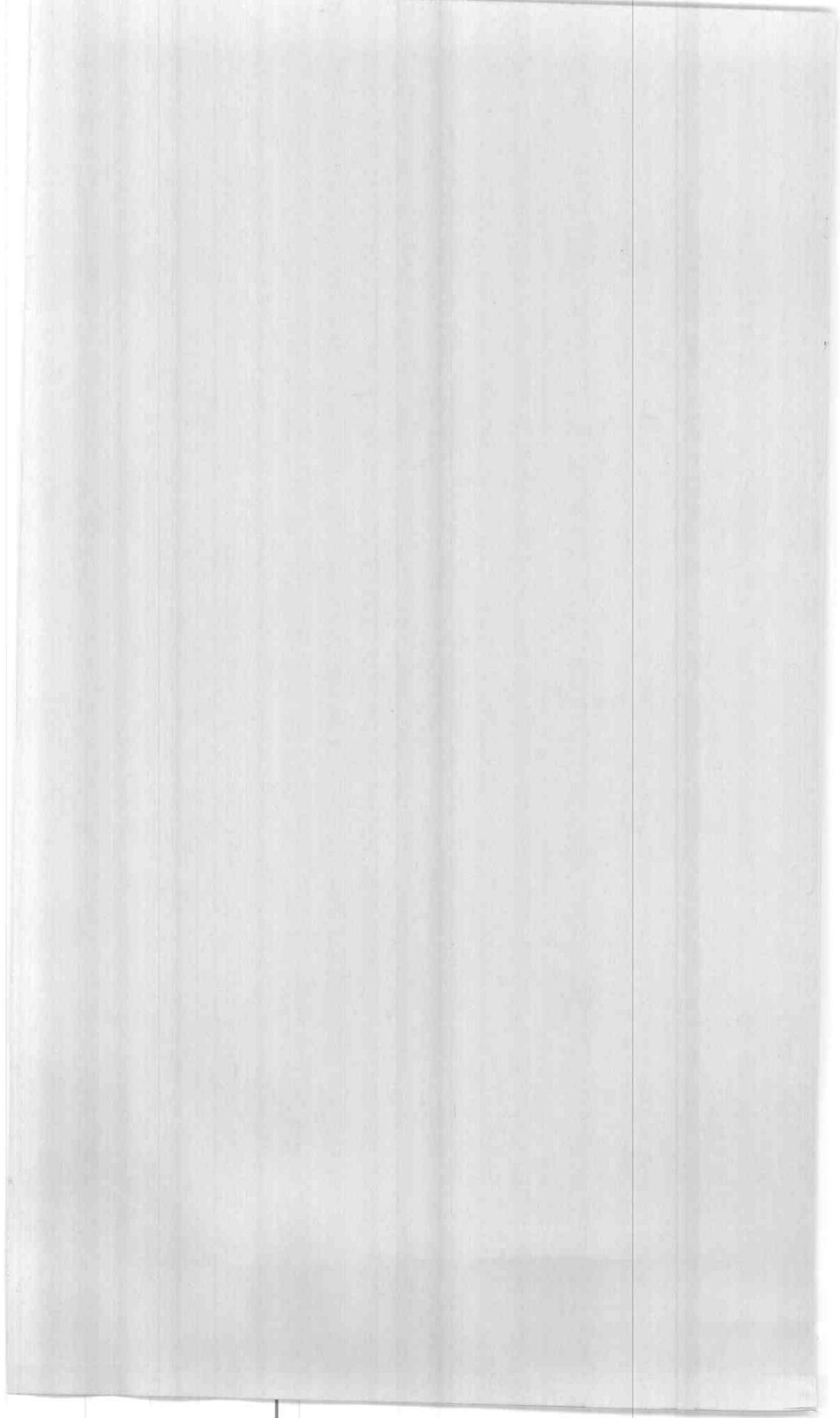


RESULTS AND DISCUSSION



IV- RESULTS AND DISCUSSION

The purpose of this work was to investigate and evaluate the antioxidant potential of some natural products on hyperlipaemia in male albino rats. The effect of wheat germ oil (W.G.O), rosemary (RM) and evening primrose (E.P.R) was studied. Also, the mixtures of W.G.O plus E.P.R and RM plus E.P.R were evaluated. To achieve this goal, two main experiments were conducted. The first experiment was the prophylactic effect of these treatments against hyperlipaemia and the second concerned the curative effect of the prementioned agents on hyperlipaemic rats.

1. Prophylactic effect of different treatments against hyperlipaemia:

This experiment was lasted for 8 weeks. Rats were fed on hypercholesterolemic diet as well as the different treatments under investigation in combination with this diet.

1.1. Prophylactic effect of different natural antioxidants on lipid profile:

The protective effect of W.G.O, RM, E.P.R and the mixture of W.G.O. plus E.P.R and RM plus E.P.R against hyperlipaemia are shown in Table's (2-6) and Fig's (1-4).

Table (2) and Fig. (1) revealed the effect of different treatments on serum total lipids. It is clearly shown that the value of total lipids in the first group (-ve control) were not affected during the experimental period of 8 weeks. In the (+ve) control group, which rats fed only on the hyperlipaemic diet, serum lipids were very highly significant increased (24.8%) compared to the 0-time value. In the groups of rats which received the different treatments during the induction of hyperlipaemia, serum lipids

revealed a dissimilar changes during the experimental period. These values were increased in the order 19.9% , 32.8%, 13.8% , 20.9% and 12.7% for W.G.O, RM, E.P.R, W.G.O + E.P.R and RM + E.P.R, respectively.

Table (3) and Fig. (2) reveal the prophylactic effect of different treatments against hypertriglyceridemia. Serum triglycerides in the (-ve) control group did not affected during the 8 weeks of treatment. On the other hand, in the (+ve) control group, triglycerides were increased by 123.6%. The prophylactic effect against hypertriglyceridemia was pronounced in the group of rats received RM which triglycerides were increased by 59.5% while it was 73.7% in W.G.O group and 76.8% in E.P.R group. The mixture of RM plus E.P.R was more effective (35.6%) than those of W.G.O plus E.P.R (47.5%).

The effect of different antioxidant on serum total cholesterol, HDL-cholesterol and T.cholesterol/HDL-cholesterol was shown in Tables (4-6) and Figs. (3, 4). Serum total cholesterol was increased by 60% in the (+ve) control group, while it was increased only by 1.8% in the (-ve) control group. The anti hypercholesterolemic effect of the different antioxidants was clearly observed. It is in order E.P.R (20.0%), RM (20.8%) and W.G.O (73.7%), while in the (+ve) control group, serum total cholesterol was increased by 60.0% after 8 weeks. The effect of RM plus E.P.R was more potential (1.3%) than those of W.G.O plus E.P.R (9.4%).

Also, administration of W.G.O caused an appreciated prophylactic effect on HDL-cholesterol, whereas its value was increased by 83.6% after 8 weeks of treatment while this value was 33.3%, 24.2%, 22.3% and 4.9% in cases of RM, E.P.R, W.G.O plus E.P.R and RM plus E.P.R,

Table(2): Prophylactic Effect of Different Treatments on s.Total Lipids (mg/dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. Total Lipids (mg/dl)					
	0 Time	4 Weeks	% Var.	8 weeks	% Var.	
1 Control (Normal Diet)	343.2 ± 23.5	338.8 ± 36.8 †	1.3 ↓	350.3 ± 40.8 †	2.0 ↑	
2 Hyperlipaemic Diet (HD)	343.0 ± 32.1	391.5 ± 39.9 *	14.1 ↑	428.2 ± 28.0 ***	۲۴,۸ ↑	
3 HD+W.G.O	342.2 ± 28.3	377.0 ± 61.9 †	10.2 ↑	410.3 ± 14.3 ***	۱۹,۹ ↑	
4 HD+RM	335.3 ± 14.7	349.5 ± 60.0 †	4.2 ↑	445.3 ± 54.7 **	۳۲,۸ ↑	
5 HD+E.P.R	360.3 ± 28.7	334.0 ± 38.0 †	7.3 ↓	410.0 ± 74.4 †	۱۳,۸ ↑	
6 HD+W.G.O+E.P.R	360.5 ± 24.7	311.2 ± 30.5 **	13.7 ↓	435.8 ± 63.2 *	۲,۹ ↑	
7 HD+RM+E.P.R	361.8 ± 19.3	301.5 ± 68.0 *	16.7 ↓	407.6 ± 76.2 †	۱۲,۷ ↑	

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

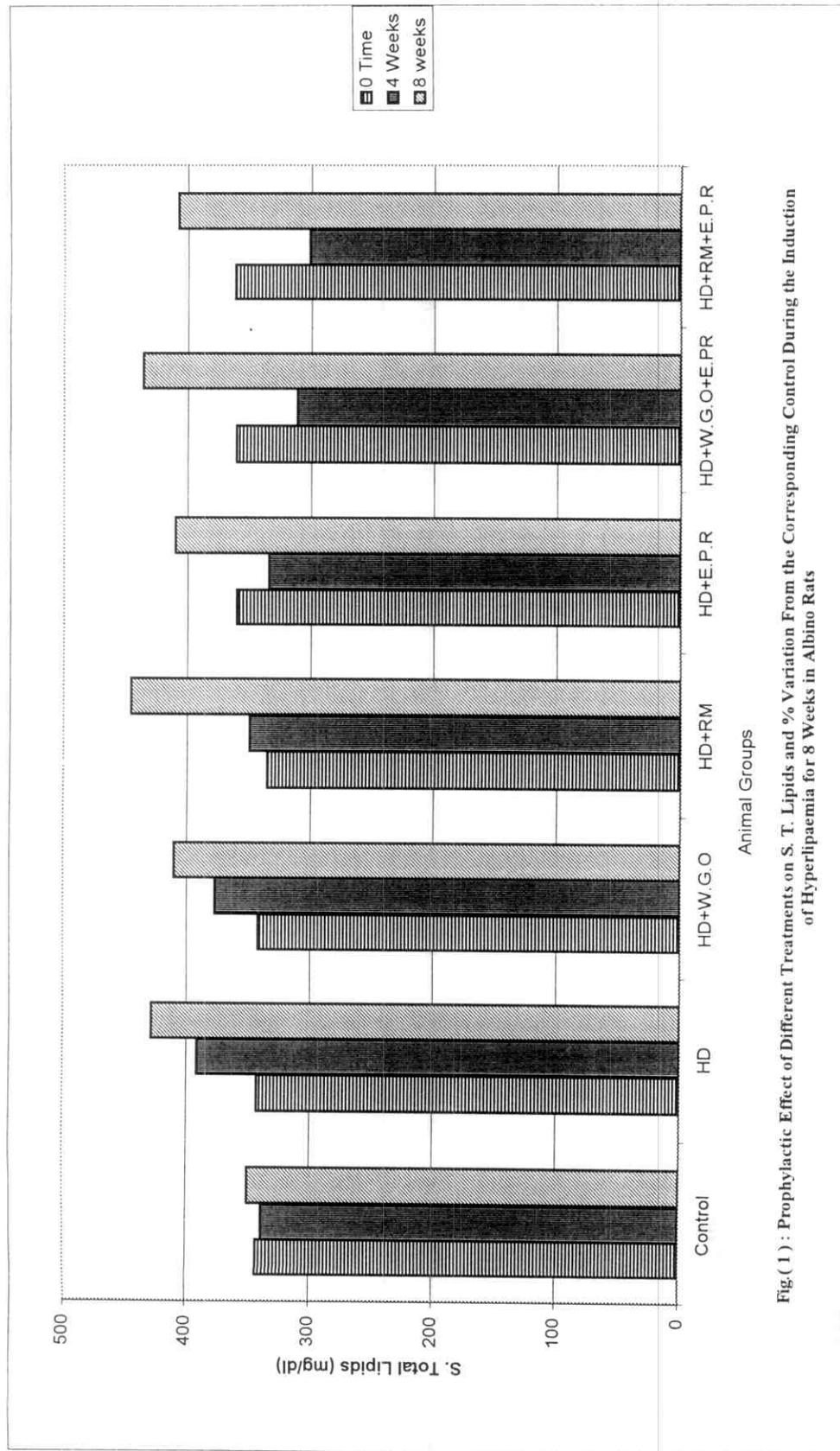


Fig.(1) : Prophylactic Effect of Different Treatments on S. T. Lipids and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(3): Prophylactic Effect of Different Treatments on s.Triglycerides{mg/dl} and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. Triglycerides{mg/dl}				
	0 Time	4 Weeks	% Var.	8 weeks	% Var.
1 Control (Normal Diet)	58.1 ± 7.21	57.0 ± 11.3 †	1.9 ↓	60.5 ± 8.26 †	4.1 ↑
2 Hyperlipaemic Diet (HD)	55.2 ± 6.24	60.7 ± 11.4 †	9.9 ↑	123.4 ± 12.2 ***	123.6 ↑
3 HD+W.G.O	52.4 ± 7.39	54.6 ± 8.38 †	4.2 ↑	91.0 ± 4.76 ***	73.7 ↑
4 HD+RM	46.4 ± 10.5	54.8 ± 5.80 †	18.1 ↑	74.0 ± 13.5 **	59.5 ↑
5 HD+E.P.R	47.8 ± 11.7	57.9 ± 12.1 †	21.1 ↑	84.5 ± 4.78 ***	76.8 ↑
6 HD+W.G.O+E.P.R	55.6 ± 7.90	61.6 ± 13.2 †	10.8 ↑	82.0 ± 12.8 **	47.5 ↑
7 HD+RM+E.P.R	60.1 ± 2.29	70.0 ± 8.16 *	16.5 ↑	81.5 ± 5.96 ***	35.6 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

Table(4): Prophylactic Effect of Different Treatments on s. T .Cholesterol{mg/dl} and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups		S. T. Cholesterol{mg/dl}				
		0 Time	4 Weeks	% Var.	8 weeks	% Var.
1	Control (Normal Diet)	54.9 ± 6.16	51.3 ± 8.83 †	6.6 ↓	55.9 ± 4.34 †	1.8 ↑
2	Hyperlipaemic Diet (HD)	45.7 ± 8.28	71.7 ± 11.1 **	56.9 ↑	73.1 ± 8.16 ***	60.0 ↑
3	HD+W.G.O	48.6 ± 7.93	65.7 ± 6.30 **	35.2 ↑	84.4 ± 6.00 ***	73.7 ↑
4	HD+RM	56.2 ± 8.27	76.7 ± 8.10 **	36.5 ↑	67.9 ± 8.32 *	20.8 ↑
5	HD+E.P.R	54.9 ± 6.25	71.0 ± 12.7 **	29.3 ↑	65.9 ± 11.4 *	20.0 ↑
6	HD+W.G.O+E.P.R	53.2 ± 7.93	70.5 ± 9.46 **	32.5 ↑	58.2 ± 3.76 †	9.4 ↑
7	HD+RM+E.P.R	52.6 ± 4.42	60.6 ± 6.03 *	15.2 ↑	53.3 ± 10.6 †	1.3 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

Table(5): Prophylactic Effect of Different Treatments on s.HDL.Cholesterol{mg/dl} and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups		S.HDL.Cholesterol{mg/dl}				
		0 Time	4 Weeks	% Var.	8 weeks	% Var.
1	Control (Normal Diet)	46.7 ± 2.59	43.5 ± 5.32 †	6.9 ↓	46.5 ± 4.99 †	0.43 ↓
2	Hyperlipaemic Diet (HD)	38.1 ± 4.90	42.6 ± 13.7 †	11.8 ↑	49.1 ± 5.35 **	28.9 ↑
3	HD+W.G.O	37.8 ± 3.49	55.1 ± 3.89 ***	45.8 ↑	69.4 ± 8.77 ***	83.6 ↑
4	HD+RM	41.1 ± 4.80	52.1 ± 11.9 *	26.8 ↑	54.8 ± 6.73 **	33.3 ↑
5	HD+E.P.R	41.8 ± 5.80	45.6 ± 10.4 †	9.1 ↑	51.9 ± 3.12 **	24.2 ↑
6	HD+W.G.O+E.P.R	40.0 ± 6.00	37.2 ± 6.59 †	7.0 ↓	48.9 ± 6.93 *	22.3 ↑
7	HD+RM+E.P.R	42.8 ± 9.20	42.9 ± 6.40 †	0.23 ↑	44.9 ± 5.83 †	4.9 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

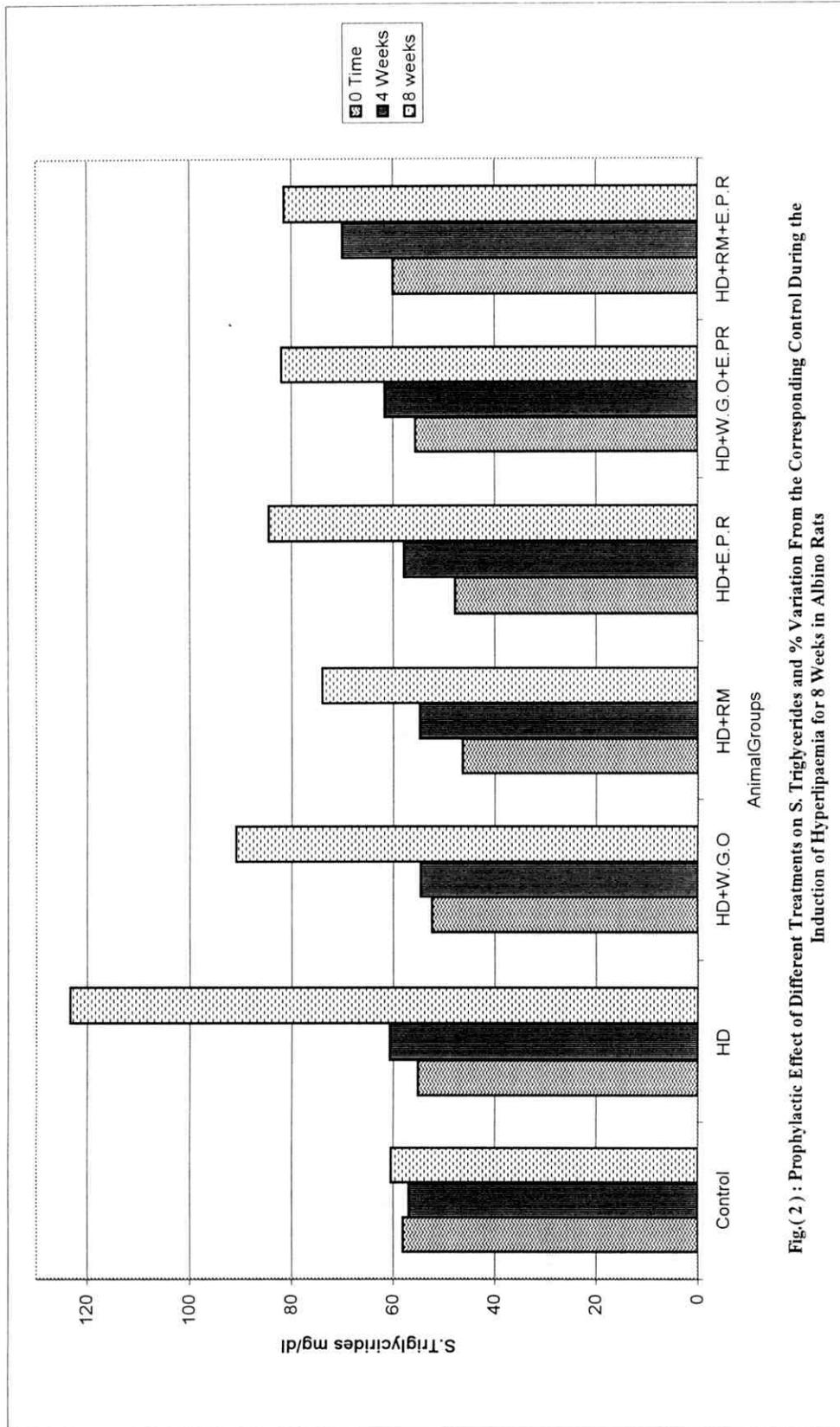


Fig.(2) : Prophylactic Effect of Different Treatments on S. Triglycerides and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(6): Prophylactic of Different Treatments on s. T. Cholesterol / HDL Cholesterol and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. T. Cholesterol / HDL Cholesterol				
	0 Time	4 Weeks	% Var.	8 weeks	% Var.
1 Control (Normal Diet)	1.18 ± 0.08	1.18 ± 0.16 †	00.0	1.20 ± 0.11 †	1.69 †
2 Hyperlipaemic Diet (HD)	1.20 ± 0.05	1.68 ± 0.25 ***	40.0 †	1.49 ± 0.11 ***	24.2 †
3 HD+W.G.O	1.29 ± 0.12	1.19 ± 0.18 †	7.7 ↓	1.22 ± 0.08 †	5.4 ↓
4 HD+RM	1.37 ± 0.06	1.47 ± 0.14 †	7.3 †	1.24 ± 0.19 †	9.5 ↓
5 HD+E.P.R	1.31 ± 0.18	1.56 ± 0.39 †	3.8 †	1.27 ± 0.20 †	3.1 ↓
6 HD+W.G.O+E.P.R	1.33 ± 0.15	1.90 ± 0.23 **	42.9 †	1.19 ± 0.21 †	10.5 ↓
7 HD+RM+E.P.R	1.23 ± 0.15	1.41 ± 0.19 *	14.6 †	1.19 ± 0.12 †	3.3 ↓

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001



Animal Groups

Fig.(4) : Prophylactic Effect of Different Treatments on S. T. Cholesterol/HDL Cholesterol and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

respectively (Table 5 and Fig. 3).

The risk ratio values (T.cholesterol/HDL-cholesterol) were shown in table (6) and Fig. (4). The ameliorative effect of different antioxidants was clearly observed compared with the +ve control group.

In general, E.P.R seems to be the more potent agent, which its prophylactic effect against hyperlipaemia was more pronounced.

1.2. Effect on hepato-renal function.

The hyperlipidemic diet fed to rats caused a marked elevation in s. ALT, s.AST and s.ALP by 303.6%, 49.3% and 44.0%, respectively (Tables 7-9 & Fig's 5-7). These data proved that the hyperlipaemic diet caused a significant effect on liver leading to liver dysfunction. All groups of animals who received different antioxidants belonging the hyperlipaemic diet s.ALT and s.ALP levels were significantly increased. s.AST levels were also increased in all treated groups except those who received W.G.O., RM and W.G.O. plus E.P.R.

Tables (10-13) and Fig's (8-9) reveals the effect of different antioxidants on serum total proteins, albumin, globulin and Alb/Glob. Serum total proteins were significantly decreased in the group of animals fed on th hyperlipaemic diet whereas albumin was slightly decreased. There are some fluctuations in the levels of serum proteins and albumin in all groups received the specific diet plus antioxidants during the 8 weeks of experiment.

Despite that there were some significant values in the level of serum creatinine and urea in all treated groups (Tables 14,15 and Fig's 10,11), these obtained results were still in the normal range of the respected parameters in albino rats and not considered as pathological values.

Table(7): Prophylactic Effect of Different Treatments on s. A LT(u/ml) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Animal Groups	S. A LT(u/ml)				
	0 Time	4 Weeks	% Var.	8 weeks	% Var.
1 Control (Normal Diet)	31.5 ± 6.19	32.9 ± 3.54 †	4.4 †	34.4 ± 2.61 †	9.2 †
2 Hyperlipaemic Diet (HD)	28.0 ± 2.83	61.6 ± 4.41 ***	120.†	113.0 ± 8.66 ***	303.6 †
3 HD+W.G.O	31.0 ± 3.08	52.0 ± 5.42 ***	67.6 †	59.3 ± 8.14 ***	91.3 †
4 HD+RM	29.6 ± 3.58	56.3 ± 5.31 ***	90.2 †	77.7 ± 4.62 ***	162.5 †
5 HD+E.P.R	27.8 ± 5.02	54.8 ± 7.18 ***	97.1 †	79.3 ± 3.77 ***	185.3 †
6 HD+W.G.O+E.P.R	28.8 ± 3.40	56.2 ± 5.47 ***	95.1 †	76.0 ± 6.38 ***	163.9 †
7 HD+RM+E.P.R	28.2 ± 3.19	53.0 ± 7.79 ***	87.9 †	95.0 ± 5.20 ***	236.9 †

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

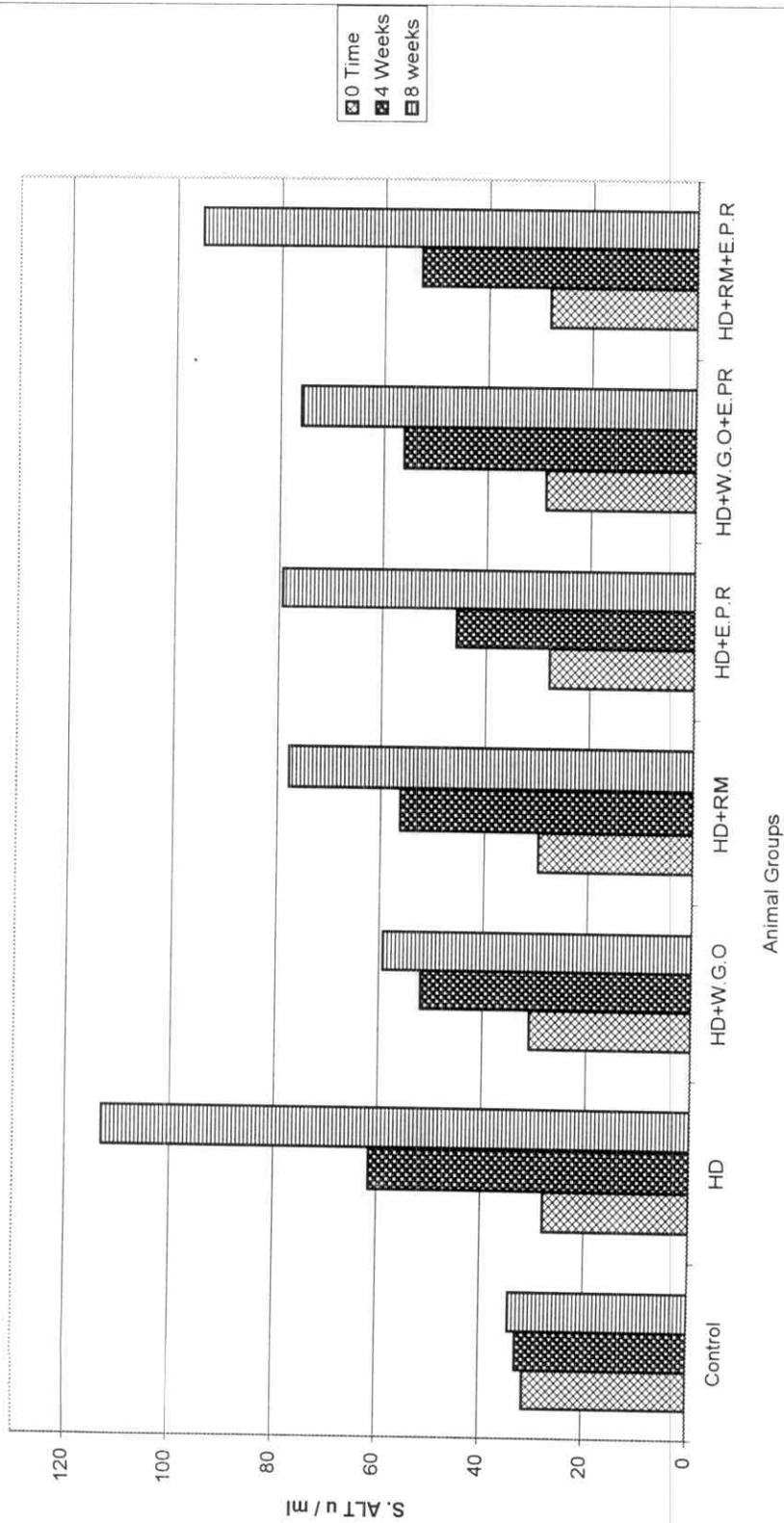


Fig.(5) : Prophylactic Effect of Different Treatments on S. ALT and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(8): Prophylactic Effect of Different Treatments on s.AST (U / ml) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Animal Groups	S.AST (U / ml)				% Var.
	0 Time	4 Weeks	8 weeks	% Var.	
1 Control (Normal Diet)	68.3 ± 12.8	69.1 ± 4.16 †	66.3 ± 10.3 †	1.2 ↑	2.9 ↓
2 Hyperlipaemic Diet (HD)	71.8 ± 8.06	99.2 ± 11.8 **	107.2 ± 13.2 ***	38.2 ↑	49.3 ↑
3 HD+W.G.O	68.5 ± 13.1	89.9 ± 5.24 **	69.8 ± 3.20 †	31.2 ↑	1.9 ↑
4 HD+RM	69.2 ± 9.47	86.0 ± 11.3 **	66.5 ± 11.7 †	24.3 ↑	3.9 ↓
5 HD+E.P.R	60.6 ± 4.83	85.6 ± 9.02 ***	77.3 ± 3.20 ***	41.3 ↑	27.6 ↑
6 HD+W.G.O+E.P.R	71.4 ± 4.51	89.0 ± 14.5 **	72.5 ± 9.88 †	24.6 ↑	1.5 ↑
7 HD+RM+E.P.R	74.6 ± 1.95	85.7 ± 9.18 **	65.6 ± 13.9 †	14.9 ↑	12.1 ↓

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

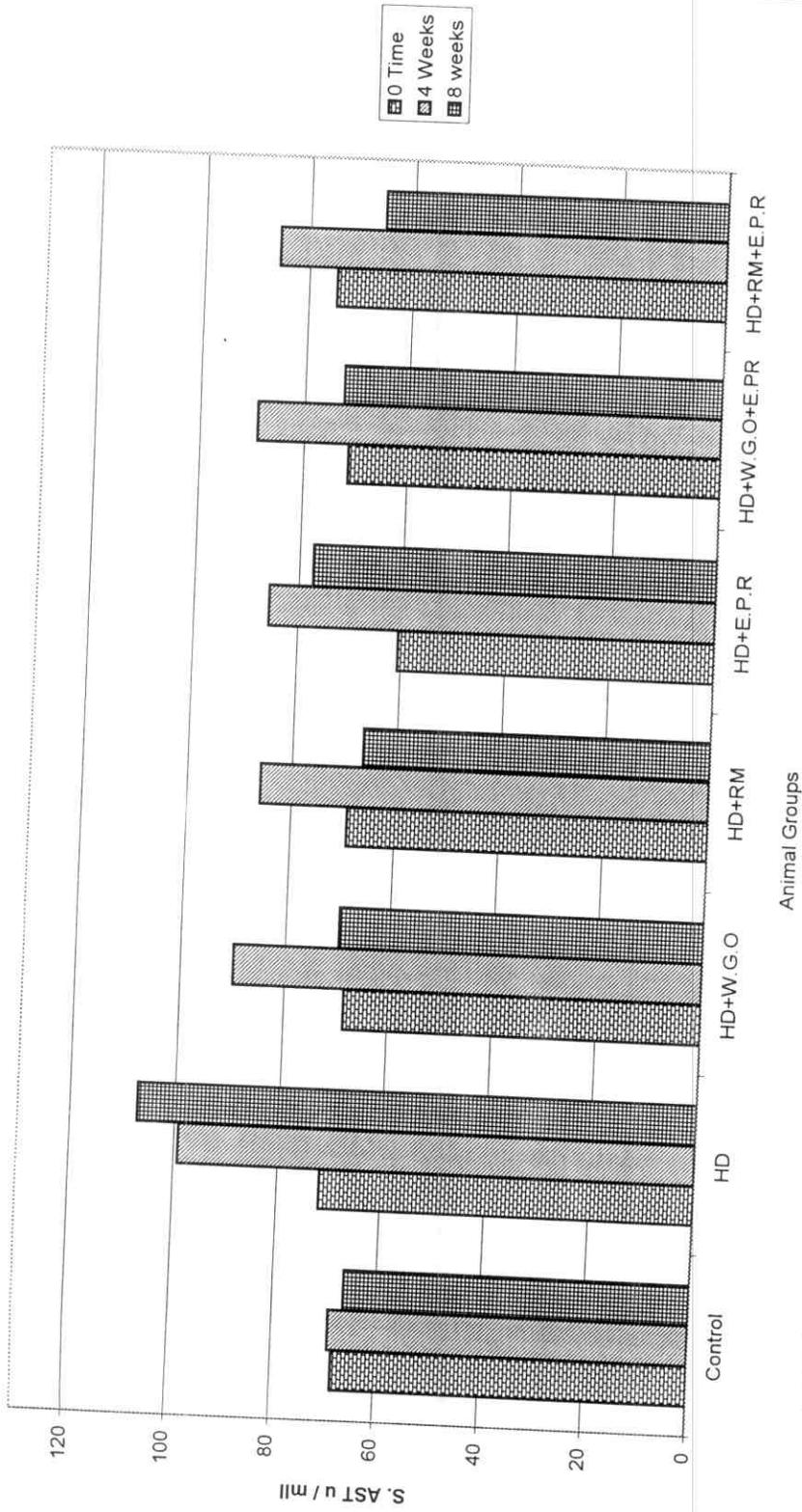


Fig.(6) : Prophylactic Effect of Different Treatments on S. AST and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(9): Prophylactic Effect of Different Treatments on s.Alk.P (U/L) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Animal Groups	S.Alk.P (U/L)			
	0 Time	4 Weeks	8 weeks	% Var.
1 Control (Normal Diet)	378.3 ± 51.7	378.3 ± 80.4 †	377.5 ± 61.8 †	0.00
2 Hyperlipaemic Diet (HD)	328.1 ± 40.4	486.7 ± 62.4 ***	472.5 ± 61.6 *	48.3 †
3 HD+W.G.O	410.2 ± 44.4	504.5 ± 34.2 **	457.0 ± 28.5 *	23.0 †
4 HD+RM	387.8 ± 45.4	495.3 ± 71.6 **	625.3 ± 33.0 ***	27.7 †
5 HD+E.P.R	405.4 ± 42.4	394.8 ± 73.0 †	536.8 ± 79.8 **	2.6 ↓
6 HD+W.G.O+E.P.R	351.0 ± 30.3	453.7 ± 93.1 **	461.7 ± 21.1 ***	29.3 †
7 HD+RM+E.P.R	364.4 ± 68.5	318.8 ± 52.9 †	507.3 ± 52.0 **	12.5 ↓

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

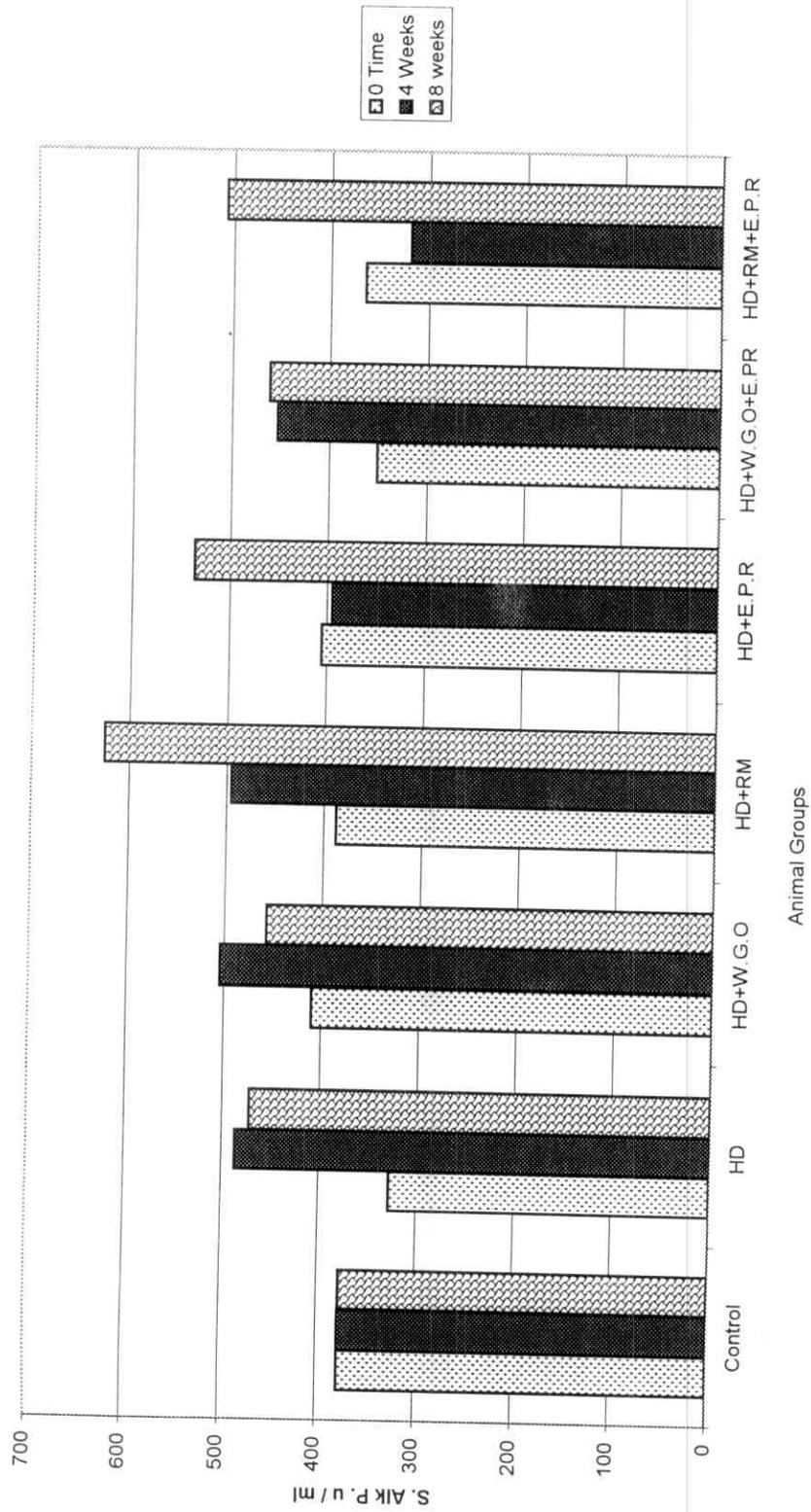


Fig.(7) : Prophylactic Effect of Different Treatments on S. ALK and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(10): Prophylactic Effect of Different Treatments on s T. Protein (gm/dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. T. Protein (gm/dl)					
	0 Time	4 Weeks	% Var.	8 weeks	% Var.	
1 Control (Normal Diet)	6.65 ± 0.48	6.65 ± 0.33 †	0.0	6.50 ± 0.41 †	2.2 ↓	
2 Hyperlipaemic Diet (HD)	5.82 ± 0.41	5.54 ± 0.39 †	4.8 ↓	5.35 ± 0.55 *	8.0 ↓	
3 HD+W.G.O	5.83 ± 0.22	5.97 ± 1.23 †	2.4 ↑	5.89 ± 0.73 †	1.0 ↑	
4 HD+RM	6.73 ± 0.70	5.67 ± 0.43 **	15.7 ↓	5.15 ± 0.39 ***	23.5 ↓	
5 HD+E.P.R	6.00 ± 0.36	6.08 ± 1.10 †	1.3 ↑	5.07 ± 0.44 ***	15.5 ↓	
6 HD+W.G.O+E.P.R	7.03 ± 0.72	5.64 ± 0.66 **	19.8 ↓	5.28 ± 0.49 ***	24.9 ↓	
7 HD+RM+E.P.R	5.52 ± 0.51	5.85 ± 0.65 †	5.8 ↑	5.26 ± 0.36 †	4.7 ↓	

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

Table(11): Prophylactic Effect of Different Treatments on s. Albumin (gm/dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Animal Groups	S. Albumin (gm/dl)					
	0 Time	4 Weeks	% Var.	8 weeks	% Var.	% Var.
1 Control (Normal Diet)	3.80 ± 0.16	3.66 ± 0.48 †	3.7 ↓	3.61 ± 0.15 †	5.0 ↓	5.0 ↓
2 Hyperlipaemic Diet (HD)	3.48 ± 0.08	3.09 ± 0.31 **	11.2 ↓	3.45 ± 0.30 †	0.86 ↓	0.86 ↓
3 HD+W.G.O	3.23 ± 0.21	3.22 ± 0.67 †	0.31 ↓	4.44 ± 0.27 ***	37.4 ↑	37.4 ↑
4 HD+RM	4.03 ± 0.15	3.31 ± 0.49 **	17.9 ↓	3.13 ± 0.16 ***	22.3 ↓	22.3 ↓
5 HD+E.P.R	3.62 ± 0.48	2.88 ± 0.43 *	20.4 ↓	3.34 ± 0.46 †	7.7 ↓	7.7 ↓
6 HD+W.G.O+E.P.R	3.90 ± 0.37	3.23 ± 0.44 *	17.2 ↓	4.14 ± 0.23 †	6.1 ↑	6.1 ↑
7 HD+RM+E.P.R	3.16 ± 0.30	3.25 ± 0.47 †	2.8 ↑	3.96 ± 0.21 †	25.3 ↑	25.3 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

Table(12): Prophylactic Effect of Different Treatments on s. Globulin (gm/dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. Globulin (gm/dl)				
	0 Time	4 Weeks	% Var.	8 weeks	% Var.
1 Control (Normal Diet)	2.85 ± 0.63	2.99 ± 0.28 †	4.9 ↑	2.84 ± 0.43 †	0.35 ↓
2 Hyperlipaemic Diet (HD)	2.34 ± 0.42	2.45 ± 0.47 †	4.7 ↑	1.90 ± 0.83 †	18.8 ↓
3 HD+W.G.O	2.60 ± 0.22	2.75 ± 0.42 †	5.7 ↑	1.45 ± 0.66 *	44.2 ↓
4 HD+RM	2.70 ± 0.50	2.36 ± 0.34 †	12.5 ↓	2.02 ± 0.29 *	25.1 ↓
5 HD+E.P.R	2.38 ± 0.32	3.20 ± 0.30 ***	34.4 ↓	1.73 ± 0.46 **	27.3 ↓
6 HD+W.G.O+E.P.R	3.13 ± 0.39	2.41 ± 0.37 **	23.0 ↓	1.14 ± 0.29 ***	63.5 ↓
7 HD+RM+E.P.R	2.36 ± 0.42	2.60 ± 0.35 †	10.1 ↑	1.30 ± 0.20 **	44.9 ↓

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

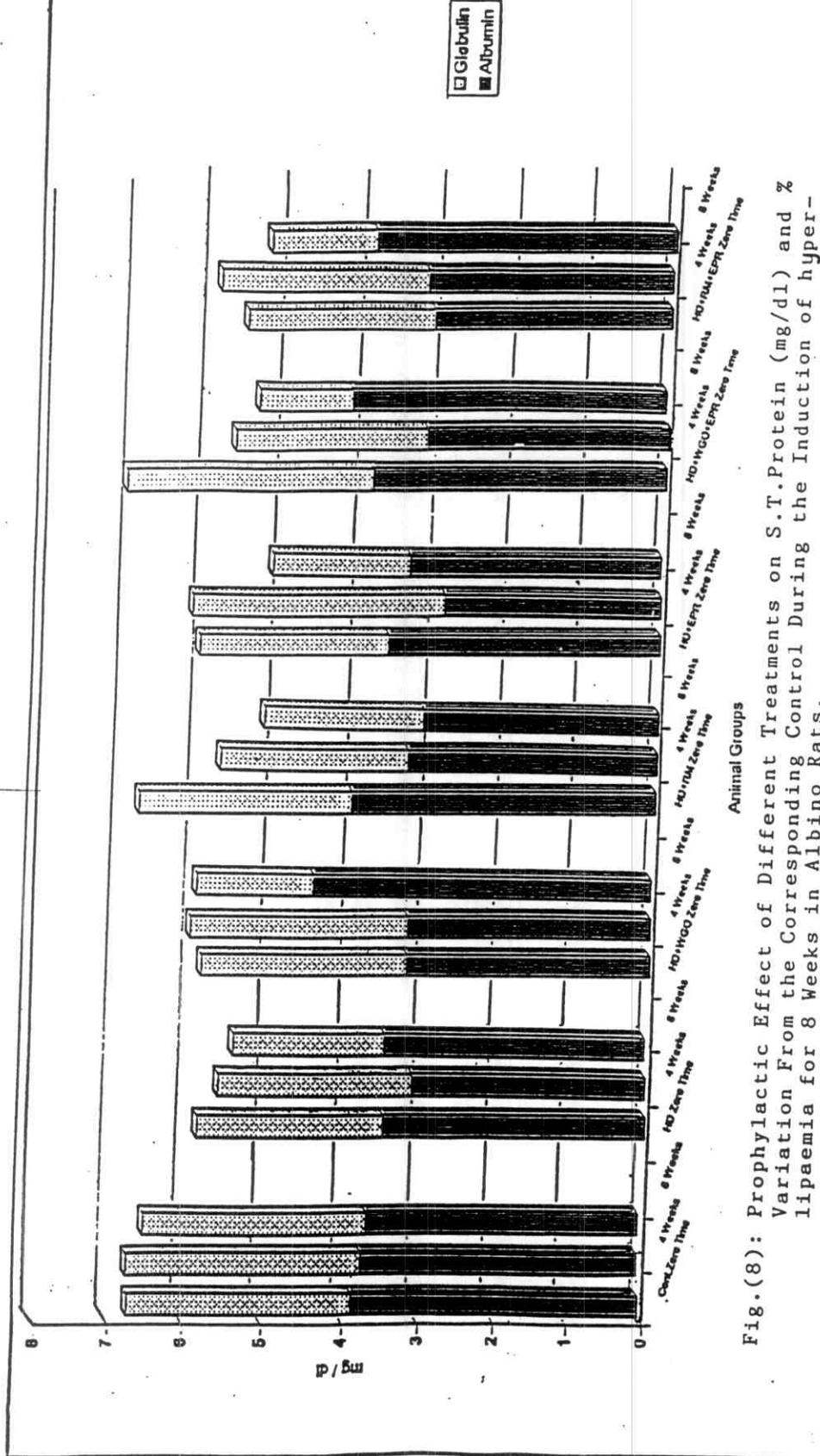


Fig.(8): Prophylactic Effect of Different Treatments on S.T.Protein (mg/dl) and % Variation From the Corresponding Control During the Induction of hyperlipaemia for 8 Weeks in Albino Rats.

Table(13): Prophylactic Effect of Different Treatments on s. Alb/Glob and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. Alb/Glob				
	0 Time	4 Weeks	% Var.	8 weeks	% Var.
1 Control (Normal Diet)	1.33 ± 0.30	1.22 ± 0.26 †	8.2 ↓	1.29 ± 0.23 †	3.0 ↓
2 Hyperlipaemic Diet (HD)	1.49 ± 0.15	1.26 ± 0.46 †	15.4 ↓	1.82 ± 0.15 **	22.3 ↑
3 HD+W.G.O	1.24 ± 0.17	1.17 ± 0.18 †	5.6 ↓	3.06 ± 0.56 **	146.8 ↑
4 HD+RM	1.49 ± 0.16	1.40 ± 0.36 †	6.0 ↓	1.55 ± 0.20 †	4.0 ↑
5 HD+E.P.R	1.52 ± 0.25	0.90 ± 0.03 ***	40.7 ↓	1.93 ± 0.77 †	26.9 ↑
6 HD+W.G.O+E.P.R	1.25 ± 0.03	1.34 ± 0.34 †	7.2 ↑	3.87 ± 0.88 ***	209.6 ↑
7 HD+RM+E.P.R	1.34 ± 0.30	1.25 ± 0.30 †	6.7 ↓	3.05 ± 0.52 ***	127.6 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

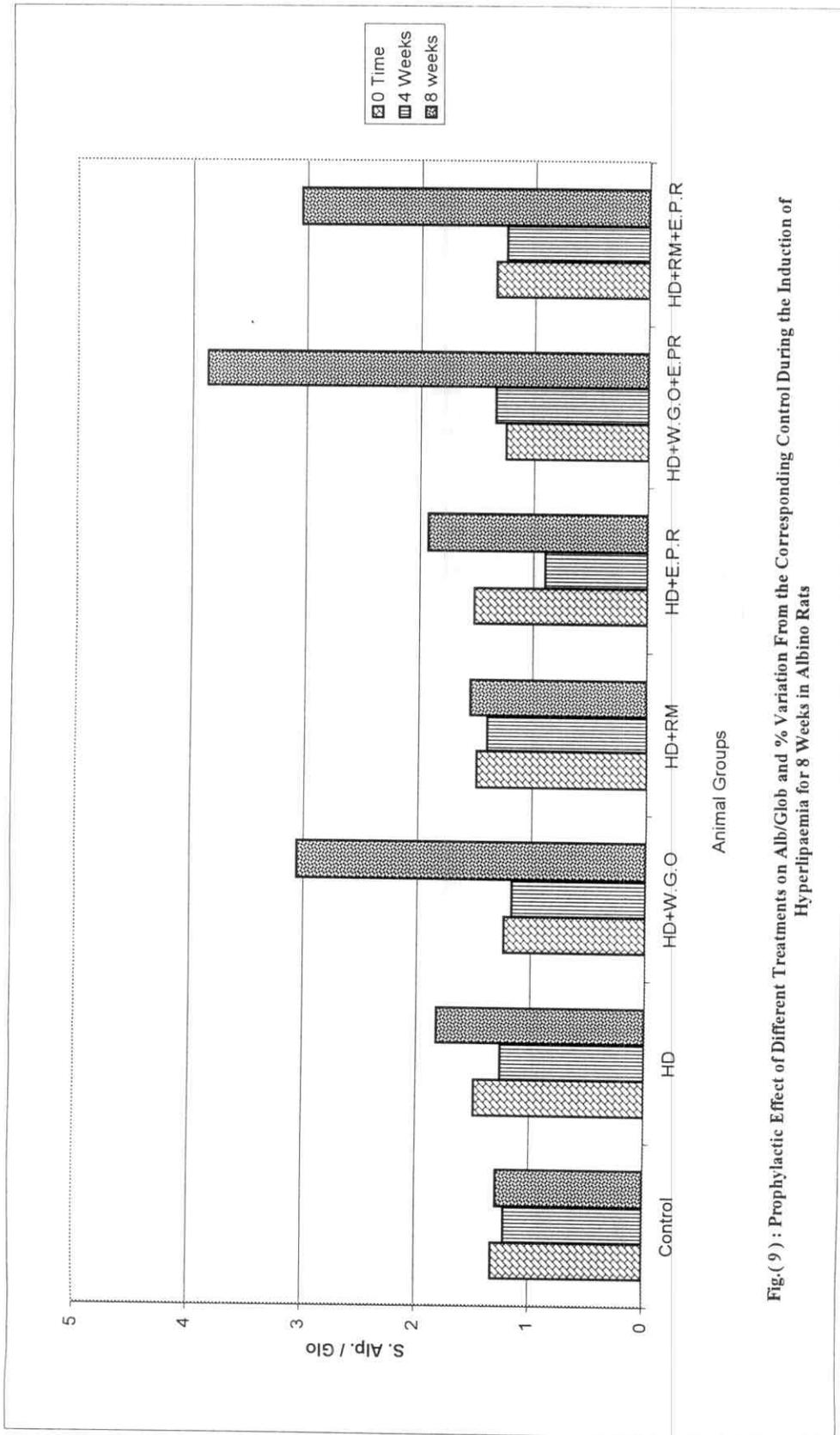


Fig.(9) : Prophylactic Effect of Different Treatments on Alb/Glob and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(14): Prophylactic Effect of Different Treatments on s. Creatinine (mg/ dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	S. Creatinine (mg/ dl)				% Var.
	0 Time	4 Weeks	8 weeks	% Var.	
1 Control (Normal Diet)	0.53 ± 0.04	0.53 ± 0.13 †	0.57 ± 0.04 †	0.0	7.5 †
2 Hyperlipaemic Diet (HD)	0.58 ± 0.02	0.64 ± 0.12 †	0.58 ± 0.06 †	10.3 †	0.00
3 HD+W.G.O	0.53 ± 0.03	0.69 ± 0.11 **	0.65 ± 0.07 *	30.2 †	22.6 †
4 HD+RM	0.53 ± 0.03	0.62 ± 0.03 ***	0.58 ± 0.07 *	16.9 †	9.4 †
5 HD+E.P.R	0.53 ± 0.02	0.70 ± 0.06 ***	0.65 ± 0.09 †	32.1 †	22.6 †
6 HD+W.G.O+E.P.R	0.53 ± 0.03	0.68 ± 0.07 ***	0.62 ± 0.03 ***	28.3 †	17.0 †
7 HD+RM+E.P.R	0.58 ± 0.02	0.61 ± 0.02 *	0.64 ± 0.04 *	5.1 †	10.3 †

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

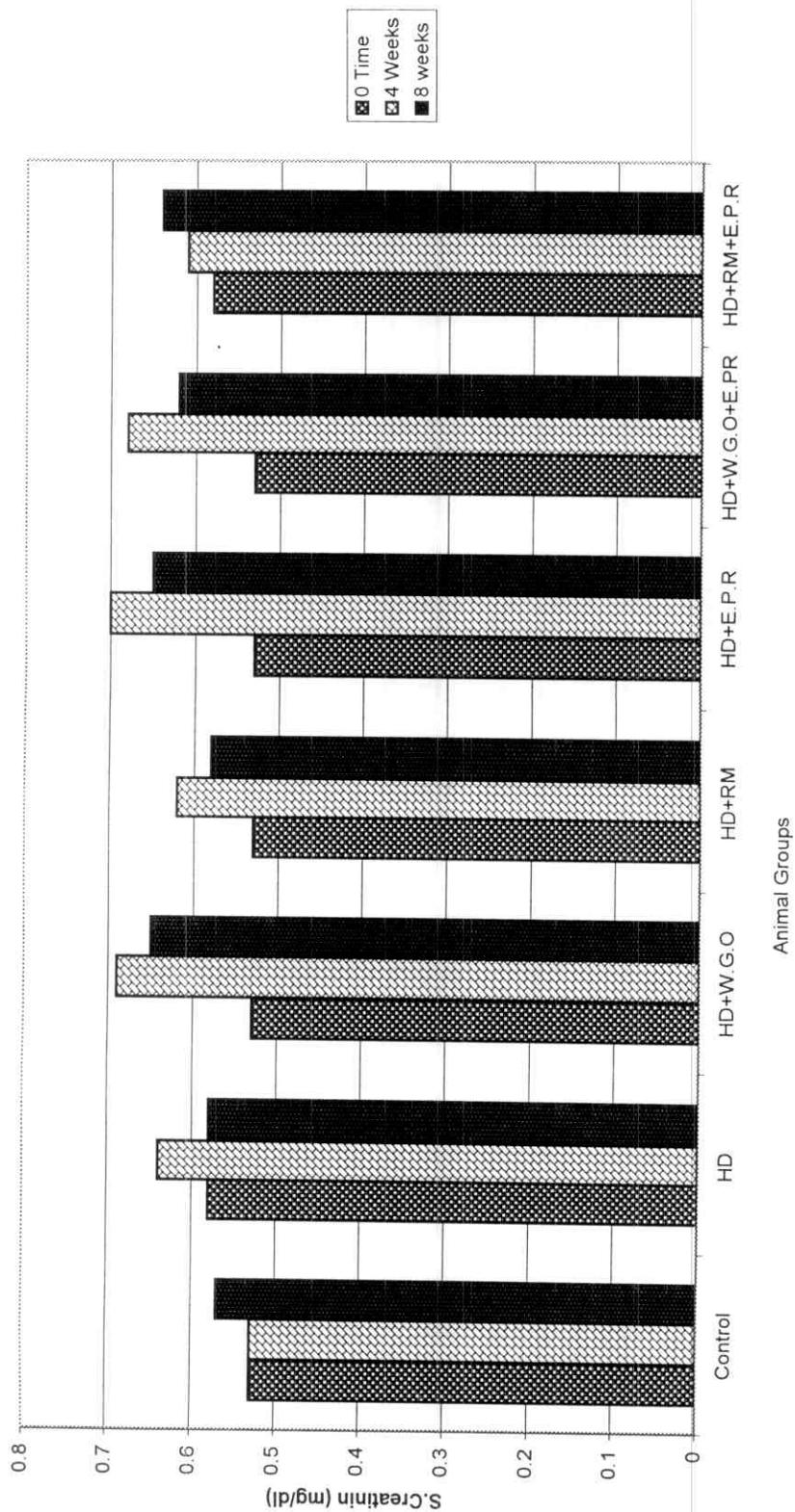


Fig.(10) : Prophylactic Effect of Different Treatments on S. Creatinin and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(15): Prophylactic Effect of Different Treatments on bl. Urea (mg/dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

	Animal Groups	Bl. Urea (mg/dl)				% Var.
		0 Time	4 Weeks	8 weeks	% Var.	
1	Control (Normal Diet)	32.1 ± 5.00	34.0 ± 5.20 †	33.8 ± 0.54 †	5.9 ↑	5.2 ↑
2	Hyperlipaemic Diet (HD)	27.2 ± 1.14	24.2 ± 4.20 †	33.7 ± 0.78 ***	11.0 ↓	23.9 ↑
3	HD+W.G.O	31.8 ± 6.25	21.6 ± 4.45 **	35.5 ± 0.56 †	32.1 ↓	11.6 ↑
4	HD+RM	32.1 ± 1.67	30.7 ± 5.89 †	34.3 ± 0.88 **	4.3 ↓	6.8 ↑
5	HD+E.P.R	31.6 ± 4.04	28.8 ± 5.04 †	37.4 ± 4.26 **	8.8 ↓	18.3 ↑
6	HD+W.G.O+E.P.R	26.4 ± 4.01	29.3 ± 3.00 †	36.7 ± 2.55 ***	10.9 ↑	39.0 ↑
7	HD+RM+E.P.R	30.4 ± 4.52	27.4 ± 3.29 †	35.9 ± 1.34 **	9.8 ↓	18.0 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

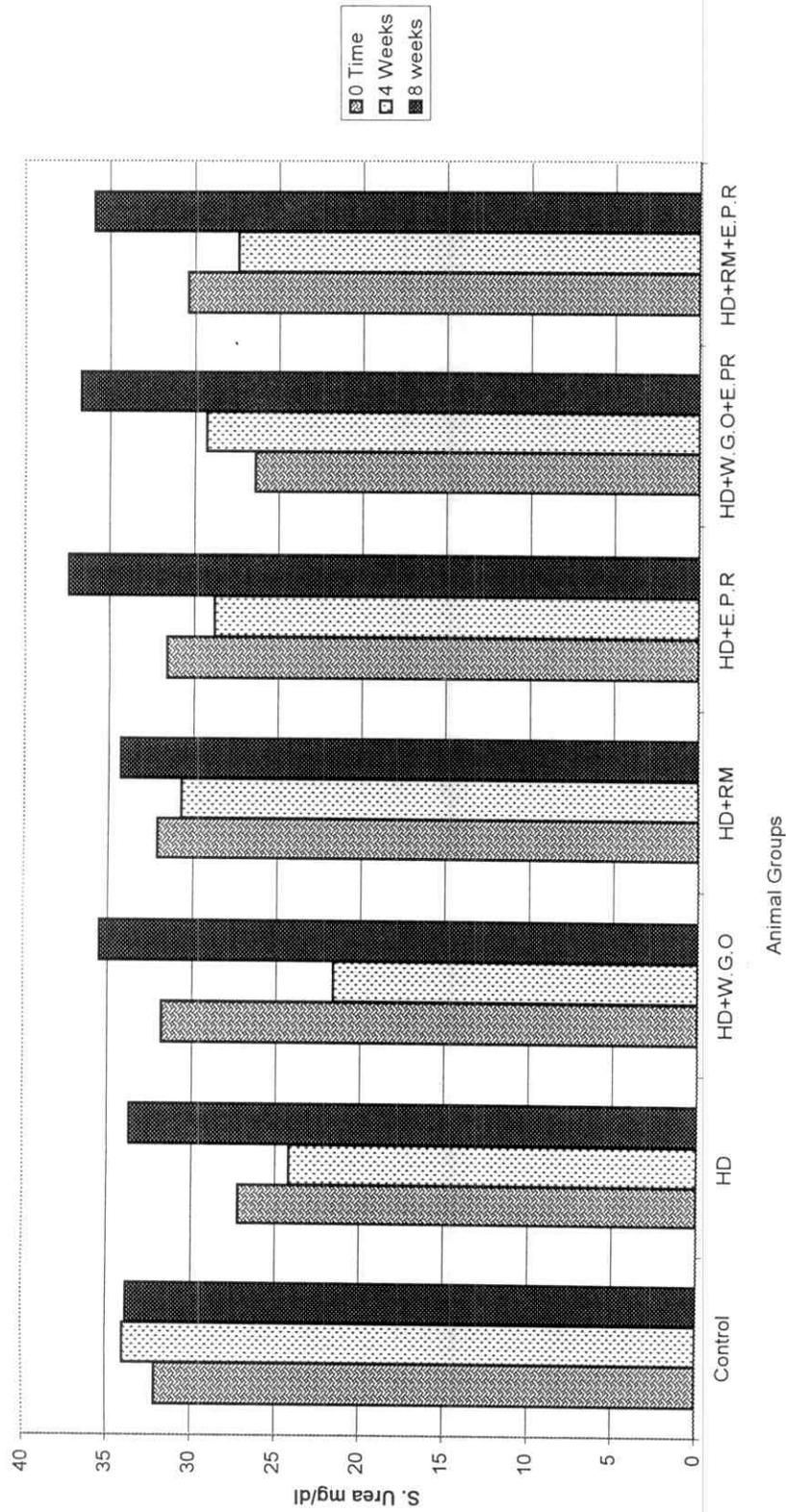


Fig.(11) : Prophylactic Effect of Different Treatments on S. Urea and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(16) : Prophylactic Effect of Differents Treatments on s.Uric acid (mg/dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups		S. Uric acid (mg/dl)				
		0 Time	4 Weeks	% Var.	8 weeks	% Var.
1	Control (Normal Diet)	1.98 ± 0.39	2.10 ± 0.32 †	6.1 †	1.99 ± 0.48 †	0.51 †
2	Hyperlipaemic Diet (HD)	1.55 ± 0.28	0.90 ± 0.08 ***	41.9 ↓	1.77 ± 0.43 *	14.2 †
3	HD+W.G.O	1.62 ± 0.37	1.58 ± 0.33 †	2.4 ↓	3.06 ± 0.19 **	88.9 †
4	HD+RM	1.70 ± 0.41	0.99 ± 0.18 **	41.7 ↓	2.39 ± 0.60 *	40.5 †
5	HD+E.P.R	1.68 ± 0.23	0.91 ± 0.26 ***	45.8 ↓	3.11 ± 0.62 **	85.1 †
6	HD+W.G.O+E.P.R	1.47 ± 0.22	1.04 ± 0.03 ***	29.2 ↓	3.29 ± 0.18 ***	123.8 †
7	HD+RM+E.P.R	1.46 ± 0.29	1.03 ± 0.10 **	29.4 ↓	2.75 ± 0.58 **	88.3 †

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

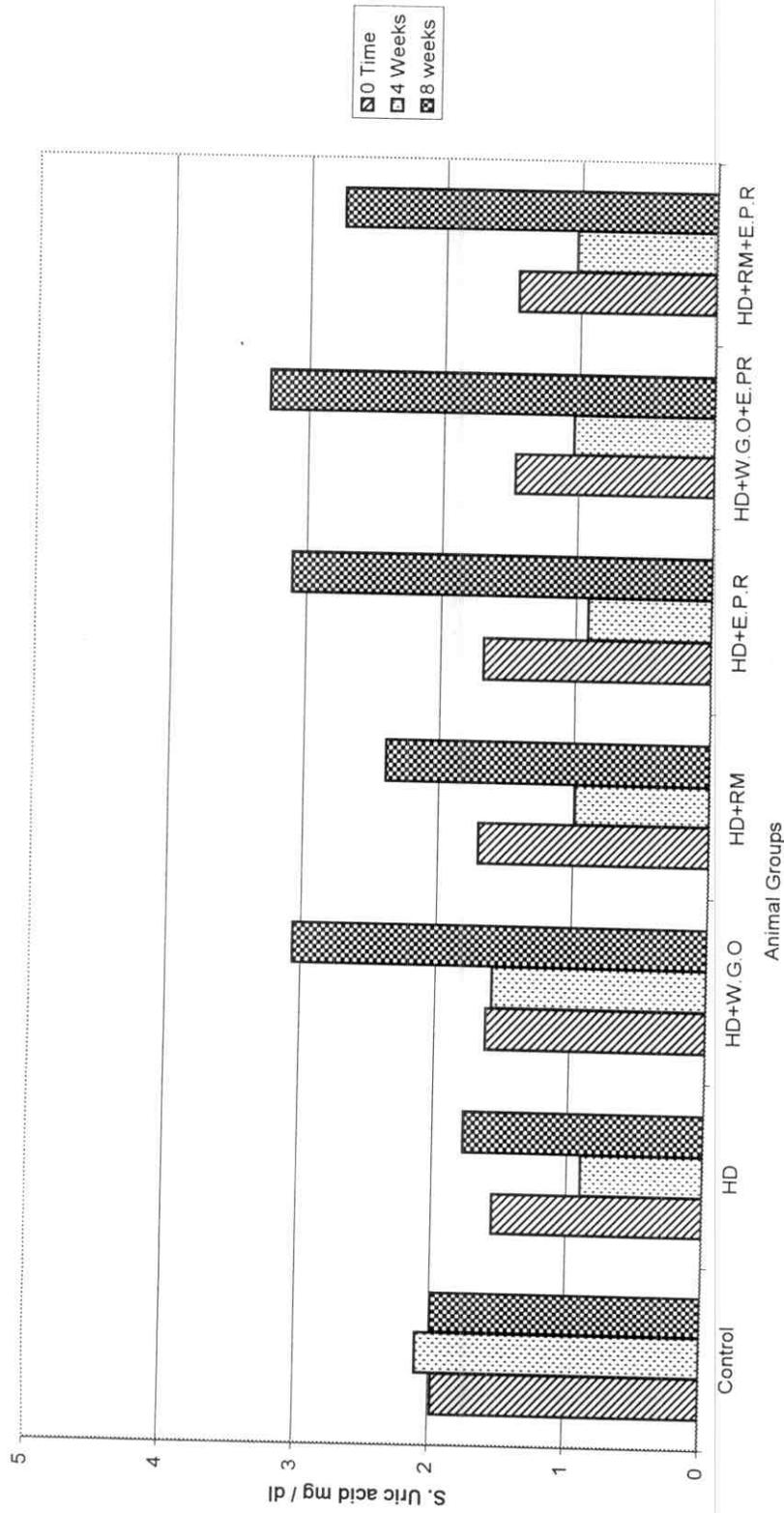


Fig.(12) : Prophylactic Effect of Different Treatments on S. Uric Acid and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Data in Table (16) and Fig. (12) reveals the effect of different treatments on serum uric acid. The hyperlipaemic diet was tended to elevate serum uric acid levels in all groups and the different antioxidants had no effect on these elevated levels.

From these results, it is clearly shown that neither the hyperlipaemic diet nor the different antioxidants had any effect on kidney function. On the other hand, the hyperlipidaemic diet was significantly affected liver function tests. The different antioxidants had a remarkable ameliorative effect on these functions, especially W.G.O.

1.3. Effect on antioxidant markers.

To evaluate the lipid peroxidation processes, erythrocyte superoxide dismutase (SOD), plasma malonodialdehyde (MDA) and reduced glutathione (GSH) were performed. These parameters are considered to be a markers for measurement of antioxidant potency.

Table (17) and Fig. (13) indicated that MDA was highly elevated by 71.5% from the pre-treatment value in the groups of rats which were received the hyperlipaemic diet, whereas in normal rats, the values of MDA did not affected through the 8 weeks of treatment. The addition of different antioxidants to the hyperlipaemic diet caused a significant increased in MDA activity by 11.9%, 25.6%, 26.2%, 44.2% and 55.4% in the groups treated with EPR, EPR plus WGO, WGO, RM + EPR and RM, respectively.

Since erythrocyte superoxide dismutase (SOD) was calculated as u/mgHb, so blood haemoglobin was also assayed (Tables 18, 19 and Fig's 14,15). SOD concentration was not affected in rats which were fed on the normal diet. On the other hand, animals fed on the hyperlipaemic diet showed a marked elevation in SOD calculated by 78.2% after 8 weeks.

This value was 15.8%, 16.6%, 26.6%, 45.2% and 71.7% for the groups of animals which fed on the hyperlipaemic diet plus EPR, WGO + EPR, RM, WGO and RM + EPR, respectively.

Table (20) and Fig. (16) revealed the effect of different treatments on reduced glutathione (GSH) . It is clearly shown that there is no any effect on GSH in the negative control group, while in the positive control group where rats fed on the hyperlipaemic diet, GSH was markedly decreased by 59.4%. In the other groups of rats which were fed on the hyperlipaemic diet plus the different antioxidants, this value was 11.1%, 17.4%, 26.0%, 35.0% and 60.9% for groups of RM, RM plus EPR, WGO, EPR and WGO plus EPR, respectively.

From the data obtained in this experiment, it is clearly deduced that all of the three antioxidants and their mixture had a clear and remarkable ameliorative effect against hyperlipaemia. The effect of EPR was more pronounced than WGO and RM. These results are agreed with those of **Erdinçler *et al.*, 1997; Abou-Safi *et al.*, 2002 and Dawood *et al.*, 2002.** The discussion of these results well held-up together with the results of the second experiment.

Table(17): Prophylactic Effect of Different Treatments on pl. MDA (n mol/ ml) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	Pl. MDA (n mol/ ml)			
	0 Time	4 Weeks	% Var.	8 weeks
1 Control (Normal Diet)	7.21 ± 1.54	7.59 ± 0.59 †	5.2 †	7.03 ± 1.22 †
2 Hyperlipaemic Diet (HD)	8.57 ± 1.48	12.2 ± 2.03 **	42.3 †	14.7 ± 0.82 ***
3 HD+W.G.O	8.00 ± 1.79	8.24 ± 1.58 †	3.0 †	10.1 ± 0.53 *
4 HD+RM	6.69 ± 1.01	7.84 ± 0.85 *	17.1 †	10.4 ± 0.88 ***
5 HD+E.P.R	9.38 ± 0.62	8.09 ± 1.19 *	13.7 †	10.5 ± 0.88 *
6 HD+W.G.O+E.P.R	8.36 ± 0.95	7.88 ± 0.80 †	5.7 †	10.5 ± 1.73 *
7 HD+RM+E.P.R	8.04 ± 1.94	8.28 ± 0.98 †	2.9 †	11.6 ± 1.43 **

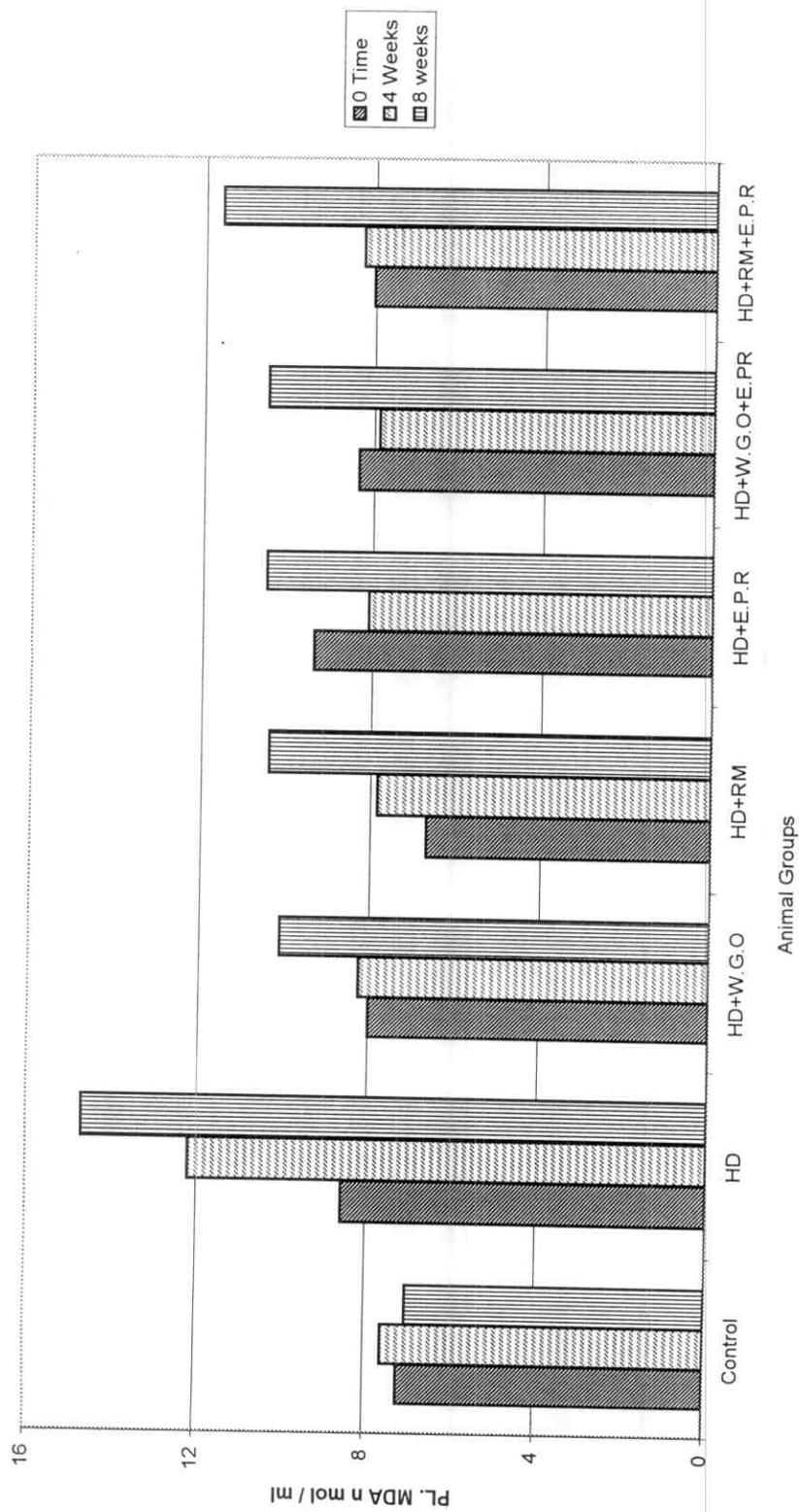
Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001



Fig(13) : Prophylactic Effect of Different Treatments on Pl. MDA and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(18): Prophylactic Effect of Different Treatments on Superoxid dismutase (u / mg Hb) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	Superoxid dismutase (u / mg Hb)				% Var.
	0 Time	4 Weeks	8 weeks	% Var.	
1 Control (Normal Diet)	4.40 ± 1.04	4.12 ± 0.79 †	4.54 ± 0.92 †	6.3 ↓	3.2 ↑
2 Hyperlipaemic Diet (HD)	3.54 ± 0.49	5.95 ± 1.35 **	6.31 ± 0.78 ***	68.1 ↑	78.2 ↑
3 HD+W.G.O	3.87 ± 0.16	4.70 ± 0.70 **	5.62 ± 0.79 ***	21.4 ↑	45.2 ↑
4 HD+RM	4.29 ± 0.75	4.63 ± 0.83 †	5.43 ± 1.32 *	7.93 ↑	26.6 ↑
5 HD+E.P.R	4.04 ± 1.00	4.28 ± 0.57 †	4.68 ± 1.10 †	5.94 ↑	15.8 ↑
6 HD+W.G.O+E.P.R	3.98 ± 0.36	4.10 ± 0.70 †	4.64 ± 0.48 **	3.02 ↑	16.6 ↑
7 HD+RM+E.P.R	3.36 ± 0.65	5.01 ± 1.73 *	5.77 ± 0.51 ***	49.1 ↑	71.7 ↑

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

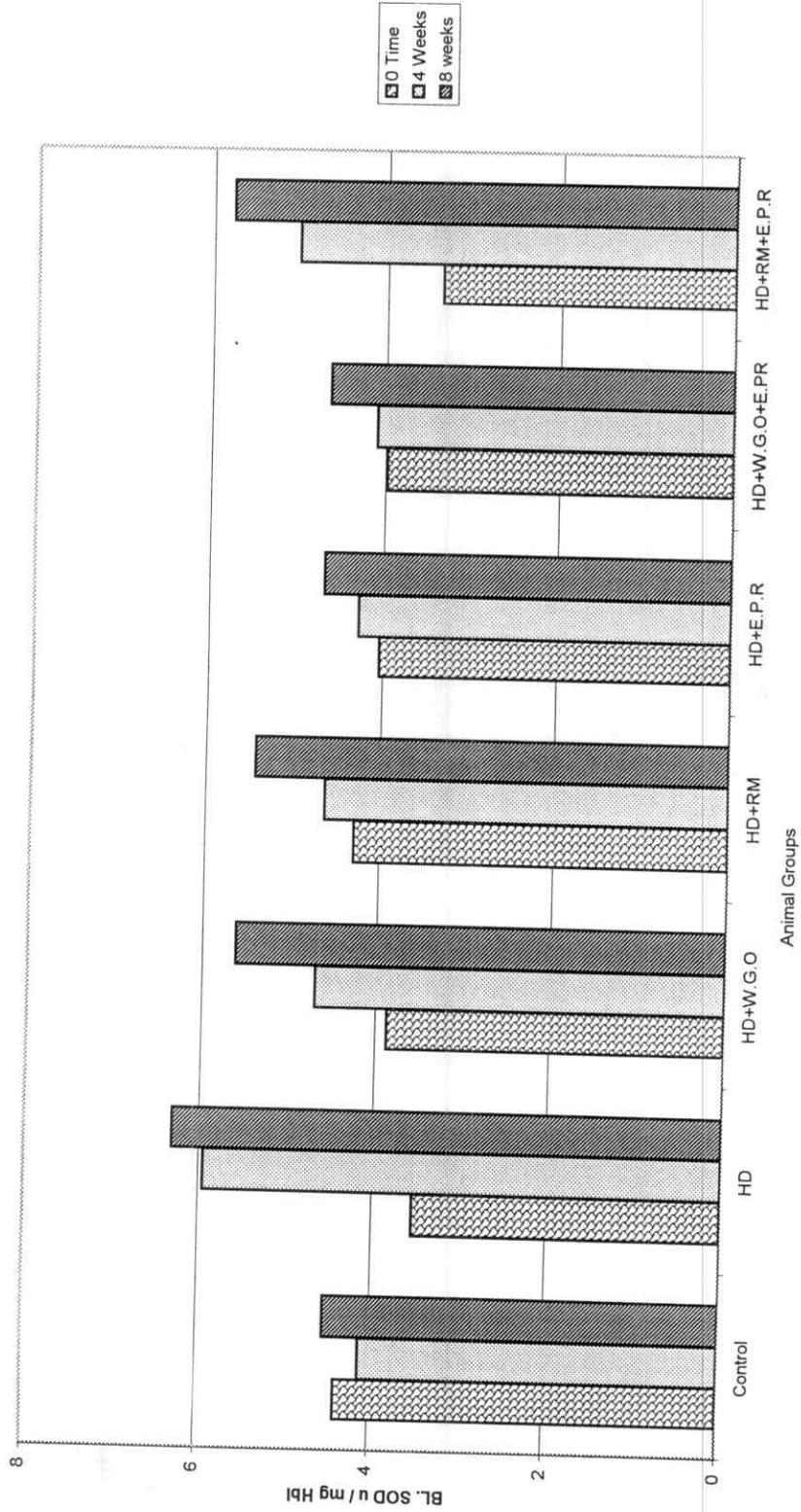


Fig (14) : Prophylactic Effect of Different Treatments on Superoxide Dismutase and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(19) : Prophylactic Effect of Different Treatments on Haemoglobin (gm/ dl) and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups	Haemoglobin (gm/ dl)				% Var.
	0 Time	4 Weeks	8 weeks	% Var.	
1 Control (Normal Diet)	14.5 ± 1.20	14.0 ± 2.79 †	14.6 ± 2.05 †	3.4 ↓	0.68 ↑
2 Hyperlipaemic Diet (HD)	14.9 ± 0.98	16.1 ± 2.63 †	14.3 ± 0.93 †	8.1 ↑	4.0 ↓
3 HD+W.G.O	14.7 ± 1.66	15.3 ± 3.73 †	13.4 ± 1.02 †	4.1 ↑	8.8 ↓
4 HD+RM	15.3 ± 1.23	15.2 ± 0.98 †	13.9 ± 3.48 †	0.65 ↓	9.2 ↓
5 HD+E.P.R	15.5 ± 0.70	15.4 ± 0.99 †	14.2 ± 3.18 *	0.65 ↓	8.4 ↓
6 HD+W.G.O+E.P.R	14.5 ± 0.22	14.7 ± 2.06 †	11.4 ± 0.88 ***	1.4 ↑	21.4 ↓
7 HD+RM+E.P.R	14.7 ± 0.20	13.1 ± 1.77 *	12.4 ± 1.33 **	10.9 ↓	15.6 ↓

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at P > 0.1

* Significant difference from the corresponding control at P < 0.05

** Highly significant difference from the corresponding control at P < 0.01

*** Very highly significant difference from the corresponding control at P < 0.001

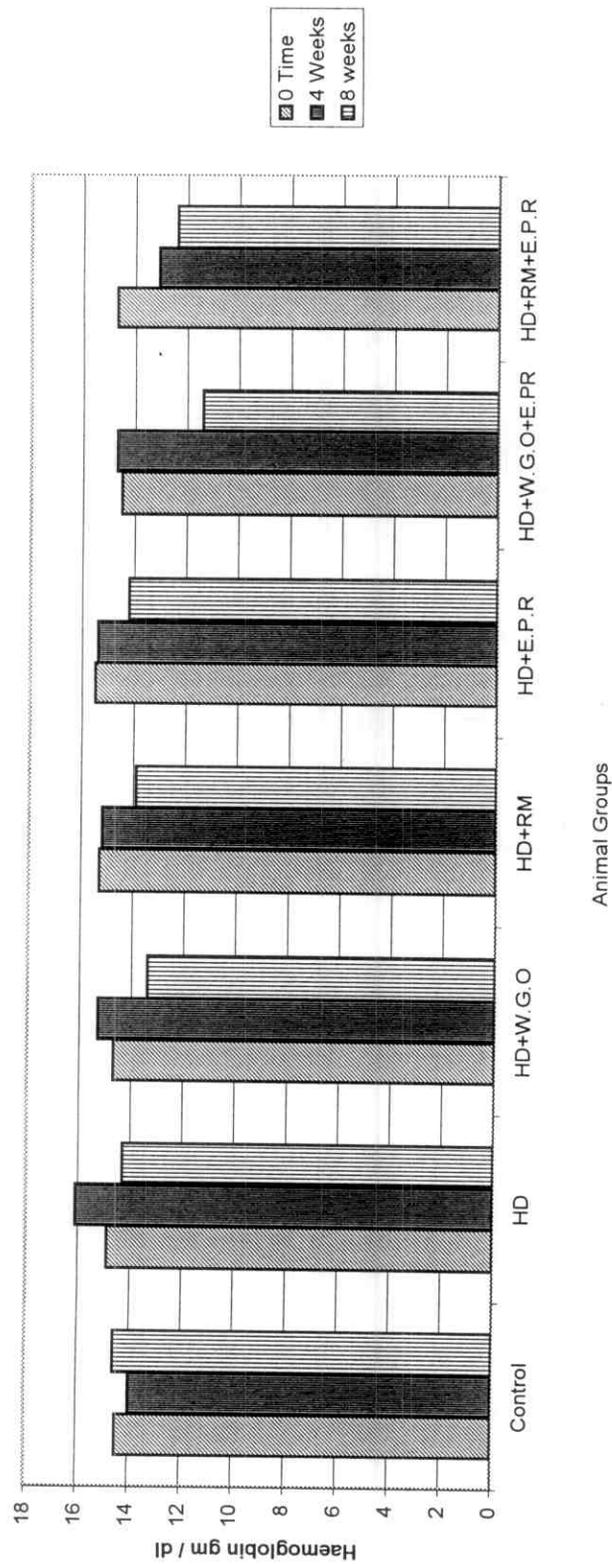


Fig.(15) : Prophylactic Effect of Different Treatments on Haemoglobin and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

Table(20): Prophylactic Effect of Different Treatments on bl. Reduced Glut.(mg/dl). and %Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats .

Animal Groups		Bl. Reduced Glut.(mg/dl).				% Var.
		0 Time	4 Weeks	% Var.	8 weeks	
1	Control (Normal Diet)	25.9 ± 3.07	24.1 ± 3.91 †	6.9 ↓	26.0 ± 6.39 †	0.39 ↑
2	Hyperlipaemic Diet (HD)	33.3 ± 3.70	23.3 ± 2.06 ***	30.0 ↓	13.5 ± 3.10 ***	59.4 ↓
3	HD+W.G.O	31.5 ± 5.28	29.2 ± 6.13 †	7.3 ↓	23.3 ± 3.41 **	26.0 ↓
4	HD+RM	24.1 ± 2.47	27.9 ± 6.63 †	15.7 ↑	21.4 ± 5.26 †	11.1 ↓
5	HD+E.P.R	32.0 ± 4.00	24.4 ± 2.91 *	23.7 ↓	20.8 ± 4.86 **	35.0 ↓
6	HD+W.G.O+E.P.R	28.2 ± 2.48	24.3 ± 2.81 *	13.8 ↓	11.0 ± 2.75 ***	60.9 ↓
7	HD+RM+E.P.R	21.8 ± 5.40	22.5 ± 1.84 †	3.2 ↑	18.0 ± 4.45 †	17.43 ↓

Each value represents the mean of 8 rats ± S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

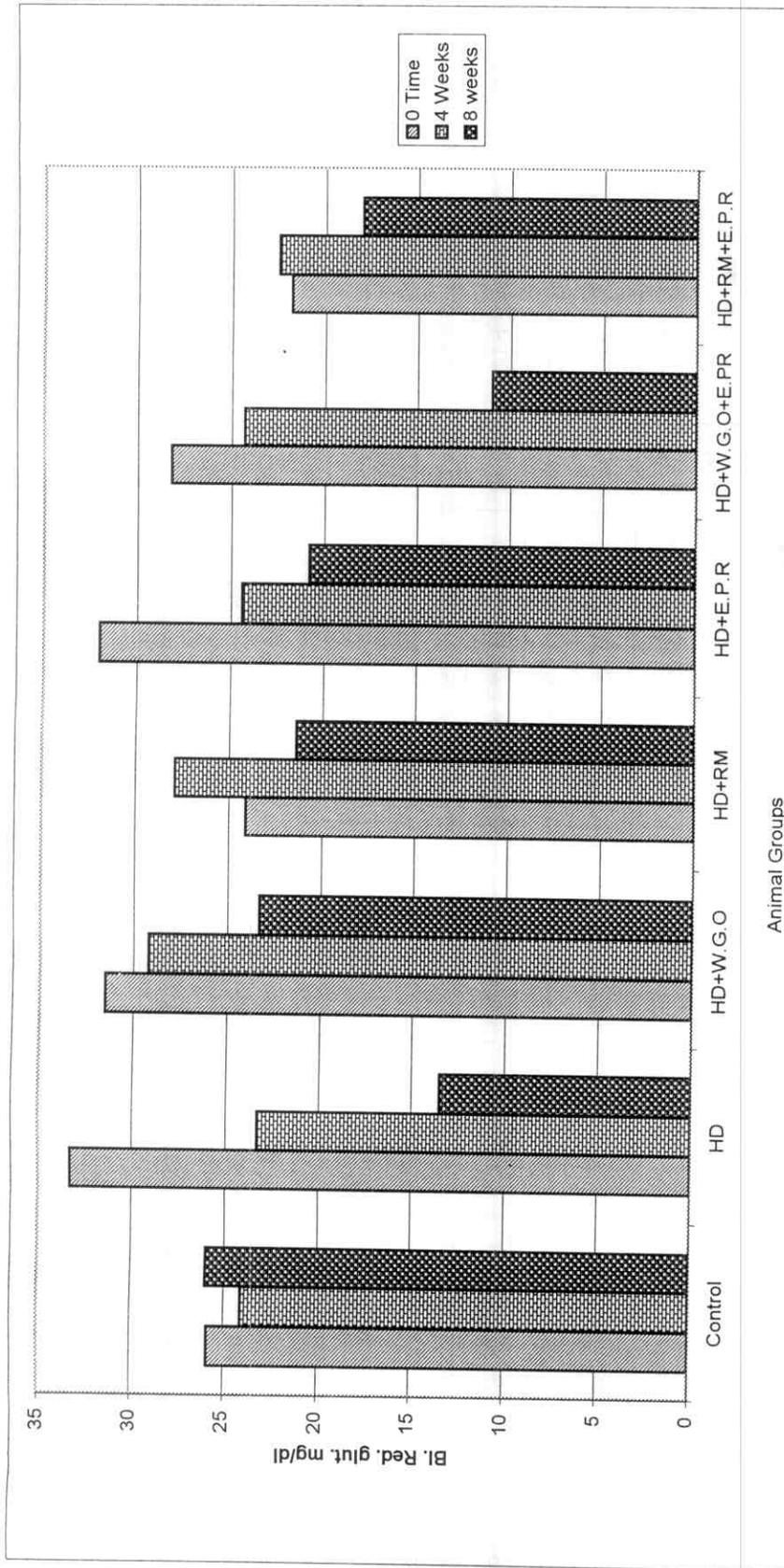


Fig.(16) : Prophylactic Effect of Different Treatments on Bl. Reduced Glut. and % Variation From the Corresponding Control During the Induction of Hyperlipaemia for 8 Weeks in Albino Rats

2. Curative effect of different treatments on hyperlipaemic rats.

In this experiment, rats were fed on the hyperlipaemic diet for 10 weeks and then treated with the different antioxidants for 4 weeks. Lovastatin was used as reference standard hypolipaemic agent.

2.1. Induction of hyperlipaemia.

Table (21) illustrated the values of different parameters under investigation before and after induction of hyperlipaemia, whereas rats were fed on cholesterol and fat-rich diet for 10 weeks. Serum total lipids, triglycerides, total cholesterol and HDL-cholesterol were significantly increased by 46.1%, 217.3%, 81.2% and 79.6%, respectively. On the other hand the risk ratio of total cholesterol/HDL-cholesterol was not affected.

Lipid fractions are metabolized and absorbed as lipoproteins. Liver play an important role in regulation of lipid metabolism. Increased of lipid fractions in serum is mainly related to partial impairment of liver cells. Since these fractions are controlled by the liver through the absorption of lipoproteins from the intestine, so these elevations may due to the increase absorption of lipoproteins from the intestine (**Ingram, 1993**).

Induction of hyperlipaemia causes liver dysfunction which proved by the increase in s.ALT, s.AST and s.ALP by 87.7%, 68.8% and 24.3%, respectively. Serum proteins were slightly increased. Induction of hyperlipaemia did not affect renal function, but serum uric acid was elevated by 11.6% table (21).

After the induction of hyperlipaemia, superoxide dismutase and malondialdehyde were significantly increased by 45.3% and 41.8%,

respectively, while reduced glutathione was significantly decreased by 49.8% table (21).

Hyperlipaemia produced high levels of blood SOD and plasma MDA "as the end product of lipid peroxidation", concomitant with reduced levels of GSH indicating depression in the antioxidant system.

These results are in closely agreement with those of **Kok et al., 1991; Erdinler et al., 1997; Abou-Safi et al., 2002 and Abdel-Maksoud et al., 2002**. Cholesterol, one of the main risk factors, is suggested to exert a pro-oxidant effect . In hypercholesterolemia, the cholesterol content of erythrocytes, platelets, polymorphonuclear leukocytes and endothelial cells increases. This increase is reported to activate these cells and cause the enhanced production of oxygen free radicals (**Kok et al., 1991**). Stress, also, intensifies the release of stress hormones and their auto-oxidation yields free radicals with lipid peroxidation. Lipid oxidation and the generation of free radicals are considered to be a natural phenomena in biological system. The formation of reactive free radicals is mediated by a number of agents and mechanisms such as xenobiotic metabolism. The free radical formed are highly reactive with molecular oxygen forming peroxy radicals and hydroperoxides and thus initiating a chain reaction. Pro-oxidant states cause cellular lesions in all major organs by damaging cellular components and cell function.

Bermond, 1990 reported that the free radicals have been implicated in the etiology of several genetic as well as acquired metabolic disorders. One of these diseases is hyperlipidaemia which favors the formation of free radicals, leading to arterial damage and platelet aggregation.

Cholesterol oxidation products have received a lot of attention because of their involvement in the development of coronary artery disease. Typical oxidation products include cholestanetriol, enantiomeric-5,6-epoxides, 7-ketocholesterol, isomeric-7-hydroxycholesterol and 25-hydroxycholesterol (**Smith et al., 1967**). Cholesterol oxidation products are readily absorbed and incorporated into high-, low-, and very low-density lipoproteins. Cholestanetriol and 25-hydroxy cholesterol have been reported to be the most atherogenic product (**Taylor et al., 1979**). The same authors added that cholesterol oxidation products may have mutagenic and carcinogenic potential. Their studies indicate that the oxidation products of cholesterol and not cholesterol itself, are responsible for the observed cytotoxic and atherogenic effects.

The cytotoxic and atherogenic effects of oxysterols are significant in the initiation of atherosclerosis, which supports the role of oxysterols in cardiovascular disease. Cholesterol oxidation products are known to cause endothelial injury (**Addis and Park, 1998**). The atherosclerotic plaques can be initiated when the vascular endothelium is damaged, such as, through oxidative injury or mechanical stress. Subsequent cellular reactions involving activated monocytes and macrophages that secrete O_2^- , H_2O_2 and hydrolytic enzymes could injure neighboring cells and lead to more endothelial damage.

As a result of the increased amounts of free radicals during hyperlipaemia, the reduced glutathione (GSH) will consume and convert to the oxidized form (GSSG), so in case of hyperlipaemia, GSH was significantly decreased. On the other hand, lipid oxidation processes activate SOD enzyme which responsible for the converting of O_2^- to H_2O_2 , so the activity of this enzyme was increased in hyperlipaemic

animals. Due to the increase of lipid peroxides, the concentration of MDA is also increased (determined as thiobarbituric acid reactive substances). **Halliwell, 1991** described the derangements of cell metabolism that results in depletion of GSH and increased lipid peroxidation which evaluated as MDA. Through these processes the activity of SOD will increased.

2.2 The curative effect of different treatments on lipid profile:

The effect of W.G.O, RM, E.P.R and Lovastatin on serum total lipids, triglycerides, total cholesterol, HDL-cholesterol and risk ratio in hyperlipaemic rats is present in Table (22) and Fig's (17-20). The reference standard hypolipaemic agent, lovastatin, revealed an appreciated effects on the different lipid parameters of hyperlipaemic rats after treatment for 4 weeks. All parameters were decreased by 22.3%, 66.3%, 12.5%, 6.71% and 6.30% for total lipids, triglycerides, total cholesterol, HDL-cholesterol and risk ratio, respectively.

All the three antioxidants had a remarkable hypolipidaemic effect. This effect is in order E.P.R > RM > W.G.O. In the group of rats which were treated with E.P.R for 4 weeks, serum total lipids, triglycerides, total cholesterol, HDL-cholesterol and risk ratio were decreased by 9.92%, 55.5%, 26.7%, 20.5% and 7.38%, respectively.

RM did not altered the concentration of serum total lipids (insignificant increase by 2.17%), but decreased the level of triglycerides, total cholesterol and risk ratio by 24.9%, 9.62% and 1.61%, respectively. On the other hand HDL-cholesterol was significantly increased by 11.1%. W.G.O decreases serum triglycerides by 41.3% while risk ratio was increased by 16.5%. The level of total lipids, total cholesterol and HDL-cholesterol was increased by 5.02%, 29.3% and 10.5%, respectively.

2.3. Effect on hepato-renal function.

Table (23) and Fig's (21-23) reveals the effect of different treatments on liver function. This effect is highly appreciated after 4 weeks of treatment. Lovastatin reduced ALT by 48.7%, AST by 27.5% and ALP by 18.8%. E.P.R was significantly decrease the activity of ALT, AST and ALP by 45.1%, 24.6% and 27.1%, respectively. RM was also decreased these paramerters level by 37.1%, 20.7% and 43.1%, while W.G.O decrease these values by 32.7%, 19.9% and 16.8%, respectively.

It was observed that there is no dramatic changes in the level of serum proteins during the course of experiment (Table 24 and Fig.24,25). Concerning the effect of different treatments on kidney function, it was clearly shown from Table (25) and Fig. (26-28) that all the changes observed in serum creatinine, urea and uric acid were still in the normal range and not indicated as a pathological case.

2.4. Effect on antioxidant markers

Table (26) and Fig's (29-32) reveals the effect of different treatments on blood haemoglobin, SOD, MDA and GSH. Blood Hb was significantly increased by 10.3% and 10.0% in the groups of rats which were treated with lovastatin and RM respectively. While this value was increased only by 3.88% in the case of E.P.R and decreased by 2.19% for W.G.O. group. SOD concentration was significantly decreased by 25.9%, 20.1%, 19.0% and 17.1% in the groups of lovastatin, E.P.R, RM and W.G.O, respectively. The values of MDA were also decreased by 31.9%,23.6%, 11.7% and 8.55% in the groups of lovastatin, W.G.O, RM and E.P.R, respectively.

W.G.O decreases the value of GSH by 27.6%, RM decreases this value by 24.2%, E.P.R by 22.9% and lovastatin by 14.0%.

In general, it is clearly shown from the results that all the antioxidants used had an ameliorative effect. Their potency is in order E.P.R. > RM > W.G.O. These data agreed with those of (**Kappus, 1991; Stampfer *et al.*, 1993; Madhavi *et al.*, 1995**).

The antioxidant potency of W.G.O is due to the presence of α -tocopherol, phytic acid, selenium and carotins. α -Tocopherol is the most active form of vit. E which considered as a principle component of the secondary defense mechanisms against free radical mediated cellular injuries. In fact it is the only natural physiological lipid soluble antioxidant that can inhibit lipid peroxidation in cell membranes (**Kappus, 1991**). In biological systems, vit E plays an important role against several diseases such as several cancers, cardiovascular disease, platelet aggregation and decreased the risk of hyperlipaemia (**Gaby and Machilin, 1991; Stampfer *et al.*, 1993**).

In rosemary (RM) leaf extract, approximately 90% of the antioxidant activity has been attributed to carnosol, a phenolic diterpene, rosemarid diphenol, rosemarinic acid, carnosic acid, rosemanol, isorosemanol and epirosemanol (**Loliger, 1991**). Plant phenolic compounds such as flavonoids, sterols and various terpene related compounds are potent antioxidants (**Nakatani, 2000**). Many of these components have different therapeutic properties and are known for their anticarcinogenic, antimutagenic, antihyperlipidaemic and cardioprotective activities (**Stampfer *et al.*, 1993**).

Barnes *et al.*, 2002 reported that RM reduced the lipid peroxides content in biological cells.

The antioxidant activity of E.P.R is due to the presence of polyunsaturated fatty acids (PUFA), especially γ -linoleic acid or Omega-6 (**Newal *et al.*, 1996**). PUFA's have long been recommended for lowering plasma cholesterol levels and consequently lowering the risk of atherosclerosis (**Gey, 1995**).

Murray, 2000 reported that the antioxidant potential of E.P.R and the other natural products that containing vit. E, vit. C and GSH may dispose the toxic free radicals via scavenging them and suppressing their formation. The production of oxygen species is increased greatly when the ingestion of the antioxidants are diminished.

The primary biological role of antioxidants is to prevent the damage that reactive free radicals can cause to cellular components. Various biochemical defense mechanisms involving enzymes, trace elements and antioxidants protect the cellular components from oxidative damage. Antioxidants effectively retard the onset of lipid oxidation. In general, antioxidants are effective at very low levels. At higher levels most of them behave as pro-oxidants because of their involvement in the initiation reactions (**Madhavi *et al.*, 1995**).

Antioxidants are chemical compounds that capable to donating hydrogen radicals and reduces the primary radicals to non-radical chemical species and are thus converted to oxidized antioxidant radicals. (**Gordon 1990**) reported that antioxidants are of 2 types; primary or chain-breaking and secondary or preventive. Primary antioxidants, when present in trace amounts, can react with peroxy radicals before they react with further unsaturated lipid molecules and convert them to more stable products. Secondary antioxidants are compounds that retard the rate of chain initiation by various mechanisms other than the pathway followed

by the primary antioxidants. The secondary antioxidants reduce the rate of auto-oxidation of lipids by such processes as binding metal ions, scavenging oxygen, decomposing hydroperoxides to non-radical products and deactivating singlet oxygen. They usually require the presence of another minor component for effective action. Typical examples are reducing agents such as tocopherols and other phenolics. It is important to note that the best known substances that possess antioxidant properties are phenolics, which fall into the category of primary antioxidants. The same author added that at high concentrations, phenolic antioxidants suffer from loss of activity and become pro-oxidants due to their participation in the initiation process.

In general we highly recommend the intake of natural antioxidants, separately or in combinations, in order to avoid different hazards which can happen due to the presence of free radicals but with caution as these compounds become pro-oxidants at high concentrations. Another major reason for such a caution is the lack of data showing that long-term intake of mega doses of these antioxidants is not likely to induce only adverse effects in human.

Table (21): Arithmetic Mean Values \pm S. D and % Changes from the Corresponding Control of Different Biochemical Parameters Before and After Induction of Hyperlipaemia in Albino Rats.

Parameter	Normal Level	Hyperlipaemia Level	% Change
S. Total lipids ¹	349.5 \pm 24.5	510.7 \pm 80.2 **	\uparrow 46.1
S. Triglycerides ¹	53.7 \pm 7.60	170.4 \pm 33.5 ***	\uparrow 217.3
S. T. Cholesterol ¹	51.0 \pm 7.03	92.4 \pm 8.41 ***	\uparrow 81.2
S. HDL - Cholesterol ¹	41.2 \pm 5.25	74.0 \pm 7.09 ***	\uparrow 79.6
T. Cholesterol / HDL - Cholest.	1.24 \pm 0.15	1.25 \pm 0.11 †	\uparrow 0.81
S. ALT ²	29.3 \pm 3.90	55.0 \pm 4.43 ***	\uparrow 87.7
S. AST ²	69.2 \pm 7.82	116.8 \pm 11.2 ***	\uparrow 68.8
S. ALP ³	375.0 \pm 46.2	466.0 \pm 66.4 *	\uparrow 24.3
S. T. Protein ⁴	6.23 \pm 0.49	6.47 \pm 0.43 †	\uparrow 3.85
S. Albumin ⁴	3.60 \pm 0.25	3.68 \pm 0.21 †	\uparrow 2.22
S. Globulin ⁴	2.63 \pm 0.41	2.79 \pm 0.36 †	\uparrow 6.08
Alb. / Glob.	1.37 \pm 0.19	1.32 \pm 0.20 †	\downarrow 3.65
S. Creatinine ¹	0.54 \pm 0.03	0.55 \pm 0.01 †	\uparrow 1.85
S. Urea ¹	30.2 \pm 3.80	29.9 \pm 2.66 †	\downarrow 0.99
S. Uric Acid ¹	1.64 \pm 0.31	1.83 \pm 0.15 *	\uparrow 11.6
Bl.- Haemoglobin ⁴	14.8 \pm 0.88	13.1 \pm 0.84 *	\downarrow 11.5
SOD ⁵	4.75 \pm 1.10	6.90 \pm 0.85 **	\uparrow 45.3
MDA ⁶	8.04 \pm 1.33	11.4 \pm 1.64 **	\uparrow 41.8
GSH ¹	28.1 \pm 3.77	14.1 \pm 2.17 ***	\downarrow 49.8

1. mg / dl
2. U / ml
3. U / L

4. gm / dl
5. U / mg Hb
6. n mol / ml

Table (22) : Arithmetic Mean Values \pm S.D and % Changes from the Corresponding Control of Serum Lipid Pattern in Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Natural Antioxidants.

Treatments	Total Lipids (mg/dl)			Triglycerides (mg/dl)			Total Cholesterol (mg/dl)			HDL- Cholesterol (mg/dl)			T. Cholesterol / HDL- Cholesterol				
	a	b	%	a	b	%	a	b	%	a	b	%	a	b	%		
Neg. Control	339.5	348.7	↑	58.5	55.6	↓	52.4	53.6	↑	46.7	47.0	↑	1.12	1.14	↑		
	\pm 53.3	\pm 25.5	2.71	\pm 3.79	\pm 6.46	4.96	\pm 6.69	\pm 7.49	2.29	\pm 5.79	\pm 4.28	0.64	\pm 0.08	\pm 0.11	1.79		
Pos. Control	511.2	552.5	↑	198.8	187.2	↓	96.0	106.3	↑	73.8	79.8	↑	1.30	1.33	↑		
	\pm 78.1	\pm 63.5	8.08	\pm 40.9	\pm 33.5	5.83	\pm 8.63	\pm 18.0	10.7	\pm 6.34	\pm 11.9	8.13	\pm 0.04	\pm 0.23	2.31		
W. G.O	488.4	512.9	↑	196.5	115.4	↓	81.7	105.6	↑	67.6	74.7	↑	1.21	1.41	↑		
	\pm 73.1	\pm 77.0	5.02	\pm 49.0	\pm 19.3	41.3	\pm 6.06	\pm 17.1	29.3	\pm 5.71	\pm 14.8	10.5	\pm 0.06	\pm 0.27	16.5		
RM	538.9	550.6	↑	134.0	100.6	↓	98.8	89.3	↓	79.6	70.8	↓	1.24	1.26	↑		
	\pm 79.7	\pm 63.6	2.17	\pm 29.1	\pm 13.4	24.9	\pm 7.08	\pm 8.52	9.62	\pm 4.37	\pm 5.38	11.1	\pm 0.11	\pm 0.14	1.61		
E.P.R	498.9	449.4	↓	140.8	62.6	↓	98.3	72.1	↓	80.4	63.9	↓	1.22	1.13	↓		
	\pm 97.0	\pm 27.2	9.92	\pm 24.0	\pm 8.91	55.5	\pm 10.2	\pm 15.1	26.7	\pm 9.59	\pm 8.41	20.5	\pm 0.03	\pm 0.09	7.38		
Red Yeast Rice (Lovastatin)	516.1	400.8	↓	182.0	61.4	↓	87.4	76.5	↓	68.6	64.0	↓	1.27	1.19	↓		
	\pm 73.1	\pm 31.1	22.3	\pm 24.7	\pm 12.5	66.3	\pm 10.1	\pm 11.3	12.5	\pm 9.47	\pm 12.6	6.71	\pm 0.13	\pm 0.11	6.30		
			Initial Value: 349.5 \pm 24.5			Initial Value: 53.7 \pm 7.60			Initial Value: 51.0 \pm 7.03			Initial Value: 41.2 \pm 5.25			Initial Value: 1.24 \pm 0.15		

Each value represents the mean of 8 rats \pm S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

a) Before Treatments

b) After Treatments

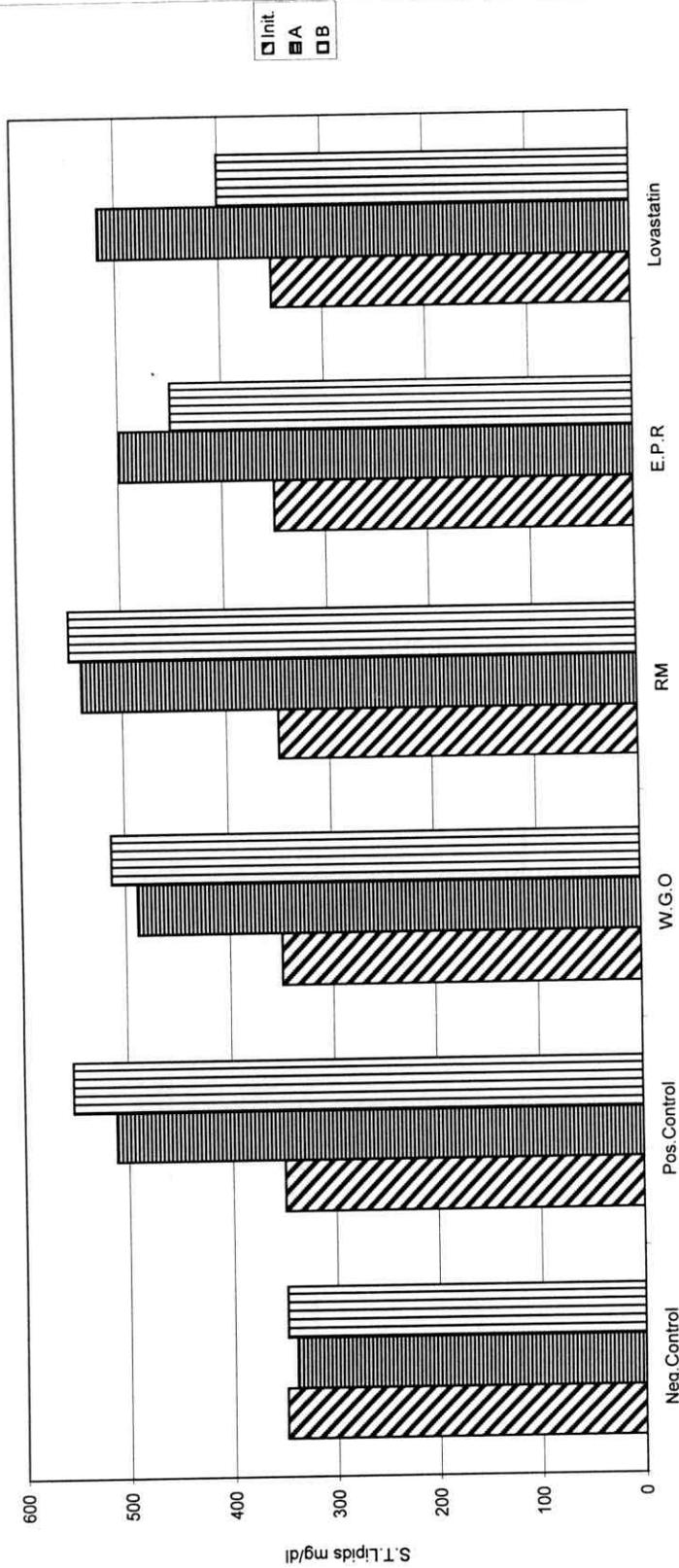


Fig. (17): Arithmetic Mean Values From The Corresponding Control of Serum T. lipids in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

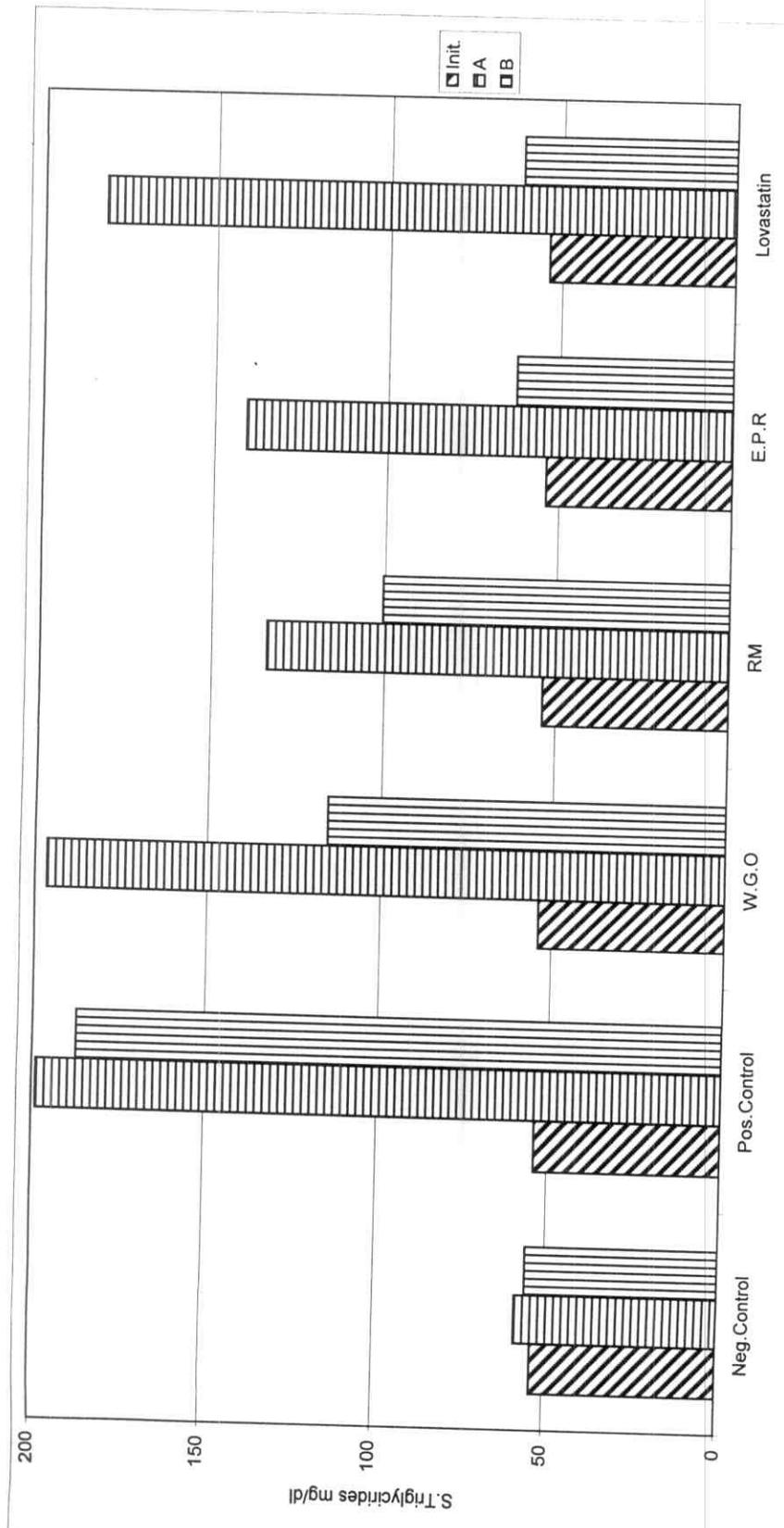


Fig.(18): Arithmetic Mean Values From The Corresponding Control of Serum Triglycerides in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

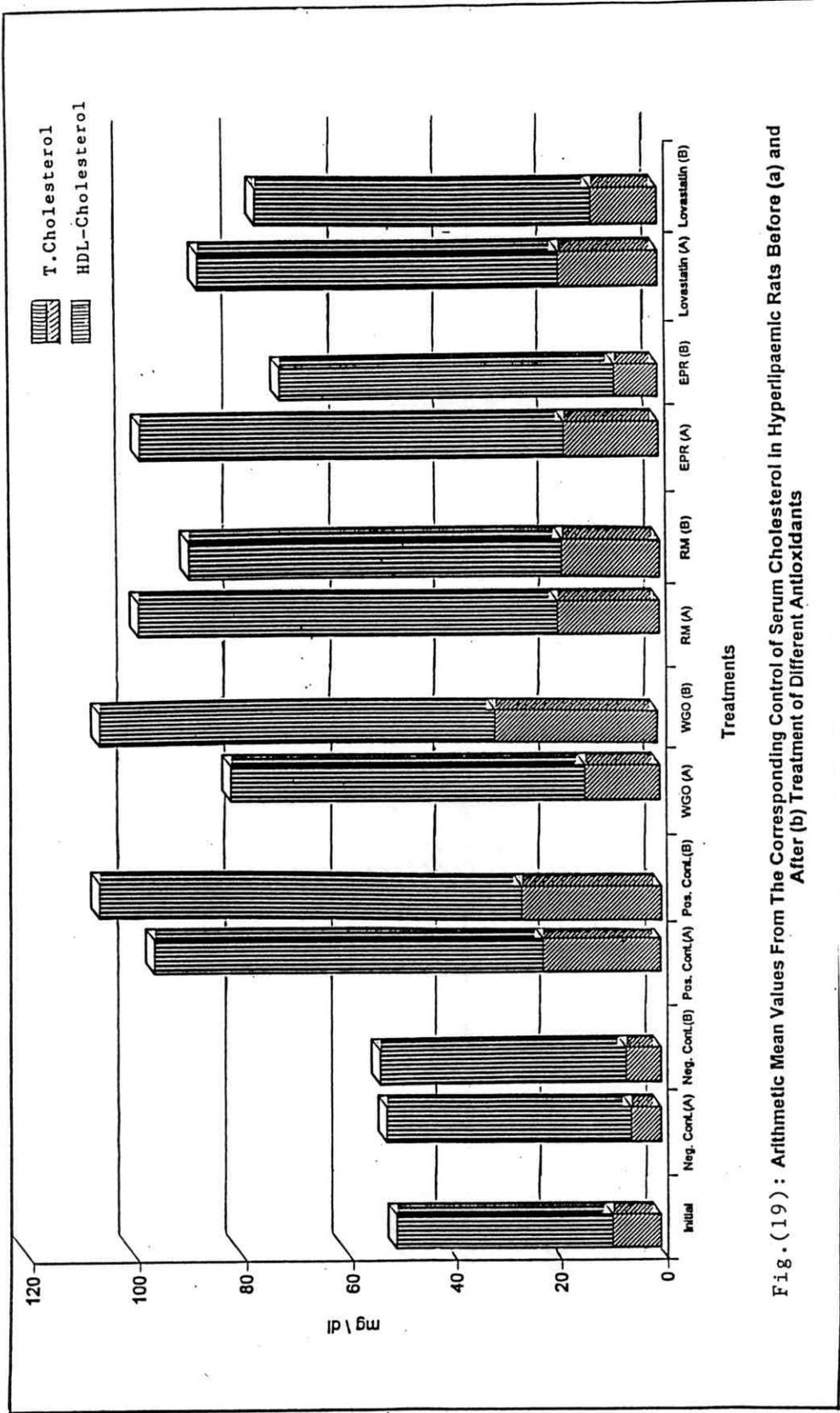


Fig. (19) : Arithmetic Mean Values From The Corresponding Control of Serum Cholesterol in Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Antioxidants

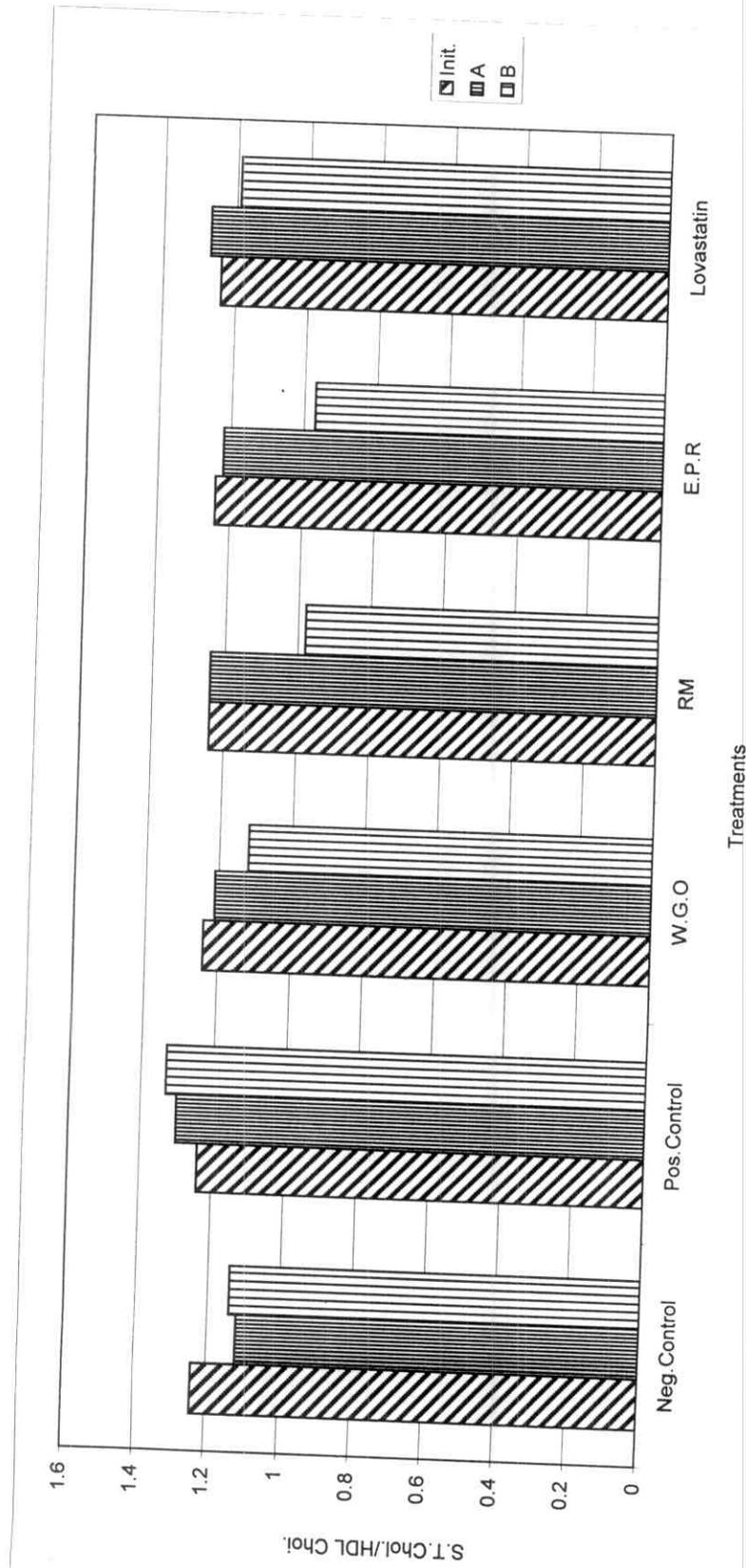


Fig.(20): Arithmetic Mean Values From The Corresponding Control of Serum T.Cholesterol / HDL Cholesterol in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

Table (23) : Arithmetic Mean Values \pm S.D and % Changes from the Corresponding Control of Serum ALT, AST and ALP in Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Natural Antioxidants.

Treatments	ALT (u / ml)			AST (u / ml)			ALP (u / ml)		
	a	b	%	a	b	%	a	b	%
Neg. Control	35.0	34.0	↓	59.0	57.1	↓	350.0	354.4	↑
	\pm 6.38	\pm 2.71	2.86	\pm 1.90	\pm 6.03	3.22	\pm 86.4	\pm 35.0	1.26
Pos. Control	51.4	46.0	↓	119.0	121.2	↑	454.1	593.7	↑
	\pm 5.55	\pm 4.89	10.5	\pm 8.26	\pm 5.61	1.85	\pm 40.6	\pm 76.5	30.7
W. G.O	54.7	36.8	↓	120.7	96.7	↓	473.7	394.0	↓
	\pm 1.09	\pm 3.34	32.7	\pm 10.8	\pm 5.73	19.9	\pm 72.4	\pm 40.8	16.8
RM	55.8	35.1	↓	116.6	92.5	↓	472.1	268.8	↓
	\pm 6.05	\pm 1.28	37.1	\pm 17.6	\pm 6.61	20.7	\pm 83.8	\pm 21.5	43.1
E.P.R	56.7	31.1	↓	112.4	84.8	↓	478.5	348.8	↓
	\pm 5.67	\pm 2.88	45.1	\pm 15.8	\pm 4.66	24.6	\pm 71.4	\pm 63.8	27.1
Red Yeast Rice (Lovastatin)	56.5	29.0	↓	115.1	83.5	↓	451.8	367.0	↓
	\pm 3.78	\pm 4.76	48.7	\pm 3.76	\pm 3.23	27.5	\pm 63.8	\pm 24.6	18.8
<i>Initial Value: 29.3 \pm 3.90</i>			<i>Initial Value: 69.2 \pm 7.82</i>			<i>Initial Value: 375.0 \pm 46.2</i>			

Each value represents the mean of 8 rats \pm S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

a) Before Treatments

b) After Treatments

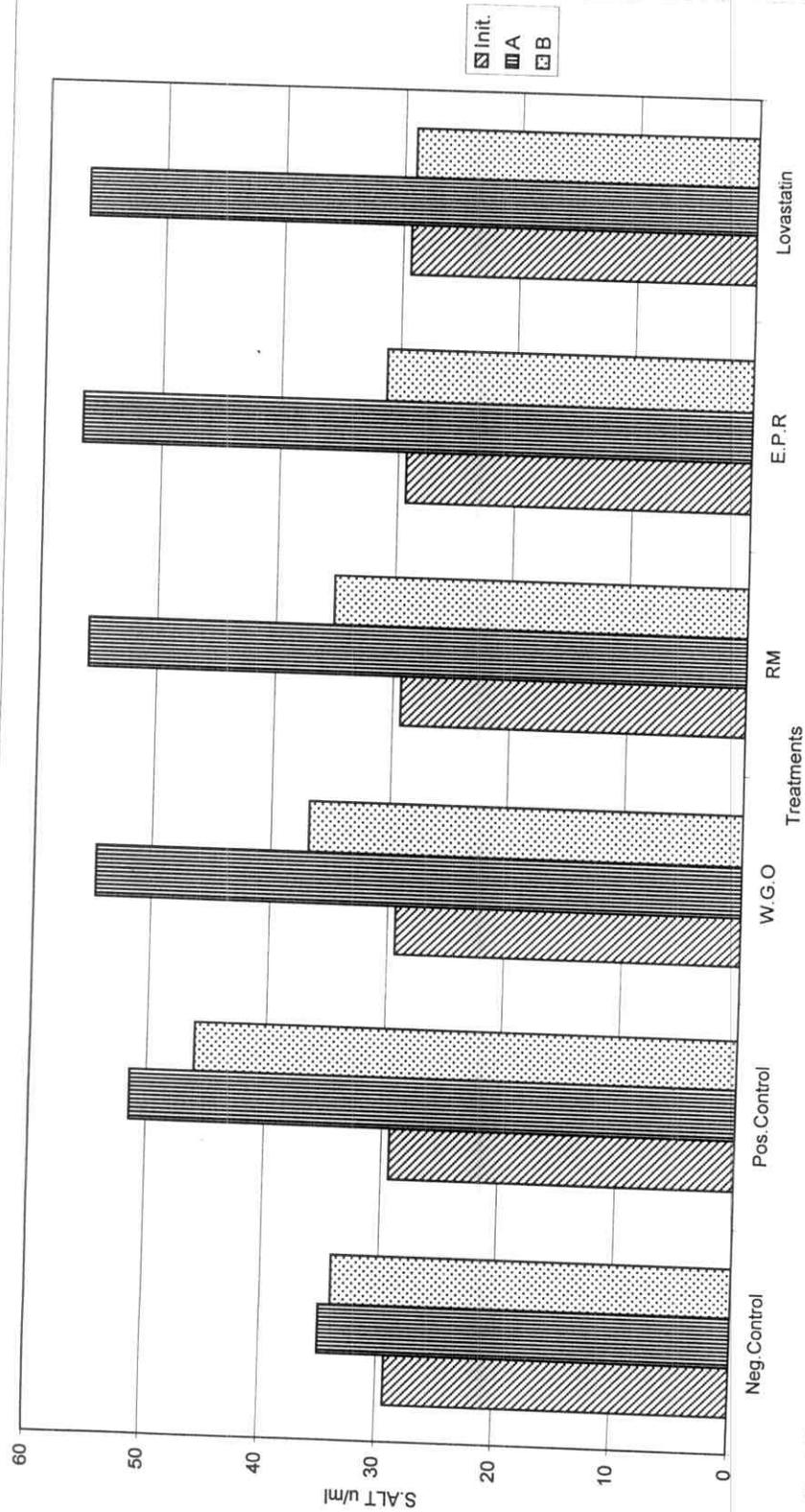


Fig.(21) : Arithmetic Mean Values From The Corresponding Control of Serum ALT (U/ml) in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

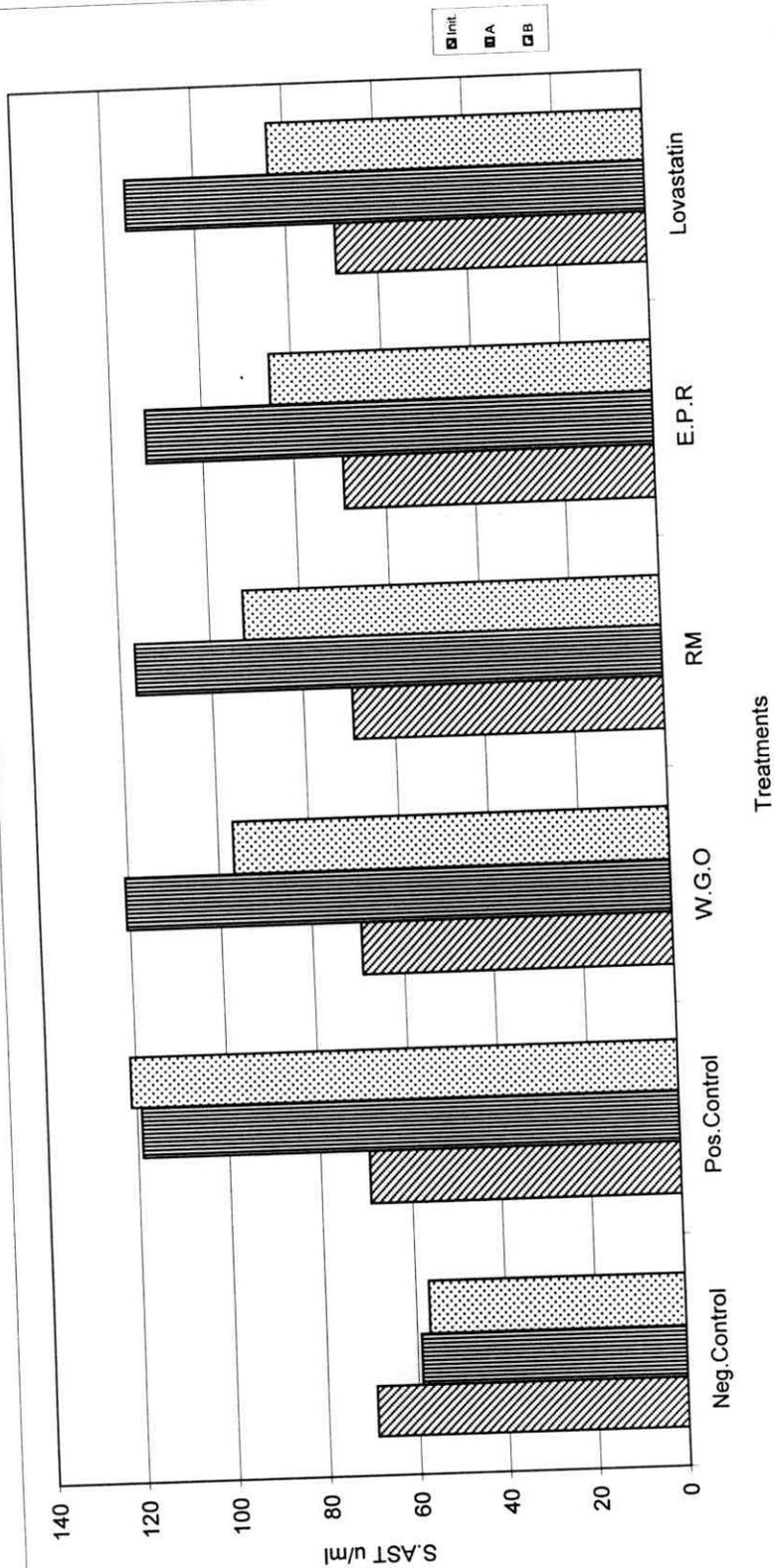


Fig.(22): Arithmetic Mean Values From The Corresponding Control of Serum AST (U/ml) in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

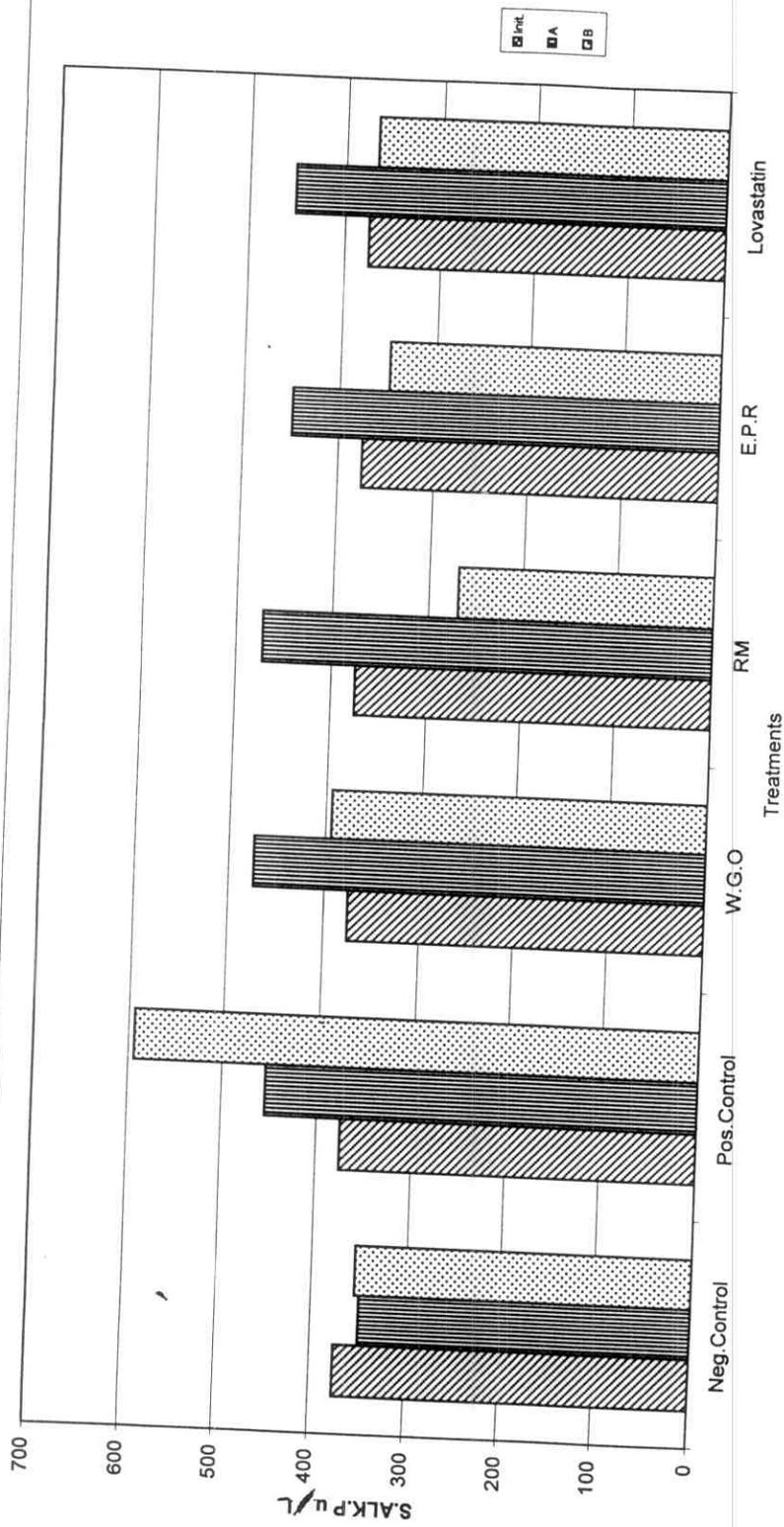


Fig.(23) : Arithmetic Mean Values From The Corresponding Control of Serum Alk. P (U/L) in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

Table (24) : Arithmetic Mean Values \pm S.D and % Changes from the Corresponding Control of Serum Proteins in Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Natural Antioxidants

Treatments	Total Protein (gm/dl)			Albumin (gm/dl)			Globulin (gm/dl)			Alb. / Glob.				
	a	b	%	a	b	%	a	b	%	a	b	%		
Neg. Control	6.48	6.61	↑	3.45	3.51	↑	3.03	3.10	↑	1.14	1.13	↓		
	\pm 0.79	\pm 0.96	2.01	\pm 0.36	\pm 0.64	1.74	\pm 0.37	\pm 0.39	2.31	\pm 0.28	\pm 0.14	0.88		
Pos. Control	6.13	6.86	↑	3.57	3.58	↑	2.56	3.28	↑	1.39	1.09	↓		
	\pm 0.48	\pm 0.68	11.9	\pm 0.23	\pm 0.25	0.28	\pm 0.41	\pm 0.59	28.1	\pm 0.30	\pm 0.17	21.6		
W. G.O	6.73	6.64	↓	3.79	3.64	↓	2.94	3.00	↑	1.29	1.21	↓		
	\pm 0.36	\pm 0.64	1.34	\pm 0.30	\pm 0.18	3.96	\pm 0.20	\pm 0.74	2.04	\pm 0.10	\pm 0.30	6.20		
RM	6.63	7.22	↑	3.71	3.80	↑	2.92	3.42	↑	1.27	1.11	↓		
	\pm 0.33	\pm 0.53	8.90	\pm 0.18	\pm 0.23	2.43	\pm 0.30	\pm 0.48	17.1	\pm 0.15	\pm 0.16	12.6		
E.P.R	6.23	6.68	↑	3.57	3.73	↑	2.66	2.95	↑	1.34	1.26	↓		
	\pm 0.59	\pm 0.37	7.22	\pm 0.23	\pm 0.17	4.48	\pm 0.57	\pm 0.36	10.9	\pm 0.27	\pm 0.18	5.97		
Red Yeast Rice (Lovastatin)	6.62	6.50	↓	3.79	3.78	↓	2.83	2.72	↓	1.34	1.39	↑		
	\pm 0.37	\pm 0.50	1.81	\pm 0.15	\pm 0.27	0.26	\pm 0.34	\pm 0.68	3.89	\pm 0.18	\pm 0.33	3.73		
			Initial Value: 6.23 \pm 0.49			Initial Value: 3.60 \pm 0.25			Initial Value: 2.63 \pm 0.41			Initial Value: 1.37 \pm 0.19		

Each value represents the mean of 8 rats \pm S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

a) Before Treatments

b) After Treatments

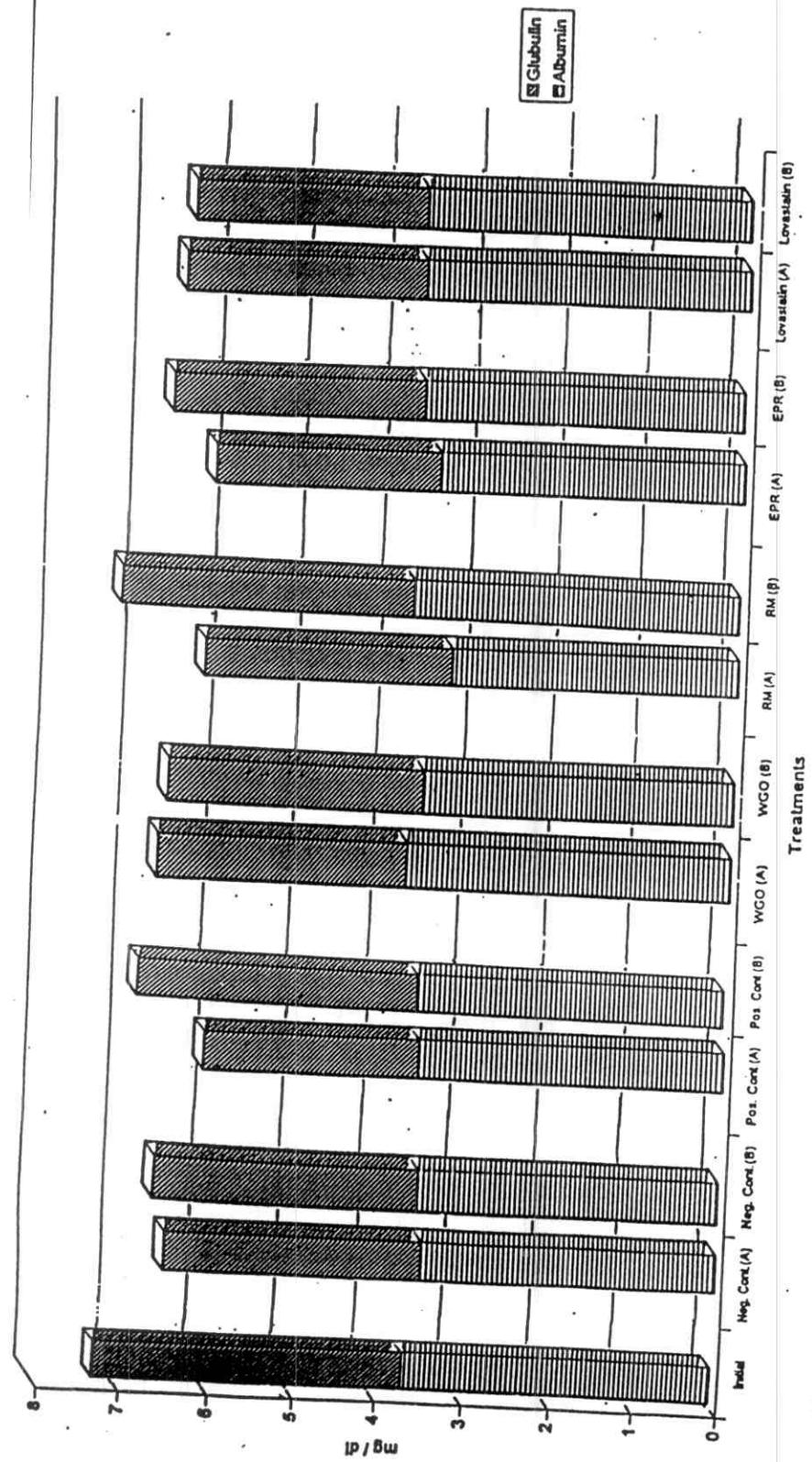


Fig. (24) : Arithmetic Mean Values From The Corresponding Control of Serum Proteins In Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Antioxidants

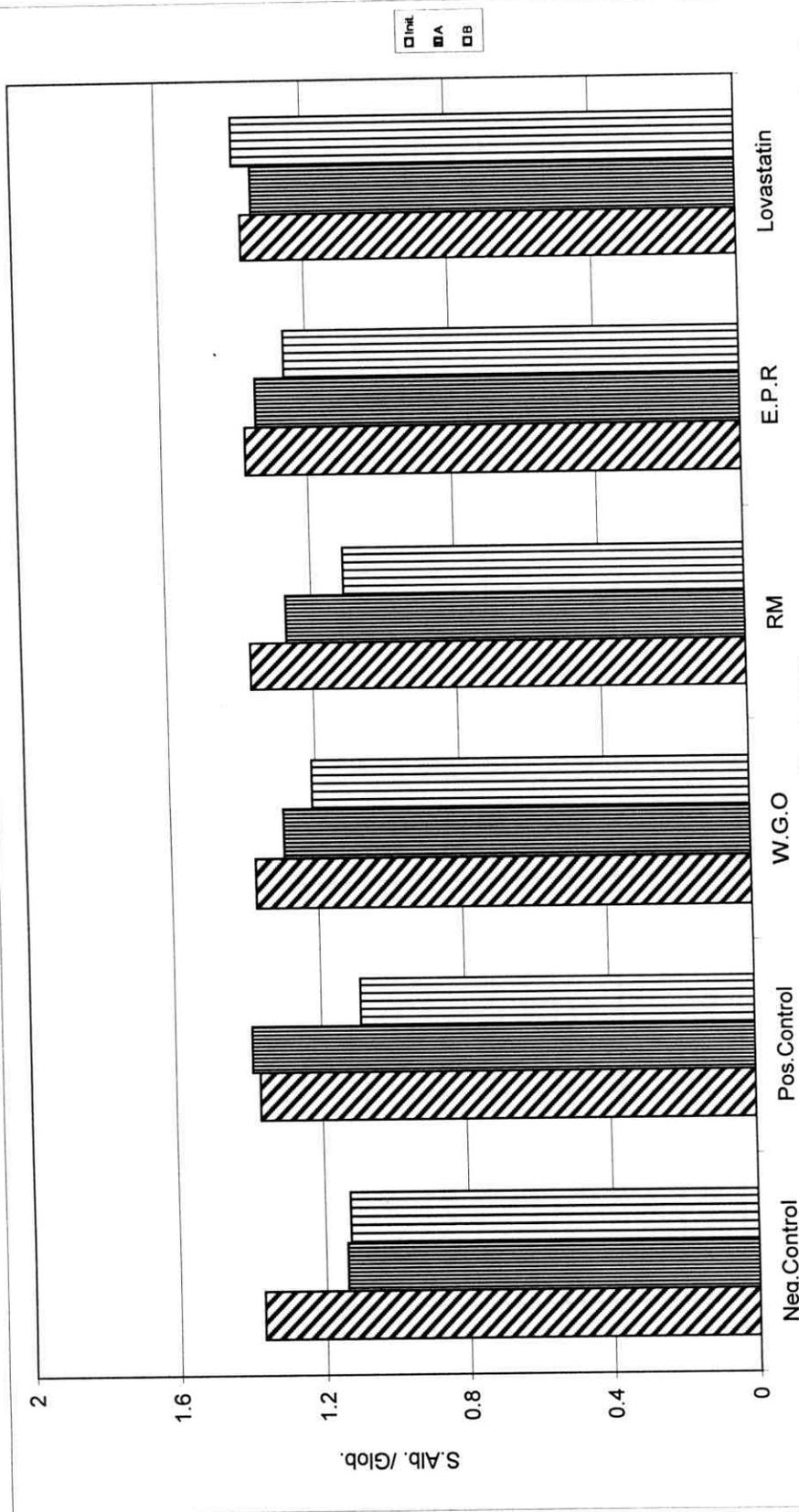


Fig.(25): Arithmetic Mean Values From The Corresponding Control of Serum Alb. / Glob. in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

Table (25) : Arithmetic Mean Values \pm S.D and % Changes from the Corresponding Control of Serum Creatinine, Urea and Uric Acid in Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Natural Antioxidants.

Treatments	Creatinine (mg / dl)			Urea (mg / dl)			Uric Acid (mg / dl)				
	a	b	%	a	b	%	a	b	%		
Neg. Control	0.55	0.54	↓	30.2	31.3	↑	2.00	2.05	↑		
	\pm 0.01	\pm 0.12	1.82	\pm 3.81	\pm 2.50	3.64	\pm 0.20	\pm 0.55	2.50		
Pos. Control	0.54	0.59	↑	27.8	23.1	↓	1.72	1.82	↑		
	\pm 0.01	\pm 0.13	9.26	\pm 2.08	\pm 3.01	16.9	\pm 0.18	\pm 0.26	5.81		
W. G.O	0.55	0.60	↑	30.4	32.3	↑	1.83	2.09	↑		
	\pm 0.01	\pm 0.07	9.09	\pm 2.88	\pm 3.18	6.25	\pm 0.15	\pm 0.19	14.2		
RM	0.55	0.64	↑	27.6	31.1	↑	1.81	2.06	↑		
	\pm 0.01	\pm 0.06	16.4	\pm 2.18	\pm 1.76	12.7	\pm 0.17	\pm 0.41	13.8		
E.P.R	0.55	0.63	↑	31.9	29.8	↓	1.85	1.95	↑		
	\pm 0.01	\pm 0.06	14.5	\pm 5.11	\pm 4.24	6.58	\pm 0.14	\pm 0.33	5.41		
Red Yeast Rice (Lovastatin)	0.56	0.49	↓	31.6	25.5	↓	1.92	1.98	↑		
	\pm 0.01	\pm 0.05	12.5	\pm 1.04	\pm 2.88	19.3	\pm 0.12	\pm 0.48	3.12		
			Initial Value: 0.54 \pm 0.03			Initial Value: 30.2 \pm 3.80			Initial Value: 1.64 \pm 0.31		

Each value represents the mean of 8 rats \pm S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

a) Before Treatments

b) After Treatments

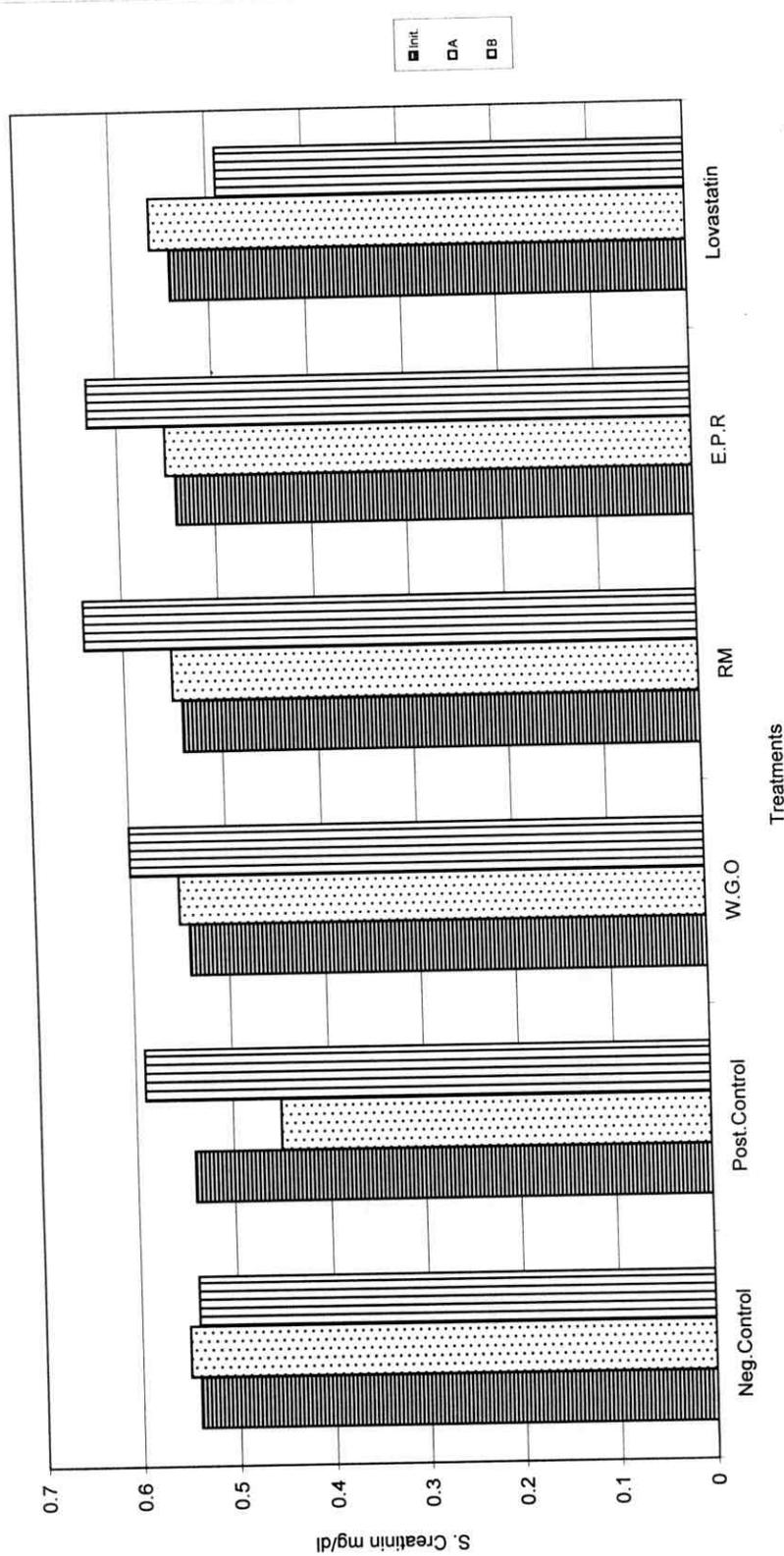


Fig.(26): Arithmetic Mean Values From The Corresponding Control of Serum Creatinin in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

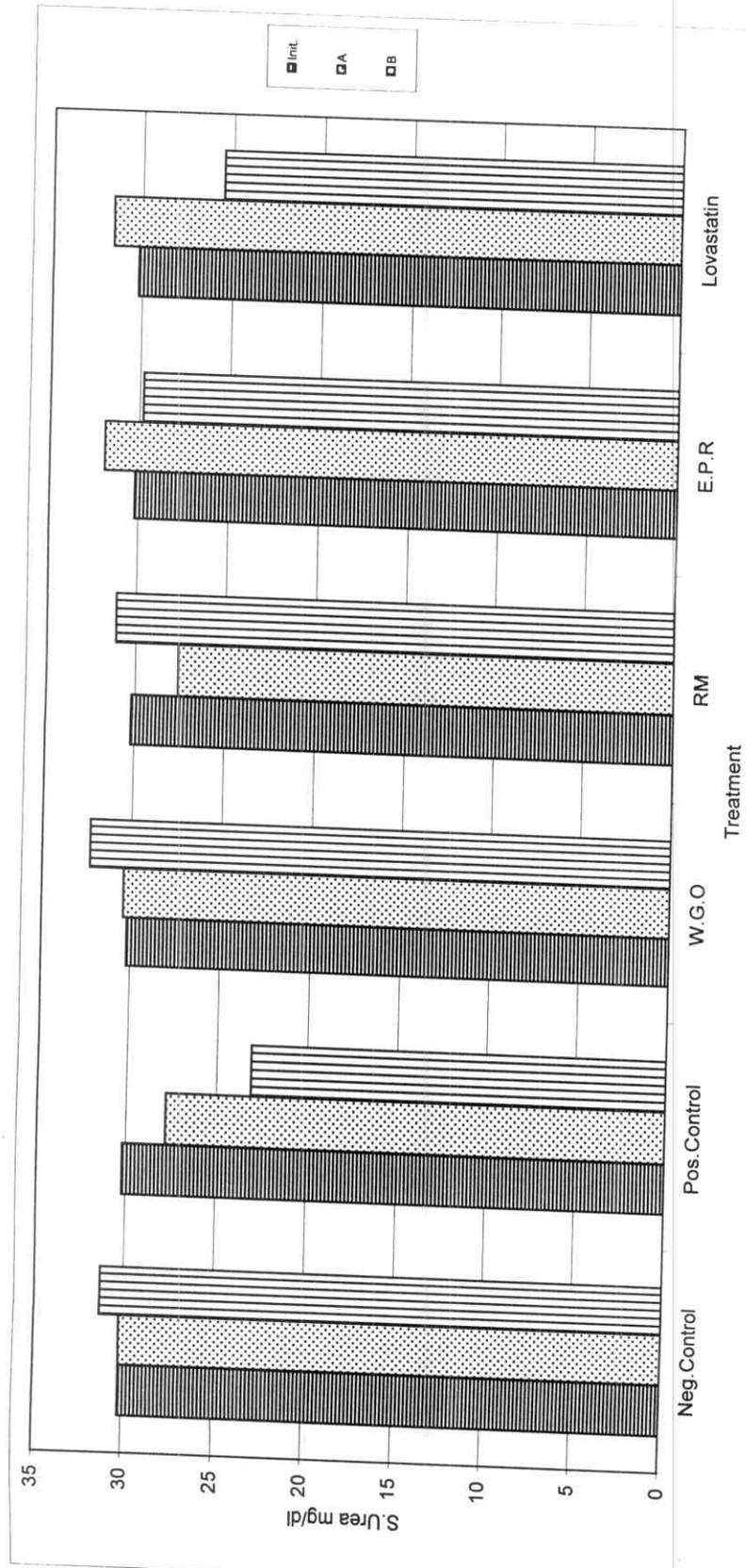


Fig.(27): Arithmetic Mean Values From The Corresponding Control of Serum Urea in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

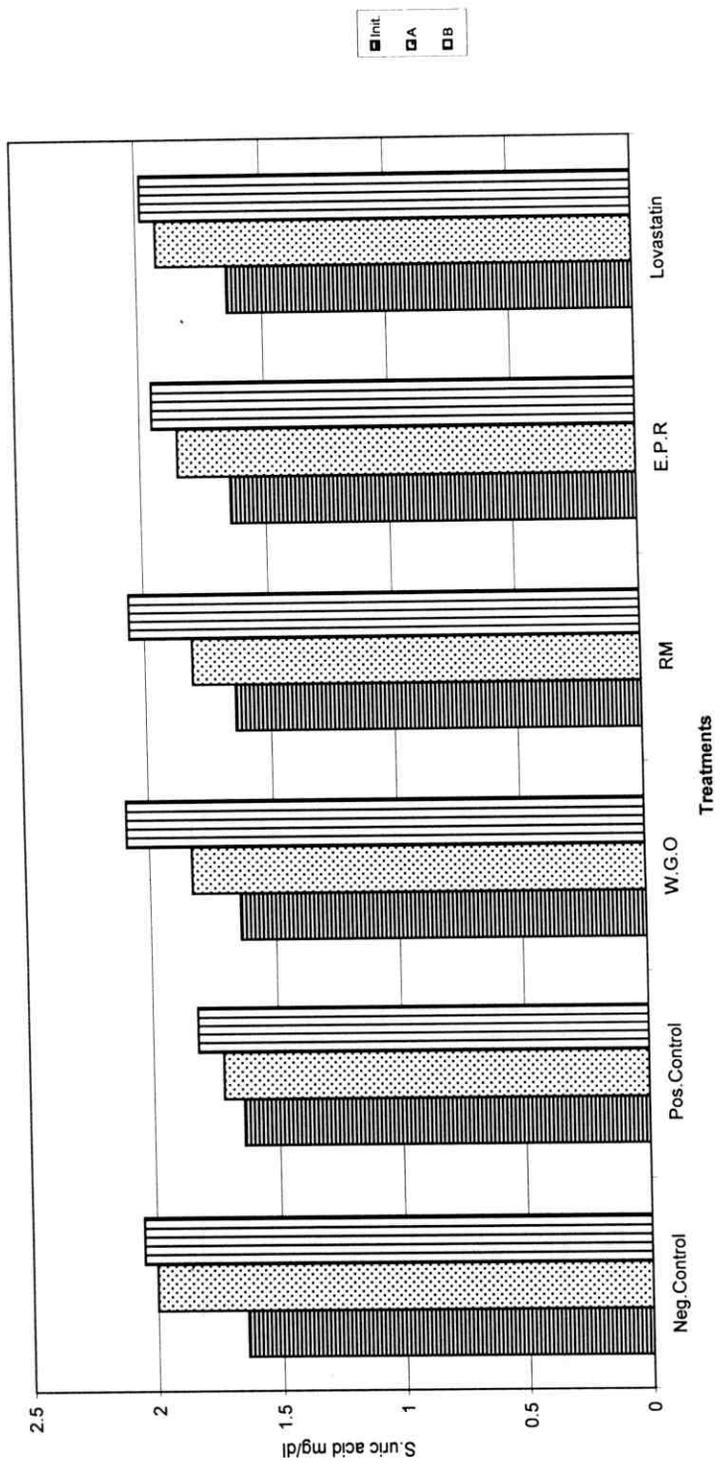


Fig.(28): Arithmetic Mean Values From The Corresponding Control of Serum Uric Acid in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

Table (26) : Arithmetic Mean Values \pm S.D and % Changes from the Corresponding Control of Hb, SOD, MDA and Glut. in Hyperlipaemic Rats Before (a) and After (b) Treatment of Different Natural Antioxidants

Treatments	Haemoglobin (g/dl)			SOD (u/mg Hb)			MDA (n mol/ ml)			Red. Glut. (mg/dl)				
	a	b	%	a	b	%	a	b	%	a	b	%		
Neg. Control	14.5 \pm 0.94	13.9 \pm 0.50	↓ 4.14	4.50 \pm 0.64	4.60 \pm 0.63	↑ 2.22	7.40 \pm 0.24	7.50 \pm 0.22	↑ 1.35	25.4 \pm 4.49	24.9 \pm 0.92	↓ 1.97		
Pos. Control	13.3 \pm 0.90	13.1 \pm 1.44	↓ 1.50	6.54 \pm 0.82	7.70 \pm 0.93	↑ 17.7	11.5 \pm 2.88	13.4 \pm 1.61	↑ 16.5	12.7 \pm 3.15	12.5 \pm 0.56	↓ 1.57		
W. G.O	13.7 \pm 0.49	13.4 \pm 0.77	↓ 2.19	6.73 \pm 0.52	5.58 \pm 0.53	↓ 17.1	10.0 \pm 1.35	7.64 \pm 0.53	↓ 23.6	14.5 \pm 2.72	10.5 \pm 0.70	↓ 27.6		
RM	13.0 \pm 0.89	14.3 \pm 0.98	↑ 10.0	7.00 \pm 0.73	5.67 \pm 0.49	↓ 19.0	11.3 \pm 2.30	9.98 \pm 0.34	↓ 11.7	15.3 \pm 2.30	11.6 \pm 0.85	↓ 24.2		
E.P.R	12.9 \pm 0.68	13.4 \pm 0.77	↑ 3.88	7.00 \pm 1.21	5.59 \pm 0.43	↓ 20.1	11.7 \pm 0.38	10.7 \pm 0.99	↓ 8.55	14.0 \pm 2.07	10.8 \pm 0.73	↓ 22.9		
Red Yeast Rice (Lovastatin)	12.6 \pm 1.22	13.9 \pm 1.20	↑ 10.3	7.23 \pm 0.95	5.36 \pm 0.50	↓ 25.9	12.3 \pm 1.30	8.37 \pm 0.98	↓ 31.9	14.3 \pm 0.63	12.3 \pm 0.92	↓ 14.0		
			Initial Value: 14.8 \pm 0.88			Initial Value: 4.75 \pm 1.10			Initial Value: 8.04 \pm 1.33			Initial Value: 28.1 \pm 3.77		

Results & Discussion

Each value represents the mean of 8 rats \pm S.D

† Insignificant difference from the corresponding control at $P > 0.1$

* Significant difference from the corresponding control at $P < 0.05$

** Highly significant difference from the corresponding control at $P < 0.01$

*** Very highly significant difference from the corresponding control at $P < 0.001$

a) Before Treatments

b) After Treatments

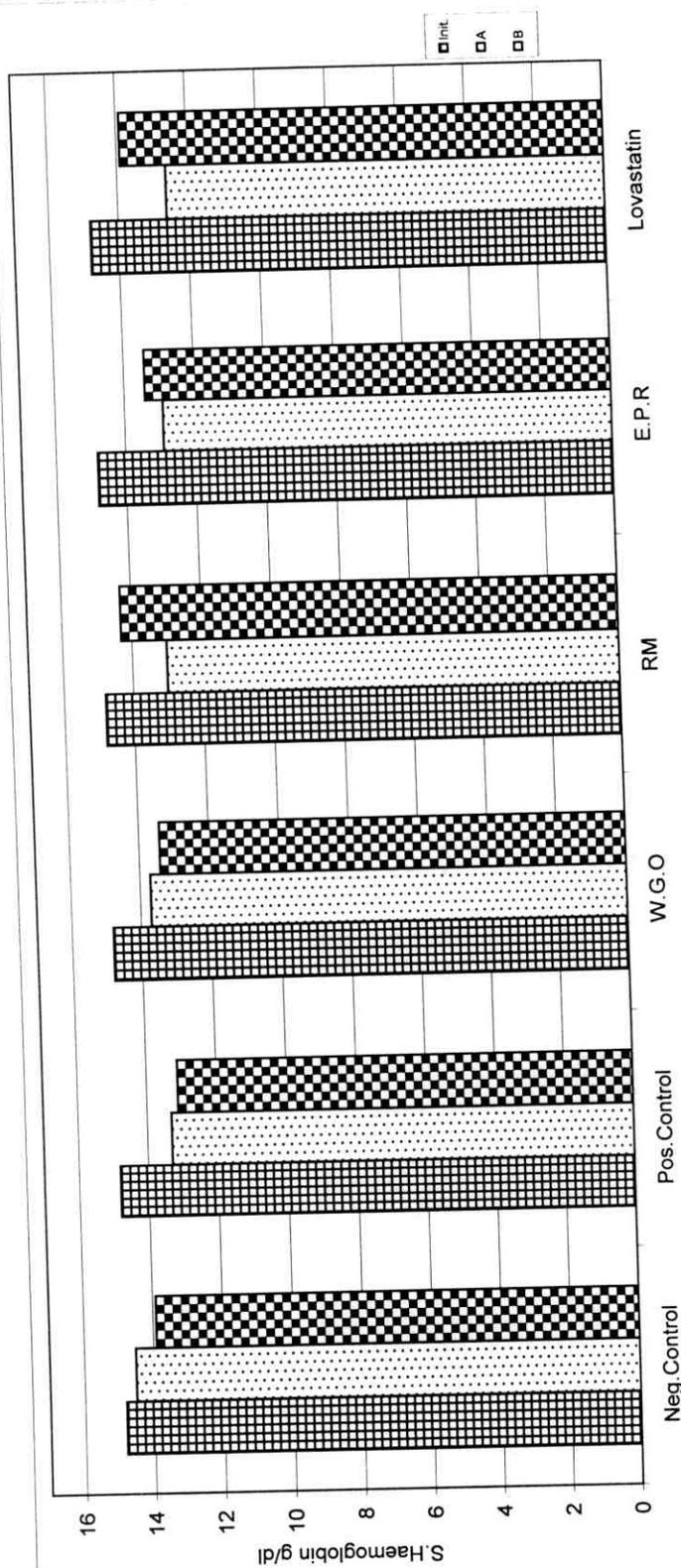


Fig.(29) : Arithmetic Mean Values From The Corresponding Control of Serum Haemoglobin in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

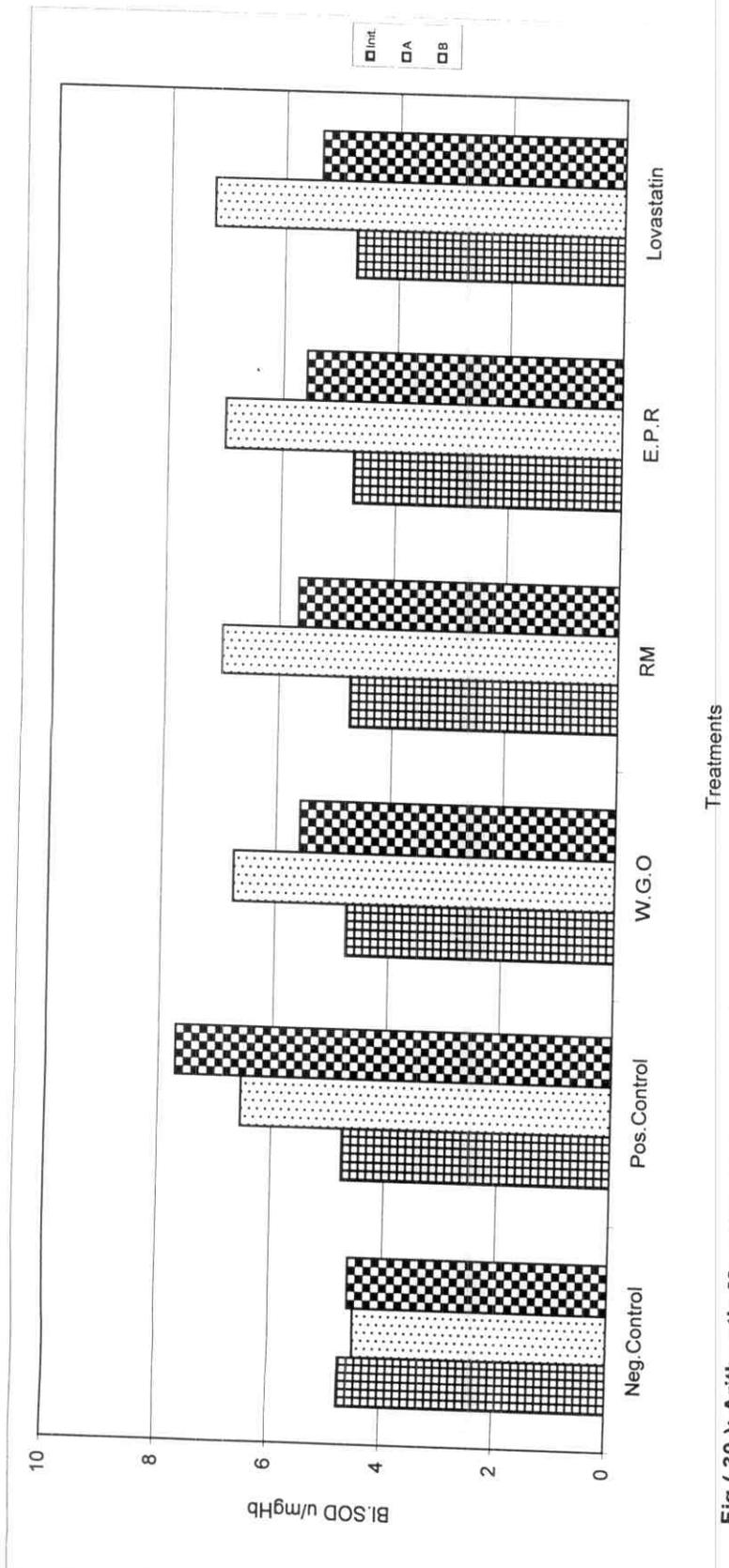


Fig. (30): Arithmetic Mean Values From The Corresponding Control of Blood SOD in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

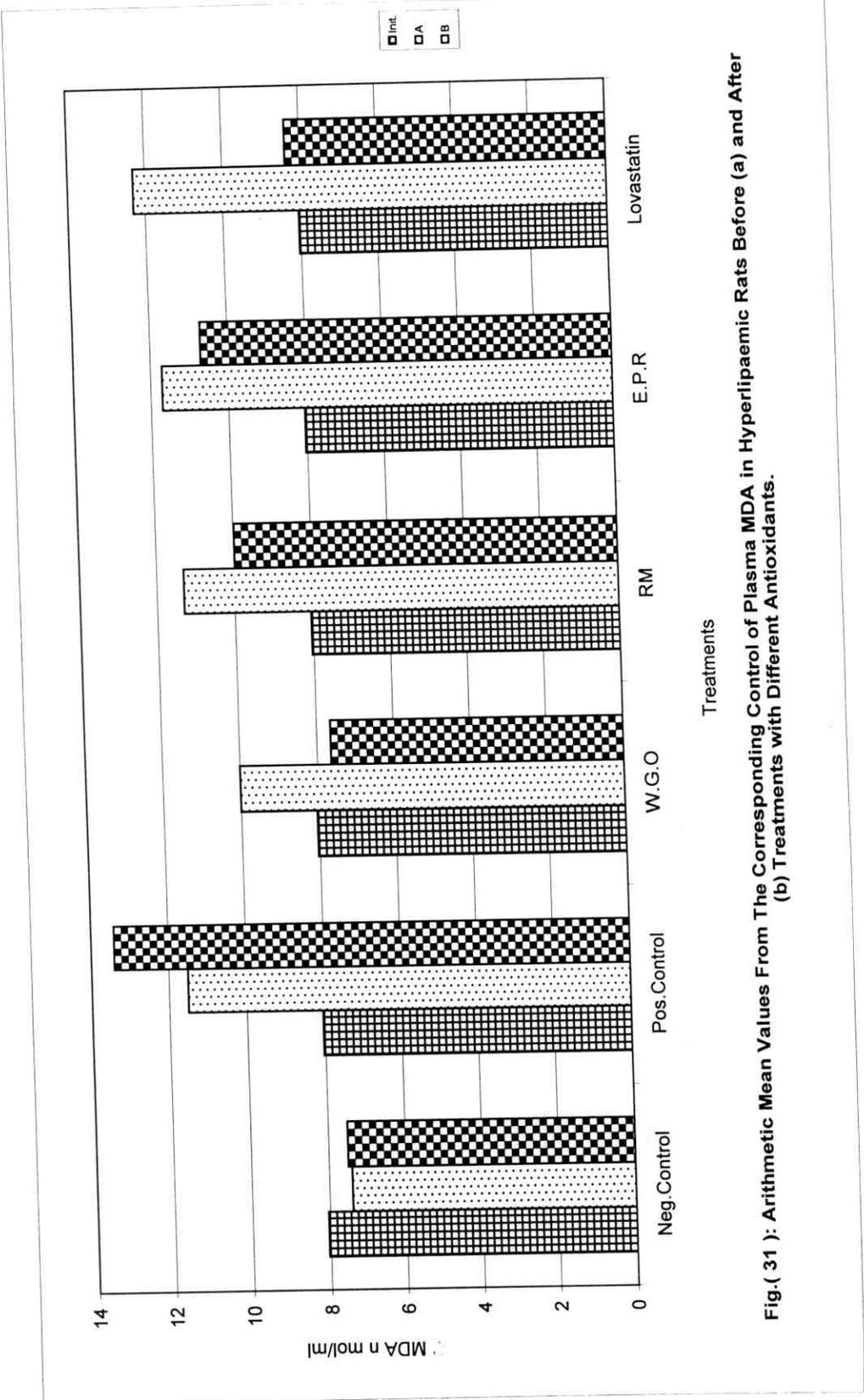


Fig.(31): Arithmetic Mean Values From The Corresponding Control of Plasma MDA in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.

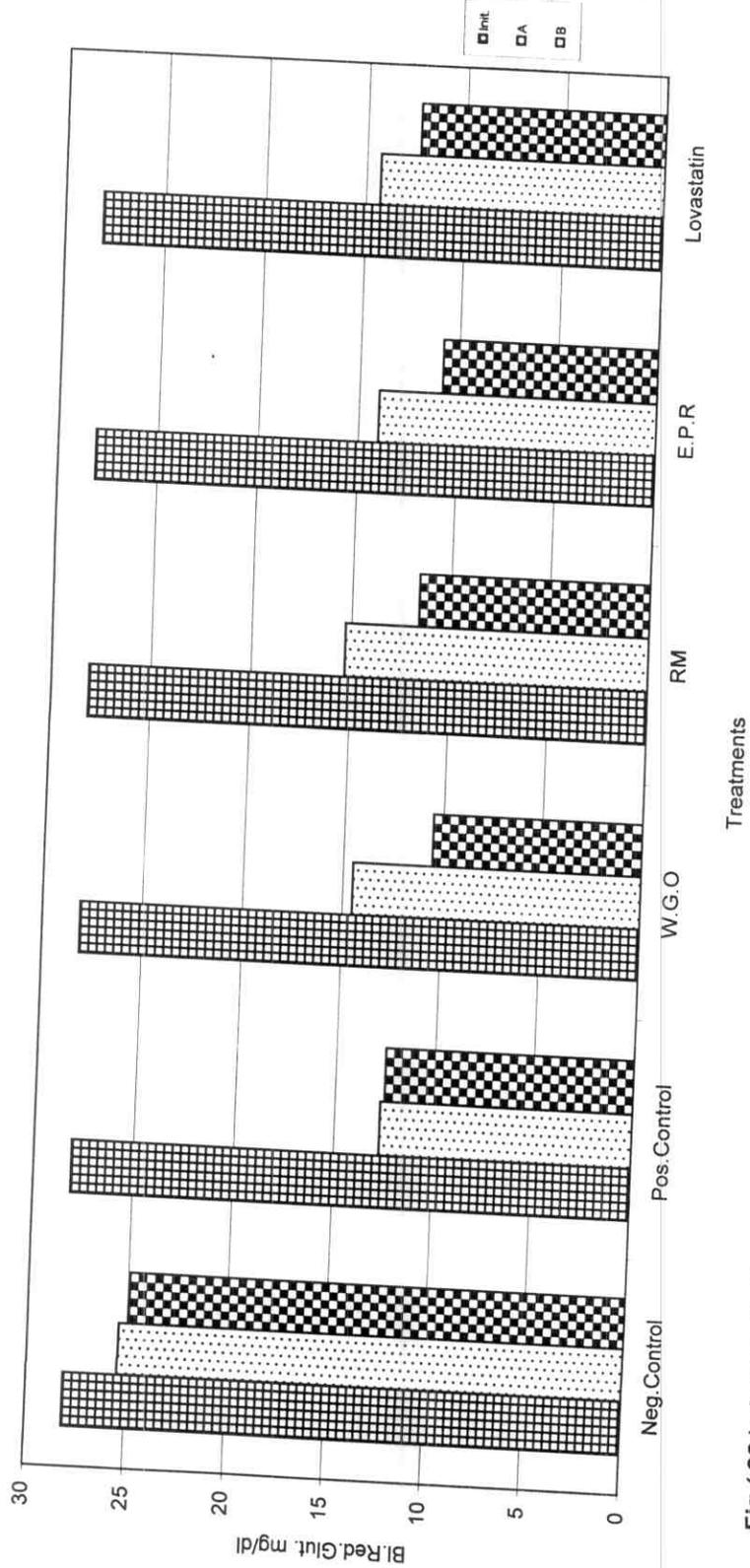


Fig.(32) : Arithmetic Mean Values From The Corresponding Control of Blood Red. Glut. in Hyperlipaemic Rats Before (a) and After (b) Treatments with Different Antioxidants.