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Antioxidant and Anticancer property of *Semecarpus anacardium* extract in B16F10 melanoma cell lines and 7, 12- Dimethylbenz(a) anthracene (DMBA) induced skin carcinogenesis

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Abstract

The present study was designed to investigate the *in vitro* and *in vivo* antioxidant and anticancer effect of *S. anacardium* nut methanolic extract and its fractions against B16F10 melanoma cell lines and 7, 12- dimethylbenz(a)anthracene (DMBA)-croton oil induced skin cancer in Swiss albino mice. The DPPH radical scavenging and superoxide radical scavenging assays were higher in methanolic extract followed by hexane, ethyl acetate and acetone. The B16F10 melanoma cell line treated with the hexane fraction have exhibited higher activity for caspase-3 and caspase-9 followed by the crude extract. The DNA of B16F10 melanoma cell lines treated with *S. anacardium* crude extract and n-hexane fraction showed a marked DNA fragmentation compared to the untreated one. *In vivo* studies showed a significant reduction in the number of tumor burden per mouse following oral treatment with *S. anacardium* methanolic extract. These findings suggest that *S. anacardium* have the potential to prevent tumor and also enhanced the antioxidant activity. This study concludes that the purification of bioactive compounds from *S. anacardium* nut responsible for anticancer effects would help to develop novel drugs in the field of pharmaceutical industries in the future

Keywords: B16F10 cell lines, Histopathology, *Semecarpus anacardium*, Swiss Albino mice.

Introduction

Skin acts as a barrier and gives protection against exogenous influence like UV radiations, toxic chemicals and infectious or mechanical stress (Fuchs and Ragavan, 2002). Skin regulates body temperature, involves in vitamin D production and also actively takes part in immune system. Skin cancer generally develops in the epidermis and tumors are easily visible, which helps in the early identification of tumor. Skin cancer contributes 30% of total cancer diagnosed worldwide and 90% of skin cancer is due to solar UV radiation (Armstrong and Kricker, 2001). Skin cancer is more common in southern latitudes of northern hemisphere (Wagner and Casciato, 2000). The World Health Organization (WHO) estimates that as many as 65,161 people die in a year worldwide due to malignant skin cancer. The paradoxical case of basal cell carcinoma in India revels that the rate of change in female population is rapidly increased growth predilection and higher percentage of pigmented lesions in Indians (Sumir Kumar *et al.,* 2014).

Free radicals are substances which are formed during metabolism or through the action of ionizing radiation which are very short lived with half lives of milli/micro/nanoseconds (Eg: superoxide, hydrogen peroxide and nitric oxide). These free radicals include reactive oxygen species (ROS) and reactive nitrogen species (RNS) which are produced in a well-regulated manner to maintain homeostasis at the cellular level in the normal healthy tissues and play an important role as signaling molecules. Increased production of ROS is associated with the onset of a variety of diseases including cancer (Leboritz*et al.,* 1996). A free radical can damage the specific site of DNA leading to breaking of strands, or it might delay the repair before replication occurs, leading to mutations (Cheesman and Slater, 1993).

Antioxidants are substances that neutralize free radicals or their actions. Therefore, it is important to maintain a balance between the prooxidant (free radicals) and antioxidants and this can be achieved by the production of antioxidant either in the body itself or obtained from the environment. Imbalance in this will result in oxidative stress, which may results in tissue injury and subsequent diseases (Sies, 1991). A number of known antioxidants are present in plants and these compounds play an important role in preventing the free radical induced diseases such as cancer and atherosclerosis (Teresita *et al.,* 1991). In alternative medicine, medicinal plant preparations have found widespread use particularly in the case of diseases not amenable to treatment by modern method (Dhalla, 2006). *Semecarpus anacardium* is one of the medicinal plants used in ayurveda and siddha medical system which is distributed in sub-himalayan, tropical and central parts of India. It belongs to the family Anacardiaceae. *S. anacardium* is a potent drug and traditionally used for neuritis, arthritis, leprosy, helminthic infections and it possess anticancer, antibacterial and anti-inflammatory properties (Chopra *et al.,*1958; Sharma and Chatuvedi, 1965). The cytotoxic effect of *S. anacardium* in colo-320 tumor cells have been reported (Smit *et al.,* 1995). The chloroform extract of *S. anacardium* nuts showed an activity of 150% T/C in a P388 test system in mice, at a dose of 50 mg/kg (Gothoskar*et al.,* 1971). Hembree *et al.*(1978) found that a fraction of the aqueous methanolic extract of the nuts was active against Eagles 9KB nasopharynx carcinoma cell cultures. Even though the anticancer property of *S. anacardium* was reported earlier for different cancer types, study against B16 melanoma cell line and its efficiency against DMBA-croton oil induced skin carcinogenesis in Swiss albino mice is scanty. Hence, the present study is focused on the activity of methanolic extract and the fractions of *S. anacardium* nut against B16F10 melanoma cell lines. Further, 7, 12- dimethylbenz(a)anthracene (DMBA)-croton oil induced skin cancer in Swiss albino mice was investigated.

Materials and Methods

Collection of plants

The *S. anacardium* nuts and Croton tiglium seeds were bought from local market and authenticated by Dr. S. Karupasamy, Associate Professor, Dept. of Botany, The Madura College, Madurai, Tamil Nadu, India.

Preparation of extract

Extraction of *S. anacardium* was carried out by the method of Sowmyalakshmi*et al.*(2005) and Weimin *et al.*(2009). The dried nuts of *S. anacardium* were washed in distilled water and crushed well. The plant material was kept in hexane for 24 h by following the cold extraction method. Then it was filtered and the residues were dried. About 25 g of the dried residue was then used for methanolic extraction in soxlet apparatus for 48 h. The extract was then dried in air over night and then stored at 4°C. Various fractions of the crude methanolic extract were prepared by the method of Raj and Dhall(2010). All the fractions were collected and the solvents were evaporated at room temperature. The dried fractions were collected and stored at 4°C. C. tiglium seeds were dried and crushed well. Then 25 g of the crushed seeds was extracted with 150 ml of petroleum ether for 48 h and the solvent was evaporated at room temperature. The extract rich in oil content was collected and stored at 4°C.

In vitro studies

The antioxidant activity (Prieto *et al.,* 1999), DPPH free radical scavenging activity (Blois, 1958) and superoxide (SO) radical scavenging activity (Liu, *et al.,* 1997) of different concentrations of *S. anacardium* crude methanolic extract and its fractions were analyzed. All experiments were repeated thrice. The results are reported as mean± SD. The capability to scavenge the DPPH radical was calculated using the following equation; %DPPH scavenging activity = (A control- A test) /A control× 100 Where A-control is the absorbance of the control reaction and A-test is the absorbance of the extract. The antioxidant activity of the extract was expressed as IC50 value which was defined as the concentration (µg/ml) of extracts that inhibit the formation of DPPH radicals and superoxide by 50 %. The scavenging activity of superoxide radical (%) was thus calculated by the following equation;

% of SO radical scavenged = [(A560blank - A560sample)/A560blank] x 100.

Effect of *S. anacardium* methanolic extract and its fractions on B16F10 melanoma cancer cell lines

Cell lines

B16F10 skin cancer melanoma cell lines were obtained from the National Centre for Cell Sciences (NCCS), Pune, India. The cells were maintained in DMEM with 10% FBS. The culture environment was maintained as 5% CO2 with optimum humidity at 37°C. The cells were passaged for every 4 days or after 100% confluency.

Cell viability assay

Cell viability assay was performed in triplicate as per the method of Kang *et al.*(2004). The mean of the cell viability values was compared to the control to determine the effect of the extract on cells. A graph was plotted against the % cell viability (B16F10 cells) Vs dilution of the extract. The minimum concentration of extract that was toxic to cells was recorded as the effective drug concentration and was compared to the positive control (cyclophasphamide).

Determination of caspase activity

Caspase 3 and caspase 9 activities of B16F10 melanoma cell lines were determined by chromogenic substrate based colorimetric assays (Das *et al.,* 2005) and expressed as pg µg-1 protein.

DNA fragmentation assay

DNA was extracted from B16F10 melanoma cells before and after treatment with 100 µg/ml of crude extract and n hexane fraction and separated in agarose gel electrophoresis following the method of Burton(1956) and Perandones*et al.*(1993).

In vivo studies

Experimental animal

The anticancer activity of *S. anacardium* methanolic extract in DMBA induced skin cancer

was studied in Swiss Albino mice (Balb/c). Mice were maintained under standard environmental condition (temperature 27± 2°C and 12 h light/dark cycle) and they were fed with standard laboratory feed and water ad libitum. Initial body weights of all animals were recorded.

Experimental design

A total of 24 albino mice were divided into 6 groups. Each group was provided with 4 animals. Group I: normal control mice (healthy mice); Group II: animals were treated topically in the shaven area with single dose of 104 µg DMBA in 100 µl acetone and 1% croton oil in acetone was applied thrice a week after two weeks for 12 weeks to induce skin cancer; Group IIIA: skin cancer induced mice treated with *S. anacardium* methanolic extract (200 mg/kg) dissolved in olive oil by gastric gavage from the second week till the end of experiment i.e., 12 weeks. Group IIIB: skin cancer induced mice treated with *S. anacardium* methanolic extract (200 mg/kg) dissolved in olive oil was applied topically from the second week till the end of experiment. Group IVA: Normal mice treated with *S. anacardium* methanolic extract (200 mg/kg) dissolved in olive oil by gastric gavage from the second week till the end of experiment. Group IVB: Normal mice treated with *S. anacardium* methanolic extract (200 mg/kg) dissolved in olive oil were applied topically from the second week till the end of experiment. Premalatha and Sachdanandam(2007) standardized that the oral administration of *S. anacardium* nut extract (200 mg kg−1 body weight) was found as highly effective in inducing phase I and phase II biotransformation enzymes. Hence, this concentration of *S. anacardium* methanolic extract was used for the present study.

Histopathological studies

The animals were sacrificed and the skin region was removed. The skin was fixed with 10% formalin for 12h, then embedded in paraffin and cut into 5 µm thick sections and stained using hematoxylin-eosin dye and finally mounted in diphenyl xylene (Ila *et al.,* 2010). The sections were then observed under microscope for histopathological changes in skin architecture and their photomicrographs were taken.

In vivo antioxidant assay

After twelve weeks of experiment, the skin and liver samples were sacrificed from each mice group for *in vivo* antioxidant assay. The biochemical parameters such as the glutathione (GSH) and catalase were analysed using the method of Moron *et al.*(1979) and Aebi (1984) respectively.

Results

In vitro antioxidant studies

Total antioxidant potential assay

The total antioxidant capacity of the methanol extract of *S. anacardium* and its fractions are shown in Fig. 1. Total antioxidant capacity of the extracts is expressed as the number of equivalents of ascorbic acid. At 100 µg/ml concentration, crude methanolic extract showed a significant antioxidant activity (56.25±3.83 µg) followed by hexane (30.52±1.95 µg), ethyl acetate (14.72±0.53 µg) and acetone (5.16±0.12 µg) fractions.

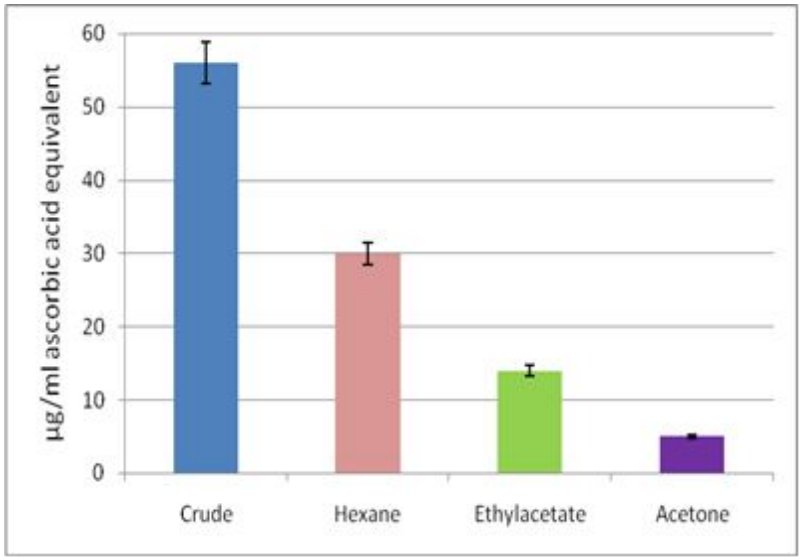

Fig. 1. Total antioxidant assay of *S. anacardium* nut crude methanolic extract and its fractions.

Free radical scavenging activity

The DPPH and superoxide radial scavenging activity of *S. anacardium* crude methanolic extract and its fractions viz., hexane, ethyl acetate and acetone are presented in Table 1. Among the tested samples, the crude methanolic extract showed a highest scavenging activity, followed by hexane, ethyl acetate and acetone fractions for both DPPH and superoxide radical scavenging activity. The IC50 value of crude methanolic extract and it fractions of hexane, ethyl acetate and acetone are 9.10±0.33, 21.10±1.10, 22.70±1.25 and 61.00±3.72 µg/ml for DPPH and 19.50±0.79, 22.00±1.43, 30.40±2.10 and 77.80±4.96 µg/ml for superoxide respectively. A dose dependent increase in the scavenging activity of the samples was observed.

Table 1: Free radial scavenging activity of crude methanolic extract and fractions of *S.anacardium* nut.

% of inhibition								
Concentration (µg/ml)	Crude		Hexane		Ethyl acetate		Acetone	
	DPPH	SO	DPPH	SO	DPPH	SO	DPPH	SO
5	39.73±1.33	24.30±1.02	36.80±1.47	23.89±0.86	30.82±1.23	22.20±0.99	12.12±0.66	7.40±0.14
10	52.66±2.45	34.30±1.39	40.96±2.11	33.30±1.95	39.50±2.37	31.40±1.63	16.15±0.93	21.90±1.12
25	54.23±1.99	59.30±3.23	53.86±2.44	55.00±2.05	52.70±3.51	48.90±2.75	21.30±1.16	29.40±1.43
50	57.30±2.89	67.40±4.22	58.28±2.85	56.30±3.24	54.26±3.62	53.60±3.18	44.80±2.90	40.30±3.41
100	67.43±4.68	72.80±5.94	64.27±4.18	63.40±3.72	63.12±3.93	62.90±4.13	64.40±5.29	53.90±2.58
150	78.32±3.82	78.20±3.94	68.58±2.63	72.18±5.66	68.45±4.80	69.65±3.71	67.70±4.93	58.70±2.83
200	82.40±6.91	80.50±5.62	76.24±4.44	79.87±5.92	70.60±3.18	73.45±4.42	69.20±3.64	61.50±3.99
IC50 (µg/ml)	9.10±0.33	19.50±0.79	21.10±1.10	22.00±1.43	22.70±1.25	30.40±2.10	61.00±3.72	77.80±4.96

Effect of *S. anacardium* methanolic extract and its fractions on B16F10 melanoma cancer cell lines

Cell viability assay

The anticancer activity of *S. anacardium* methanolic crude extract and its fractions were analyzed using B16F10 melanoma cell lines following MTT assay. The percentage of viable cells after treatment with different concentrations of the extract is shown in Fig. 2. The viability of the cells decreased in dose dependent manner. Among the tested samples, the hexane fraction and crude extract have shown greater cytotoxicity compared to the ethyl acetate and acetone fractions.

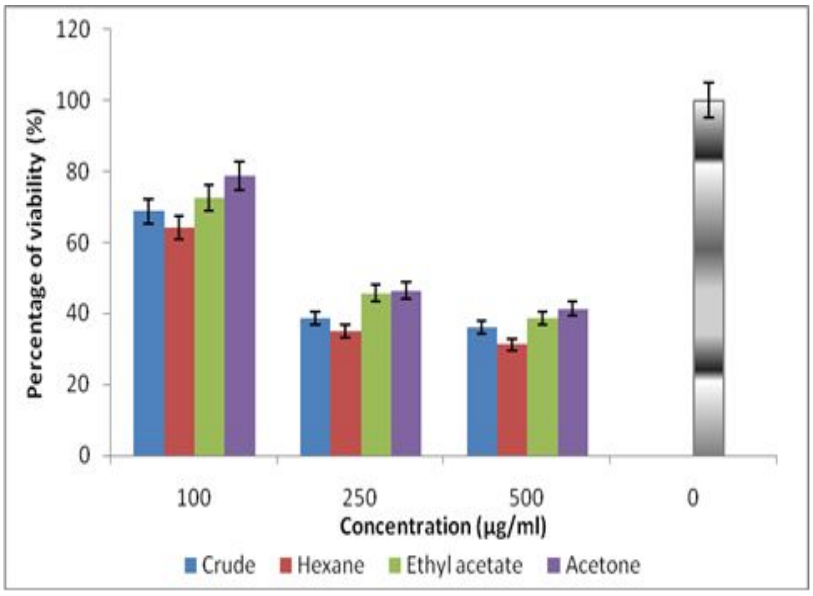

Fig. 2. Percentage of viable B16F10 melanoma cells treated with *S. anacardium* nut crude methanolic extract and its Fractions

Caspase activity

The expression of caspase-3 and caspase-9 is shown in Table 2. A significant increase in the activity of caspases in the B16F10 melanoma cell lines was observed following treatment with different concentration of the extracts. However, the B16F10 melanoma cell line treated with the hexane fraction have exhibited higher activity for caspase-3 and caspase-9 followed by the crude extract.

Table 2: Activity of caspase 3 and caspase 9 in B16F10 melanoma cell lines after treatment. Values are mean ± SD (n=3).

Samples (µg/ml)	Caspase – 3 (pg/ µg)	Caspase – 9 (pg/µg)
Control	56 ± 4.2	65 ± 3.4
Hexane		
10	68 ± 4.7	78 ± 4.6
100	127 ± 9.2	135 ± 4.9
500	145 ± 11.5	154 ± 5.5
Crude		
100	72 ± 3.1	82 ± 1.9
500	99 ± 8.8	111 ± 7.1

DNA fragmentation assay

The DNA of *S. anacardium* crude extract and n-hexane fraction treated B16F10 melanoma cell line is shown in Fig. 3. The DNA of B16F10 melanoma cell line without any treatment (lane 1) have not shown fragmentation and those that were treated with *S. anacardium* crude extract (lane 2) and n-hexane fraction (lane 3) showed a marked DNA fragmentation.

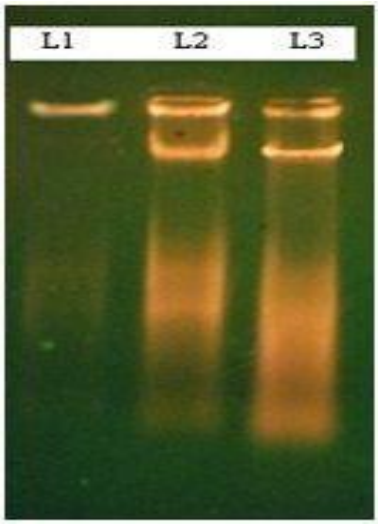

Fig. 3. DNA fragmentation assay. L1 –B16F10 melanoma cell DNA without treatment; L2 –B16F10 melanoma cell DNA treated with 100µg/ml of crude extract; L3 –B16F10 melanoma cell DNA treated

In vivo studies

Skin carcinogenesis

The developments of tumor and *S. anacardium* treated mice are shown in Fig.4. The final body weight of the mice was reduced in group II compared to *S. anacardium* treated mice (Table 3). Topical application of DMBA followed by croton oil in Swiss albino mice have developed skin papillomas, which started to appear from the 4th week in group II and 8th and 7th weeks for group IIIA and IIIB respectively. For the mice in group II, tumor incidence reached 100% at the end of 12th week and the total number of papillomas recorded was 33 (Table. 3). The tumor yield per mouse and tumor burden was 8.25±0.75. In group IIIA, tumor incidence was 50%, the tumor yield was 1.00±0.5 and the tumor burden was recorded as 2.00. In group IIIB, the tumor incidence was 75%, tumor yield was 1.75± 0.25 and the tumor burden was 2.33±0.33. The mice of group I - normal, group IVA – treated with *S. anacardium* orally alone and group IVB – treated with *S. anacardium* topically alone did not showed any tumor incidence.

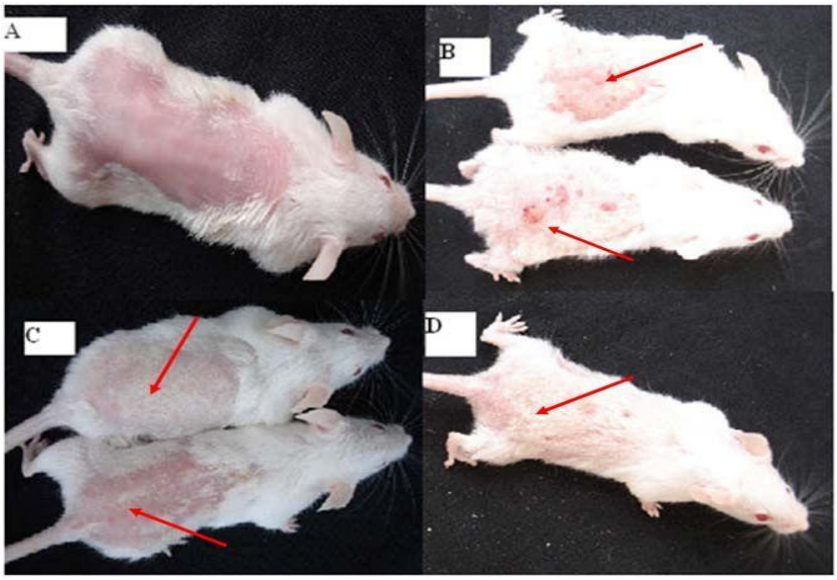

Fig. 4. Development of tumor and *S. anacardium* treated mice. A –Control – mice receiving normal diet and no treatment; B – Mice treated with DMBA/croton oil alone; C – Mice treated with DMBA/croton oil and orally provided *S. anacardium*; D – mice treated with DMBA/croton oil and topically applied *S. anacardium*with 100µg/ml of n-hexane fraction

Table 3: Effect of *S.anacardium* nut extract on DMBA/croton oil induced skin carcinogenesis in Swiss albino mice. Values are mean ± SD (n=4).

Groups	Total no. of animals	Body weight		Total no. of tumors developed	% of tumor incidence	Tumor yield	Tumor burden
		Initial (g)	Final (g)				
I	4	26.30±0.51	34.08±1.90	-	-	-	-
II	4	26.10±0.60	30.05±1.70	33	100	8.25±0.75	8.25±0.75
III A	4	25.98±0.55	33.81±2.10	4	50	1.00±0.5	2.00
III B	4	26.60±0.43	32.73±2.30	7	75	1.75±0.25	2.33±0.33
IV A	4	26.20±0.37	34.40±1.90	-	-	-	-
IV B	4	26.42±0.45	34.20±2.20	-	-	-	-

Histopathology

The group I mice which served as a control showed a normal epithelium and normal distribution of cells (Fig. 5A). The group II mice, treated with DMBA-croton oil alone showed prominent features of hyperkeratosis, acanthosis and papillomatosis (Fig. 5B). However, the group IIIA and IIIB mice treated with carcinogen and also received the oral treatment and topical application of *S. anacardium* methanolic extract respectively showed near normal epidermis (Fig. 5C and 5D). The mice in group IVA and IVB have received only the oral treatment and topical application of *S. anacardium* methanolic extract showed normal epidermis.

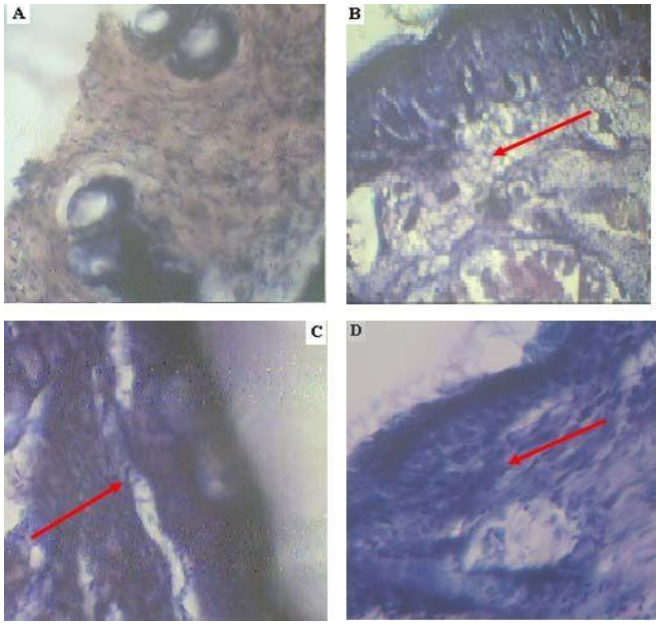

Fig. 5. Hematoxylin and eosin staining of cross section of mouse skin. A-control; B-DMBA-croton oil alone; C-DMBA-croton oil and orally provided *S. anacardium*; D- DMBA-croton oil and topical applied *S. anacardium*.

Estimation of glutathione (GSH)

The expression of GSH in the skin and liver of different treatment groups mice is shown in Fig. 6A. GSH level decreased sharply to 0.32±0.01 µ mol/g tissue in skin and 0.54±0.03 µ mol/g tissue in liver of group II, i.e., carcinogen alone treated group. Group IIIA and IIIB showed an increased level of GSH expression of about 1.80±0.08 and 1.77±0.06 µ mol/g tissue respectively for skin and 2.32±0.15 and 2.27±0.09 µ mol/g tissue respectively for liver, comparing with group II. On the other hand, mice in group IVA and IVB showed normal GSH expression as that of untreated control.

Estimation of catalase (CAT)

The expression of catalase in the skin and liver of different treatment groups mice are shown in Fig. 6B. A decreased level of expression of catalase i.e., 5.54±0.27 and 8.23±0.41 nmol/mg protein in skin and liver respectively was noticed in group II and the level of the catalase was increased in the *S. anacardium* treated mice i.e., 8.92±0.46 and 14.25±0.98 nmol/mg protein in orally treated mice skin and liver respectively. The topically treated mice skin and liver have shown 7.33±0.39 and 13.12±1.10 nmol/mg protein respectively. Mice belonging to group IV A and IV B have exhibited normal catalase level as that of untreated control.

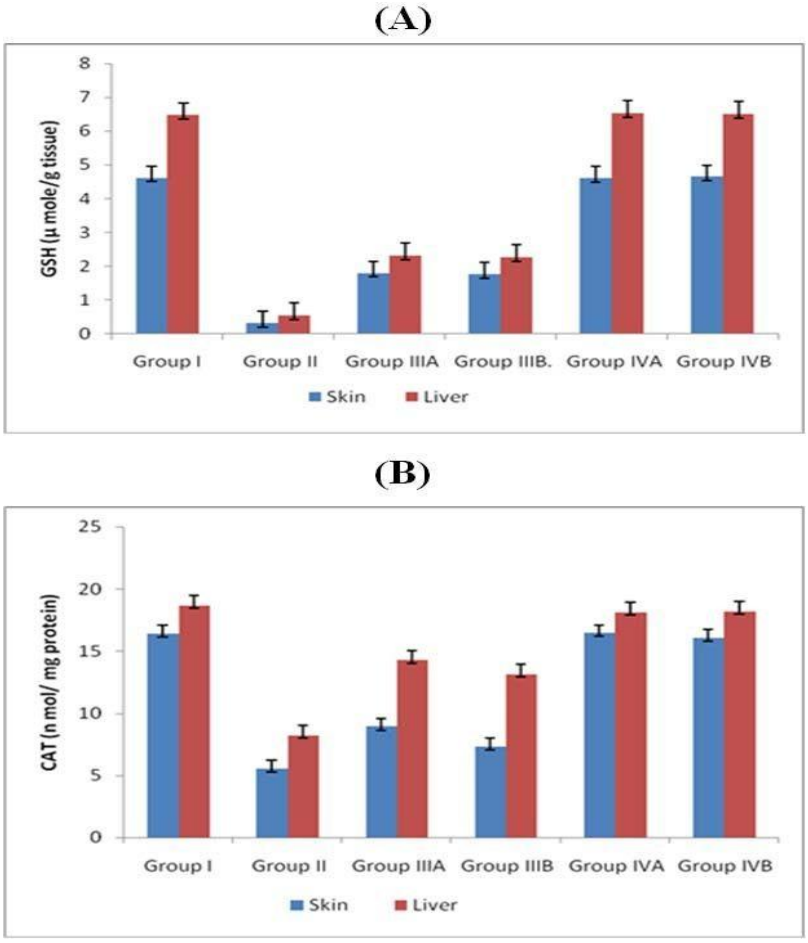

Fig. 6. Variation in the GSH (A) and catalase (B) level in the skin and liver during DMBA induced skin tumorigenesis in different groups of mice.

Discussion

In recent years, the natural and plant derived compounds have received greater attraction in the discovery of drugs for cancer treatment. Cancer chemoprevention by phytochemicals appears to be one of the most feasible approaches for cancer control. Vegetables, fruits, spices, teas, herbs, and medicinal plant metabolites such as carotenoids, phenolic compounds, and terpenoids, have been proven to suppress experimental carcinogenesis in various organs (Wattenberg, 1985; Borek, 2001).

The results of antioxidant assay showed that the *S. anacardium* crude methanolic extract and its fractions are rich in antioxidants. It showed IC50 values even in low concentrations. It was reported that *S. anacardium* nut shells containsbiflavanoids, tetrahydromentoflavone, jeediflavone, semecarpuflavone, galluflavone, nallaflavone, semearpentin and anacarduflavone(Mona *et al.,* 2010). The free radical scavenging property of *S. anacardium* may be due to its rich flavonoid contents. Studies of cancer treatment in experimental animals have

assessed the impact of a wide variety of flavonoids and a selected few isoflavones for their efficacy in inhibiting cancer. *S. anacardium* crude methanolic extract and its hexane fraction were tested efficient against B16F10 melanoma cell lines, showing its higher cytotoxicity. Weimin et al. 2009 reported that the hexane fractions of *S. anacardium* lehyam showed high cytotoxic activity and caspase activity in breast cancer cell line. They isolated the active compounds (7:Z, 10:Z)-3 pentadeca-7,10-dienyl-benzene-1,2,-diol and (8:Z)-3-pentadec-10-enyl-benzene-1,2-diol found to be in large concentration in the hexane fraction and responsible for the anticancer property. Similarly, Raveendran *et al.*(2009) isolated an anticancer catechol i.e., 3-[8'(Z),11'(Z)-pentadecadienyl] catechol from *S. anacardium*, which actively inhibited tumor cells and the cytotoxicity was due to induction of apoptotic pathway.

The caspases level was found to increase in a dose dependent level in the B16F10 cell line. DNA fragmentation assay of B16F10 cell line treated with the hexane fraction and crude extract showed DNA fragmentations which confirmed the cell are killed by apoptotic pathway. It was reported that the anticancer potential in DMBA induced skin cancer of a synthetic coumarin was due to induction of caspase mediated apoptosis (Soumya *et al.,* 2009).

DMBA-croton oil induced skin cancer in animal model showed the post treatment effect of *S. anacardium* methanolic extract; an active reduction in tumor number was observed compared to the negative control i.e., group II. Also, the antioxidant enzymes, glutathione and catalase were found to be increased in the skin and liver samples of the *S. anacardium* treated mice models. The orally treated animals showed better results than the topically treated animals. The histopathology study also clearly showed the occurrence of hyperkeratosis, acanthosis and papillomatosis in the skin epidermis of DMBA-croton oil treated animals. The animals received oral and topical treatment showed near normal epidermis and this indicated that the *S. anacardium* extract effectively inhibited the tumor formation. This tumor inhibiting property may be due to the presence of flavonoids, as they have been reported to impart antiproliferative action on several cancer cells (Birt *et al.,* 2001). Premalatha *et al.*(1997) reported that the animal model induced with aflotoxin mediated hepatocarcinoma treated with *S. anacardium* nut milk extract significantly increased the antioxidant enzymes and decreased the level of LDH, indicating that *S. anacardium* can be treated for hepatic cancers. Ila *et al.*(2010)reported that Saffaron, a plant derived compound found to inhibit the DMBA induced skin cancer and brought up the antioxidant level comparing with that of disease control.

Conclusion

S.anacardium methanolic extract has significant application against B16F10 melanoma cell lines and in DMBA induced skin carcinogenesis. The anticancer property of *S. anacardium* extract is due to its rich antioxidant contents and free radical scavenging activity. However, further investigation is necessary for identifying its molecular mechanism of cytotoxic effect.

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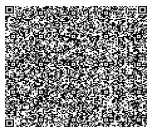	Website: www.ijcrims.com
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