen (1O2) is associated with several degenerative diseases such as diabetes, cancer, cardiovascular diseases, Alzheimer's disease, and inflammation [1, 2], alteration in the balance between production of free radicals and the scavenging activities of antioxidants causes oxidative stress [3, 4].

QUICK RESPONSE CODE

DOI: 10.13040/IJPSR.0975-8232.15(10).2991-98

This article can be accessed online on www.ijpsr.com

DOI link: https://doi.org/10.13040/IJPSR.0975-8232.15(10).2991-98

Antioxidants are well known for their role in deactivating free radicals that have damaging roles in biological cells [5]. Polyphenolic compounds are the most prevalent antioxidants seen in plants and they are mainly categorized into flavonoids, phenolic acids, stilbenes, and lignans [6]. Evidences from epidemiologic studies have revealed that, consumption of leafy vegetables was associated with reduced risk of diseases due to their antioxidant properties.

Moringa oleifera is widely cultivated in Southeast Asia, mainly in India, Thailand, and the Philippines [7]. The compounds of *Moringa oleifera*, in addition to their essential medicinal properties, act as effective antioxidants and inhibitors of bacterial and fungal growth. *Moringa oleifera* is rich in phenolic compounds to inhibit oxidation in food [8, 9]

considerably. There are 11 phenolic acids are present in the leaves of *Moringa oleifera* (gallic acid, caffeic acid, chlorogenic acid, o-coumaric acid, p-coumaric acid, ellagic acid, gentisic acid, sinapic acid and syringic acid) [10, 11], flavonoids (primarily flavonol and glycoside: quercetin, rhamnetin, campferol, apigenin, and myricetin), and their derivatives (p-coumaroylquinic acids and their isomers, feruloylquinic, and caffeoylquinic) [12]. The leaves of *Moringa oleifera* traditionally have various biological activities, including hypocholesterolemic agent [13], regulation of thyroid hormone status [14] and antidiabetic [15], gastric ulcers [16], antitumor [17], and hypotensive agent [18]. Antioxidant properties of any natural compound helps to maintain health when continuously taken in dietary foods, spices, or drugs [19].

Ehrlich's ascites carcinoma originally appeared as spontaneous breast cancer in female mice and used as a subcutaneously transplantable tumour in the mouse system [20]. Ehrlich's ascites carcinoma is an undifferentiated carcinoma, which is hyper diploid in nature and it has the characteristics of rapid proliferation, high transplantation, short life span, and 100% malignancy with no tumour-specific transplantation antigen [21]. The resemblance of Ehrlich's ascites carcinoma and human tumours are more sensitive to chemotherapy can be used as an efficient model in *in-vivo* anticancer studies. The present study shows the antioxidant activity of ethanolic leaf extracts of *Moringa oleifera* Lam. (ELMO) against Ehrlich ascites carcinoma (EAC) in albino mice.

MATERIAL AND METHODS:

Plant Material: The leaves of Moringa oleifera Lam collected in the month of January 2019 from Anantapuramu district, Andhra Pradesh, India. The plant was identified and accession number (134/A) was confirmed by a taxonomist from SK university anantapuramu. The leaves were dried under the shade and powdered with a mechanical grinder and stored in an air-tight container.

Preparation of the Extract: 150g of *Moringa oleifera* leaves powder was soaked in 90% ethanol and 10% distilled water for 24 hours in a percolator, after 24 hours it was allowed to percolate, and the extract was collected in Petri dishes. The extract was concentrated in a vacuum using a rotary flash evaporator (40°C) and there was a net yield of 23.00 g of the concentrated extract (17.80 w/w %).

Animals used: Swiss albino mice, weighing 20-25g, male, were procured from the animal house of the Basaveshwara Medical College and Hospital, Chitradurga, Karnataka, India. The number of animals used in the present study was 116 and All the animals kept in standard polypropylene cages under standard conditions: temperature (24±10C), relative humidity (40-45%), and a 12:12 light: dark cycle. All the animals were fed with standard rodent diet (Amruth Rat Feed, manufactured and supplied by Pranav Agro Industries, Pune, India), and water ad libitum. The animals were allowed to acclimatize to laboratory conditions 48h before we start the experiment, and the experimental protocol was duly approved by the institutional animal ethical committee (Reg. no.1284/ac/09/CPCSEA).

Transplantation of Tumours: The Ehrlich's ascites carcinoma cells obtained from the Amala Cancer Research Institute, Thrissur, and Kerala, India and were maintained in vivo in male Swiss albino mice by intraperitoneal (i.p.) transplantation of 3 × 106 cells per mouse every 10^{th} day. Ascitic fluid was drawn from Ehrlich ascites carcinoma tumour-bearing mice around 7 to 8 days of tumour-bearing cells, and animals received 0.2 ml of tumour cell suspension containing 3×106 tumour cells intraperitoneally. Ehrlich ascites carcinoma cells were collected from male Swiss donor albino mice of 20-25 g body weight and suspended in sterile saline (0.9% NaCl, Almotaheda pharma) and fixed number of viable cells (usually 3× 106 cells/mice) were implanted in the peritoneal cavity of each recipient mouse [22]. 0.5 ml of Ehrlich ascites carcinoma was withdrawn by a sterile disposable syringe, diluted with 4.5 ml of normal saline (0.9%Nacl), 0.2 ml of diluted EAC was injected i.p. in each recipient mouse then tumour cells were allowed to multiply within the peritoneal cavity.

Antioxidant Activity in Ehrlich Ascites Carcinoma Model: In this study total of 24 male albino mice were taken, divided into four groups, each group contains 6 animals (n=6).

Group I: Normal Control (Non-EAC bearing mice). This group consists of 6 Swiss albino mice;

all mice received 5.0 ml of normal saline/kg body weight orally by gastric intubation daily for 15 days.

Group II: Ehrlich ascites carcinoma Control (EAC-bearing mice). This group consists of 6 Swiss albino mice induced with Ehrlich ascites carcinoma. About 3x 106 EAC tumour cells were injected intraperitoneally into healthy mice. All the mice received daily 5.0 ml of normal saline/kg body weight orally by gastric intubation for 15 days.

Group III: Protective group (ELMO treated-EAC bearing mice). This group consists of 6 Swiss albino mice; each animal-treated ELMO (500 mg / 5ml / kg body wt.) was mixed in 5.0ml of a warm aqueous solution, given orally into gastric intubation once a day for 4 days and on the 5th day 3x106 Ehrlich ascites carcinoma tumour cells were injected intraperitoneally. Again ELMO (500, mg / 5ml /kg body wt.) was given orally to each animal for 11 days.

Group IV: Curative group (EAC bearing- ELMO treated mice). This group consists of 6 Swiss healthy albino male mice were injected about 3x 106 EAC tumour cells intraperitoneally and have received 5.0 ml of normal saline/kg body weight orally by gastric intubation daily for 4 days. 5th day onwards, 5.0 ml warm aqueous solution of ELMO (500 mg /kg body weight) was given orally for 15 days.

Body Weight: The body weights of experimental mice measured both in the treated and control group at the beginning of the experiment (day 0) and sequentially on every 5th day during the treatment period.

Tumour Volume: All the Animals were dissected and ascitic fluid was collected from the peritoneal cavity, volume of the fluid was measured by taking it in a graduated centrifuge tube, and packed cell volume was determined by centrifuging at 1000 rpm for 5 min.

Estimation of Antioxidant: On the 15th day, body weights and abdominal circumferences were recorded in all the control protective and curative groups, and then the mice were anesthetized and sacrificed. The ascitic fluid was collected immediately in clean dry graduated tubes by puncturing the abdomen. The volumes of fluid were noted and blood samples from each mouse collected from the eyes by Sino-orbital puncture of mice using micro-capillary tubes [23]. The blood samples were withdrawn in clean, dry test tubes containing ethylene diamine tetra acetic acid (EDTA) and centrifuged at 3000 rpm for 15 minutes. The supernatant plasma was separated and stored deep-frozen at -20°C until assayed and the remnants (the packed RBCs) were stored in a deep freezer at -20°C until assayed. The mice were dissected, and liver tissues removed directly, bloodstains of liver tissues were removed by smooth blotting using blotting paper and immediately transferred into a clean pre-weighed beaker. Then one part of the liver samples homogenized in 0.9% NaCl (1g of the liver tissue added to 10ml of 0.9% NaCl i.e., 1:10 W/V) using Teflon mortar and the homogenates was stored at –20°C until assayed.

Estimation of Lipid Peroxidation (LPO): The levels of thiobarbituric acid reactive substances (TBARS) in the liver and plasma were measured by the method of Yoshioka *et al.* [24] as a marker for lipid peroxidation. Blood plasma in the case of liver: 10% (w/v), i.e. 1 g of liver tissue was homogenized in 10 ml saline 0.9% NaCl. In a 10 ml centrifuge tube, 0.5 ml of the sample was mixed with 2.5 ml of Trichloroacetic acid (TCA), and 1 ml of TBA added to the solution, then incubation was done for 30 minutes in a boiling water bath, followed by rapid cooling.

The standard solution was prepared by adding 1.9ml of Trichloroacetic acid, and 1ml of TBA. The blank was prepared by mixing 2 ml of Trichloroacetic acid with 1 ml of TBA. 4ml of n-butyl alcohol added to all the tubes, and after shaking, tubes were centrifuged at 3000 rpm for 10 minutes, the n-butanol layer transferred to the cuvette and the optical density (OD) measured at 532nm.

Estimation of Reduced GSH: The liver tissue and plasma Glutathione were determined by the method of Beutler and Kelly [25], virtually all the non-protein sulfhydryl groups of tissues are in the form of reduced Glutathione and 0.2 ml of tissue homogenate was mixed with 1.8 mL of EDTA

solution, 3.0 ml precipitating reagent (after precipitating proteins with TCA) was added to this solution, mixed thoroughly, and kept for 5 min before centrifugation. 4.0 ml of 0.3-M disodium hydrogen phosphate solution and 1.0 ml of DTNB (5, 5-dithiol bis 2-nitro benzoic acid) reagent were added to 2.0 ml of the filtrate, and the absorbance read at 412nm.

Assay of Superoxide Dismutase: The activity of Superoxide dismutase in liver tissue and plasma was assayed by the method of Minami [26], the assay mixture contained blood (packed RBC's), and liver tissue homogenate was 10 % w/v, 0.9 % NaCl using Teflon mortar homogenizer, ethanol, and chloroform (2:1 v/v) for removal of hemoglobin, 20 g. of cacodylic sodium buffer and five ml of diethylene diamine Penta acetic acid (DPTA) was dissolved in 2L distilled water. The pH of the buffer was adjusted to 8.2 by adding Tris hydroxyl methylene amino methane using a pH meter and the volume was completed to 100 ml distilled water: Then, 0.0801gm of Nitro –blue tetrazolium dissolved in distilled water and the volume was adjusted to 100 ml, 16 ml Triton x-100 was added to 100 ml of distilled water, 0.011 gm Pyrogallol was dissolved in 10 ml 1 N HCl and being used as a stock solution .1 ml of the stock solution was completed to 10 ml with distil water for being used in the assay. Stopper solution of 16 ml Triton x-100 was added to 8.2 ml of formic acid and the volume was completed to 100 ml by distilling water.

RESULTS: Antitumor activity of ELMO against EAC tumour-bearing mice was assessed by the parameters such as body weight, abdominal circumference, and tumour volume and the results are shown in **Fig. 1**. The tumour volume and body weight were significantly ($p < 0.01$) increased in EAC control animals when compared with normal control animals.

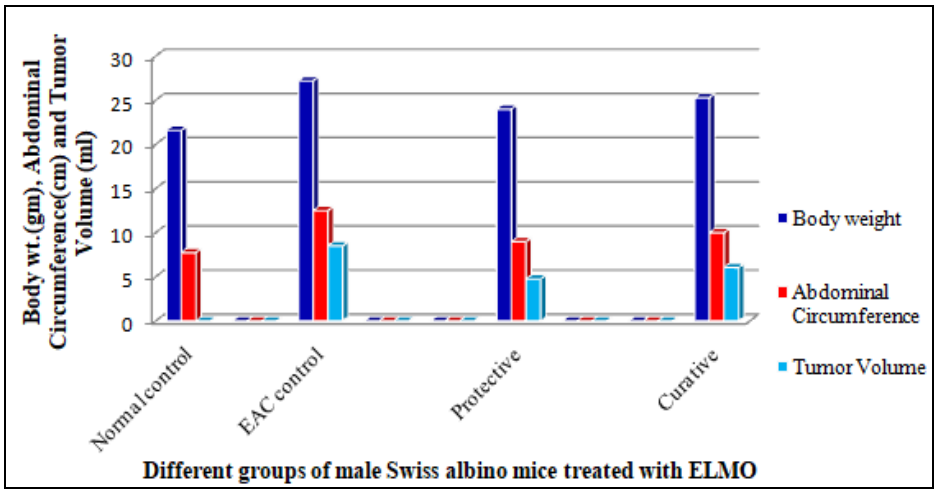

FIG. 1: BODYWEIGHT, ABDOMINAL CIRCUMFERENCE, AND TUMOUR VOLUME OF DIFFERENT GROUPS OF EAC-BEARING MICE. Administration of ELMO at the dose of 500 mg/kg body wt. significantly ($p < 0.05$) decreased body weight, abdominal circumference, and tumour volume.

TABLE 1: ANTIOXIDANT EFFECT OF *MORINGA OLEIFERA* ON BLOOD AND LIVER TISSUE IN EAC-BEARING MICE

Group	TBARS (mM/L)		GSH (mg/ dl)		SOD (µg/ ml)	
	Plasma	Liver	Plasma	Liver	Plasma	Liver
Normal control	19.84 ± 1.89	166.84 ± 5.28	2.73 ± 0.22	10.72 ± 0.71	7.45 ± 0.42	3.81 ± 0.29
EAC control	33.84 ± 1.67	190.33 ± 3.15	1.02 ± 0.22*	8.49 ± 0. 54*	2.56 ± 0.30	1.23 ± 0.08
Protective	24.27 ± 3.80*	170.99 ± 5.79**	2.68 ± 0.07***	9.82 ± 0.28***	4.66 ± 0.28*	3.55 ± 0.51***
Curative	30.08 ± 2.65*	173.83± 6.63*	2.49 ± 0.89***	9.62 ± 0.26**	4.79 ± 0.84*	3.13 ± 0.09***

Data was expressed as Mean ± S.E.M. (n = 6) animals in each group, *p<0.01, **p<0.001, ***p<0.0001 vs EAC control and Normal control.

Antitumor Effect of ELMO on TBARS Levels: ROS formed in cancer tissues results in lipid peroxidation and subsequently to increase in Malondialdehyde (MDA) level. **Fig. 2** depicts the levels of TBARS in the plasma and liver tissue of experimental animals. In the present study, the

levels of Malondialdehyde were significantly ($p < 0.01$) increased in Ehrlich's ascites carcinoma control animals compared to normal control animals. After treatment with ELMO at 500 mg/kg body weight the protective and control groups showed significantly ($p < 0.001$) reduced Malondialdehyde levels compared to Ehrlich's ascites carcinoma control animals, and results are expressed as mmol/L.

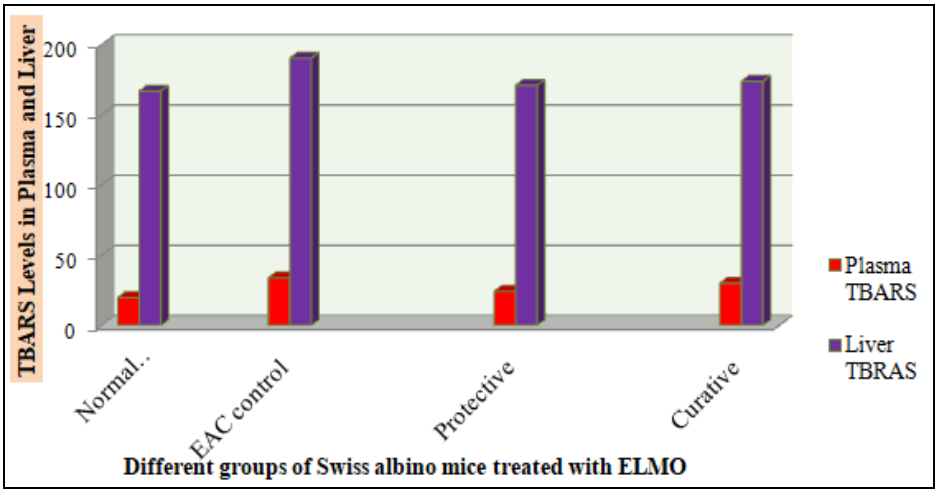

FIG. 2: PLASMA AND LIVER THIOBARBITURIC ACID REACTIVE SUBSTANCES (TBARS) OF NORMAL AND DIFFERENT GROUPS OF EAC-BEARING MICE

Antitumor Effect of ELMO on GSH Levels: Fig. 3 depicts the levels of glutathione in the plasma and liver tissue of experimental animals. In the present study the levels of glutathione was significantly ($p < 0.01$) reduced in Ehrlich's ascites carcinoma control animals compared to normal control animals. After treatment with ELMO at 500 mg/kg body weight of the protective and control groups showed significantly ($p < 0.0001$) increased levels of glutathione compared to Ehrlich's ascites carcinoma control animals, and the results were expressed as mg/dl.

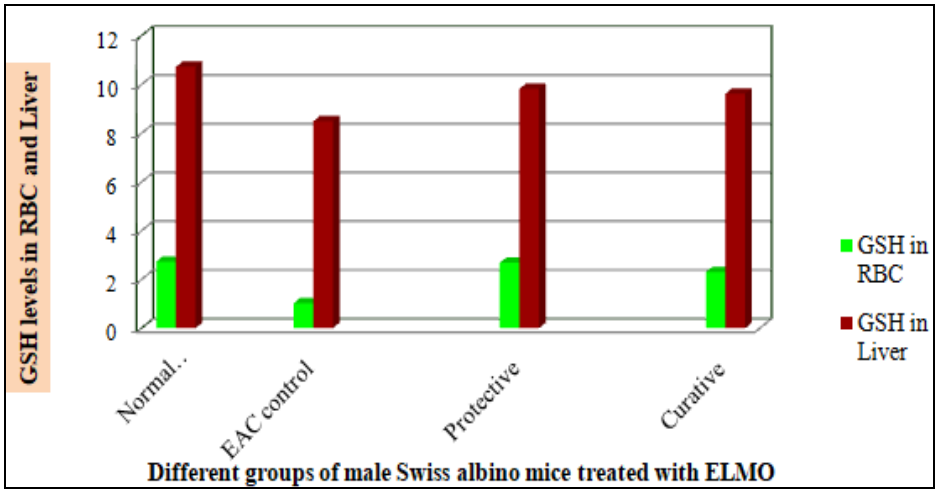

FIG. 3: GLUTATHIONE (GSH) IN PLASMA AND LIVER TISSUE OF NORMAL AND DIFFERENT GROUPS OF EAC-BEARING MICE

Antitumor Effect of ELMO on SOD Levels: Fig. 4 depicts the levels of Superoxide dismutase in the plasma and liver tissue of experimental animals. In the present study, the levels of SOD were significantly ($p < 0.01$) reduced in Ehrlich's ascites carcinoma control animals compared to normal control animals. After treatment with ELMO at 500 mg/kg body weight the protective and control group both showed significantly ($p < 0.0001$) increased Superoxide dismutase levels compared to Ehrlich's ascites carcinoma control animals and the results were expressed as mg/dl.

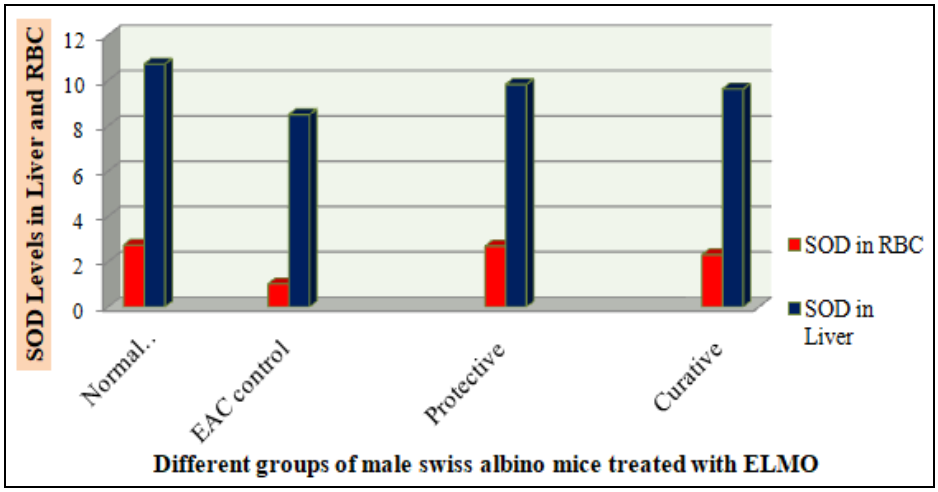

FIG. 4: SUPEROXIDE DISMUTASE (SOD) IN PLASMA AND LIVER TISSUE OF NORMAL AND DIFFERENT GROUPS OF EAC-BEARING MICE

DISCUSSION: The present study in EAC tumour-bearing mice carried out to evaluate the antitumor activity and antioxidant status of ethanolic leaf extract of *Moringa oleifera* (ELMO). We observed in Ehrlich's ascites carcinoma tumour-bearing mice a rapid increase in ascitic tumour volume, and ascitic fluid is the direct nutritional source for tumour cells, and a rapid increase in ascitic fluid with tumour growth would be a means to meet the nutritional requirement of tumour cells [27].

Animals treated with ELMO inhibit the tumour volume, abdominal circumference, body weight, and viable tumour cells, and increased the life span of the tumour-bearing mice. The reliable criteria for judging the value of any anticancer drug are the prolongation of the life span of animals [28].

It was concluded that ELMO by decreasing the nutritional fluid volume, arrests the tumour growth of Ehrlich's ascites carcinoma -bearing mice. Thus, ELMO has antitumor activity against Ehrlich's ascites carcinoma bearing mice. In mice bearing Ehrlich's ascites carcinoma tumour showed rapid increase in ascitic tumour volume. Ascitic fluid is the direct nutritional source for tumour cells, and a rapid increase in ascitic fluid with tumour growth would be a means to meet the nutritional requirement of tumour cells [27].

Malondialdehyde (MDA) is formed during oxidative degeneration as a product of free oxygen radicals [29], and was accepted as an indicator of lipid peroxidation [30]. Malondialdehyde, the end product of lipid peroxidation, was reported to be higher in cancer tissues than in non-diseased organs [31]. The findings of the present study indicate that, TBARS levels in the tested cancerous tissues are higher than those in normal tissues. The results of the present study are in agreement with the published data [32, 33], and emphasizes the reduction in free radical yield and the subsequent decrease in harm and damage to the cell membrane and a decrease in Malondialdehyde production.

Glutathione, a potent inhibitor of the neoplastic process, plays a vital role in the endogenous antioxidant system. It is found in exceptionally high concentrations in the liver and is known to have a crucial function in the protective process. Excessive production of free radicals results in oxidative stress, which damages the macromolecules in *in-vivo* lipid peroxidation [34].

It was also reported that the presence of tumours in the human body or in experimental animals affects many functions of the vital organs, especially the liver, even the tumour site does not interfere directly with organ function [35].

In our study, GSH levels in experimental mice were significantly lower than the Ehrlich's ascites carcinoma control mice. Superoxide dismutase catalyzes the diminution of superoxide into H_2O_2, which should eliminate by glutathione Peroxidase and, or catalase [36]. Further, it was reported that, a decrease in Superoxide dismutase activity in Ehrlich's ascites carcinoma bearing mice may be due to the loss of Mn2+-containing Superoxide dismutase activity in EAC cells, and the loss of mitochondria, leading to a decrease in total Superoxide dismutase activity in Liver and plasma,

similar findings were observed in our present study in Ehrlich's ascites carcinoma bearing mice. The administration of ELMO showed a significantly increased Superoxide dismutase a and was reported that, plant-derived extracts containing antioxidant principles showed cytotoxicity toward tumour cells [37] and antitumor activity in experimental animals [38]. Antitumor activity of these antioxidants is either through induction of apoptosis [39] or by inhibition of neovascularisation [40]. The implication of free radicals in tumours is well documented [41]. The free radical hypothesis supported the fact that the antioxidant effectively inhibits the tumour, and the observed properties attributed to the antioxidant and antitumor principles present in the plant extract.

CONCLUSION: In conclusion, the present study demonstrates that the ethanolic leaves extract of *Moringa oleifera* (ELMO) increases the life span of Ehrlich's ascites carcinoma tumour-bearing mice and decreases the lipid peroxidation and augmenting the endogenous antioxidant enzymes in the liver and plasma. These parameters suggest that the ethanolic leaves extract of *Moringa oleifera* (ELMO) exhibits potential antioxidant and antitumor effects.

ACKNOWLEDGMENT: The authors are grateful to Malwanchal University and Basaveshwara Medical College and Hospital for giving the opportunity to conduct this study.

CONFLICT OF INTEREST: The authors declare no conflict of interest.

REFERENCES:

1. Ranjith US, Roopitha B and Jacob CM: Isolation anti-diabetic and antioxidant evaluation of aqueous extract of *Cansjera rheedei* leaves. Asian J Pharma Clin Res 2013; 6: 228-34.
2. Roberts RA, Laskin DL, Smith CV, Robertson FM, Allen EMG and Doorn JA: Nitrative and oxidative stress in toxicology and disease. Toxicological Sciences. Toxicol Sci 2009; 112: 4-16.
3. Adesanoye OA, Adekunle AE, Adewale OB, Mbagwu AE, Delima AA and Adefegha SA: Chemoprotective effect of *Vernonia amygdalina* Del. (Asteraceae) against 2-acetylaminofluorene-induced hepatotoxicity in rats. Toxicol Ind Health 2016; 32: 47–58.
4. Adeyanju A, Molehin OR, Ige E, Adeleye L and Omoniyi O: Sildenafil, a phosphodiesterase-5- inhibitor decreased the oxidative stress induced by carbon tetrachloride in the kidney of rats: a preliminary study. Journal of Applied Pharmaceutical Science 2018; 8: 106–111.
5. Sreeramulu D, Reddy CV, Chauhan A, Balakrishna N and Raghunath M: Natural antioxidant activity of commonly consumed plant foods in India: effect of domestic processing, Oxid Med Cell Longev 2013; 1-12.
6. Pandey KB and Rizvi SI: Plant polyphenols as dietary antioxidants in human health and disease. Oxid Med Cell Longev 2009; 2: 270–278
7. Sebranek JG, Sewalt VG, Robbins, KL and Houser TA: Comparison of a natural rosemary extract and BHA/BHT for relative antioxidant effectiveness in pork sausage. Meat Sci 2005; 69: 289–296.
8. Keokamnerd T, Acton J, Ha, I and Dawson P: Effect of commercial rosemary oleoresin preparations on ground chicken thigh meat quality packaged in a high-oxygen atmosphere. Poult Sci 2008; 87: 170–179.
9. Sabale V, Patel V, Paranjape A, Arya C, Sakarkar SN and Sabale PM: *Moringa oleifera* (Drumstick): An overview. Pharmacogn Rev 2008; 2: 7–13.
10. Prakash D, Suri S, Upadhyay G and Singh BN: Total phenol, antioxidant and free radical scavenging activities of some medicinal plants. IJFSN 2007; 58: 18–28.
11. Singh BN, Singh R, Prakash D, Dhakarey R, Upadhyay G and Singh H: Oxidative DNA damage protective activity, antioxidant and anti-quorum sensing potentials of *Moringa oleifera*. Food Chem Toxicol 2009; 47: 1109–1116.
12. Rani NZA, Husain K and Kumolosasi E: Moringa genus: a review of phytochemistry and pharmacology. Front Pharmacol 2018; 9: 108.
13. Mehta K, Balaraman R, Amin AH, Bafna PA and Gulati OD: J Ethnopharmacol 2003; 86: 191-195.
14. Tahiliani P: A Kar Role of *Moringa oleifera* leaf extract in the regulation of thyroid hormone status in adult male and female rats. Pharmacological Research 2000; 41: 319-323.
15. Makonnen E, Hunde A and Damecha G: Hypoglycaemic effect of *Moringa stenopetala* aqueous extract in rabbits. Phytotherapy Research 1997; 11: 147-148.
16. Pal SK, PK Mukherjee and Saha BP: Studies on the antiulcer activity of *Moringa oleifera* leaf extract on gastric ulcer models in rats. Phytoth Res 1995; 9: 463-65.
17. Bharali R, J Tabassum and MRH Azad: Chemomodulatory effect of *Moringa oleifera*, Lam, on hepatic carcinogen metabolizing enzymes, antioxidant parameters, and skin papilloma genesis in mice. Asian Pacific Journal of Cancer Prevention 2003; 4: 131-139.
18. Faizi S, Siddiqui BS, Saleem R, Siddiqui S, Aftab K, Gilani AH: Fully acetylated carbamate and hypotensive thiocarbamate glycosides from *Moringa oleifera*. Phytochemistry 1995; 38: 957-963.
19. Singh RP, Padmanathi B and Rao AR: Modulatory influence of *Adhatoda vesica* (Justicia adhatoda) leaf extract on the enzymes of xenobiotic metabolism antioxidant status and lipid peroxidation in mice. Molecular and Cell Biochemistry 2000; 213: 99-109.
20. Olinescu A: Ehrlich ascitic tumor; experimental model. Biology of the laboratory animals and comparative oncology. Oncology Institute; Cluj Napoca 1992; 19.
21. Kaleoğlu O and İşli N: Ehrlich-Lettre Ascites Tumor. Tıp Fakültesi Mecm 1977; 40: 978-84.
22. Salem FS and Badr MO: Neamat – Allah, A. N. Biochemical and pathological studies on the effects of levamisole and chlorambucil on Ehrlich ascites carcinoma bearing mice. Vet Italiana 2011; 47: 89-95.
23. Riley V: Adaptation of orbital bleeding technique to rapid serial blood studies. Proc Exp Biol Med 1960; 104: 751 – 754.
24. Yoshioka T, Kawada K, Shimada T and Mori M: Lipid peroxidation in maternal and cord blood and protective

mechanism against activated oxygen toxicity in the blood. Am J Obstet Gynecol 1979; 135: 372–376.
25. Beutler E and Kelly BM: The effect of sodium nitrate on red cell glutathione. Experientia 1963; 18: 96-97.
26. Minami M and Yoshikawa H: A simplified assay method of superoxide dismutase activity for clinical use. Clin Chim Acta 1979; 92: 337–342.
27. Prasad SB and Giri A: Antitumor effect of cisplatin against murine ascites Dalton's lymphoma. Indian J Exp Biol 1994; 32: 155-1562.
28. Clarkson BD and Burchenal JH: Preliminary screening of antineoplastic drugs. Prog Clin Cancer 1965; 1: 625-629.
29. Valenzuela A: The biological significance of determination in the assessment of tissue oxidative stress. Life Sci 1990; 48: 301.
30. Neilsen F, Mikkelsen BB, Neilsen JB, Andersen HR and Grandjean P: Plasma malondialdehyde as biomarker for oxidative stress reference interval and effects of lifestyle factors. Clin Chemist 1997; 47: 1209-1214.
31. Yagi K: Lipid peroxides and human diseases. Chem Phys Lip-ids 1987; 45: 337–351.
32. Louw DF, Bose R and Sima A: A Neurosurgery 1997; 41: 1146-1150.
33. De Cavanagh EM, Honegger AE, Hofer E, Bordenave RH, Bul-lorsky EO, Chasseing NA and Fraga C: Cancer 2002; 94: 3247-3251.
34. Sinclair AJ, Barnett AH and Lunie J: Free radical and auto-oxidant systems in health and disease Br. J Hosp Med 1990; 43: 334-344.
35. DeWys WD: Pathophysiology of cancer cachexia: current understanding and areas for future research. Cancer Res 1982; 42: 721-726.
36. Rushmore TH and Packet CB: Glutathione–s-transferase, structure regulation, and therapeutic implication. J Biol Chem 1993; 268: 11475-11478.
37. Jiau-Jian L and Larry WO: Overexpression of manganese-containing superoxide dismutase confers resistance to the cytotoxicity of tumor necrosis factor and/or hyperthermia. Cancer Res 1977; 57: 1991-1998.
38. Ruby AJ, Kuttan G, Babu KD, Rajasekaran KN and Kuttan R: Anti-tumor and antioxidant activity of natural curcuminoids. Cancer Lett 1995; 94: 783-789.
39. Ming L, Jill CP, Jingfang JN, Edward C and Brash E: Antioxidant action *via* p53 mediated apoptosis. Cancer Res 1998; 58: 1723-1729.
40. Putul M, Sunit CP and Ritha B: Neovascularisation offers a new perspective to glutamine-related therapy. Ind J Exp Biol 2000; 38: 88-90.
41. Ravid A and Korean R: The role of reactive oxygen species in the anticancer activity of vitamin D. Anticancer Res 2003; 164: 357-367.

How to cite this article:
Ramanath B and Kumar RRV: Antioxidant and antitumor effects of *Moringa oleifera* lam against Ehrlich's ascites carcinoma bearing mice. Int J Pharm Sci & Res 2024; 15(10): 2991-98. doi: 10.13040/IJPSR.0975-8232.15(10).2991-98.