

## HYPOLIPIDAEMIC EFFECT OF *CONVOLVULUS MICROPHYLLUS* IN CHOLESTEROL FED GERBILS (*MERIONES HURRINAE JERDON*)

M. CHATURVEDI, P.C. MALI and V.P. DIXIT

Reproductive Physiology Section, Department of Zoology, University of Rajasthan, Jaipur - 302 004, India. The hypolipidaemic activities of *Convolvulus microphyllus* has been observed in domestic animals (rats, rabbits and rhesus monkeys). The 50% ethanolic extract of *Convolvulus microphyllus* (whole plant) when administered to cholesterol fed gerbils, reduced serum cholesterol level, LDL cholesterol, triglycerides and phospholipids significantly after 90 days. The possible mechanism of action is discussed.

**Keywords :** Cholesterol; *Convolvulus microphyllus*; Gerbils; Hypolipidaemic; Triglycerides.

### Introduction

The change in lipids and lipoprotein profile leading to development of cardiovascular disease in oral contraceptive users<sup>1,2</sup>. It is known that a diet containing a high level of fat and cholesterol by itself increases risk of atherosclerosis complications<sup>3</sup>.

*Convolvulus microphyllus*, also known as Sunkh pushpi, has been used for prevention of atheroma in rats and rabbits. The present investigation is designed to evaluate whether 50% EtOH of *C. microphyllus* could be used for reducing the serum cholesterol, LDL cholesterol, triglycerides and phospholipids and thus inhibits atherogenesis.

### Materials and Methods

Twenty male and female gerbils (*Meriones hurrinae Jerdon*) with average body weight of 60 - 100 gms were used. They were kept under normal conditions, fed with soaked wheat and grams and water *ad libitum*. Body weight was recorded twice in a week. Blood was taken from tail veins and analysed for cholesterol<sup>4</sup>, triglycerides<sup>5</sup>, phospholipids<sup>6</sup>, HDL<sup>7</sup> and haematological parameters.

### Hyperlipidaemic models

20 male and female gerbils (60 - 100 gms) were kept on atherogenic diet. It is comprised of wheat flour base in addition with milk powder, dried egg yolk, hydrogenated fat, butter, dried yeast, salt, sugar and vitamin mixture.

The average consumption of diet was 20 - 25 gms/gerbil/24 hrs. (60 - 100 gms/Kg b.wt.). Cholesterol in 1 ml coconut oil was administered each day for 90 days by gavage. Cholesterol feeding for 90 days resulted in hypercholesterolemia. Animals whose serum cholesterol levels were 150 mg/dl or above, were included for experimentation.

#### Experimental design :

1. Control vehicle treated
2. Atherofed
3. Hyperlipidaemic + 50% EtOH of *C. microphyllus* (100 mg/Kg)

50% EtOH of *C. microphyllus* at a dose level of 100 mg/Kg was administered orally for 90 days. On day 91, all animals were sacrificed, blood was taken directly from ventricle of heart and biochemical parameters were estimated.

## Results and Discussion

### Serum cholesterol :

Control :  $96.21 \pm 4.37$  mg/dl ; Athero  $225.16 \pm 28.79$  mg/dl ;

*C.microphyllus* :  $93.75 \pm 6.25$  mg.dl. Percent reduction 58.36%.

### LDL cholesterol :

Control :  $58.60 \pm 3.8$  mg/dl ; Athero  $144.66 \pm 50.9$  mg/dl ;

*C.microphyllus* :  $59.70 \pm 8.74$ mg/dl. Percent reduction 58.73%.

Triglyceride : Control :  $35.72 \pm 1.6$  mg/dl ; Athero  $52.75 \pm 6.75$  mg/dl ; *C.microphyllus* :  $35.87 \pm 3.09$  mg/dl.

### Phospholipids :

Control :  $61.44 \pm$  mg/dl ; Athero  $155.28 \pm 10.29$  mg/dl ;

*C.microphyllus* :  $68.73 \pm 2.07$  mg/dl.

Cholesterol and glycogen content of liver and heart muscles were significantly high in cholesterol fed animals.

### Liver cholesterol :

Control :  $10.88 \pm 0.82$  mg/gm ; Athero  $12.01 \pm 1.54$  mg/gm ;

*C.microphyllus* :  $8.12 \pm 0.62$  mg/gm.

### Heart muscle cholesterol :

Control :  $6.51 \pm 0.11$  mg/gm ; Athero  $15.57 \pm 0.89$  mg./gm ;

*C.microphyllus* :  $6.87 \pm 0.62$  mg/gm.

The relationship between dietary lipid and atherosclerosis has been a subject of investigation for over last six decades. Cholesterol fed rabbits is a useful experimental model to study this relationship. Cholesterol added diet has been shown to result in a rapid development of

hypercholesterolemia<sup>8,9</sup> and subsequently appears in the form of atheroma<sup>10</sup>. Plasma triglyceride level have been reported to increase or remain normal<sup>11</sup> decrease, in response to dietary cholesterol. Plasma triglycerides and cholesterol carry the highest risk for ischemic heart disease<sup>12</sup>, HDL and LDL cholesterol are significant variables for coronary heart disease<sup>13</sup>.

In almost all animals the atherogenic diet provoked serious atheromatous lesions localised in the thoracic and abdominal portions of the aorta. *C.microphyllus* extract feeding prevented intimal thickening and development of atherosclerosis even in the presence of hypercholesterolemia.

*C.microphyllus* extract reduced serum cholesterol and LDL cholesterol by 58% and 58%, respectively. But these levels still remain above normal values. Serum triglyceride was also decreased after *C.microphyllus* extract feeding. This decrease was due to the reduction in the LDL fraction. Cholesterol feeding to gerbils leads to the formation of cholesterol rich  $\beta$ -VLDL. *C.microphyllus* extract feeding checks this formation and resulted in the lowering of VLDL cholesterol.

Miller and Miller<sup>13</sup> have presented evidence that HDL is inversely related to total body cholesterol postulate that the mechanism of action may involve transport of cholesterol back to the liver. Glomset<sup>14</sup> showed that HDL alters the balance of non-esterified cholesterol between plasma and cells by increasing its utilization in the lecithin/cholesterol acetyl transferase (LCAT) system to form cholesteryl ester, which would move less slowly back into the cells.



### Acknowledgement

Authors are thankful to UGC, New Delhi, for financial assistance and Head, Department of Zoology for providing necessary facilities.

### References

1. London R S 1992, *Obstet. and Gyncol. Survey* 47 777
2. Ghanbarisissan M A and Leelamma 1994, *Indian J. Expt. Biol.* 32 307
3. Kannel W B and Castelli W P 1979, *Am. Int. Med.* 90 8
4. Zlatkis A Zak B, and Bozle A J 1953, *J. Lab. and Clin. Med.* 41 486
5. Gottfried S P and Rosenberg B 1973, *Clin. Chem.* 19 1077
6. Zilversmit D B and Davis A K 1950, *J. Lab. and Clin. iknvest.* 35 155
7. Burnstein M, Scholmic M R and Morphein R 1970, *J. lipid. Res.* 11 583
8. Shore V G, Shore B and Hart R G 1974, *Biochemistry* 13 1579
9. Wissler R W and Vesselinovitch D 1974, *In Atherosclerosis III* G. Schettier and A Weizel, Editors, Spring Ericrlag, Berlin. Heidelberg, 317 - 325.
10. Kritchevsky D A, Mayer A W, Tesar W C, Lojan J B, Brown R A, Davies M D and Cox H R 1954, *Am. J. Physiol.* 178 30
11. Stange E, Agostini and Papenberg J 1975, *Atherosclerosis* 22 125
12. Corlson L A and Bottiger L E 1972, *Lancet* 1 865
13. Miller G J and Miller N E 1975, *Lancet* 1 16
14. Glomset J A 1970, *Am. J. Clin. Nutr.* 23 1129