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Effect of lead on some haematological and biochemical characteristics of *Clarias gariepinus* dietary supplemented with lycopene and vitamin E

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ASTRACT

The present study aimed to investigate the potential protective effects of lycopene (9 mg/kg) in comparison with vitamin E (50 mg/kg) against the impacts of lead (Pb) toxicity(7 mg/l: 1/4 of 96 h LC50) on fishes Pb exposed for 15 and 30 days. Pb impacts were evaluated in terms of haematological and biochemical characteristics. The activities of aspartate aminotransferase and alanine aminotransferase in serum were significantly increased due to Pb. Treatment with Pb caused a significant increase in mean corpuscular haemoglobin concentration, serum glucose, total lipid,urea and creatinine and Lipid peroxidation in liver,kidney and gills tissues. On the other hand, Pb significantly caused decline in serum total protein,albumin, blood haemoglobin, red blood cell count, haematocrit value, mean corpuscular volume and mean corpuscular haemoglobin . It was observed that supplementation of lycopene and vitamin E decreases the toxic effect of lead.

Keywords: Fishes, Lead, Lycopene, Vitamin E, Haematological, Biochemical

INTRODUCTION

Fish are widely used to evaluate the health of aquatic ecosystems because pollutants build up in the food chain and are responsible for adverse effects and death in the aquatic systems (Yousuf and El-Shahawi, 1999; Farkas *et al.,* 2002). The studies carried out on various fishes have shown that heavy metals may alter the physiological activities and biochemical parameters both in tissues and in blood (Tort and Torres, 1988; Canli, 1995; Basa and Usha Rani, 2003). The level of heavy metals in the water and in the sediment of some parts of the river Nile is higher than the tolerance levels or limits set by the Egyptian General Authority for Standards and Quality Control (Anwar, 2003).

Lead (Pb) is one of the most dangerous pollutants in our environment which accumulates in the body due to its low rate of elimination (Harrison and Winchest, 1971). Lead enters aquatic systems from urban, mining and agricultural runoff, atmospheric precipitation, plating process, the use of phosphate fertilizers and gasoline containing lead that leaks from fishery boats and a variety of natural sources, including erosion and volcanic emissions (Denny *et al.*, 1987; Handy, 1994). Several reports have indicated that Pb can cause neurological, hematological, gastrointestinal, reproductive, circul-atory, immunological, histopa-thological and histochemical changes all of them related to the dose and time of exposure to Pb (Falke and Xwennis, 1990; Royce *et al*., 1990; Park *et al*., 2006; Patrick, 2006; Ademuyiwa *et al*., 2007; Berrahal *et al*., 2007; Farrag *et al*., 2007; Abdallah *et al*., 2010). Also,Lead has many undesired effects, including behavioral (Shafiq-ur-Rehman, 1991), respiratory (Hillam and Ozkan, 1986), visual (Winneke *et al*., 1988), growth retardation (Shukla *et al*., 1991), renal (Vyskocil *et al*., 1989, 1991), hepatic (Honchel *et al*., 1991; Hao *et al*., 2002) and reproductive dysfunction (Marchlewicz *et al*., 1993).and it was

reported that lead increased the level of lipid peroxidation (Upasani *et al*., 2001). Lead for example causes renal failure and liver damage (Emmerson, 1973).

Haematological variables are environmentally sensitive and so are used to determine the effects of external stressors and toxic substances (Wendelaar Bonga, 1997). In fish, exposure to chemical pollutants can induce either increases or decreases in haematological levels. Damage to blood and heamapoietic organs in fish may be induced by changes in environmental conditions (Dewilde and Hauston, 1967; Gardner and Yevich, 1969) or water born pollutants (Reichenbach-klink, 1966; Gardner and Yevich, 1969). Blood cell morphology, distribution and indices such as red blood cells (RBCs), hemoglobin (Hb), package cell volume (PCV), mean corpuscular hemoglobin concentration (MCHC), platelets, white blood cells count (WBCs), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH) are good indicators of systemic response to external stress (Srivastava and Choudhay, 2010).

The biochemical parameters in fish are valid for physiopathological evaluation and sensitive for detecting potential adverse effects and relatively early events of pollutant damage (Juneja, and Mahajan, 1983; Ranzani-Paiva *et al.,* 1999; Almeida *et al.,* 2002; Matos *et al.,* 2007; Osman *et al.,* 2010). Many studies have investigated changes in many physiological and biochemical blood indices induced by environmental conditions and the presence of contaminants (Kori-Siakpere, 2006; Maheswaran *et al.,* 2008 and Ololade and Oginni, 2010).

Antioxidants are substances that may protect cells from the damage caused by unstable molecules known as free radicals. Antioxidants interact with and stabilize free radicals and may prevent some of the damage free radicals might otherwise cause. Free radical damage may lead to cancer. Examples of antioxidants include beta-carotene, lycopene, vitamins C, E, A and other substances (Sies, 1997).Vitamin E is a fat-soluble vitamin that exists in 8 forms. Each form has its own biological activity, which is the measure of potency or functional use in the body (Traber and Packer, 1995). VE has also been shown to play a role in immune function, in DNA repair, and other metabolic process (U.S. Department of Agriculture, 2004). VE functions to protect membrane lipids from damage (Frei, 1991).

Lycopene, a fat soluble carotenoid, is a precursor of b-carotene (Sandmann, 1994) and has at least twice the antioxidant capacity of β -carotene (Di Mascio *et al*., 1989). These naturally occurring antioxidants play important roles in animal health by inactivating harmful free radicals produced through normal cellular activity and from various stressors (El-Demerdash *et al.*, 2004).

MATERIALS AND METHODS

Specimens collection and treatment manipulation

Eighty healthy fish of The Nile catfish, *Clarias gariepinus* (200-300 g) in weight, (33-37cm) in length were caught from the River Nile at Assiut, Egypt. Fishes immediately were transported to the fish laboratory in the Department of Zoology, Faculty of Science, Assiut University.The experimental fishes were reared in aerated glass tanks (160 L capacity) and divided into 8 groups(10 fish /tank) and acclimatized for two weeks before being used in the experimental study. The experimental fish fed pellets at a rate of 3% of wet weight twice daily. Dead fish were removed and recorded daily .Faeces and residual food were aspirated regularly. The water temperature, p*H* and dissolved oxygen (DO) concentration were measured daily (26±.4 ºC, 7.25±.36 pH and 6.12±.19 mg L-1 DO).

Preparation of tomato paste to adjust the lycopene dose

Tomatoes used for the experiment were obtained from the local market. Fresh peeled, deseeded tomatoes were pulped well to a smooth consistency in a warring blender. The lycopene content in tomato paste was estimated spectrophotometrically according to the methods of Ranganna (1976) and Choudhari and Ananthanarayan (2007). The lycopene concentration in the tomato paste was 30.028 mg/100 g. (Okajima *et al.* 1998). Based on the review of Xianquan *et al*. (2005), such concentration could not be affected by current conditions of diet preparation and storage of a short time (37ºC for 4 weeks). In addition to lycopene, tomato paste composition include water, proteins, carbohydrates, fibres, calcium, potassium, zinc, copper, manganese, iron, vitamin C, vitamin E, b-carotenoids and other phytonutrients.

Experimental design

Fishes were weighed, measured and classified randomly into 8 groups (10 fish/tank) according to dose of lead, tomato paste in terms of lycopene, vitamin E and their combinations (Table 1). The diets (maize and soy bean, 15 g/kg fish) were pelleted after addition of vitamin E and tomato paste doses for the treated groups and the addition of suitable amounts of molasses and water. The diets were dried at room temperature and stored in small bags for fish feeding.

Table 1: The fish groups exposed to lead (7mg/l) and lycopene (9mg/kg body weight) and vitamin E (50 mg/kg body weight) and their combinations.

Treatments	C	VE	LYC	LYC+VE	Pb	Pb+VE	Pb+LYC	Pb+LYC+VE
Lead (mg/L)	0	0	0	0	7	7	7	7
Vitamin E (mg/kg)	0	50	0	50	0	50	0	50
Lycopene (mg/kg)	0	0	9	9	0	0	9	9

C= control, VE= vitamin E, LYC= lycopene and Pb= lead dose

Stock solution (1,000 ppm) of lead as lead nitrate Pb $(NO_3)2$ was prepared and stored in clean glass bottles and diluted to concentration of 7 mg/l. Such low sublethal lead concentration (1/4 of 96 h LC_{50}) was chosen according to levels monitored by Adeyemo *et al*., (2007). Lead doses were prepared and added constantly to the aquarium for 4 weeks. The test water was replaced daily with the required amount of stock solution to prevent deterioration of water quality and replenish cadmium levels. Tomato paste was added to the diet in concentration of 30 g/kg BW (9 mg lycopene/kg BW). Dose response of lycopene was described previously by Rodriguez *et al*. (2004). Also, vitamin E (α -tocopherol) was supplemented in 50 mg/kg BW. Such vitamin E concentration was chosen according to levels monitored by Ortun˜o *et al*. (2001). It is worthy to mention that vitamin E (α tocopherol) in tomato paste was estimated to be 38.67 ± 2.29 mg/100 g tomato paste dry weight with no effect by industrial processing (Capanoglu *et al.,* 2008).

Blood analyses

After 15 and 30 day periods, blood samples of the control and treated fish (4 fish/treatment) were collected from caudal vein of the fish in a small plastic tubes containing heparin solution (0.2 ml/ml blood) as anticoagulant. These blood samples were used for determining erythrocyte count (Dacie and Lewis 1984) using haemocytometer. Haemoglobin (Hb) was estimated where it was converted into red cyanometh-aemoglobin under the influence of potassium ferricyanide and potassium cyanide (Vankampen 1961). Haemoglobin level was determined by using suitable kits (Diamond Diagnostics, Egypt) according to

Stoskopf (1993). Haematocrit value (Hct) was calculated according to the formulae mentioned by Stoskopf (1993). Mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were calculated according to Cloes (1980).

Some other blood samples were collected and left to coagulate for 15–20 min at 4 prior to centrifugation or 20 min at 3,000 rpm to separate serum. The fresh serum was subjected to biochemical analysis. Serum glucose (mg/l) was determined, using assay kits supplied by (Spectrum Diagnostics, Egypt). Total protein (g/100 ml) content and total lipids (g/l) contents were determined colorimetrically using assay kits supplied by Diamond Diagnostics, Egypt. Activities of aspartate aminotransferase (AST, U/I) and alanine aminotransferase (ALT, U/I) were determined colorimetrically using assay kits (Spectrum Diagnostics, Egypt) according to Reitman and Frankel (1957). The samples were measured by spectrophotometer (Ultroscopec 3100 Pro). Also, serum urea and creatinine were estimated using kits supplied from Biomerieux (France).

Lipid peroxidation measurement

Lipid peroxidation was indirectly measured according to the method of Ohkawa *et al*. (1979) (thiobarbituric acid reactive substance, TBARS, test). Ten (w/v) tissue homogenate from liver,kidney and gills was used (this homogenate contained 1% v/v dimethyl sulfoxide to prevent further oxidation). Tissue homogenate aliquots of 0.2 ml was added to 0.2 ml 8.1% w/v sodium dodecyl sulphate solution followed by 1.5 ml 20% v/v acetic acid and 1.5 ml 0.8% thiobarbituric acid. The mixture was made up to 4.0 ml with distilled water and heated to 95ºC for 1 h. The samples were cooled, centrifuged at 2,000 rpm for 10 min. and measured at 532 nm using spectrophotometer (Ultroscopec 3100 Pro). The results were expressed as nmol malondialdehyde formation per g tissue.

Statistical analysis

The basic statistics, means, standard errors and ranges of the measured parameters were estimated. The patterns of variation due to lead, lycopene and vitamin E doses and their combinations were studied by three- and four-way analysis of variance using the SPSS package (SPSS 1998) at the 0.05 significance level. Levene's test of equality of error variance of the dependent variables was applied, with rejection of the null hypothesis for raw, log-transformed and SQRT-transformed data. So, the homogeneity of variance was assumed for raw data. The pattern of variations was also recorded by one-way analysis of variance, revealing significant difference due to lead, lycopene and vitamin E (P\0.0001); The Tukey-HSD test was considered for multiple comparisons.

RESULTS

Haematological parameters

Haematological parameters the normal values of red blood cell (RBCs) count, haemoglobin content and haematocrit value of *C. gariepinus* for 15 and 30 days periods are given in Tables (2 & 3) and Figs. 1-6. The lead and lycopene main effects were highly significant (P\0.0001) in both periods for RBCs, Hb and Hct .No significant main effect of vitamin E was recorded in both periods for RBCs, Hb and Hct. Pb-VE main effect was highly significant (P<0.0001) in both periods. Pb-LYC-VE interaction main effect was significant in both exposure periods. The time of exposure main effect was not significant and a positive correlations were recorded between similar RBCs, Hb and Hct in the two periods (r RBCs= 0.959, r Hb = 0.961 and r Hct= 0.978).

The normal value of mean corpuscular value (MCV), mean

corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) of *C. gariepinus* in the two periods are given in Tables 2 & 3. The lead main effect was highly significant (P<0.0001) for MCV and MCHC in the two periods. Lycopene main effect was significant only for MCV. No significant main effect of vitamin E was recorded at both periods for the three previous parameters. Pb-VE main effect was significant in both periods for MCV and for MCHC at the 15-day period. Pb-LYC main effect was significant in both periods for MCV and for MCHC at the 30-day period. Pb-LYC-VE interaction showed a significant effect for MCV and MCHC at the 30-day period and for MCH at the 15-day period. LYC time of exposure effect was not significant in MCH and MCHC and significant in MCV.

Table 2: The basic data(N=4) of blood constituent parameters of *C. gariepinus* exposed to lead (Pb),Vitamin E(VE),Lycopene (LYC) and their combinations for 15 day.

Treatments / Parameters	Control	LYC	VE	LYC+VE
	Mean ±SE (Min-Max)	Mean ±SE (Min-Max)	Mean ±SE (Min-Max)	Mean ±SE (Min-Max)
HB (g/100ml)	17.08±.34 ^{C} (16.46-17.71)	18.94±.06 ^{D} (18.82-19.08)	17.28±.02 ^{C} (17.23-17.33)	17.3±.19 ^{C} (16.94-17.67)
RBC x (10^6 /mm^3)	2.46±.02 ^{CD} (2.41-2.51)	2.63±.01 ^{E} (2.61-2.66)	2.52±.02 ^{D} (2.46-2. 59)	2.47±.02 ^{D} (2.42-2.53)
Hct (%)	37.31±.15 ^{C} (36.09-38.59)	42.54±.27 ^{D} (42.03-43.05)	39.32±.53 ^{C} (38.25-40.8)	38.36±.53 ^{C} (37.24-39.48)
MCV (µm^3/cell)	151.51±.92^{CD} (149.75-153.74)	151.51±1.19 ^{E} (158.27-163.68)	155.77±2.4 ^{DE} (150.96-160.62)	154.99±1.79^{DE} (150.76-159.3)
MCH (µg/cell)	69.36±1.1 ^{BC} (66.84-71.78)	71.89±.2 ^{C} (71.4-72.35)	68.45±.72 ^{AB} (66.71-70.24)	69.92±.8 ^{BC} (67.96-71.91)
MCHC(%)	45.77±.54 ^{AB} (44.48-47.13)	44.53±.32 ^{AB} (43.85-45.32)	43.95±.53 ^{A} (42.47-45.04)	45.11±.14 ^{AB} (44.75-45.48)
AST	57.9±1.27 ^{A} (55.34-61.22)	87.56±.82 ^{C} (85.44-89.16)	69.1±2.59 ^{AB} (63.21-74.68)	72.27±1.15 ^{B} (69.19-74.14)
ALT	26.95±.86 ^{C} (24.62-28.77)	17.91±.25 ^{A} (17.22-18.33)	24.45±.74 ^{BC} (22.75-26.27)	21.54±.7 ^{AB} (19.63-22.81)
TP	4.8±.05 ^{BC} (4.71-4.95)	6.51±.17 ^{E} (6.15-6.99)	5.8±.1 ^{DE} (5.65-6.11)	5.93±.03 ^{DE} (5.84-5.98)
TL	9.47±.12 ^{E} (9.12-9.66)	11.75±.006 ^{G} (11.74-11.77)	11.42±.1 ^{FG} (11.22-11.7)	11.12±.04 ^{F} (11-11.23)
ALB	1.7±.06 ^{CDE} (1.54-1.86)	1.79±.07 ^{DE} (1.59-1.93)	1.78±.02 ^{DE} (1.71-1.85)	2.09±.24 ^{E} (1.71-2.8)
GLU	85.25±1.39 ^{C} (82.87-88.71)	64.62±1.22 ^{B} (61.97-67.19)	70.39±2.23 ^{B} (65.14-75.9)	52.32±1.58 ^{A} (49.22-55.86)
Urea	15.42±.08 ^{AB} (15.21-15.61)	16.33±.08 ^{B} (16.11-16.53)	18.41±.09 ^{B} (18.18-18.65)	12.39±.06 ^{A} (12.23-12.56
CREAT	.44±.01 ^{B} (.41-.46)	.44±.009 ^{B} (.42-.46)	.33±.01 ^{A} (.32-.36)	.41±.01 ^{B} (.39-.44)
LPO(K)	6.75±.01 ^{D} (6.71-6.79)	3.43±.03 ^{B} (3.35-3.51)	4.57±.04 ^{C} (4.46-4.68)	2.74±.07 ^{A} (2.55-2.93)
LPO(L)	5.34±.03 ^{C} (5.27- 5.41)	1.9 ±.18 ^{A} (1.55-2.43)	3.18±.01 ^{B} (3.15-3.22)	1.52±.04 ^{A} (1.42-1.64)
LPO(G)	3.68±.02 ^{D} (3.63-3.74)	1.57±.02 ^{B} (1.52-1.62)	2.51±.02 ^{C} (2.47-2.56)	1.3±.01 ^{A} (1.26-1.34)

Different letters indicate significance at p<0.05 Red blood cells (RBCs), Haemoglobin (Hb), Hematocrit (Hct), Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH), Mean corpuscular haemoglobin concentration (MCHC), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Total protein (TP), Total lipid (TL), Albumin (Alb), Glucose (GLU), Urea and Creatinine (Creat), Lipid peroxidation in kidney LPO (K), Lipid peroxidation in Liver LPO (L), Lipid peroxidation in Gills LPO (G).

Table 2: Cont

Parameters / Treatments	Pb	Pb+LYC	Pb+VE	Pb+LYC+VE
	Mean ± SE (Min-Max)	Mean ± SE (Min-Max)	Mean ± SE (Min-Max)	Mean ± SE (Min-Max)
HB (g/100ml)	14.81±.15 ^{A} (14.51-15.11)	16.28±..18 ^{B} (15.94-16.63)	15.37±.06 ^{A} (15.23-15.51)	15.31±.05 ^{A} (15.2-15.42)
RBC x (10^6 /mm^3)	2.15±.02 ^{A} (2.1-2.22)	2.37±.02 ^{BC} (2.31-2.43)	2.19±.01 ^{A} (2.16-2.23)	2.31±.006 ^{B} (2.3-2.33)
Hct (%)	28.83±.68 ^{A} (27.46-30.2)	34.63±.63 ^{B} (33.21-36.07)	30.7±.51 ^{A} (29.7-32.02)	33.6±.05 ^{B} (33.5-33.7)
MCV (μm^3/cell)	133.64±2.93 ^{A} (127.21-140.56)	146.12±2.03 ^{BC} (141.75-150.55)	139.87±1.62 ^{AB} (136.81-143.58)	145.16±.48 ^{BC} (143.77-145.88)
MCH (μg/cell)	68.66±.95 ^{AB} (66.48-71.03)	68.71±.19 ^{AB} (68.33-69.08)	70.02±.21 ^{BC} (69.55-70.5)	66.13±.21 ^{A} (65.73-66.75)
MCHC(%)	51.41±.67 ^{C} (50.03-52.84)	47.05±.78 ^{B} (45.38-48.73)	50.08±.73 ^{C} (48.43-51.36)	45.55±.15 ^{AB} (45.26-45.88)
AST	191.05±.91 ^{F} (188.4-192.33)	149.14±2.39 ^{E} (142.51-153.65)	132.14±5.08 ^{D} (121.38-142.66)	127.22±4.54 ^{D} (113.92-134.18)
ALT	49.9±1.62 ^{E} (45.38-53.01)	34.98±.96 ^{D} (33.45-37.87)	38.34±1.22 ^{D} (35.08-40.49)	34.07±.68 ^{D} (32.44-35.22)
TP	3.27±.13 ^{A} (3.09-3.67)	5.05±.09 ^{CD} (4.88-5.3)	4.5±.31 ^{BC} (3.87-5.31)	4.23±0.1 ^{B} (4.03-4.49)
TL	5.54±.16 ^{A} (5.11-5.91)	8.36±.09 ^{D} (8.21-8.57)	6.92±.1 ^{B} (6.75-7.18)	7.55±.01 ^{C} (7.51-7.59)
ALB	.98±.009 ^{A} (.96-1)	1.24±.02 ^{AB} (1.17-1.28)	1.29±.02 ^{ABC} (1.23-1.35)	1.59±.01 ^{BCD} (1.55-1.63)
GLU	117.62±1.98 ^{E} (112.03-121.24)	102.05±1.46 ^{D} (99.4-105.44)	107.71±1.73 ^{D} (103.5-112.01)	99.78±2.48 ^{D} (93.66-105.22)
Urea	29.79±1.13 ^{D} (27.15-32.61)	32.05±1.21 ^{D} (29.13-34.97)	29.1±1.1 ^{D} (26.17-31.43)	24.42±1.007 ^{C} (22.23-26.83)
CREAT	.87±.009 ^{D} (.85-.89)	.83±.009 ^{D} (.81-.85)	.65±.01 ^{C} (.63-.68)	.62±.01 ^{C} (.6-.64)
LPO(K)	49.25±1.85 ^{F} (46-54.5)	12.05±.5 ^{D} (10.85-13.3)	16.83±.47 ^{E} (16.15-18.2)	6.87±.3 ^{C} (6.4-7.75)
LPO(L)	26.52±.82 ^{E} (25.05-28.5)	7.86±.62 ^{C} (6.6-9.45)	13.12±.34 ^{D} (12.15-13.6)	5.86±.29 ^{C} (5.2-6.55)
LPO(G)	24.37±.9 ^{D} (23.05-27)	6.57±.61 ^{C} (5.1-7.9)	8.83±.82 ^{C} (7.3-11)	2.8±.05 ^{B} (2.65-2.9)

Different letters indicate significance at $p<0.05$ Red blood cells (RBCs), Haemoglobin (Hb), Hematocrit (Hct), Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH), Mean corpuscular haemoglobin concentration (MCHC), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Total protein (TP), Total lipid (TL), Albumin (Alb),Glucose (GLU), Urea and Creatinine (Creat), Lipid peroxidation in kidney LPO(K), Lipid peroxidation in Liver LPO(L), Lipid peroxidation in Gills LPO (G).

Table 3: The basic data (N=4) of blood constituent parameters of *C. gariepinus* exposed to lead (Pb), Vitamin E (VE), Lycopene (LYC) and their combinations for 30 day.

Parameters / Treatments	Control	LYC	VE	LYC+VE
	Mean ± SE (Min-Max)	Mean ±SE (Min-Max)	Mean ±SE (Min-Max)	Mean ±SE (Min-Max)
HB (g/100ml)	18.28±.38 ^{C} (17.59-18.98)	19.47±.07 ^{D} (19.32-19. 62)	18.53±.1 ^{C} (18.32-18. 74)	18.56±.18 ^{C} (18.22-18.91)
RBC x (10^6 /mm^3)	2.57±.02 ^{C} (2.51- 2.64)	2.71±.006 ^{D} (2.7-2.73)	2.57±.007 ^{C} (2.56-2.59)	2.6±.01 ^{C} (2.56-2.64)
Hct (%)	40.57±.63 ^{C} (39.05-41.65)	44.76±.05 ^{D} (44.64-44.88)	40.93±.14 ^{C} (40.59-41.28)	41.79±.41 ^{C} (40.79-42.8)
MCV (µm^3/cell)	157.56±1.82 ^{C} (154.47-162.69)	165.01±.44 ^{D} (163.69-165.6)	159.27±.25 ^{CD} (158.55-159.72)	160.75±1.52 ^{CD} (157.53-164.61)
MCH (µg/cell)	71±1.24 ^{A} (68.1-73.94)	71.77±.32 ^{A} (70.95-72.39)	72.09±.26 ^{A} (71.56-72.72)	71.4±.81 ^{A} (69.2-72.73)
MCHC (%)	45.05±.34 ^{AB} (44.08-45.63)	43.49±.1 ^{A} (43.27-43.71)	44.49±.73 ^{A} (42.32-45.6)	44.41±.22 ^{A} (43.92-44.9)
AST	63.98±3.47 ^{A} (53.65-68.59)	88.62±1.21 ^{B} (86.22-91.87)	65.73±.73 ^{A} (64.12-67.55)	76.51±.48 ^{AB} (75.32-77.65)
ALT	26.16±1.21 ^{C} (22.94-28.32)	14.11±.27 ^{A} (13.51-14.82)	16.11±.17 ^{AB} (15.77-16.43)	19.25±.84 ^{B} (17.99-21.68)
TP	5.14±.01 ^{CD} (5.1-5.19)	6.55±.13 ^{E} (6.18-6.77)	6.34±.008 ^{E} (6.33-6.37)	6.05±.05 ^{E} (5.92-6.16)
TL	10.79±.22 ^{DE} (10.22-11.18)	19.37±.29 ^{G} (18.86-20.23)	15.74±.08 ^{F} (15.5-15.89)	11.53±.03 ^{E} (11.45-11.62)
ALB	1.77±.04 ^{C} (1.69-1.89)	2.22±.05 ^{D} (2.12-2.37)	1.74±.08 ^{C} (1.55-1.92)	2.25±.09 ^{D} (1.99-2.41)
GLU	62.4±1.74 ^{C} (59.93-67.42)	51.06±.9 ^{B} (49.04-53.28)	34.16±1.42 ^{A} (31.6-38.2)	45.22±.9 ^{B} (43.1-47.15)
Urea	12.61±.14 ^{A} (12.27-12.96)	16.69±.22 ^{B} (16.2-17.18)	18.68±.2 ^{B} (18.17-19.15)	10.57±.12 ^{A} (10.28-10.87)
CREAT	.46±.007 ^{A} (.45-.48)	.46±.01 ^{A} (.43-.49)	.36±.01 ^{A} (.33-.39)	.4±.01 ^{A} (.38-.43)
LPO(K)	5.24±.02 ^{D} (5.19-5.29)	2.81±.01 ^{C} (2.78-2.84)	2.21±.02 ^{B} (2.15-2.27)	1.19±.02 ^{A} (1.15-1.23)
LPO(L)	3.83±.02 ^{D} (3.79-3.89)	1.42 ±.1 ^{B} (1.24-1.67)	2.51±.07 ^{C} (2.33-2.71)	1.13±.01 ^{A} (1.1-1.17)
LPO(G)	3.16±.02 ^{D} (3.12-3.21)	1.52±.02 ^{B} (1.47-1.58)	2.44±.02 ^{C} (2.39-2.49)	1.23±.01 ^{A} (1.19-1.26)

Different letters indicate significance at $p<0.05$ Red blood cells (RBCs), Haemoglobin (Hb), Hematocrit (Hct), Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH), Mean corpuscular haemoglobin concentration (MCHC), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Total protein (TP), Total lipid (TL), Albumin (Alb), Glucose (GLU), Urea and Creatinine (Creat), Lipid peroxidation in kidney LPO(K), Lipid peroxidation in Liver LPO(L), Lipid peroxidation in Gills LPO (G).

Table 3: Cont

Parameters / Treatments	Pb	Pb+LYC	Pb+VE	Pb+LYC+VE
	Mean ±SE (Min-Max)	Mean ± SE (Min-Max)	Mean ± SE (Min-Max)	Mean ± SE (Min-Max)
HB (g/100ml)	14.39±.06 ^{A} (14.26-14.53)	17.17±.09 ^{B} (16.98-17.37)	16.85±.1 ^{B} (16.65-17.05)	17.28±.05 ^{B} (17.17-17. 42)
RBC x (10^6 /mm^3)	2.06±.03 ^{A} (2-2.15)	2.45±.02 ^{B} (2.42-2.51)	2.41±.01 ^{B} (2.38-2.45)	2.42±.007 ^{B} (2.41-2.44)
Hct (%)	26.66±.17 ^{A} (26.24-27.09)	36.79±.3 ^{B} (36.18-37.4)	36.36±.15 ^{B} (36.02-36.71)	36±.23 ^{B} (35.44-36.56)
MCV (μm^3/cell)	129.39±2.13 ^{A} (123.67-133.7)	150.01±1.04 ^{B} (148.48-153.08)	150.58±.69 ^{B} (149.05-152.12)	148.6±1.11 ^{B} (146.88-151.7)
MCH (μg/cell)	70.26±.67 ^{A} (69.02-71.55)	70.03±.45 ^{A} (69.2-71.27)	69.77±.43 ^{A} (68.72-70.83)	71.34±.16 ^{A} (70.98-71.78)
MCHC(%)	53. 98±.24 ^{E} (53. 51-54.45)	46.68±.11 ^{CD} (46.44-46.93)	46.33±.1 ^{BC} (46.1-46.56)	48.01±.3 ^{D} (47.31-48.6)
AST	214.79±6.94 ^{F} (195.2-227.98)	146.75±4.65 ^{E} (134.12-154.31)	107.67±.68 ^{C} (106.14-109.12)	124.32±1.71 ^{D} (120.39-128.29)
ALT	81.81±.99 ^{E} (79.55-84.34)	29.81±1.86 ^{C} (26.25-35.07)	35.85±.44 ^{D} (34.91-36.98)	28.02±.21 ^{C} (27.65-28.44)
TP	3.11±.01 ^{A} (3.08-3.14)	5.3±.14 ^{D} (4.96-5.63)	4.74±.21 ^{BC} (4.13-5.11)	4.37±.12 ^{B} (4.02-4.54)
TL	4.27±.2 ^{A} (3.82-4.74)	9.99±.06 ^{CD} (9.85-10.11)	7.51±.01 ^{B} (7.48-7.54)	9.43±.22 ^{C} (8.87-9.89)
ALB	.75±.01 ^{A} (.73-.78)	1.03±.004 ^{B} (1.02-1.04)	1.18±.009 ^{B} (1.17-1.21)	1.55±.03 ^{C} (1.46-1.63)
GLU	122.09±2.23 ^{F} (117.48-127.45)	72.31±1.45 ^{D} (68.99-75.88)	103.36±1.93 ^{E} (99.93-107)	96.33±2.85 ^{E} (88.32-101.66)
Urea	43.84±.79 ^{E} (41.92-45.77)	42.26±.86 ^{E} (40.13-44.37)	34.47±1.1 ^{D} (32.11-37.17)	23.12±1.57 ^{C} (19.16-26.72)
CREAT	1.08±.07 ^{C} (.96-1.3)	1.09±.03 ^{C} (.99-1.16)	.92±.02 ^{B} (.86-.99)	.76±.02 ^{B} (.71-.82)
LPO(K)	26.54±.23 ^{H} (26.12-27.05)	9.53±.01 ^{F} (9.49-9.57)	12.54±.02 ^{G} (12.48-12.58)	6.39±.03 ^{E} (6.32-6.46)
LPO(L)	25.22±.03 ^{H} (25.15-25.29))	8.17±.02 ^{F} (8.13-8.23)	11.29±.07 ^{G} (11.14-11.45)	4.97±.05 ^{E} (4.85-5.13)
LPO(G)	23.53±.02 ^{H} (23.57-23.59)	6.46±.01 ^{F} (6.42-6.51)	9.46±.02 ^{G} (9.42-9.53)	3.35±.009 ^{E} (3.33-3.37)

Different letters indicate significance at $p<0.05$ Red blood cells (RBCs), Haemoglobin (Hb), Hematocrit (Hct), Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH), Mean corpuscular haemoglobin concentration (MCHC), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT),Total protein (TP),Total lipid (TL), Albumin (Alb), Glucose (GLU), Urea and Creatinine (Creat), Lipid peroxidation in kidney LPO(K), Lipid peroxidation in Liver LPO(L), Lipid peroxidation in Gills LPO (G).

The time of exposure main effect was not significant and a positive correlations were recorded between similar MCV and MCH in the two periods (r MCV=0.926, rMCH=0.647 and rMCHC= 0.930). Diet supplementation with tomato paste and/or vitamin E improved haematological parameters in comparison with the control.

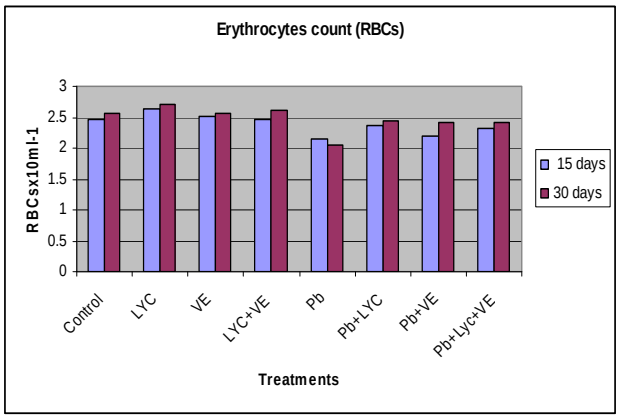

Fig.1: The variability of erythrocytes count x (10^6/mm^3) in different treatments for 15 and 30 days.

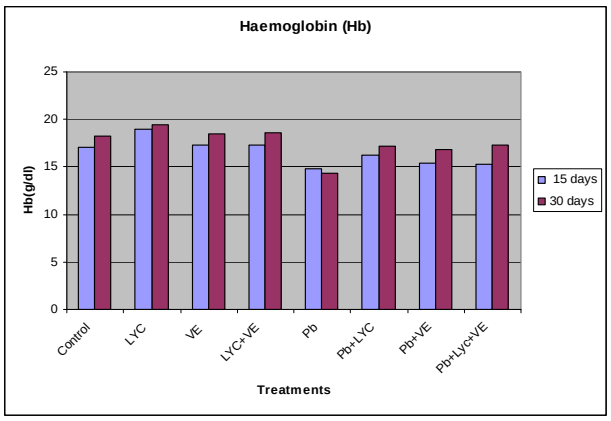

Fig. 2: The variability of haemoglobin content (g/100ml) in different treatments for 15 and 30 days.

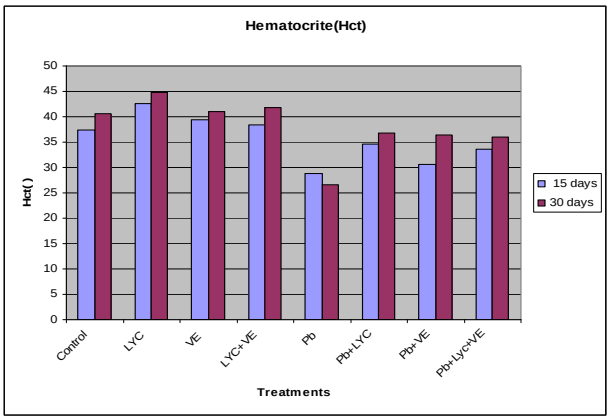

Fig. 3: The variability of haematocrit value (%) in different treatments for 15 and 30 days.

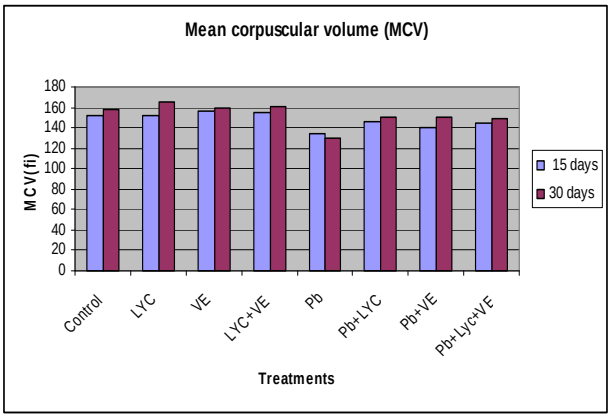

Fig. 4: The variability of mean corpuscular volume (MCV) (µm^3/cell) in different treatments for 15 and 30 days

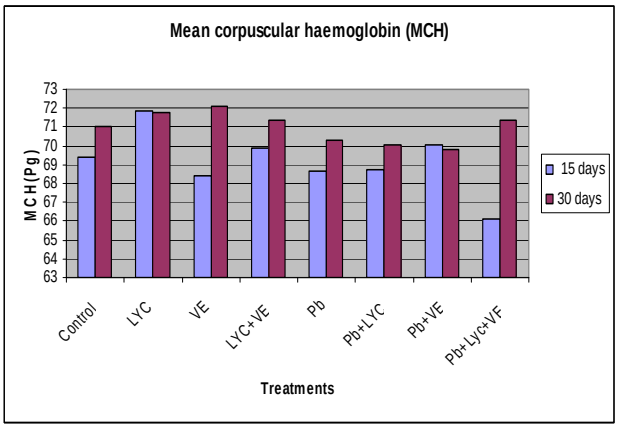

Fig. 5: The variability of mean corpuscular haemoglobin (MCH) (µg/cell) in different treatments for 15 and 30 days.

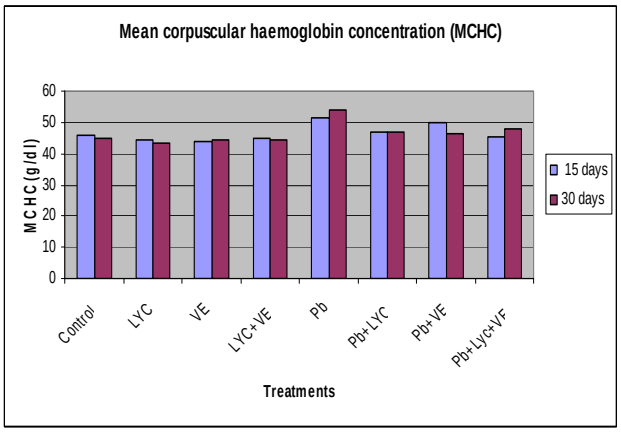

Fig. 6: The variability of mean corpuscular haemoglobin concentration (MCHC) (g/dl) in different treatments for 15 and 30 days.

Biochemical parameters

Glucose level

The normal glucose level of *Clarias gariepinus* for 15 and 30 days periods are given in Tables (2 & 3) and Fig. 7. Lead main effect was significantly increased in both periods ($P<0.0001$). In addition, the main effects of Pb, Lycopene, VE and their interactions were significant in the two periods.

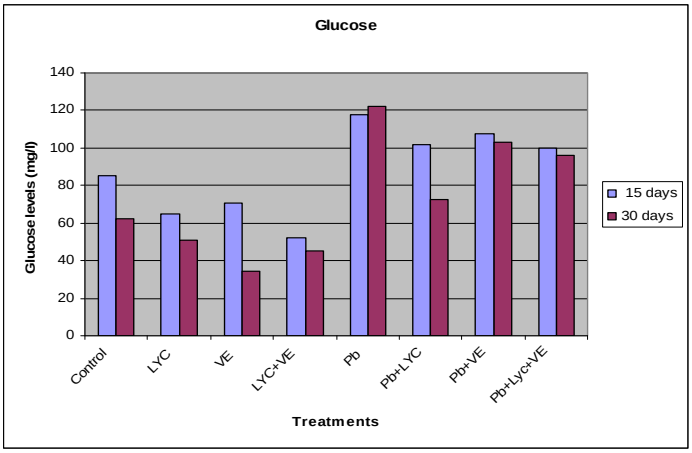

Fig. 7: The variability of glucose (mg/L) concentrations in different treatments for 15 and 30 days.

Total protein level

The total protein level of *Clarias gariepinus* for 15 and 30 days periods are given in Tables (2 & 3) and Fig. 8. Lead reflects highly significant decrease in total protein at both periods (P<0.0001). The main effect of lycopene and /or vitamin E was significant in the two periods. Pb-LYC-VE main effect was significant at the second period. The time of exposure main effect was not significant and a positive correlation was obtained between the total protein levels in the two periods (r TP =.940).

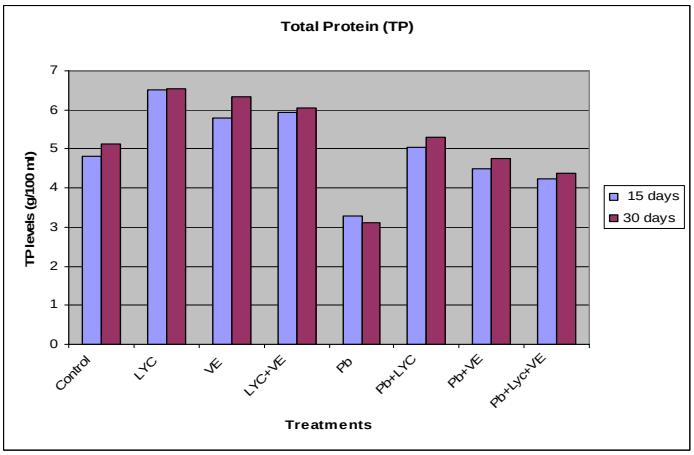

Fig. 8: The variability of total protein (g/100ml) concentrations in different treatments for 15 and 30 days.

Total lipids level

The total lipids of *Clarias gariepinus* for 15 and 30 days periods are given in Tables (2 & 3) and Fig. 9. The lead main effect was highly significant (P<0.0001) in both exposure periods. Lycopene main effect was highly significant in both exposure periods. Vitamin E main effect was highly significant in both exposure periods. Pb-LYC-VE main effect was highly significant in both exposure periods. The time of exposure main effect was significant and a positive correlation was obtained between the total lipid levels in the two periods (r TL =.995).

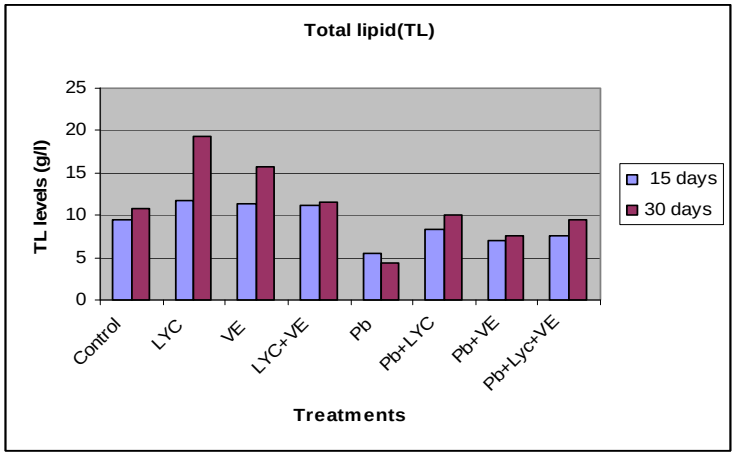

Fig. 9: The variability of total lipids (g/L) concentrations in different treatments for 15 and 30 days.

AST and ALT levels

The normal values of aspartate amino transferase (AST) and alanine amino transferase (ALT) activity percentage of *Clarias gariepinus* in the two periods are given in Tables (2 & 3) and Figs. 10,11. Lead showed highly significant (P<0.0001) increase in AST and ALT activities in the two periods. The main effect of Lycopene was highly significant in AST and ALT activities in the two periods. No significant main effect of vitamin E was recorded except for ALT at the second period. Pb-LYC-VE interaction showed a significant effect except for ALT at the second period in the two periods. The time of exposure main effect was highly significant for ALT. and a positive correlations were obtained between AST activity at 15 and 30 days in the two periods (r AST=.982, rALT=.986). The diet supplementation with lycopene and /or vitamin E to Pb-treated fish decreased significantly the activities of AST and ALT serum to the control level.

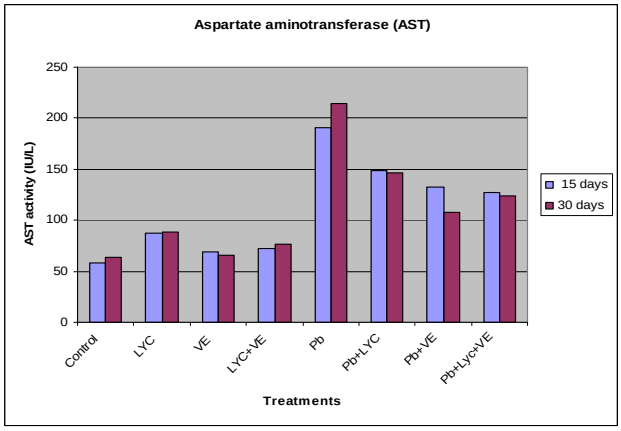

Fig. 10: The variability of aspartate aminotransferase (AST) activity (IU/L) in different treatments for 15 and 30 days.

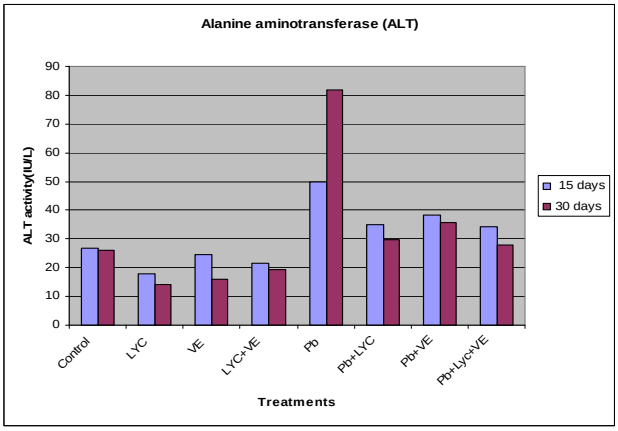

Fig. 11: The variability of alanine aminotransferase (AST) activity (IU/L) in different treatments for 15 and 30 days.

Albumin level

The normal values of albumin (Alb) of *Clarias gariepinus* in the two periods are given in Tables (2 & 3) and Fig 12. The lead effect was highly significant (P<0.0001) decrease in both periods. No significant main effect of vitamin E was recorded at both periods. Lycopene and LYC-VE interaction was significant only at the second period. Pb-

LYC-VE interaction main effect was not significant in both exposure periods. The time of exposure main effect was not significant except Pb, LYC and Pb-VE time of exposure main effect was significant and a positive correlation was obtained between albumin levels in the two periods (r ALB=.914).

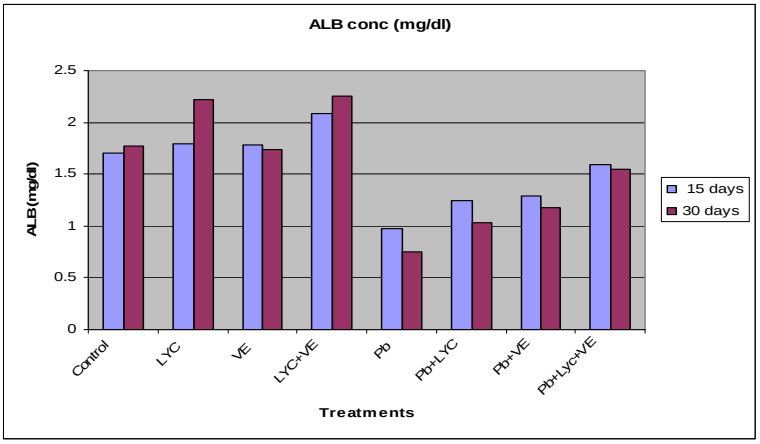

Fig. 12: The variability of Albumin concentrations (mg/dl) in different treatments for 15 and 30 days.

Urea level

The normal values of Urea level of *Clarias gariepinus* in the two periods are given in Tables (2 & 3) and Fig. 13. The lead effect was highly significant (P<0.0001) in the two periods .Lycopene and vitamin E main effects were significant only at the second period. The main effect of Pb-LYC was highly significant in both exposure periods (P<0.0001). The main effect of Pb-VE was highly significant in both exposure periods (P<0.0001). The main effect of Pb-LYC-VE was highly significant in both exposure periods (P<0.0001) and a positive correlation was obtained between Urea levels in the two periods (r urea=.982). The time of exposure main effect was significant except Pb-LYC-VE time.

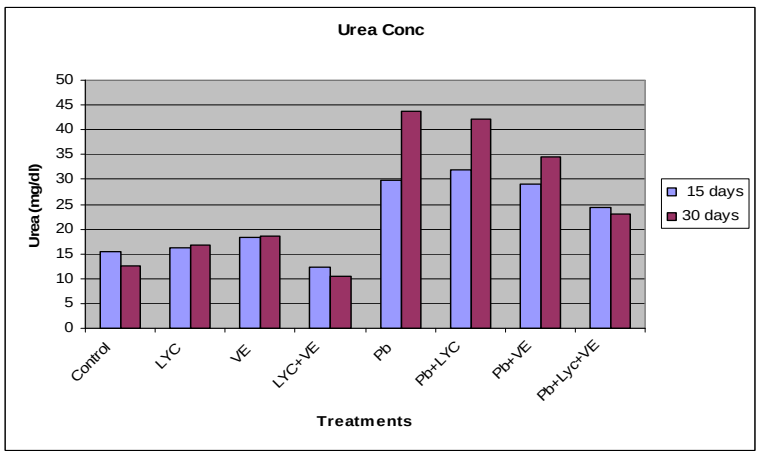

Fig. 13: The variability of Urea concentrations (Mg/dl) in different treatments for 15 and 30 days.

Creatinine level

The normal values of Creatinine level of *Clarias gariepinus* in the two periods are given in Tables (2 & 3) and Fig. 14. The lead effect was highly significant (P<0.0001) in the two periods. Vitamin E main effect was significant only at the first period. Lycopene and LYC-VE interaction was not significant at the two periods. The main effect of Pb-LYC was highly significant in both exposure periods (P<0.0001). The main

effect of Pb-VE was highly significant in both exposure periods (P<0.0001). The main effect of Pb-LYC-VE was highly significant in both exposure periods (P<0.0001), and a positive correlation was obtained between Creatinine levels in the two periods (r =.961). The time of exposure main effect was not significant.

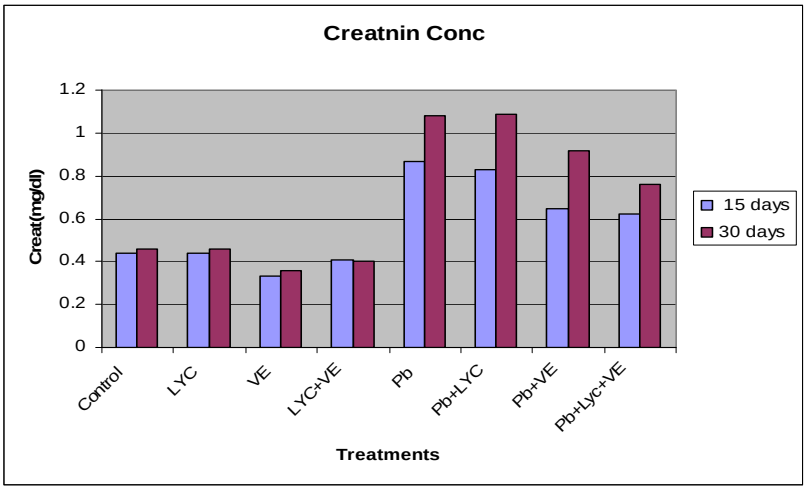

Fig. 14: The variability of Creatinine concentrations (Mg/dl) in different treatments for 15 and 30 days.

Lipid peroxidation measurement

The results of lipid peroxidation (LPO) in liver, kidney and gills of *Clarias gariepinus* in the two periods are given in Tables (2 & 3) and Figs. 15, 16 &17. The main effects of Pb, LYC, VE and their interactions were highly significant (P<0.0001) for liver, kidney and gills in the two periods. Similary. The time of exposure main effect was highly significant (P<0.0001), r(LPO)L=1.000, r(LPO)k=1.000, r(LPO)g=1.000. The level of lipid peroxidation (LPO) for liver, kidney and gills was significantly (P<0.0001) decreased in Pb-exposed fishes fed diets supplemented with Vitamin E and/or Lycopene.

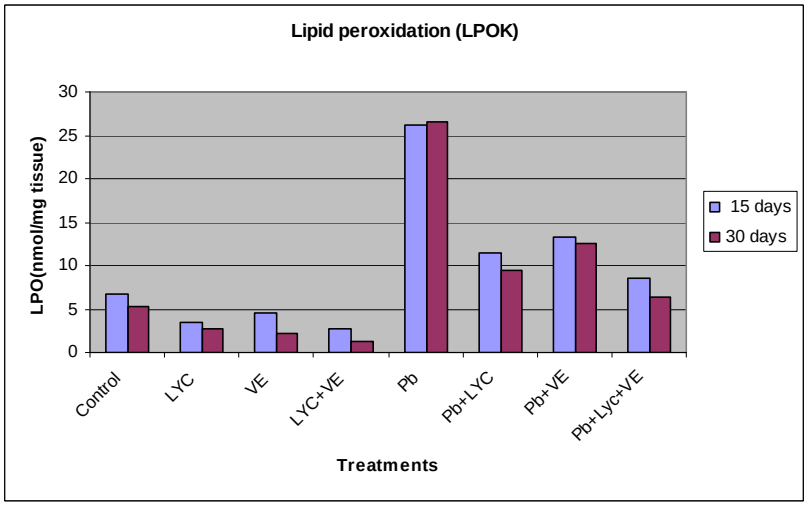

Fig. 15: The variability of lipid peroxidation levels in Kidney (nmol/mg tissue) in different treatments.

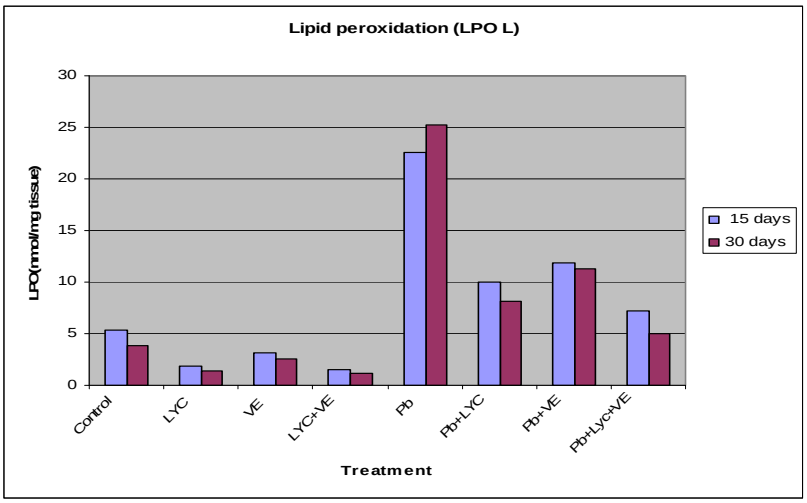

Fig. 16: The variability of lipid peroxidation levels in Liver (nmol/mg tissue) in different treatments

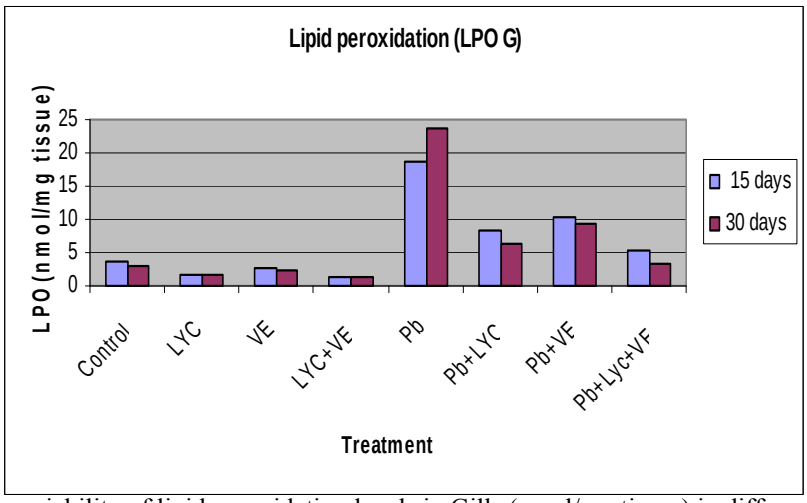

Fig. 17: The variability of lipid peroxidation levels in Gills (nmol/mg tissue) in different treatments.

DISCUSSION

The Pb-exposed fish showed a significant reduction in their RBCs, Hb and Hct in comparison with those exposed to Pb with supplementation of lycopene and vitamin E. These results are in agreement with those of Adeyemo (2007) and Alkahemal -Balawi *et al*., 2011) who found a significant reduction in RBCs and Hb African cat fish *(Clarias gariepinus*) after exposure to lead. Oluah and Omerebel (2010) found a significant decrease in Hb value of *Clarias gariepinus* after exposure to Lead. These alterations were attributed to direct or feedback responses of structural damage to RBC membranes resulting in haemolysis and impairment in haemoglobin synthesis, stress-related release of RBCs from the spleen and hypoxia, induced by exposure to lead (Shah, 2006). These Pb-induced decrease in haematological parameters may be also due disequilibrium of the osmotic pressure inside and outside the blood cell (Heath 1995).

The Pb-induced changes towards increase or decrease in MCV, MCH and MCHC values were reported in the present work. Similar findings were recorded by Adeyemo (2007) who found asignificant increase in MCV, MCH and MCHC values of *C. gariepinus* after exposure to Lead. Shah, (2006) found asignificant increase in MCV, MCH and MCHC values of tench (*Tinca tinca*)

after exposure to Lead. Oluah and Omerebel (2010) found decrease in MCV, MCH and MCHC after exposure of *Clarias gariepinus* to lead. Similar findings were recorded by Ololade and Ogini (2009) after exposure of *Clarias gariepinus* to Zinc. These chemicals-induced alterations in MCV, MCH and MCHC were attributed to direct or feedback responses of structural damage to RBC membranes resulting in haemolysis and impairment in haemoglobin synthesis, stress-related release of RBCs from the spleen and hypoxia (Marei *et al*., 1998; Shah, 2006).

In the present study the increase of serum glucose (hyperglycemia) was revealed in *Clarias gariepinus* exposed to sublethal concentration of lead. This result is in agreement with that of Alkahemal-Balawi *et al*. (2011) who found increasing in glucose level (hyperglycemia) of *Clarias gariepinus* after exposure to lead. Similar findings were observed by Martinez *et al.,* 2004 and Çiftçi *et al*. (2008) working on *Prochilodus lineatus* and *Anguilla anguilla* respectively using lead. Martins *et al*. (2007) reported a reduced blood glucose level after stress in Atlantic halibut (*Hippoglossus hippoglossus* L.) fed vitamin E supplemented diets as in the present study.

The present investigation showed decrease in the serum total protein level (hypoproteinemia) after exposure to lead. Similar result was recorded by Martinez *et al*. (2004) after exposure of *Prochilodus lineatus* to lead. Çiftçi *et al*. (2008) reported a decreased total protein level after exposure of *Anguilla Anguilla to lead.* Such decrease of total protein may be due to destruction of protein-synthesizing subcellular structures, inhibition of hepatic synthesis of blood protein as a result of heavy metals-protein interaction or due to stimulated protein catabolism to provide extra energy requirement to overcome the stress in the polluted medium (Fontana *et al.*, 1998).

The present investigation showed decrease in the serum total lipid level. Similar result was recorded by Martinez *et al.* (2004) in *C. gariepinus* after exposure to lead. This may be due to the increase in secretion of catechloamines and corticosteroids a result of pollutant stress, which enhanced metabolic rate and in turn reduced metabolic reserves (Fayed *et al*., 2001).

In the present study,decrease in albumin level was recorded in Pb-treated *Clarias gariepinus*. The same result was obtained by Mahmoud *et al.* (2012) by exposure of *Clarias gariepinus* to mercury. Similar result was obtained by Liao *et al.,(*1986), who referred to the decline in albumin level, which render the acute turpentine induced inflammation in the presence of no significant change in the concentration of total protein in plasma. Also, they postulated that such situation was accompanied by a corresponding decline in the relative abundance of albumin mRNA in liver. Similar findings were recorded by Mekkawy *et al*., 1996; Hasheesh *et al*., 2000; El Fayoumi and Abd Allah (2003), who worked on *O. niloticus, Chrysichthyes auratus, Cyprinus carpio*, and Rainbow trout, respectively. Such results referred to the disturbance in impacts of heavy metals and pesticides on the plasma colloidal osmotic pressure and transportation mechanisms of fatty acids and hormones. Also, the decreased albumin levels (hypoalbuninemia) reflect the active inflammation and serious hepatic and renal damage. The latter damage can not prevent albumin from the blood into urine and being lost. These results may be attributed to liver necrosis (because of toxicant) which, led to leakage from liver into the blood and/or tactual inhibition of liver enzymes.

The current Pb-induced increase in AST and ALT activities can be

confirmed by similar results reported by Olojo *et al*. (2012) who found increasing in AST and ALT levels of *C. gariepinus* after exposure to lead . It has been reported that alterations in enzymes activities in the serum directly indicates major pathologic changes in cell membrane Permeability or hepatic cell rupture (Benjamin, 1978). The diet supplementation with Vitamin E and/ or tomato paste to Pb-treated fish diet led to a significant decreases in the activity of AST and ALT serum. These results are in agreement with those of El-Komy and Hassan (2005) who observed decrease in the activity of AST and ALT of thioacetamide-treated male rat fed tomato-juice as supplemented diet. Kalender *et al*. (2005) and Ogur *et al*. (2005) also recorded decrease in activity of AST and ALT after diazinon-induced and nitrate-induced stress in male rats fed vitamin E as supplemented diets.

In the present study, significant increases in serum urea and creatinine were recorded.These results are confirmed by those of Mahmoud *et al*. (2012) who reported an increase in the previous parameters in *Clarias gariepinus* after exposure to mercury. Zaki *et al*. (2009) found an increase in the urea and creatinine in *Oreochromis niloticus* due to cadmium exposure. Hadi *et al*. (2009) reported that the increase of creatinine level might be induced by glomerular insufficiency, increased muscle tissue catabolism or the impairment of carbohydrates metabolism .The diet supplementation with Vitamin E and /or lycopene to Pb-treated fish decreased levels of serum urea and creatinine to the control level. These results are in agreement with those of Karahan *et al*. (2005) who observed adecrease in the levels of serum urea and creatinin of gentamicin –treated rats fed tomato juice as supplemented diet.

In the current work, Lead exposure significantly stimulated the levels of oxidized lipids in the form of malondialdehyde (MDA) in the liver, kidney and gills .These results strongly indicated the destructive effect due to an increase of reactive oxygen substances followed by lipid peroxidation .These results are in agreement with those of El-Sokkary *et al*. (2003, 2005) who found asignificant increase in the oxidized lipids in lead- treated animals. The rise in lipid peroxidation level in liver means amodification in the physical characterisitics of cell membrane (Ursini *et al*., 1991). Since lipid peroxidation leads to hydrolysis of phospholipids into hydroperoxy fatty acids (Salgo *et al*., 1993). The level of lipid peroxidation was significantly decreased in Pb-exposed fishes fed diets supplemented with vitamin E and lycopene. Similar results for vitamin E were observed on fish species such as red sea bream, *Pagellus bogaraveo* (Murata and Yamauchi,1989), channel catfish, *Ictalurus punctatus* (Gatlin *et al*.,1992), African catfish ,*Clarias gariepinus* (Baker and Davies,1996 and 1997), sea bass ,*Dicentrarchus labrax* (Gatta *et al*., 2000), Atlantic Salmon, *Salmo salar* (Scaife *et al*., 2000) ,juvenile gilthead seabream ,*Sparus aurata L*. (Mourente *et al*., 2002), rainbow trout ,*Salmo gairdneri* (Chaiyapechara *et al*., 2003), hybrid tilapia, *Oreochromis niloticus* x *O. aureus* (Huang *et al*., 2003 and 2004), grouper, *Epinephelus malabaricus* (Yu-Hung and Shi-Yun, 2005) and red hybrid tilapia, *Oreochromis sp*.(Wang *et al*., 2006).

Lycopene was found by many authors to improve stress-induced lipid peroxidation in rats (Bhuvaneswaria *et al* 2001, Velmurugan *et al*., 2001 and 2002, El-Demerdash *et al*. 2004, El-Komy and Hassan, 2005, Moreira *et al*., 2005, Atessahin *et al*., 2006 and Yilmaz *et al* ., 2006).

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ARABIC SUMMERY

تأثير الرصاص على بعض الخصائص الدموية والبيوكيميائية لسمكة كلاريس جاريبينس مع إضافة الليكوبين وفيتامين هـ

اسامة محمد محمود [1] ـ عبدالباسط مسعود عبيد[2] ـ سلوى منصور محمد[2]

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2- قسم علم الحيوان – كلية العلوم – جامعة جوب الوادي .

تهدف الدراسة الحالية إلى بحث التأثير الوقائي لليكوبين وفيتامين هـ ضد التأثيرات الضارة للرصاص بعد تعرض الأسماك للرصاص لمدة 15 ، 30 يوم .

لقد درست تأثيرات الرصاص بدلالة الخصائص الدموية والبيوكيميائية ولقد وجدت زيادة معنوية في نشاط إنزيمات الكبد (ALT-AST) ومتوسط تركيز الهيموجلوبين في كريات الدم الحمراء ، جلوكوز مصل الدم، معدل الدهون الكلي ، اليوريا ، الكرياتينين ، موكسدات الدهون. ووجد نقص معنوى في معدل البروتين الكلي، الألبيومين، الهيموجلوبين، عدد كرات الدم الحمراء، الهيماتوكريت، متوسط حجم كريات الدم الحمراء، متوسط هيموجلوبين كرات الدم الحمراء ولقد لوحظ تقليل التأثيرات الضارة للرصاص بإضافة الليكوبين وفيتامين هـ.