

Comparative Effects of *Purslane* seed oil (PSO) and 5-Flourourasil on Ehrlich ascites carcinoma (EAC) in female albino mice

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ABSTRACT

The present study was to evaluate anti-cancer effects of *Purslane* seed oil (PSO) (200g/kg. diet), 5-Flourourasil (20mg/kg b.w. i.p) and their combinatorial formulation in female mice induced by Ehrlich ascites cells for 21 consecutive days prior. Ten days after intraperitoneal inoculation of tumor EAC cells in mice, PSO was supplemented at (200g/kg. diet) daily for 21 consecutive days. On the 22th day, the mice were sacrificed for the estimation of tumor growth (tumor volume), and biochemical parameters (glucose, insulin, alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), lipid peroxides (TBARS), protein thiols (Pr-SHs), reduced glutathione (GSH), superoxide dismutase (SOD), glutathione peroxidase (GPx), total cholesterol (TC), triglycerides (TG), HDL-C, LDL-C, 17 β -estradiol and progesterone). The results of this study also showed that administration of *Purslane* seed oil (PSO), 5-Flourourasil both individually and in combination for 21 days to the carcinoma induced mice demonstrated a significant ($P<0.01$) decrease in tumor volume and a significant ($P<0.01$) improvement in biochemical parameters and life span as compared to the EAC control mice than either agent alone. On the other hand, the results clearly suggest that the combination of PSO and 5-Flourourasil produced higher antioxidant activities on experimental EAC control as well as 5-Flourourasil mice than their individual influences.

Keywords: *Purslane* seed oil (PSO), 5-Flourourasil, breast cancer, Ehrlich ascites cells and antioxidants

INTRODUCTION

Cancer is an unnatural cell growth, where they can lose their natural function and spread through the blood, at all the body. Breast cancer is the more commonly diagnosed in industrialized countries and has the highest death toll [1]. Oxidative stress is involved in the process development of cancer and tumors; due to that ROS can damage the macromolecules as lipids which react with metals (as free iron and copper) and produce aldehydes and synthesize malondialdehyde inducing mutations [2] or cause breaks in the double chain, produce modifications in guanine and thymine bases, and sister chromatid exchanges [3]. Humans have evolved with antioxidant systems to protect against free radicals and ROS. These systems include some antioxidants produced in the body (endogenous) and others obtained from the diet (exogenous) [4]. The first include (a) enzymatic defenses, such as glutathione

peroxidase, catalase, and superoxide dismutase, which metabolize superoxide, hydrogen peroxide, and lipid peroxides, thus preventing most of the formation of the toxic ROS [2]. Plants vegetables and spices used in folk and traditional medicine have gained wide acceptance as one of the main sources of prophylactic and chemopreventive drug discovery and development [5, 6]. It is widely accepted that a diet rich in fruits and plants are rich sources of different kinds of antioxidants, phenolic compounds are the most studied and have been recognized to possess a wide range of properties including antioxidant, antibacterial, anti-inflammatory, hepatoprotective and anticarcinogenic actions [5]. Many of the biological functions of flavonoids, phenolic, catechins, curcumin, resveratrol and genistein compounds have been attributed to their free radical scavenging, metal ion chelating and antioxidant activities [6, 7]. Several medicinal plants have been implicated in the mechanisms of

chemoprevention which refers to the use chemical substances of natural origin or synthetic to reverse, retard or delay the multistage carcinogenesis process [6]. One of such plants, purslane, in Arabic '*Rejlah*, (*Portulaca oleraceae* L.) occurs in the Arabian has been examined in some animal studies and clinical studies for its hepatoprotective [8] and hypolipidemic effects [9]. *Purslane* is reported to be rich in α -linolenic acid and β -carotene and used as a health food for patients with cardiovascular diseases [9]. It contains several types of vitamins and minerals [10], fatty acids [11], glutathione, glutamic acid, aspartic acid dopamine, dopa, coumarins, flavonoids, alkaloids, saponins, and anthocyanin [12].

Plant-derived oils are rich sources of volatile terpenoids and phenolic compounds [13]. The essential oil and lipid-soluble compounds are known to have potential to prevent obesity and have been used in aromatherapy for obese middle-aged women. Some volatile compounds extracted from plants may have antioxidant activity that could mitigate obesity-related complications, including atherosclerosis and some cancers [13–16]. Not surprisingly, plants such as purslane contain high levels of unsaturated fatty acids and poly-phenols [9, 13], which are excellent scavengers of reactive and represent a promising anti-tumor effects. In vivo tests have been conducted with purslane leaves to determine for example, its hepatoprotective [8], hypolipidemic, hypoglycemic and antioxidant activity [7]. But there are no reports about antitumor of *purslane* seed oil. The present study aimed to evaluate the possible antitumor effect *Purslane* seed oil (PSO) and 5-Fluorouracil in the form of combinatorial formulation against Ehrlich ascites carcinoma (EAC) in female albino mice.

MATERIALS AND METHODS

Plant material

Plant materials of purslane were collected from Horbit Village, El-Sharkyea Governorate, Egypt. The plant material was identified, authenticated taxonomically by Dr. Heba El-Gezawy, Pharmacognosy department, faculty of Pharmacy, October 6 University. Voucher specimens were kept in the department of Pharmacognosy, Faculty of Pharmacy, October 6 University. The seed were cleaned, dried under direct sunlight and powdered by a mechanical grinder.

Extraction of Fixed oil

After being cleaned by hand carefully to remove the foreign materials such as other seeds, stones and small stalks, purslane seed were dried at 50°C for 12h in an oven, and then crushed into powder in a grinder with a size range of 0.55-1.0mm. The resulted powder was kept in a vacuum dryer until use. Purslane ground samples were mixed with hexane (1:10, *m/V*) at (60-80°C) using a Soxhlet apparatus. This process of extraction was repeated for 6h, the hexane distilled out by distillation assembly, then concentrated by hot plate drying and air-drying at temperature of 40±2 °C.

Mice

This experiment was conducted in accordance with guidelines established by the Animal Care and use Committee of October 6 University. Adult mice weighing around 25 ± 2gms were purchased from Faculty of Veterinary Medicine, Cairo University. They were individually housed in cages in an air-conditioned room with a temperature of 22 ± 2°C, a relative humidity of 60%, and an 8:00 to 20:00 light cycle. During the acclimatization period, each animal was raised on a regular diet *adlibitum*.

Chemicals

5-fluorouracil was from Merck Ltd., Germany. All the other reagents used were of analytical grade and were obtained commercially.

Experimental design

EAC cells were obtained from Cancer institution, Cairo. The cells maintained in vivo in Swiss albino mice by intraperitoneal transplantation and later tumor cells were injected intraperitoneally (2x10⁶ cells per mouse) to animals of all groups except the first group [17].

The animals were divided into 5 groups consisting of 8 animals, two controls groups and three treatment groups:

Group (1): Control (normal diet).

Group (2): EAC control (tumor bearing mice (TB)) (supplemented normal diet).

Group (3): EAC (tumor bearing mice (TB)) + (normal diet/ *purslane* seed oil (200g/kg. diet) daily for 3 weeks after subcutaneous implantation of EAC.

Group (4): EAC (tumor bearing mice (TB)) + 5-fluorouracil (20mg/kg) was given by intraperitoneal injection on alternate days for 3 weeks after subcutaneous implantation of EAC.

Group (5): EAC (tumor bearing mice (TB)) + normal diet/*purslane* seed oil (PSO) (200g/kg. diet) + 5-fluorouracil (20mg/kg, i.p.) was injected on alternate days for 3 weeks after subcutaneous implantation of EAC.

Groups 1 and 2 ingested a normal diet free from PSO. Also, supplemented diets for groups 3 and 5 were prepared by combining normal meal (800g) with 200g of *purslane* seed oil in a mechanical mixer [18]. Diets were stored in airtight containers at 4°C in a refrigerator. Peroxide content of the diets did not change during the storage period [19].

Effect of PSO, 5-fluorouracil and PSO, 5-fluorouracil in combination on tumor volume and weight

On 31th day, after 24h of dose, 8 mice from each group were dissected and the ascites fluid was collected from peritoneal cavity. The volume was measured by taking it in a graduated centrifuge tube. The tumor weight was measured by taking the weight of mice before and after collection of ascites fluid from peritoneal cavity [20, 21].

At the end of the study, all mice were sacrificed blood was collected, centrifuged, and plasma was used freshly for estimation of plasma glucose [22]. The plasma insulin, progesterone and 17β -estradiol concentration were measured using the insulin ELISA kit (Shibayagi Co. Japan) [23-25], respectively), as well as transaminases (L-alanine and L-aspartate) [26], alkaline phosphatase (ALP) [27]. Also, lactate dehydrogenase (LDH) [28], TBARS, Pr-SHs and GSH levels in blood and hepatic were done by the methods described by Buhl and Jackson [29], Uchiyama and Mihara [30], Koster, et al., [31] and Chanarin [32], respectively. Blood and liver Superoxide dismutase (SOD) and glutathione peroxidase (GPx) activities were carried out Paglia and Valentine [33], Marklund and Marklund [34], respectively. Plasma triglyceride, total cholesterol and HDL- cholesterol were determined using commercially available kits (Asan and Youngdong Pharmaceutical Co., Korea) [35-37]. Plasma LDL-cholesterol level was calculated from Falholt et al [38] formula (LDL-cholesterol = total cholesterol - triglycerides/5 - HDL-cholesterol).

Statistical analysis

All the grouped data were statistically evaluated with SPSS/11 software. Hypothesis testing methods included one way analysis of variance (ANOVA) followed by least significant difference (LSD) test. *P* values of less than 0.05 were considered to indicate statistical significance. All the results were expressed as mean \pm SD for eight separate determinations.

RESULTS AND DISCUSSION

Supplementation (PSO) 200g/kg.diet and injection of 5-fluorouracil on tumour volume and weight to mice resulted in a significant decrease in tumour volume and weigh compared to the group that received subcutaneous implantation of EAC (table 1). The decrease in tumour volume and weight in group of mice which supplemented POS and 5-fluorouracil in combination (Group 5) more pronounced than their supplemented each one individual (Groups 3 and 4).

Subcutaneous implantation of EAC into the right thigh of the lower limb of mice resulted in a significant decrease in plasma glucose and insulin compared to the normal control group (table 2) ($p < 0.01$). Supplementation of PSO and intraperitoneal administration of 5-fluorouracil to mice resulted in a

significant increase in plasma glucose and insulin compared to the group that received subcutaneous implantation of EAC ($p < 0.05$). The increase in plasma glucose and insulin was a significant in the group that was treated with PSO+ 5-fluorouracil compared to the groups that received PSO and 5-fluorouracil individual ($p < 0.05$).

Tables 3-5 showed that subcutaneous implantation of EAC into the right thigh of the lower limb of mice resulted in a significant increase in plasma AST, ALT, ALP, LDH and TBARS as well as decrease in plasma Pr-SHs, blood GSH, SOD and GPx compared to the normal control group ($p < 0.01$). Supplementation of PSO and intraperitoneal administration of 5-fluorouracil to mice resulted in a significant decrease in plasma AST, ALT, ALP, LDH and TBARS as well as decrease in plasma Pr-SHs, blood GSH, SOD and GPx compared to the group that received subcutaneous implantation of EAC ($p < 0.05$). The effect of PSO + 5-fluorouracil in combination is more pronounced than when PSO and 5-fluorouracil supplemented individually ($p < 0.05$).

Table 6 showed that subcutaneous implantation of EAC into the right thigh of the lower limb of mice resulted in a significant decrease in plasma cholesterol (TC), triglycerides (TG), HDL-C and LDL-C compared to the normal control group ($p < 0.01$). Supplementation of PSO and intraperitoneal administration of 5-fluorouracil to mice resulted in a significant increase in plasma TC, TG, HDL-C and LDL-C compared to the group that received subcutaneous implantation of EAC ($p < 0.01$). The effect of PSO+ 5-fluorouracil in combination is more pronounced than when PSO and 5-fluorouracil supplemented individually ($p < 0.01$).

Table 7 showed that subcutaneous implantation of EAC into the right thigh of the lower limb of mice resulted in a significant decrease in plasma 17β -estradiol and progesterone compared to the normal control group ($p < 0.01$). Supplementation of PSO and intraperitoneal administration of 5-fluorouracil to mice resulted in a significant increase in plasma estrogen and progesterone compared to the group that received subcutaneous implantation of EAC ($p < 0.01$). The effect of PSO+ 5-fluorouracil in combination is more pronounced than when PSO and 5-fluorouracil supplemented individually ($p < 0.01$).

Table 1. Effect of PSO, 5-fluorouracil and PSO, 5-fluorouracil in combination on tumor weight

No.	Groups	Tumor Volume (ml)	Tumor weight (gm)
(I)	Normal (Non-tumor bearing mice (NTB))	0.0 \pm 0.0	0.0 \pm 0.00
(II)	EAC control (tumor bearing mice (TB))	8.3 \pm 0.20*	6.2 \pm 0.3*
(III)	Purslane seed oil (PSO) (200g/kg.diet)	4.5 \pm 0.40@	3.0 \pm 0.1@
(IV)	5-Fluorouracil (20mg/kg b.w. i.p)	2.3 \pm 0.10@	1.60 \pm 0.2@
(V)	5-Fluorouracil + PSO	2.7 \pm 0.30@	1.95 \pm 0.20@

5-Fluorouracil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups

(III) and (V). Values are given as mean \pm SD for groups of eight animals each. * Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

Table 2. Level of plasma glucose and insulin in normal and experimental groups of mice

No.	Groups	Glucose (mg/dL)	Insulin (uIU/ml)
(I)	Normal (Non-tumor bearing mice (NTB))	110.73 ± 6.48	13.44 ± 2.05
(II)	EAC control (tumor bearing mice (TB))	67.83 ± 8.17*	5.61 ± 1.27*
(III)	Purslane seed oil (PSO) (200g/kg.diet)	95.08 ± 4.83@	9.68 ± 2.09@
(IV)	5-Flourourasil (20mg/kg b.w. i.p)	77.64 ± 5.40@	5.98 ± 1.77@
(V)	5-Flourourasil + PSO	81.1 ± 7.11@	7.38 ± 2.59@

5-Flourourasil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups (III) and (V).

Blood samples were collected. Values are given as mean ± SD for groups of eight animals each. * Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

Table 3. Level of plasma alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase (ALP) in serum of normal and experimental groups of mice

No.	Groups	ALT (U/L)	AST (U/L)	ALP (U/L)
(I)	Normal (Non-tumor bearing mice (NTB))	27.95 ± 4.70	32.88 ± 5.22	134.61 ± 10.07
(II)	EAC control (tumor bearing mice (TB))	50.16 ± 6.38*	67.08 ± 8.32*	274.36 ± 15.91*
(III)	PSO (200g/kg.diet)	33.10 ± 4.26@	31.29 ± 3.55@	166.18 ± 13.85@
(IV)	5-Flourourasil (20mg/kg b.w. i.p)	35.74 ± 6.85@	38.00 ± 5.00@	182.22 ± 13.94@
(V)	5-Flourourasil + PSO	29.54 ± 5.13@	33.19 ± 2.48@	129.50 ± 17.21@

5-Flourourasil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups (III) and (V).

Blood samples were collected. Values are given as mean ± SD for groups of eight animals each. * Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

Table 4. Levels of plasma lactate dehydrogenase (LDH), lipid peroxides (TBARS) and protein thiols (Pr-SHs) of normal and experimental groups of mice

No.	Groups	LDH (U/L)	TBARS (nmol/ml)	Pr-SHs (μmol/l)
(I)	Normal (Non-tumor bearing mice (NTB))	110.37 ± 9.18	40.52 ± 3.29	152.16 ± 9.42
(II)	EAC control (tumor bearing mice (TB))	246.05 ± 14.23*	92.44 ± 7.03*	68.90 ± 7.64*
(III)	PSO (200g/kg.diet)	121.87 ± 11.40@	45.62 ± 5.10@	117.43 ± 14.86@
(IV)	5-Flourourasil	135.60 ± 18.19@	52.18 ± 6.45@	85.40 ± 9.32@
(V)	5-Flourourasil + PSO	105.83 ± 9.36@	39.27 ± 5.32@	126.84 ± 13.50@

5-Flourourasil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups (III) and (V).

Blood samples were collected. Values are given as mean ± SD for groups of eight animals each. * Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

Table 5. Level of reduced glutathione (GSH), superoxide dismutase (SOD) and glutathione peroxidase (GPx) in blood of normal and experimental groups of mice

No.	Groups	GSH (mg%)	SOD (U/g Hb)	GPx (U/g Hb)
(I)	Normal (Non-tumor bearing mice (NTB))	25.46 ± 3.22	62.82 ± 3.29	95.74 ± 9.42
(II)	EAC control (tumor bearing mice (TB))	14.08 ± 2.97*	42.63 ± 3.95*	67.90 ± 4.38*
(III)	PSO (200g/kg.diet)	19.39 ± 3.05@	53.16 ± .39@	86.29 ± 6.14@
(IV)	5-Flourourasil (20mg/kg b.w. i.p)	12.41 ± 4.10@	43.60 ± .21@	61.05 ± 7.33@
(V)	5-Flourourasil + PSO	24.80 ± 5.05@	59.38 ± 4.17@	103.26 ± 9.08@

5-Flourourasil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups (III) and (V). Activity is expressed as: 50% of inhibition of pyrogallol autooxidation per min for SOD and the obtained values

were divided by the haemoglobin (Hb) concentration. Values are given as mean ± SD for groups of eight animals each. * Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

Table 6. Level of plasma total cholesterol (TC), triglycerides (TG), HDL-C and LDL-C of normal and experimental groups of mice

No.	Groups	TC (mg/dl)	TG (mg/dl)	HDL-C (mg/dl)	LDL-C (mg/dl)
(I)	Normal (Non-tumor bearing mice (NTB))	124.43 ± 9.61	109.16 ± 6.39	34.29 ± 5.05	68.31 ± 4.32
(II)	EAC control (tumor bearing mice (TB))	73.76 ± 10.81*	67.24 ± 5.90*	20.64 ± 3.95*	39.67 ± 6.22*
(III)	PSO (200g/kg. diet)	95.27 ± 8.13@	85.24 ± 5.96@	29.11 ± 4.37@	49.112 ± 4.60@
(IV)	5-Flourourasil (20mg/kg b.w. i.p)	81.46 ± 6.85@	59.57 ± 6.18@	25.68 ± 4.39@	43.87 ± 5.33@
(V)	5-Flourourasil + PSO	109.24 ± 11.38@	93.25 ± 10.16@	36.70 ± 5.30@	53.89 ± 6.14@

5-Flourourasil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups (III) and (V).

Values are given as mean \pm SD for groups of eight animals each. LDL-C (mg/dl) = TC-HDL-[TG / 5].

* Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

Table 7. Level of plasma estrogen and progesterone of normal and experimental groups of mice

No.	Groups	17 β -estradiol (pg/ml)	Progesterone (ng/ml)
(I)	Normal (Non-tumor bearing mice (NTB))	12.70 \pm 1.35	20.63 \pm 2.54
(II)	EAC control (tumor bearing mice (TB))	2.54 \pm 0.39*	12.58 \pm 2.03*
(III)	PSO (200g/kg.diet)	8.81 \pm 2.06@	16.25 \pm 1.77@
(IV)	5-Flourourasil (20mg/kg b.w. i.p)	10.03 \pm 1.76@	14.93 \pm 3.71@
(V)	5-Flourourasil + PSO	13.39 \pm 2.05@	18.66 \pm 3.49@

5-Flourourasil was given i.p. as a daily dose of 20mg/kg b.w. It was given to all groups except the normal one (NTB). The test PSO was orally given daily for 2 weeks at 200g/kg.diet. It was given to all groups (III) and (V). Values are given as mean \pm SD for groups of eight animals each. * Significantly different from normal group at $p < 0.01$. @ Significantly different from control group at $p < 0.05$.

The present article aimed to study the antitumor activity of *purslane* seed oil (PSO) in EAC bearing mice as well as compare its activity with 5-Flourourasil, a standard antitumor drug. Our results showed that PSO when combined with 5-Flourourasil or individual were able to significantly decrease the tumor volume and weigh as compared to that of the EAC control group. Cancer is a pathological state involving uncontrolled proliferation of tumor cells. Reduced volume and weight of tumor indicated a decrease in abnormal cell divisions, i.e. tumor proliferation [39, 40]. In this study, we observed and reported that PSO can revert or inhibit EAC induced tumor [4], which may be due to free radical scavenging property of extract in the presence of antioxidant phytochemicals [12, 13 and 16].

The present work showed that EAC implantation caused fall of blood glucose and insulin in EAC control mice. Hypoglycemia was proportional to the number of tumor cells inoculated into the host. One reason for hypoglycemia could be an augmented consumption of glucose by the cells of the tumor [42, 43]. Indeed, hypoglycemia was most expressed in mice with large tumors, i.e., with the highest tumor volume and weight due to transport of glucose through the membrane of tumor [44]. Facilitated transport of glucose is attributed to the changes of the membrane of tumor cells [45] and increase of insulin-like (glucose-lowering) substances level in the tumor cells, or produces an insulin-like (glucose-lowering) principle itself. Several authors have described higher concentration of insulin-like substances in the plasma of mice with some tumors [46, 47]. However, we have found a decrease of insulin activity in the plasma of EAC control group. Supplementation of PSO and 5-Flourourasil resulted to increase glucose and insulin levels when compare to EAC control group. According to the presented results PSO containing antioxidant phytochemicals [12, 13 and 16] inhibit EAC induced

tumor which my led to decrease the rate of glucose and insulin transport to the tumor cells.

Liver is considered to be the main organ of drug detoxifying organ, some liver marker enzyme levels were measured from serum. AST, ALT, ALP, LDH and TBARs levels were increased in EAC controlled mice, whereas Pr-SHs, GSH, SOD and GPx levels were decreased. In the present study, subcutaneous implantation of EAC into the mice resulted in a significant decrease in blood GSH, SOD and GPx as well as plasma TC, TG, HDL-C and LDL-C with a significant increase in plasma TBARs compared to the normal control group. These results were in agreement with Raju and Arockiasamy [48] who reported that the consumption of free amino acid for building the proteins of rapidly dividing tumor cells might result in the disturbance of the enzyme activity in the liver [49]. On treatment with PSO altered liver enzyme level was restored as that of the normal group.

Alterations of cholesterol metabolism, including increased cholesterol synthesis and accumulation of cholesterol esters in tumor tissues associated with a decrease of high density lipoprotein cholesterol in serum, were previously observed in different models of neoplastic cell proliferation including haematological malignancies. A number of studies had indicated that reactive oxygen species (ROS) are involved in a variety of different cellular processes ranging from apoptosis and necrosis to cell proliferation and carcinogenesis. Flavonoids and tannins are well known polyphenolic natural antioxidants. The flavonoids present in *Purslane* plant are thought to be the cause of their antitumor and anti-inflammatory effects [8, 9]. Flavonoids have a chemopreventive role in cancer by means of their effect in signal transduction in cell proliferation and angiogenesis [50]. This important property may be responsible for its antitumor activity against EAC *in vivo*. Antioxidant activity of *purslane* against different reactive oxygen and nitrogen species has already been established by the present authors [8, 9].

The present work showed that EAC implantation caused fall of plasma sex hormones; estrogen and progesterone when compare with normal control mice. EAC bearing mice associated with increase receptor population [51] and altered estrogen and progesterone

levels were brought back to normal by PSO and 5-Flourourasil treatment.

Therefore, from the present study it can be concluded that POS showed promising antitumor potential in Ehrlich ascites carcinoma bearing albino mice which can be attributed to its flavonoids content. This could serve as a stepping stone towards the discovery of newer safe and effective antitumor agents.

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