

C. Four rats were maintained on both the heavy lipid diet (10% cholesterol, 30% saturated fat) and bromocriptine (daily dosage: 1 mg/kg of body weight) over a six month period.

D. Four rats were on the heavy lipid diet (10% cholesterol, 30% saturated fat) over a period of 4 months, and then sacrificed. (One rat died during the period.)

The coronary arteries and aortas of the rats were harvested and histologically prepared by H & E technique to maximize plaque contact with elastic fibers and smooth muscle layers.

Heart tissue was examined to detect pathological change.

The results were as follows:

A. All sections were normal. No distinctive fat deposition was found in either tunica intima or tunica media.

B. All sections demonstrated a marked reduction in fat deposits compared to animals fed the high fat diet for 4 months (Group D) and demonstrated only a minimal retention of lipid deposits in the tunica media. The tunica intima was unaffected.

C. All sections were similar to Group A. No definitive fat deposits were detected in either the tunica media or tunica intima.

D. All sections showed heavy lipid deposits among the elastic elements of the tunica media. Tunica intima was thickened.

Having described the invention, what is claimed is:

1. A method of therapeutically reducing the level of cholesterol in the blood of a vertebrate animal diagnosed as having excessive total plasma cholesterol which comprises administering to said vertebrate animal an effective dosage of a prolactin-inhibiting compound sufficient to inhibit prolactin secretion and reduce the level of cholesterol in the blood by at least 15 percent.

2. A method of therapeutically reducing the level of triglycerides in the blood of a vertebrate animal diagnosed as having excessive total plasma triglycerides which comprises administering to said vertebrate animal an effective dosage of a prolactin-inhibiting compound sufficient to inhibit prolactin secretion and reduce the level of triglycerides in the blood by at least 15 percent.

3. A method of therapeutically reducing the levels of cholesterol and triglycerides in the blood of a vertebrate animal diagnosed as having excessive total plasma cholesterol and triglycerides which comprises administering to said vertebrate animal an effective dosage of a prolactin-inhibiting compound sufficient to inhibit prolactin secretion and reduce the cholesterol and triglyceride levels in the blood by at least 15 percent.

4. A method of suppressing and reducing the level of lipid plaques formed in the walls of the blood vessels of a vertebrate animal diagnosed as having an atherosclerotic condition which comprises administering to said animal an effective dosage of a prolactin-inhibiting compound sufficient to inhibit prolactin secretion and decrease the total plasma cholesterol and total triglyceride levels in the blood of the animal over a period of time sufficient to suppress and reduce the formation of

lipid plaques in the walls of the blood vessels of the animal.

5. The method of claim 4 wherein the dosage is administered over a period of at least about 25 days.

6. The method of claim 5 wherein the period of treatment ranges from about 90 days to about 180 days.

7. The method of claim 5 wherein the amount of plaque in the walls of the blood vessels of the animal is reduced by at least 15 percent.

8. The method of claim 4 wherein the dosage is administered over a period of at least 90 days, and the amount of plaque in the walls of the blood vessel of the animal is reduced by at least 15 percent.

9. The method of claim 4 wherein the prolactin-inhibiting compound is 2-bromo-alpha-ergocryptine or its salts formed from pharmaceutically acceptable acids.

10. The method of claim 4 wherein the prolactin-inhibiting compound is 2-bromo-alpha-ergocryptine or its salts formed from pharmaceutically acceptable acids, and the dosage of said prolactin-inhibiting compound is administered over a period of at least 25 days.

11. The method of claim 10 wherein the daily dosage of the prolactin-inhibiting compound ranges from about 0.02 mg/kg of body weight to about 6 mg/kg of body weight.

12. The method of claim 4 wherein the prolactin-inhibiting compound is selected from the group consisting of 6-methyl-8 beta-carbobenzyloxy-aminoethyl-10 alpha-ergoline; 1,6-dimethyl-8 beta-carbobenzyloxyaminomethyl-10 alpha-ergoline; 8-acylaminoergolines; ergocornine; 9,10-dihydroergocornine; and D-2-halo-6-alkyl-8-substituted ergolines.

13. The method of claim 4 wherein the dosage of the prolactin-inhibiting compound is administered to a human, the dosage ranges from about 2.0 mg/100 kg of body weight to about 10 mg/100 kg of body weight, and the dosage is administered over a period of at least 25 days.

14. The method of claim 13 wherein the dosage of said prolactin-inhibiting compound is administered over a period of from about 90 days to about 120 days.

15. The method of claim 13 wherein the amount of plaque in the walls of the blood vessels of the human as a result of the treatment is reduced by at least 15 percent, based on weight.

16. The method of claim 13 wherein the amount of plaque in the walls of the blood vessels of the human as a result of the treatment is reduced by at least 50 percent, based on weight.

17. The method of claim 13 wherein the prolactin-inhibiting compound is 2-bromo-alpha-ergocryptine or its salts formed from pharmaceutically acceptable acids.

18. The method of claim 13 wherein the prolactin-inhibiting compound is selected from the group consisting of 6-methyl-8 beta-carbobenzyloxy-aminoethyl-10 alpha-ergoline; 1,6-dimethyl-8 beta-carbobenzyloxyaminomethyl-10 alpha-ergoline; 8-acylaminoergolines; ergocornine; 9,10-dihydroergocornine; and D-2-halo-6-alkyl-8-substituted ergolines.

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