Research Article

www.ijrap.net

REFLECTIONS OF ABHRAK BHASMA MEDIATED HEPATOPROTECTIVE EFFECTS ON LIPIDS AND PHOSPHOLIPIDS IN SINGLE DOSE OF CCL_4 INTOXICATED RAT

Teli Parashuram[1], Jadhav Jaywant[1] and Kanase Aruna[2]*

[1]Cell Biology Section, Department of Zoology, Shivaji University, Kolhapur, Maharashtra, India

[2]Consulting Scientist, APT Research Foundation-National Toxicological Center, Pune, Maharashtra, India

Received on: 20/01/15 Revised on: 12/02/15 Accepted on: 25/03/15

***Corresponding author**

Aruna Kanase, Consulting Scientist, APT Research Foundation-National Toxicological Center, Pune, Maharashtra, India

E-mail: arunakanase@gmail.com

DOI: 10.7897/2277-4343.06258

ABSTRACT

Total lipids and phospholipids are diagnostically of relative importance in assessing the state of organisms health. They are susceptible to CCl_4 induced fatty degeneration in liver and can be used to assess hepatic damage/protection. The present study was aimed to evaluate hepatoprotective influence of abhrak bhasma on total lipids and phospholipid contents. To differentiate the silica mediated effects since abhrak bhasma is mica – silica ore derived. SiO_2 was used as silica drug control. Hepatotoxicity was induced by subcutaneous administration of single dose of 3.0 ml CCl_4/kg body wt for 24 h. 10, 20, 30 and 40 mg doses of abhrak bhasma and amorphous SiO_2 were given orally to CCl_4 intoxicated rats. CCl_4 administration to rats, caused a significant alterations in total lipids and phospholipid contents in liver, kidney and serum; indicating fatty degeneration and hence alterations in functions of liver. These altered levels of lipids and phospholipid were retrieved to near normal level by graded doses of abhrak bhasma in CCl_4 treated rat. Abhrak bhasma was more effective than SiO_2. The results indicate that hepatoprotective activity of abhrak bhasma was dose dependent. 10 mg dose being minimum effective dose and higher doses maintained lipid and phospholipid metabolism. SiO_2 indicated similar results. All the doses of abhrak bhasma potentially protect/ recover the liver fatty degeneration by fat mobilization influencing liver and serum total lipids and hepatic cell membrane protection influencing phospholipids content of liver and serum. SiO_2 higher doses also showed similar effects, but with some differences.

Keywords: Abhrak bhasma, Fatty degeneration, Hepatotoxicity, SiO_2, Phospholipids, Total lipids.

INTRODUCTION

Liver plays an important role during protection against hazards of harmful drugs, chemicals and xenobiotics. It is involved in lipid metabolism, protein metabolism and detoxification of xenobiotics[1]. Therefore total lipids and phospholipids in liver, kidney and serum are diagnostically of relative importance in assessing the state of organism's health[2]. Most of the compounds from lipids are phospholipids; which occur normally in cell membranes and lipoproteins, where they are structural and functional entity[3]. All membranous organelles contain phospholipids and the mitochondria which are the regulators of cell metabolism and energy production in the body. They also play an essential role in signal transduction, triglycerides transport and membrane related activities[3]. Carbon tetrachloride (CCl_4) is known to exert toxic effects on liver and associated effect on kidney by altering free radical mediated oxidative status. Activated metabolites of CCl_4 i.e. CCl_3 attacks easily on lipids resulting in damage to intracellular membranes and also the plasma membranes[1]. CCl_4 generated free radicals (CCl_3) lead to fatty degeneration even by single dose where accumulation of fats in centrolobular region is significant histological picture[4]. Thus both accumulation of fats in hepatocytes and failure to deposit fats may be influencing the transport of various components of lipids. It has been already shown that CCl_4 can interfere with the liver phospholipid synthesis[5]. Their alterations cause tissue dysfunctions and has been used its toxicity as hepatotoxicity model especially of centrolobular zone. Abhrak bhasma is a commonly used Ayurvedic drug against varied diseases and disorders including hepatitis. Its use as anti-aging, pro-immunity and rejuvenation agent is also popular[6]. Abhrak bhasma at 20 mg dose had been protective in inhibiting centrolobular fatty degeneration in single dose of CCl_4 induced fatty degenerative effects showing rats[4] by influencing lipases: acid, alkaline and hormone sensitive[7], lipid peroxidation[8] and glutathione contents[9]. In present work, abhrak bhasma and SiO_2 induced alterations in total lipid and phospholipid contents in liver, kidney and serum of single dose induced CCl_4 intoxicated male albino rats were evaluated. To differentiate silica influenced effects, SiO_2 was used as silica control for abhrak bhasma, since abhrak bhasma is prepared from mica-silica ore.

MATERIAL AND METHODS

Experimental Animal

Male albino rats, *Rattus norvegicus* weighing about 130-140 g each were used for experiments. They were bred and maintained in the Departmental Animal House (Reg. No. 233/CPCSEA) under standard conditions and were given standard pellet diet (prepared by Amrit feeds, Sangli, MS, India). Food and water were provided *ad libitum*.

Preparation of abhrak bhasma and SiO_2

Abhrak bhasma was prepared in the laboratory as described in Rasa Ratna Sammucchaya under the

supervision of Ayurvedacharya[6]. SiO_2 treatment was given as silica control. To study dose dependent effects of abhrak bhasma and SiO_2 on total lipids and phospholipid contents of liver, kidney and serum; different doses viz. 10, 20, 30 and 40 mg/kg body wt were administered orally with honey. Honey control rats group that were used showed data as normal rat, therefore, honey control group data is not presented.

Experimental Design

The experimental animals were divided into following groups, each comprising of six animals.
Group 1- The rats were maintained as normal without any treatment.
Group 2- Hepatotoxicity induced by single dose of 3.0 ml CCl_4/ kg body wt for 24 h sc.
Group 3- 10 mg abhrak bhasma/kg body wt was given po.
Group 4- 20 mg abhrak bhasma/kg body wt was given po.
Group 5- 30 mg abhrak bhasma/kg body wt was given po.
Group 6- 40 mg abhrak bhasma/kg body wt was given po.
Group 7- 10 mg SiO_2/kg body wt was given po.
Group 8- 20 mg SiO_2/kg body wt was given po.
Group 9- 30 mg SiO_2/kg body wt was given po.
Group 10- 40 mg SiO_2/kg body wt was given po.
Group 11- CCl_4 (3.0 ml/kg body wt) sc + 10 mg abhrak bhasma/kg body wt po for 24 h.
Group 12- CCl_4 (3.0 ml/kg body wt) sc + 20 mg abhrak bhasma/kg body wt po for 24 h.
Group 13- CCl_4 (3.0 ml/kg body wt) sc + 30 mg abhrak bhasma/kg body wt po for 24 h.
Group 14- CCl_4 (3.0 ml/kg body wt) sc + 40 mg abhrak bhasma/kg body wt po for 24 h.
Group 15- CCl_4 (3.0 ml/kg body wt) sc + 10 mg SiO_2/ kg body wt po for 24 h.
Group 16- CCl_4 (3.0 ml/kg body wt) sc + 20 mg SiO_2/kg body wt po for 24 h.
Group 17- CCl_4 (3.0 ml/kg body wt) sc + 30 mg SiO_2/kg body wt po for 24 h.
Group 18- CCl_4 (3.0 ml/kg body wt) sc + 40 mg SiO_2/kg body wt po for 24 h.

The rats were killed after 24 h by giving deep ether anesthesia and liver and kidney tissues were separated from animals and taken for biochemical estimation.

Preparation of tissue homogenates

The liver and kidney were perfused with chilled phosphate buffer saline (PBS). They were dissected out, minced and washed with PBS. The minces were then suspended in 10 mM Tris-HCl homogenizing buffer (pH 7.0). The minces were homogenized with Potter-Elvehjam homogenizer with Teflon piston at 1500 RPM with 8 up and down strokes. The liver and kidney homogenates were centrifuged in refrigerated centrifuge at 4°C for 10 minutes at 3000 × g. The supernatants were collected and used for biochemical estimation.

Collection of serum

The blood was aspirated from the left ventricle with the syringe and was allowed to clot at room temperature in test tubes. On clotting the serum samples were obtained by centrifuging the clots using table top centrifuge. The colorless samples were stored at 10°C until use (within 6 h).

Biochemical estimation

Total lipid content in serum was estimated as per Frings *et al*., (1972)[10] and phospholipid content was estimated by method of Zilversmit and Davis (1950)[11].

Statistical analysis

The results were expressed as Mean ± SEM of different groups. The significant differences between groups were evaluated by one way analysis of variance (ANOVA) followed by student 't' test. The statistical calculations were carried out with the help of XLSTAT 7.5 computer programme. Values $P < 0.05$, $P < 0.01$ and $P < 0.001$ were considered to show statistical significance.

RESULTS AND DISCUSSION

In our preparatory data (not-presented) it was observed that single dose of CCl_4 induced toxicity in male albino rat is normalized after 72 h; if not treated by any of the drug/s. But simultaneous treatments of single graded doses of abhrak bhasma normalized the liver and kidney functions[12] and histology within 24 h[13]. Therefore present experimental design included data of 24 h that shows hepatoprotection by reducing the time interval of recovery.

Table 1: Effect of abhrak bhasma and SiO_2 on total lipid contents in liver, kidney and serum of male albino rats

Groups	Liver	Kidney	Serum
	mg/ 100 g Tissue	mg/ 100 g Tissue	mg/dl
Normal	6284.54 ± 106.14	1892.34 ± 62.13	148.43 ± 6.48
10 mg AB	6187.35 ± 84.27	1874.91 ± 56.74	157.68 ± 9.52
20 mg AB	5941.57 ± 115.36	1806.37 ± 68.19	148.54 ± 8.94
30 mg AB	6114.98 ± 134.69	1799.98 ± 92.34	152.54 ±10.67
40 mg AB	6111.32 ± 89.98	1784.78 ± 96.26	153.48 ± 7.69
10 mg SiO_2	5998.67 ± 154.69	1872.96 ± 93.39	146.65 ± 11.58
20 mg SiO_2	6036.67 ± 123.17	1846.26 ± 66.88	149.35 ± 8.69
30 mg SiO_2	6311.15 ± 148.00	2009.14 ± 89.16	162.25 ± 14.25
40 mg SiO_2	6565.52 ± 187.41	2084.33 ± 72.84	175.36 ± 9.18[a]

Values are mean ± SE of 6 animals. P values: a < 0.05; b < 0.01; c < 0.001 vs normal rats

Table 2: Effect of abhrak bhasma and SiO_2 on total lipid contents in liver, kidney and serum of CCl_4 intoxicated male albino rats

Groups	Liver	Kidney	Serum
	mg/ 100 g Tissue	mg/ 100 g Tissue	mg/dl
Normal	6189.54 ± 96.25	1912.07 ± 54.33	148.65 ± 6.98
CCl_4 [3.0 ml/kgBW]sc	7223.14 ± 221.41^{b}	2228.36 ± 107.65^{a}	214.68 ± 5.99^{c}
CCl_4 + 10 mg AB	6848.32 ± 195.41^{a}	2199.11 ± 97.34^{a}	191.68 ± 8.97^{b}
CCl_4 + 20 mg AB	6429.69 ± 195.14	2016.03 ± 91.44	168.39 ± 9.41^{ay}
CCl_4 + 30 mg AB	6288.68 ± 98.87^{y}	1987.22 ± 79.39	156.36 ± 11.69^{y}
CCl_4 + 40 mg AB	6078.22 ± 189.25^{y}	1936.35 ± 96.16	153.67 ± 8.47^{z}
CCl_4 + 10 mg SiO_2	7135.35 ± 195.42^{b}	2200.18 ± 96.13^{a}	205.68 ± 13.25^{b}
CCl_4 + 20 mg SiO_2	6989.56 ± 188.17^{b}	2018.36 ± 79.16	198.71 ± 10.24^{b}
CCl_4 + 30 mg SiO_2	6598.47 ± 141.32^{ax}	2088.19 ± 68.47	168.97 ± 9.38^{y}
CCl_4 + 40 mg SiO_2	6894.58 ±196.35^{a}	2192.14 ± 92.18^{a}	190.72 ± 14.25^{b}

Values are mean ± SE of 6 animals. P values: a < 0.05; b < 0.01; c < 0.001 vs normal rats; x < 0.05; y < 0.01; z < 0.001 vs CCl_4 treated rats

Table 3: Effect of abhrak bhasma and SiO_2 on phospholipid contents in liver, kidney and serum of male albino rats

Groups	Liver	Kidney	Serum
	mg/ 100 g Tissue	mg/ 100 g Tissue	mg/dl
Normal	1624.21 ± 94.51	1402.29 ± 75.91	112.54 ± 9.36
10 mg AB	1621.25 ± 125.35	1454.21 ± 74.57	115.64 ± 5.68
20 mg AB	1612.34 ± 128.47	1425.45 ± 36.54	122.21 ± 9.64
30 mg AB	1599.29 ± 95.47	1433.36 ± 54.68	118.54 ± 4.99
40 mg AB	1612.54 ± 108.52	1498.62 ± 101.68	116.35 ± 9.68
10 mg SiO_2	1631.21 ± 109.64	1408.68 ± 89.67	111.65 ± 10.65
20 mg SiO_2	1621.33 ± 136.21	1368.44 ± 53.64	113.51 ± 5.69
30 mg SiO_2	1588.68 ± 136.24	1388.39 ± 111.55	126.14 ± 10.58
40 mg SiO_2	1561.57 ± 128.59	1337.43 ± 106.5	128.65 ± 8.57

Values are mean ± SE of 6 animals. P values: a < 0.05; b < 0.01; c < 0.001 vs normal rats

Table 4: Effect of abhrak bhasma and SiO_2 on phospholipid contents in liver, kidney and serum of CCl_4 intoxicated male albino rats

Groups	Liver	Kidney	Serum
	mg/ 100 g Tissue	mg/ 100 g Tissue	mg/dl
Normal	1641.21 ± 104.32	1442.22 ± 56.21	116.14 ± 5.64
CCl_4 [3.0 ml/kg BW]sc	1181.25 ± 154.48^{a}	1372.69 ± 111.24	168.84 ± 12.25^{b}
CCl_4 + 10 mg AB	1498.10 ± 121.14	1414.87 ± 146.31	126.54 ± 8.34^{x}
CCl_4 + 20 mg AB	1604.32 ± 94.20^{x}	1431.16 ± 69.58	124.47 ± 6.58^{x}
CCl_4 + 30 mg AB	1632.54 ± 98.68^{y}	1441.63 ± 104.25	121.01 ± 6.32^{y}
CCl_4 + 40 mg AB	1601.61 ± 131.51^{x}	1443.09 ± 96.21	122.54 ± 5.69^{y}
CCl_4 + 10 mg SiO_2	1398.22 ± 114.64	1399.99 ± 96.35	154.58 ± 9.11^{b}
CCl_4 + 20 mg SiO_2	1495.19 ± 112.48^{x}	1412.12 ± 65.64	141.25 ± 11.35
CCl_4 + 30 mg SiO_2	1598.87 ± 101.45^{y}	1408.38 ± 108.12	131.54 ± 5.57^{x}
CCl_4 + 40 mg SiO_2	1588.31 ± 108.61^{y}	1397.54 ± 109.54	134.21 ± 6.37^{y}

Values are mean ± SE of 6 animals. P values: a < 0.05; b < 0.01; c < 0.001 vs normal rats; x < 0.05; y < 0.01; z < 0.001 vs CCl_4 treated rats

Alterations in total lipid contents

Total lipid contents in liver of normal group of albino rats was 6284.54 ± 106.14 mg per 100 g tissue; which was maintained at its normal level by treatments of all studied doses of abhrak bhasma. Similarly treatments of 10 mg, 20 mg, 30 mg and 40 mg doses of SiO_2 also protected normal lipid level in liver. The results of kidney lipids showed no influence of CCl_4 or all the studied doses of abhrak bhasma. Treatments of SiO_2 also showed effects similar to the effects of abhrak bhasma in total lipid contents of kidney. In serum of normal rats total lipid contents was 148.43 ± 6.48 mg/dl. Treatment of 10 mg, 20 mg, 30 mg and 40 mg doses of abhrak bhasma maintained lipid contents. Administration of 10 mg, 20 mg and 30 mg SiO_2 doses did follow the alterations in serum lipid contents as that of abhrak bhasma. While treatment of 40 mg SiO_2 dose showed significant rise ($P < 0.05$) as compared to the lipid contents of normal rat. These results indicated that abhrak bhasma administration to albino rats maintained normal state of lipid metabolism with reference to total lipid content as compared to SiO_2 treated control groups. But SiO_2 results deflected with 40 mg dose. These alterations confirm the SiO_2 influence at highest dose studied earlier viz. liver and kidney functions[12], Lipid peroxidation and glutathione[8,9] and histological alterations[4] in same experimental conditions. Administration of CCl_4 to the normal rats caused a significant increase ($P < 0.01$) in liver total lipid contents (by 1.16 fold). Treatments of 10, 20, 30 and 40 mg abhrak bhasma simultaneously with CCl_4 to the rats counteracted and showed progressive reduction in total lipid contents towards normal level; more significantly noted with 30 mg and 40 mg doses ($P < 0.01$) which showed significantly lowered lipid contents and brought to normal levels. Treatment of similar doses of SiO_2 simultaneously with CCl_4 administration showed similar trend of response as noted with abhrak bhasma treatments but with some variation with 30 mg and 40 mg doses that showed high contents with low significance than observed in normal rat. In kidney, increase ($P < 0.05$) in total lipid contents was noted with CCl_4 administration (by 1.16 fold); which was progressively counteracted by abhrak bhasma treatments and brought it to normal level with all

the doses. Minimum required dose to achieve normal levels was 20 mg. Treatments of 10 and 20 mg SiO_2 doses showed similar response as observed with abhrak bhasma treatments with low turnover. But 30 mg and 40 mg doses of SiO_2 treatments tend to increase total lipid contents; significance being ($P < 0.05$) with 40 mg dose. Administration of 3.0 ml CCl_4/ kg body wt; exhibited significant increase ($P < 0.001$) in total lipids levels in serum (1.44 fold). Treatments of various doses of abhrak bhasma against CCl_4 induced toxicity; showed progressive reduction in the total lipid contents of serum and achieved normal levels. Similar trend was noted with the administration of various doses of SiO_2 to the CCl_4 intoxicated rats; which have showed progressive reductions in serum total lipid contents and maintained normal levels. Especially with 30 mg dose which showed significant efficacy ($P < 0.01$) as compared to other SiO_2 doses. These observations suggest that CCl_4 administration to rats caused a significant increase in total lipids in liver and serum; which indicates the fatty degeneration of liver and hence alterations in functions of liver[12]. Increase in CCl_4 induced total lipid contents seems to be mainly due to the increased mobilization of free fatty acids from peripheral depots[5]. Treatment with abhrak bhasma stimulates the recovery and protection from CCl_4 induced hepatic damage that reflects decrease of elevated level of total lipid contents in liver. The lipid lowering effects are shown with more efficiency by abhrak bhasma than SiO_2. Kidney lipid contents were altered within the normal range with abhrak bhasma which is in agreement with earlier studied kidney functions[12], lipid peroxidation and glutathione[8,9] alterations. The reduced levels of total lipid mediated through abhrak bhasma, SiO_2 treatments may be due to reduced level of triglycerides, cholesterols, phospholipids and lipoproteins in CCl_4 treated rats[5]. Recovery and protection of liver and kidney functions[12] are indicators of membrane integrity so also the membrane dependent lipid metabolisms. Abhrak bhasma and SiO_2 effectively reduced the lipid levels altered by CCl_4 metabolism. The effect exerted by abhrak bhasma was more pronounced than SiO_2. Since abhrak bhasma assume to content high levels of silica since it is derived from mica[9]. The similar changes observed with SiO_2 can be credited to silica content. Kidney lipid contents though influenced by CCl_4/ abhrak bhasma/ SiO_2 (with combined or solo administration), they were in normal range. This indicates CCl_4/ abhrak bhasma/ SiO_2 in single doses hardly affect lipid contents and lipid metabolism in kidney. Alterations in serum are indicative of transporting status of total lipids. Since kidney lipid contents are not influenced by single doses of CCl_4/ abhrak bhasma/ SiO_2, the transporting status of total lipids is indicator of alterations occurring in lipids of liver alone and that also mainly from centrolobular region. Which shows accumulation of lipids[4]. As primary response of abhrak bhasma and SiO_2 with CCl_4 retards the fat accumulation which seems to be effect of silica either in the form of abhrak bhasma/ SiO_2. (Table 1 and 2)

Alterations in phospholipid contents

Phospholipids are the structural components of bio membranes and maintain the structural integrity of the hepatocellular membrane[3]. They play role in the molecular organization and the functional activity of membrane bound enzymes[3]. They are more susceptible to CCl_4 induced free radical CCl_3 leading to lipid peroxidation than other lipid classes[5] and alter the cellular structure of membrane bound enzymes by changing the membrane phospholipids and fatty acid composition. Thus the alterations are significant indicators of hepatocellular/kidney functions. The phospholipid content in normal rat liver was 1624.21 ± 94.51 mg/100 g tissue, which was not altered significantly by all the studied doses of abhrak bhasma in single dose experimental schedule of 24 h. Treatments of all doses of SiO_2 doses to the normal rat did not alter the phospholipid contents in normal rat. Thus abhrak bhasma or SiO_2 do not influence phospholipids of liver, kidney and serum of normal rats as single dose. Significant increase ($p < 0.01$) in the phospholipid content in serum and decrease in that of liver in CCl_4 administered rat was observed (28.02 %). These altered phospholipid contents were recovered/ protected to near normal level by abhrak bhasma in CCl_4 treated rats by 10, 20, 30 and 40 mg doses. SiO_2 treatments also showed similar protective results by 10 and 20 mg doses; while phospholipid contents in liver and serum were normalized only by 30 and 40 mg SiO_2 doses. Kidney phospholipids though influenced the turnover, they are maintained in normal range either by abhrak bhasma or by SiO_2 treatment. Total protection of liver from fatty degeneration[4] as observed in same experimental set up indicated membrane integrity of hepatocytes and hence that of liver functions. CCl_4 influenced increased serum phospholipid contents were progressively and significantly decreased by abhrak bhasma treatments and was maintained to the normal level. Thus all the doses of abhrak bhasma were equally potent to protect the normal phospholipids levels in serum. All doses of SiO_2 in CCl_4 intoxicated rats decreased phospholipid contents but 30 mg and 40 mg SiO_2 doses decreased phospholipid contents (by 22.09 % and 20.51 %) significantly ($p < 0.05$). Serum phospholipid levels increased by CCl_4 were protected by all the doses of SiO_2 except 10 mg dose. Thus abhrak bhasma (all the doses) and SiO_2 20, 30 and 40 mg doses protected the serum phospholipids levels. Thus abhrak bhasma seems to be more potent having lowest effective dose of 10 mg. The results showed marked elevation in phospholipid contents in serum and decrease that of in liver of CCl_4 intoxicated rats. The decreased level of phospholipids in liver indicated the alteration and disturbance in the phospholipid metabolism after the administration of CCl_4. It can be directly related to the centrolobular necrotic region induced by CCl_4 where cellular membranes damage is evident[4] in histological architecture. It may be positively correlated with the hepatic lipogenic enzyme activity or increase in phospholipase activity[14]. CCl_4 during biotransformation is known to generate CCl_3 which lead to fatty degeneration[5]. The phospholipids from liver cell membranes damaged by

CCl_3 released into blood stream by fatty degeneration resulted in rise of serum phospholipid contents. The considerable increase in the levels of phospholipids in liver in CCl_4 + abhrak bhasma treated rats suggests that abhrak bhasma protects the fatty degeneration evidenced by histological studies[4] and hence integration hepatocyte membrane phospholipids. As stated earlier, abhrak bhasma inhibits lipid peroxidation[8], reducing CCl_3 production and hence membrane damage reduction leading to prevent the release of phospholipids. Thus alterations in liver and in serum are justified. Based on the above results it can be concluded that abhrak bhasma possess hepatoprotective potency against CCl_4 toxicity. Results obtained with abhrak bhasma were found to be more effective than those of SiO_2. A comparative histopathological study of liver and kidney from above mentioned different groups also supported the hepatoprotective efficacy of abhrak bhasma[4]. Single doses of abhrak bhasma viz. 10, 20, 30 and 40 mg given independently hardly influence phospholipid contents in normal rat liver, kidney and serum[13]. Same was true with SiO_2 doses. Against CCl_4 toxicity induced phospholipid contents the minimum protective dose for liver are 10 mg of abhrak bhasma and SiO_2. Thus both the drugs are equally potent against single dose of CCl_4 as single dose. In protection of serum phospholipid levels induced by single dose of CCl_4; the minimum effective doses of abhrak bhasma and SiO_2 differ. Abhrak bhasma shows 10 mg minimum effective dose. For the same protective effects SiO_2 dose required is 20 mg. Thus it seems abhrak bhasma is more potent as compared to SiO_2 against CCl_4 induced alterations in phospholipid content. But the histological architecture[4] studied in similar dose schedules and experimental design showed hepatocytes hypertrophied which are adaptive alterations[15-17] with 30 mg and 40 mg SiO_2 doses along with same alterations in periarterial region. This was not observed with the any of the abhrak bhasma doses[4]. The differences in abhrak bhasma and SiO_2 influence observed in histological alterations also supported by their free radical scavenging potency; where abhrak bhasma is effective from 10mg dose onwards. While SiO_2 as 40 mg though showed tendency to scavenge free radicals but none of the doses showed full potency to scavenge CCl_4 induced free radicals[8]. Thus abhrak bhasma seems to be more efficient than SiO_2 since it is not only maintaining phospholipids contents but also maintains the normal hepatic and kidney histological architecture[4]. Thus SiO_2 though it is positively modifying phospholipid metabolism in presence of CCl_4 and not influencing it in normal metabolism, it alters some histological appearances. Therefore abhrak bhasma is more efficient than SiO_2. This also differentiates metabolism of not processed silica and processed silica by Shodhan and Maran used for the preparation of abhrak bhasma as described in Ayurveda. This also differentiates role of silica as SiO_2 on different metabolisms related with CCl_4 metabolisms. (Table 3 and 4)

ACKNOWLEDGMENTS

Authors are thankful to Dr. S. S. Patil for his contribution in abhrak bhasma preparation and other constructive suggestions.

REFERENCES

1. Timbrel JA. Biotransformation of xenobiotics. In General and applied toxicology ed. By B Ballentyne, Marrs T and P Turner Pub. by MacMillan press Ltd, London; 1995. p. 79-109.
2. http://www.lifetechnologies.com, The Molecular probes, Handbook, Fatty acid analogs and phospholipids – section 13.2
3. Alberts Bruce, Alexander Johnson, Juhan Lewis, David Morgan, Martin Ratt, Keith Roberts and Peter Walter. Molecular Biology of Cell. 6th Edition; 2014.
4. Teli Parashuram, Jadhav Jaywant and Kanase Aruna. Impact of abhrak bhasma and silicon dioxide on histological architecture of liver and kidney in single dose of CCl_4 intoxicated male albino rat. Int. J. Pharma Sci. and Res 2015; 6(01): 168-173.
5. Recknagel RO. Carbon tetrachloride hepatotoxicity. Pharmacol 1967; 19: 145-208.
6. Sharma S. Rasa Ratna Samuchhaya, Published by Motilal Banrasidas, New Delhi; 1977. p. 72-108.
7. Buwa S, Patil S, Kulkarni P, Kanase A. Hepatoprotective actions of abhrak bhasma in albino rats against hepatitis induced by CCl_4. Indian J. Expt. Biol 2001; 39: 1052-1057.
8. Teli Parashuram, Jadhav Jaywant, Kanase Aruna. Comparison of abhrak bhasma and silicon dioxide efficacy against single dose of carbon tetrachloride induced hepatotoxicity in rat by evaluation of lipid peroxidation. Am. J. Pharm Health Res 2014c; 2(7): 186-196.
9. Teli Parashuram, Jadhav Jaywant, Kanase Aruna. Effect of abhrak bhasma and silicon dioxide on hepatic and renal glutathione status in rats: hepato-protection testing against single dose of CCl_4 induced hepatotoxicity. Int. J. of Pharmacol and Toxicol. 2014d; 2(2): 92-94. http://dx.doi.org/10.14419/ijpt.v2i2.3288
10. Frings CS, Frendly TW, Dunn RT, Queen CA. Improved determination of total serum lipids by the sulpho-sphovanillin reaction. Clin. Chem 1972; 18(7): 673-674.
11. Zilversmit DB, Davis AK. Micro-determination of phospholipids by TCA precipitation. J. Lab. Clin. Med 1950; 35: 155-161.
12. Teli Parashuram, Chougule Priti, Jadhav Jaywant and Kanase Aruna. Abhrak bhasma mediated alterations in liver and kidney functions in male albino rats during CCl_4 induced toxicity. Int. J. Res. Ayurveda Pharm 2013; 4(5): 696-700. http://dx.doi.org/10.7897/2277-4343.04514
13. Teli Parashuram, Jadhav Jaywant, Thorat Dattatraya, Kanase Aruna. Primary responses of graded doses of mica derived Ayurvedic drug, abhrak bhasma and silicon dioxide on liver and kidney histology in male albino rat. J. Pharmacy Research 2014b; 8(7): 877-883.
14. Lamb RG, Snyder JW, Coleman JB. New trends in the prevention of hepatocellular death. Modifiers of calcium movement and of membrane phospholipid metabolism. In: Testa B, Perrissaud D, eds., Liver Drugs: From Exp. Pharmacol. to Therap. Appl. Boca Raton, CRC Press; 1988. p. 53-66.
15. Desmet VJ. Liver tissue examination. J Hepatol 2003; 39 Suppl 1: S43-9. http://dx.doi.org/10.1016/S0168-8278(03)00138-7
16. Lee WM. Drug induced hepatotoxicity. J. Med 2003; 349: 474-485.
17. Majno G and Joris I. Cells, Tissues and Disease, Principals of General Pathology, 2nd Edi. Oxford University Press, New York and Oxford; 2002.

Cite this article as:

Teli Parashuram, Jadhav Jaywant and Kanase Aruna. Reflections of abhrak bhasma mediated hepatoprotective effects on lipids and phospholipids in single dose of ccl4 intoxicated rat. Int. J. Res. Ayurveda Pharm. 2015;6(2):285-289 http://dx.doi.org/10.7897/2277-4343.06258

Source of support: Nil, Conflict of interest: None Declared