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HEMATOLOGICAL AND BIOCHEMICAL EFFECTS OF CURCUMIN IN SCHISTOSOMA MANSONI INFESTED MICE

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ABSTRACT

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In order to investigate the effect of curcumin on treatment of Schistosoma mansoni infested mice. Forty, male CD-1 Swiss albino mice used in this experiment, divided equally into 4 groups, the first kept as control, the second supplemented with curcumin, the third infected with *S.mansoni* and the 4th infected then treated with curcumin. The results emphasized on the presence of anemia leucopenia, neutropenia and esinophilia in infested groups either treated or not with somewhat an improvement in the treated group. The biochemical parameters showed that curcumin tries to restore the increased transaminases, alkaline phosphatase, antioxidants, triglycerides and low density lipoproteins to normal. Finally, curcumin treated group still closer to the infected group more than to the normal control group.

Keywords: *Schistosoma, Curcumin, Hematology, Leucocytosis, Biochemical, Antioxidants.*

INTRODUCTION

There is no doubt that schistosomiasis is one of the major communicable diseases which affecting human and animals either domestic or wild, as it comes secondly to the malaria with socio-economic and health importance in the developing world (Bergquist and Colley 1998). Schistosomiasis is a chronic debilitating parasitic disease in tropical and subtropical countries caused by *Schistosoma* species (Gryseels *et al.*, 2006). It is affecting about 200 million people infected worldwide and almost 600 million at risk.

Schistosoma is still one of the most prevalent epidemic disease in Egypt and in other developing countries in spite of many attempts to control this parasitic infection over many years (El-Khoby *et al.,* 2000). Schistosomiasis mostly affecting the liver and intestine causing granuloma formation, fibrosis and certain necrotic changes in the hepatic tissues (Elbanhawey *et al*., 2007).

Current treatment relies on praziquantel (PZQ) (Zhang and Coultas 2013). However, praziquantel does not treat early infection or prevent reinfection (Magnussen, 2003). In addition to, numerous evidences indicates to increasing the emergence of strains of *Schistosoma mansoni* resistant to praziquantel (Melman *et al*., 2009, Van der Werf, 2003 and Zhang and Coultas 2013).

In the last few years, there is an obvious increase in searching for antiparasitic drugs from natural sources, especially from plants, which are the main source of biologically active compounds for the development of new treatments (Magalhães *et al.,* 2009 and Silva *et al.,* 2009). One of these compounds is curcumin.

Curcumin is a yellow pigment from rhizomatous plant turmeric (*Curcuma longa*) widely cultivated in tropical and subtropical regions throughout the world, (Cerny *et al*., 2011). Extensive in vitro and in vivo studies have indicated that curcumin has a potent antitumor, anti-viral, anti-oxidant, and anti-inflammatory properties (Aggarwal and Harikumar, 2009 and Tu *et al*., 2011). Moreover, several recent reports showed that curcumin exerts beneficial effects in animal models of liver toxicity, inflammation and cirrhosis (Chen and Zheng, 2008 and Fu *et al.,* 2008).

Several reports revealed that curcumin enhances the hepatic detoxification by acting as a free radical scavenger, increases the glutathione/glutathione disulfide ratio to reduce oxidative stress and inhibits the activation and nuclear translocation of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) (Leclercq *et al*., 2004 and Reyes-Gordillo *et al.,* 2007). Recent study postulated that curcumin protects against hepatic fibrosis by inactivating hematopoietic stem cells (HSCs) through activation of peroxisome proliferator-activated γ- receptor, which interrupts platelet-derived growth factor and epidermal growth factor signaling in activated HSCs (Lin and Chen, 2008).

The first studies about the curcumin effects on *Schistosoma mansoni* showed the schistosomicidal effect of the oil extract of *C. longa* against *S.mansoni* infected mice (El-Ansary *et al.,* 2007). Morias *et al.* (2013) and Allam (2009) described in vitro and in vivo Schistosomicidal activity of curcumin against *S. mansoni* adult worms. Recently, El-Agamy *et al.* (2011) showed that curcumin has a potent anti-fibrotic activity in suppressing and reversing *S. mansoni*-induced liver fibrosis.

On the basis of anti-protozoal and anti-parasitic activity of medicinal plants and natural products, the aim of the present work was to evaluate the antishistosomal activity of curcumin against *Shistosoma mansoni.*

MATERIALS and METHODS

Chemicals:
All common chemicals used were purchased from one of the following suppliers Sigma Co. (St. Loius, MO, USA). All other chemicals and reagents were of the highest purity commercially available and were purchased from the British Drug Houses (BHD), Poole Dorset, UK. The diagnostic kits are purchased from Human Company (Germany).

Experimental animals:
Twenty healthy and other Twenty infected (with eighty *Schistoma mansoni* cercaria) male CD-1 Swiss albino mice (8-10 weeks of age) were used throughout the present study and were purchased from Theodore Bilharz Research Institute (TBRI, Imbaba, Giza, Egypt). All animals were maintained on standard commercial diet and water ad libitum.

Experimental design:
Animals were divided into four groups with 10 mice in each group, as the following:

Group I: healthy control group received normal diet (non-infected non-treated).

Group II: healthy received normal diet mixed with curcumin (300 mg/kg/day) (non- infected treated) from the 15th day after receiving the animals and continue for 4weeks.

Group III: infected and received normal diet (infected non-treated).

Group IV: infected and received normal diet mixed with curcumin (300 g/kg/day) (infected treated) from the 15th day after receiving the animals and continue for 4weeks.

Five animals from each group were sacrificed for sampling at the 2nd and 4th week post treatment.

Hematological examination:
Whole blood samples were collected from retro-orbital venous plexus of mice in a clean EDTA tube for determination of erythrocytic count (RBCs), hemoglobin concentration (Hb), haematocrit value (PCV), total and differential leucocytic count according to Feldman *et al.* (2000).

Biochemical Analysis:
Serum and liver homogenate were taken for all measurements. Serum samples were collected and stored at -20 ^{o} C until used. The liver was dissected out, washed in ice-cold saline, blotted dry, and weighed. Then homogenate was prepared in phosphate buffer 0.1M, pH 7.4 and used for the biochemical analysis.

Serum hepatic enzymes:
Activities of serum Aspartate transaminase (AST) and Alanine transaminase (ALT) were assessed according to Reitmans and Frankel (1957). alkaline phosphatase (ALP) was assayed by the kinetic methods of human kits (Germany) according to EDKC (1972). Total protein and albumin were measured according to Doumas *et al.* (1981). Serum globulin was calculated by subtracting the obtained albumin value from the total protein as described by Doumas and Biggs (1972). Tumor necrosis factor-alfa (TNF-a) and Alfa- fetoprotein was assayed using a commercial ELISA kit.

Estimation of serum lipids:
Total cholesterol (TC) measured according to (Richmond 1973), Triglycerides (TG), according to Wahlefeld and Bergmeyer (1974) and HDL-C was estimated according to Warnik *et al.* (1983) by the human kits (Germany). LDL- C were estimated by the formula of, Friedewald *et al.* (1972).
LDL- C= (total cholesterol) − (HDL-C) − (triglycerides/5)

Determination of non-enzymatic antioxidants
Reduced GSH was determined according to the method of Ellman (1959) based on the formation of a yellow coloured complex with Ellman's reagent (0.0198% DTNB in 1% sodium citrate). The color developed was read at 412 nm.

Assay of antioxidants enzymes:
Superoxide dismutase was assayed spectrophotometrically according to Paoletti and Mocali (1990). This method consists of purely chemical reaction sequence that generates superoxide from molecular oxygen in the presence of EDTA, manganese (II) chloride and mercaptoethanol. NAD H oxidation is linked to the availability of superoxide anions in the medium. The decrease in absorbance at 340 nm was monitored for 20 min at 5 min, One unit of SOD activity is defined as the amount of enzyme required to inhibit the rate of NADPH oxidation of

the control by 50%. Catalase assay was carried out according to the method of Aebi (1974). One unit was defined as that amount of the enzyme which converts 1 mole substrate to product in 1 sec.

Statistical analysis:
Results were expressed as mean ±standard error (S.E.). One-way analysis of variance (ANOVA) test by SPSS 16.0 for windows was carried out to test for any differences between the mean values of all groups. A value of $p < 0.05$ was interpreted as statistically significant Tamhane and Dunlop (2000). Means at the same row followed by different letters were significantly different and the highest value was represented with the letter (a).

RESULTS

The erythrogram, in the present work, showed microcytic hypochromic anemia in groups (III & IV) all over the experimental periods (Table 1). Group (II) showed an insignificant change in RBC count as compared to control group. Both of groups (III & IV) showed leucopenia as compared to control group although supplementation of curcumin to non-infected mice improved the WBCs count compared to control group (Table 2). On regarding the differential leucocytic count, our results revealed neutropenia, lymphopenia and eosinophilia in groups (III & IV) when compared with control group (Table 2). Supplementation of curcumin to group (II) insignificantly increased neutrophil, esinophil and lymphocytic count as compared to control group.

Table 1: Erythrogram in mice infested with *S. mansoni* and or treated by curcumin (mean ± .E).
Data are presented as mean ± SE, n = 5 and values which have different letters are significantly differs from each other at $p \leq 0.05$ using ANOVA test.

	Groups / Parameters	I	II	III	IV
2nd WEEK post treatment	RBCS (X10 ^{6} / UL)	6.56 ^{a} ±0.05	6.61 ^{a} ±0.06	4.74 ^{c} ±0.06	5.21 ^{b} ±0.4
	Hb (g/dl)	12.50 ^{a}±0.07	12.57 ^{a}±0.04	7.44 ^{b} ±0.17	7.30 ^{b} ±0.33
	PCV (%)	41.38 ^{a}±0.06	40.97 ^{a}±0.04	27.01 ^{c}±0.19	30.90 ^{b} ±0.28
	MCV (fl)	63.13 ^{a} 0.44	61.98 ^{a}±0.61	57.06 ^{c}±0.82	59.34 ^{b} ±0.46
	MCH (pg)	19.07 ^{a}±0.20	19.03 ^{a}±0.21	15.73 ^{b}±0.45	14.00 ^{c} ±0.55
	MCHC (gm/dl)	30.21 ^{a}±0.17	30.70 ^{a}±0.08	27.54 ^{b}±0.58	23.63 ^{c} ±1.10
4th week post treatment	RBCS (X10 ^{6} / UL)	6.11 ^{a} ±0.05	6.09 ^{a} ±0.03	4.28 ^{c} ±0.06	4.67 ^{b} ±0.03
	Hb (g/dl)	12.74 ^{a} ±0.12	12.53 ^{a}±0.08	7.23 ^{b} ±0.07	7.28 ^{b} ±0.15
	PCV (%)	39.38 ^{a} ±0.10	39.03 ^{a}±0.09	25.15 ^{c}±0.20	27.22 ^{b} ±0.23
	MCV (fl)	64.51 ^{a} ±0.67	64.10 ^{a}±0.40	58.72 ^{b}±0.93	58.37 ^{b} ±0.91
	MCH (pg)	20.87 ^{a} ±0.23	20.58 ^{a}±0.13	16.89 ^{b}±0.27	15.62 ^{c} ±0.36
	MCHC (gm/dl)	32.35 ^{a} ±0.29	32.10 ^{a}±0.28	28.76 ^{b}±0.37	26.75 ^{c} ±0.53

I: (non-infected non-treated group). **II**: (non- infected treated group)
III: (infected non-treated group). **IV**: (infected treated group)

Table 2: Leukogram in mice infested with *S. mansoni* and or treated by curcumin (mean ± S.E).

Data are presented as mean ± SE, n = 5 and values which have different letters are significantly differs from each other at $p \leq 0.05$ using ANOVA test.

parameters	groups	I	II	III	IV
2nd week post treatment	TLC (X10 3 / UL)	8.22[a]±0.20	8.45[a]±0.09	6.74[b]±0.12	6.72[b]±0.10
	LYMPHOCYTE (X10 3 / UL)	5.19[a]±0.13	5.22[a]±0.13	4.41[b]±0.08	4.60[b]±0.14
	NEUTROPHIL (X10 3 / UL)	2.85[a]±0.14	3.10[a]±0.06	1.57[b]±0.05	1.69[b]±0.07
	ESINOPHIL (X10 3 / UL)	0.13[c]±0.01	0.20[c]±0.01	0.68[a]±0.02	0.39[b]±0.04
	MONOCYTE (X10 3 / UL)	0.03[b]±0.01	0.02[b]±0.00	0.07[a]±0.00	0.03[b]±0.00
4th week post treatment	TLC (X10 3 / UL)	6.80[ab] ±0.34	7.18[a]±0.64	5.40[b]±0.53	5.78[ab] ±0.63
	LYMPHOCYTE (X10 3 / UL)	4.84[ab] ±0.33	5.09[a] ±0.60	3.11[b]±0.66	3.96[ab] ±0.69
	NEUTROPHIL (X10 3 / UL)	1.80 [a] ±0.14	1.88 [a]±0.09	1.38[b] ±0.18	1.34 [b] ±0.14
	ESINOPHIL (X10 3 / UL)	0.12 [c] ±0.01	0.18[c] ±0.04	0.81 [a] ±0.07	0.41 [b] ±0.06
	MONOCYTE (X10 3 / UL)	0.03 [c]±0.00	0.02[c] ±0.00	0.09 [a]±0.001	0.07 [b] ±0.01

I: (non-infected non-treated group). **II**: (non- infected treated group)
III: (infected non-treated group). **IV**: (infected treated group)

The significant ($p<0.05$) increase of serum AST, ALT, and ALP levels were observed in groups (III & IV) when compared with control group (Table 3). On the other hand, group (IV) showed reduction in the serum enzymes level as compared to group (III). Serum levels of total protein, albumin and globulin were reduced significantly in groups (III & IV) as compared to control group (Table 3). However Supplementation of curcumin to infected mice (group IV) resulted in elevation of total protein, albumin and globulin levels compared with infected non treated mice. Groups (III & IV) showed significant increases of AFP and TNF-a compared with control group. However supplementation of curcumin to infected mice improved the both AFP and TNF- α levels as compared to infected non-treated mice (Table 4).

Table 3: Serum liver enzymes and proteinogram of mice infested with *S. mansoni* and or treated with curcumin (mean ±S.E).

Data are presented as mean ± SE, n = 5 and values which have different letters are significantly differs from each other at $p \leq 0.05$ using ANOVA test.

parameters	groups	I	II	III	IV
2nd week post treatment	AST (IU/L)	22.41[c]± 0.29	21.36 [d] ±0.15	39.66[a] ±0.13	28.72 [b] ±0.35
	ALT(IU/L)	19.30 [c]±0.62	17.75 [c] ±0.35	29.89 [a] ±0.63	22.71 [b] ±0.58
	ALP(IU/L)	38.42 [c] ±0.24	37.44 [d] ±0.28	58.31 [a] ±0.36	44.32 [b] ±0.23
	Total protein g/dl	6.71[a]±0.06	6.79[a]±0.09	3.59[c]±0.05	4.61 [b] ±0.09
	Albumin g/dl	2.42 [a] ±0.21	2.67 [a] ±0.11	1.19 [b] ±0.14	1.61 [b] ±0.08
	globulin g/dl	3.94 [a] ±0.19	3.84 [a] ±0.14	2.13 [c] ±0.16	2.69 [b] ±0.09
4th week post treatment	AST (IU/L)	34.56 [c] ±0.56	35.44 [c] ±.58	67.82 [a]±0.79	47.85 [b] ±0.98
	ALT(IU/L)	18.50 [d] ±0.33	21.45 [c] ±0.34	68.52 [a] ±0.49	32.26 [b] ±0.35
	ALP(IU/L)	35.67 [c] ±0.57	32.17 [d] ±0.41	72.06 [a] ±0.62	46.43 [b] ±0.49
	Total protein g/dl	6.57[a]±0.15	6.78[a]±0.12	3.87[c]±0.11	4.89 [b] ±0.32
	albumin g/dl	2.40[a]±0.14	2.49[a]±0.11	1.46 [b] ±0.08	1.66 [b] ±0.06
	globulin g/dl	3.83[a]±0.18	3.95[a]±0.14	2.27[c]±0.15	2.94[b]±0.30

I: (non-infected non-treated group). **II**: (non- infected treated group)
III: (infected non-treated group). **IV**: (infected treated group)

Table 4: Serum AFP and TNF of mice infested with *S. mansoni* and /or treated with curcumin (mean ±S.E). Data are presented as mean ± SE, n = 5 and values which have different letters are significantly differs from each other at $p \leq 0.05$ using ANOVA test.

parameters \ groups		I	II	III	IV
2nd week post treatment	AFP	7.64^{c}±0.10	7.00^{c}±0.39	38.65^{a}±0.72	30.12^{b}±0.54
	TNF	12.45^{c} ±0.30	11.48^{c} ±0.32	33.32^{a}±0.50	24.71^{b} ±0.27
4th week post treatment	AFP	8.87^{c}±0.47	8.51^{c}±0.57	50.44^{a}±0.70	41.96^{b}±0.23
	TNF	12.35^{c} ±0.40	10.76^{c} ±0.37	48.41^{a} ±0.87	40.72^{b} ±1.08

I: (non-infected non-treated group). **II**: (non- infected treated group)
III: (infected non-treated group). **IV**: (infected treated group)

Groups (III & IV) showed increased levels of serum TC &TG and LDL-C as compared to control group (Table 5). There was significant decrease in HDL-C groups (III & IV) in the first period. On the other hand, curcumin supplementation to the infected mice lowered the serum TC &TG and LDL-C concentrations as compared to infected group.

Table 5: Serum lipid profile of mice infested with *S. mansoni* and /or treated with curcumin (mean ±S.E).

parameters \ groups		I	II	III	IV
2nd week post treatment	Cholesterol (mg/dl)	84.32^{c} ±1.21	79.80^{c} ±1.77	114.85^{a} ±3.70	99.52^{b} ±1.71
	triglycerides(mg/dl)	57.54^{c} ±1.12	51.11^{c} ±3.67	79.03^{a}±2.24	68.21^{b} ±1.93
	HDL-C (mg/dl)	16.18^{a} ±0.60	17.47^{a} ±0.69	13.25^{b} ±0.68	13.30^{b} ±0.91
	LDL-C (mg/dl)	56.63^{c}±1.32	52.21^{c} ±2.07	85.78^{a} ±3.54	72.73^{b} ±1.58
4th week post treatment	Cholesterol (mg/dl)	84.26^{c} ±1.01	81.86^{c} ±1.27	140.65^{a} ±1.61	114.73^{b} ±2.08
	triglycerides(mg/dl)	46.16^{c} ±0.70	45.48^{c} ±2.59	66.82^{a} ±1.87	52.70^{b} ±1.41
	HDL-C (mg/dl)	21.13^{a} ±0.71	22.42^{a} ±1.19	19.76^{a} ±0.76	20.27^{a} ±0.83
	LDL-C (mg/dl)	53.89^{c} ±1.53	50.34^{c} ±2.41	107.52^{a} ±1.20	83.91^{b} ±2.21

Data are presented as mean ± SE, n = 5 and values which have different letters are significantly differs from each other at $p \leq 0.05$ using ANOVA test.
I: (non-infected non-treated group). **II**: (non- infected treated group)
III: (infected non-treated group). **IV**: (infected treated group)

Results shown in (table 6) indicated that the content of reduced glutathione was significantly ($P < 0.05$) decreased in hepatic tissue of group (III) compared to control group (Table 6). Supplementation of diet with curcumin (group II) caused a significant ($P < 0.05$) increase in the content of reduced glutathione compared with both of control and infected non-treated groups (group I and group III). Infected mice supplemented with curcumin (group IV) partially restored the content of reduced glutathione to the normal values. Tables (6) showed that the activities of liver tissue SOD and CAT of group (III) significantly decreased ($P < 0.05$) as compared to control group. Treatment of non-infected mice with curcumin (group II) significantly increased the activities of liver tissue SOD and CAT as compared to that of group (III).

Table 6: Levels of GSH, SOD and CAT in liver tissue homogenates of mice infested with *S. mansoni* and /or treated with curcumin (mean ±S.E).

Data are presented as mean ± SE, n = 5 and values which have different letters are significantly differs from each other at $p \leq 0.05$ using ANOVA test.

parameters	groups	I	II	III	IV
2nd week post treatment	GSH	30.49 ^{b} ±0.29	32.44^{a}±0.30	13.48 ^{d} ±0.23	24.21 ^{c} ±0.21
	SOD	23.60^{a}±0.21	24.23^{a}±0.49	14.32 ^{c} ±0.24	17.55 ^{b} ±0.29
	CAT	67.33^{b}±0.27	70.47^{a}±0.40	53.39 ^{d} ±0.54	61.58 ^{c} ±0.40
4th week post treatment	GSH	34.70^{b}±0.29	35.81^{a}±0.33	18.77^{d}±0.41	25.71^{c}±0.23
	SOD	21.71^{a}±0.31	20.02^{b}±0.44	11.81^{d}±0.22	14.94^{c}±0. 27
	CAT	70.82^{a}±1.23	72.01^{a}±0.83	48.91 ^{b} ±1.02	51.75 ^{b} ±1.80

I: (non-infected non-treated group). **II**: (non- infected treated group)
III: (infected non-treated group). **IV**: (infected treated group)

DISCUSSION

The erythrogram, in the present work, showed microcytic hypochromic anemia in groups (III & IV) all over the experimental periods (Table 1). This sign marked from a significant decrease in all of the erythrocytic parameters (the mean values of RBCs count, PCV, Hb concentration, MCV, MCH and MCHC) in groups (III & IV all over the experiment as compared to control group, such decrease was more outstanding in infected non treated group (group III) than infected treated group (group IV). Our results are in agreement with that obtained by Abd EL-Mottaleb *et al*. (2008) and Nahla *et al*. (2008), who recorded a significant decrease in the erthrocytic count and blood indices accompanied with schistosoma infection.

On the contrary our results are disagree with that reported by Bugarski *et al*. (2006) who mentioned insignificant changes in any of the erythrogram parameters. This may be attributed to difference in parasitis species and the dose of parasitic infestation.

Administration of curcumin, to non-infected animal group (II) showed an insignificant change in RBC count as compared to control group. The infected mice treated with curcumin, showed an improvement in erythrogram values (PCV, MCV, MCH and MCHC) as compared to infected non-treated group. Sharma *et al*. (2011) proved that curcumin administration to infected mice improved the erythrocytic count, Hb and blood indices.

Decrease in RBCS count may be returned to the reduction in erythropoiesis in bone marrow and faster rate of destruction of peripheral RBC in spleen (Coles 1986). Decrease in Hb can be related to reduction in size of RBC, impaired biosynthesis of heam in bone marrow or due to reduction in the rate of formation of RBCS. Sturrock *et al*. (1996) attributed the presence of anemia to chronic blood loss that result from the bleeding induced by migration of worms through intestinal wall or due to blood consumption by adult schistosomes.

Table (2) illustrates total (TLC) and differential leukocyte count (Lymphocyte, Neutrophil, Esinophil and Monocytic count) in control and experimental groups of animals. Both of infected non-treated and infected treated groups showed leucopenia as compared to control group although supplementation of curcumin to non-infected mice improved the WBCs count compared to control group. These results agree with El-sheikha *et al*. (2008) who recorded significant decrease in total leucocytic count in infected mice with Schistosomiasis. In contrast to this result, Abd EL-Mottaleb *et al.* (2008) and Willingham *et al.* (1998) who noticed non-significant change in total leucocytic count in all experimental groups. Allam 2009 demonstrated that infected treated mice showed insignificant alteration in total leukocytic count. The difference may be due to difference of infestation dose and or experimental period. Supplementation of curcumin to non-infected mice showed insignificant increase in total leucocytes.

These results agree with Antony *et al.* (1999) who proved that Curcuma longa extract administration increased the total lecuocytic count in Balb/c mice due to the immune-stimulating activity of Curcumin.

On regarding the differential leucocytic count, our results revealed neutropenia, lymphopenia and eosinophilia in groups (III & IV) when compared with control group (Table 2). Similar results were obtained by Bugarski *et al.* (2006) who reported a significant neutropenia and esinophilia. Also these results are in accordance with that obtained by Abd EL-Mottaleb *et al*. (2008), Sharma *et al.* 2011, Ver cruysse *et al.* (1988) and Nahla *et al.* (2008) who found neutropenia, lymphopenia and eosinophilia accompanied parasitic infection. Simultaneously, a significant neutropenia and lymphopenia were observed, which could be ascribed to the recruitment of these cells to the site of the infection (Bugarski *et al*., 2006). Otherwhile Abd EL-Mottaleb *et al*. (2008) mentioned that the eosinophilia may be due to the powerful defense reaction and allergic manifestation against *Schistosoma mansoni* and their eggs. From the same side, animals were primarily characterized by the appearance of eosinophilia, which was not unexpected since eosinophilia is the most frequent response to helminths Klion and Nutman (2004).

Supplementation of curcumin to non-infected mice insignificantly increased neutrophil, esinophil and lymphocytic count as compared to control group. The results showed insignificant changes of neutrophil, leucocyte and eosinophil in infected treated mice group when compared to infected non-treated group. Sharma *et al*. 2011 recorded that curcumin may stabilize the cell membrane and restore various blood variables.

Hepatic damage can affect the metabolic processes in the body due to the role of liver in general metabolism. Enzymes are necessary for normal cellular metabolism including that of the liver (Rajamanickam and Muthuswamy, 2008). Hepatoprotective activity of curcumin was evaluated on *Schistosoma mansoni* infected mice by estimation of serum hepatic enzymes. Hepatic cells appear to participate in a variety of enzymatic metabolic activities. Infection of *Schistosoma mansoni* damages the hepatic cells leading to a significant increase in serum levels of AST, ALT, and ALP respectively (Table 3). The significant ($p<0.05$) increase of serum AST, ALT, and ALP levels were observed in group (III) when compared with control group. On the other hand, group (IV) showed reduction in the serum enzymes level as compared to group (III).

These results are in agreement with previously reported by El-Gowhary *et al*. (1993). Allam (2009) reported that, infected mice treated with curcumin restore the hepatic ALT and AST activities that were decreased by *S. mansoni* infection. This amelioration in the activities of liver enzymes could be attributed to the reduction in hepatic granuloma size and fibrosis as well as absence of necrotic hepatic tissue in infected treated mice (Allam, 2009). Apparently it appears that the membrane damage seems to be the prime culprit for the marked increase in the serum marker enzymes, AST, ALT, and ALP (Naik *et al*., 2011).

Serum levels of total protein, albumin and globulin were reduced significantly in group (III) as compared to control group (Table 3). However Supplementation of curcumin to infected mice (group IV) resulted in elevation of total protein, albumin and globulin levels compared with infected non treated mice. Similar observations were noticed by El-Ansary *et al.* (2007) and El-Emam *et al.* (2011). These results supported through the work of El-Heig *et al*. (1977) who recorded a marked decrease of protein content in *S. mansoni* infected mice.

Alfa-fetoprotein (AFP) is a glycoprotein, of unknown function, normally produced during neonatal development by the liver and in small concentrations by the gastrointestinal tract (Abelev *et al*., 1963). Abnormal serum level of AFP has been reported in patients with liver cirrhosis and hepatocellular carcinoma (Gupta *et al.,* 2003). So our work was extended to observe the effect of both *Schistosoma* infection and administration of curcumin on serum alfa-fetoprotein (AFP) and, tumor necrosis factor-alfa (TNF- α) (Table 4). Infected non treated mice showed significant increases of AFP and TNF-a compared with control group. However supplementation of curcumin to infected mice improved the both AFP and TNF- α levels as compared to infected non-treated mice. These results agreed with that observed by El-Rigal *et al*. (2011), Who recorded elevated level of AFP in sera of *S. mansoni* infected mice which may be considered as an index for liver fibrosis related to Schistosomiasis.

Tumor necrosis factor-alpha (TNF-α) is a cytokine involved in systemic inflammation and is a member of a group of cytokines that stimulate the acute phase reaction. Torben and Hailu (2007) stated that increased level of this inflammatory cytokine after egg excretion may be an indication of its effect in complications of Schistosomiasis, it is capable of inducing tissue injury and fibrosis. The results showed in table (4) indicated that, TNF-α is increased in serum of infected non treated mice compared to control group. The infected mice group treated with curcumin showed an improvement in serum TNF-α level compared with infected non treated mice. These results agree with that obtained by El-Rigal *et al.* 2011 and Allam 2009 who observed that infected

mice treated with curcumin revealed low serum level tumor necrosis factor alpha (TNF-a).

In order to investigate the hypolipidemic effects of curcumin on *S. mansoni* infected mice, quantitative assay of lipid profile was conducted by measuring the concentrations of serum total cholesterol (TC) & triglycerides (TG) and lipoproteins HDL-C and LDL-C. The infected non trated mice showed increased levels of serum TC &TG and LDL-C as compared to control group (Table 5). There was significant decrease in HDL-C in the first period in groups (III & IV). On the other hand, curcumin supplementation to the infected mice lowered the serum TC & TG and LDL-C concentrations as compared to infected group. These results are in accordance with that obtained by Arafa (2005) while opposite to that reported by Baum *et al*. (2007).

Similar observation were recorded by Godkar *et al*. (1996) who investigated that supplementation of curcumin in diet of Swiss mice caused a marked decrease of serum TC &TG level. The mechanism by which curcumin decreased serum cholesterol in previous study is not known. One hypothesis is that curcumin prevents the increase in serum cholesterol in the animal studies by inhibiting dietary cholesterol absorption (Arafa, 2005). Curcumin was reported to cause a little increase of plasma HDL-C in rats (Arafa, 2005). Otherwise Kang and Chen (2009) provide a novel insights into the roles and mechanisms of curcumin in lowering the level of LDL-C that include curcumin suppressed LDL-R receptor gene expression in activated hepatic stellate cells.

Reduced glutathion (GSH) are thought to play a vital role in protecting cells against reactive oxygen. During the metabolic action of GSH, its sulfhydryl group becomes oxidized resulting with the formation of corresponding disulfide compound, GSSG. Thus depletion of GSH content is associated with an increase in GSSG concentration resulting with the depletion in GSH/GSSG ratio. The content of reduced glutathione was significantly ($P < 0.05$) decreased in hepatic tissue of infected non treated mice group compared to control group (Table 6). Supplementation of diet with curcumin (group II) caused a significant ($P < 0.05$) increase in the content of reduced glutathione compared with both of control and infected non-treated groups (group I and group III). Infected mice supplemented with curcumin (group IV) partially restored the content of reduced glutathione to the normal values. Similar protective effect of curcumin pretreatment that showed a powerful antioxidant effect; it notably elevated GSH concentration and attenuated cellular ALT and AST released from hepatocytes reported by Naik *et al.* (2004). The decreases in GSH level of infected mice is in agreement with the findings of Leelank and Bansal (1996), who reported GSH depletion decreases the GSH/GSSG ratio and production of free radicals. These free radicals interact with membrane lipids leading to the production of lipid hydroperoxides.

Tables (6) showed that the activities of liver tissue SOD and CAT of infected non treated mice significantly decreased ($P < 0.05$) as compared to control group. Treatment of non-infected mice with curcumin (group II) significantly increased the activities of liver tissue SOD and CAT as compared to that of infected mice. In addition, a significant recovery relating to liver tissue SOD and CAT was observed in infected mice supplemented curcumin (El-Demerdash *et al*., 2009). Also, Rizk (1998) and Allam (2009) reported that catalase activity was enhanced in infected mice treated with curcumin. The antioxidant enzymes superoxide dismutase and catalase play an important role in keeping homeostasis and protection against oxidative damage by removing the toxic free radicals in vivo (EI Shenawy *et al*., 2008 and Jia *et al*., 2009). Recently, Rizk *et al.* (2012) noticed that the reduction in catalase activity could be attributed to its utilization in scavenging the free radicals overload which generated during Schistosomiasis. A decrease of SOD activity can be resulted from increased removal of superoxide anions (Sharma *et al.,* 2005). The levels of antioxidant enzymes are known to be elevated in cells in response to free radical production (Bandyopadhyay *et al.*, 1999).

These results coincide with that of Cerny *et al*. (2011) who observed plasma catalase activity as a marker of oxidative stress was 2.4-fold elevated as compared to control and this level further increased to 3-fold following curcumin treatment. Priyadarsini (1997) and Masuda *et al*. (1999) indicated that the exact mechanism of antioxidant activity of curcumin is not clear, while it is known to react with glutathione and also undergo dimerization by interacting with free radicals. Naik *et al*. (2011) and Kurup *et al*. (2007) attributed the antioxidant property of curcumin extract to the presence of chemical groups like hydroxyl methoxy and 1,3-diketone conjugated diene system. Naik *et al*. (2011) believed that the antioxidant activity of curcumin might be directly or indirectly associated with the maintenance or preservation of membrane integrity, which might help to prevent the elevation of serum marker enzymes observed during inflammation.

According to our results we concluded that curcumin, could not be used as an anti-parasitic whereas it only improves the alterations of hematological, biochemical, antioxidants parameters previously induced in schistosoma mansoni infected mice.

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تأثير الكركم على صورة الدم، والكيمياء الحيوية في الفئران المصابة بالبلهارسيا المعوية

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هدفت هذه الدراسة إلى دراسة تأثير الكركم على علاج البلهارسيا في الفئران المصابة بالبلهارسيا. واستخدم في هذه التجربة أربعين فأرا قسمت بالتساوي إلى أربع مجموعات ، أبقيت الأولي كمجموعة ضابطة ، والثانية تم تجريعها الكركم ، والثالثة والرابعة تم إصابتهم بالبلهارسيا المعوية مع علاج الرابعة بالكركم. أكدت نتائجنا على وجود فقر الدم ، نقص في الكريات البيضاء ، نقص الخلايا المتعادلة و الخلايا الحامضية في المجموعات المصابة سواء المعاملة أو غير المعاملة بالكركم مع تحسن في المجموعة التي تلقت العلاج. وأظهرت القياسات البيوكيميائية تحسن في مستويات الالانين امينو ترانسيفيريز، الاسبرتيت امينو ترانسيفيريز ، الألكالين فوسفاتيز ، البروتين الكلي ، الزلال ، الألفا فيتو بروتين ، عامل نخر الورم. كما اظهرت نتائج صورة الدهون تحسن في الكوليسترول الكلي والجليسريدات الثلاثية والبروتينات الدهنية عالية الكثافة والبروتينات الدهنية منخفضة الكثافة بشكل ملحوظ في الفئران المصابة التي عولجت مقارنة مع الفئران المصابة وغير معالجة. وبنفس الوقت أظهرت النتائج تحسنا في بعض مضادات الأكسدة (أنزيم السوبر اكسيد دسمياتيز ، وانزيم الكاتاليز ومواد الجلوتاثيون المختزل) في الفئران المصابة التي عولجت بالكركم.