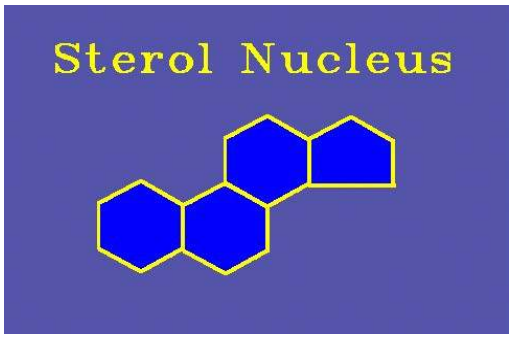
IV. THE CHOLESTEROL CAPER

"An ounce of prevention gathers no bucks."

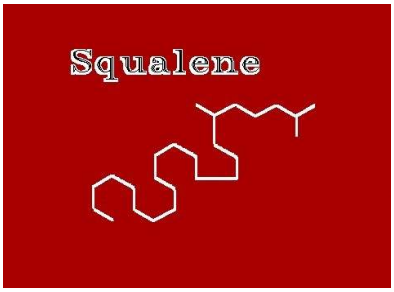
-Willy Sutton's brother.



Ah, the jetés, the arabesques, the fandangos make Baryshnikov look like a rank amateur! Americans *dance* around the cholesterol issue, and the pop media carries almost as much cholesterol in its pages as the readers carry in their arteries. Full-on front cover bleats on the cholesterol problem (Oops! Make that "crisis") list all the band aid remedies (cut the eggs, hold the bacon), but seldom is vegetarianism mentioned. The dreaded "V" word remains on the editorial hit although it would seem sensible that people who want their serum cholesterol to drop should just stop eating the stuff.

So what is cholesterol and why is everyone saying such bad things about it?

Cholesterol is a sterol molecule and a survivor in the three billion year molecular evolution that led to the first cells. It's synthesized by most animal cells from Coenzyme A (a molecule made of Pantothenic acid and a complicated carbon chain) which links to acetate, a two carbon molecule related to vinegar. After repeated enzymatic transfers the acetate molecules form into a long carbon chain which, because of its bonding angles, looks like this:



From here, even if one is not a biochemist, it's pretty obvious what happens next:

Straight chain carbon compounds assemble easily, but once they form rings the energy needed to disassemble them goes up drastically. Several species of bacteria which have time to split molecules nobody else wants can break down the sterol nucleus into carbon dioxide and water[1], but for animals it's not worth the trouble. Animals can get rid of cholesterol only by dumping it, modified slightly, as a bile acid in the stool.

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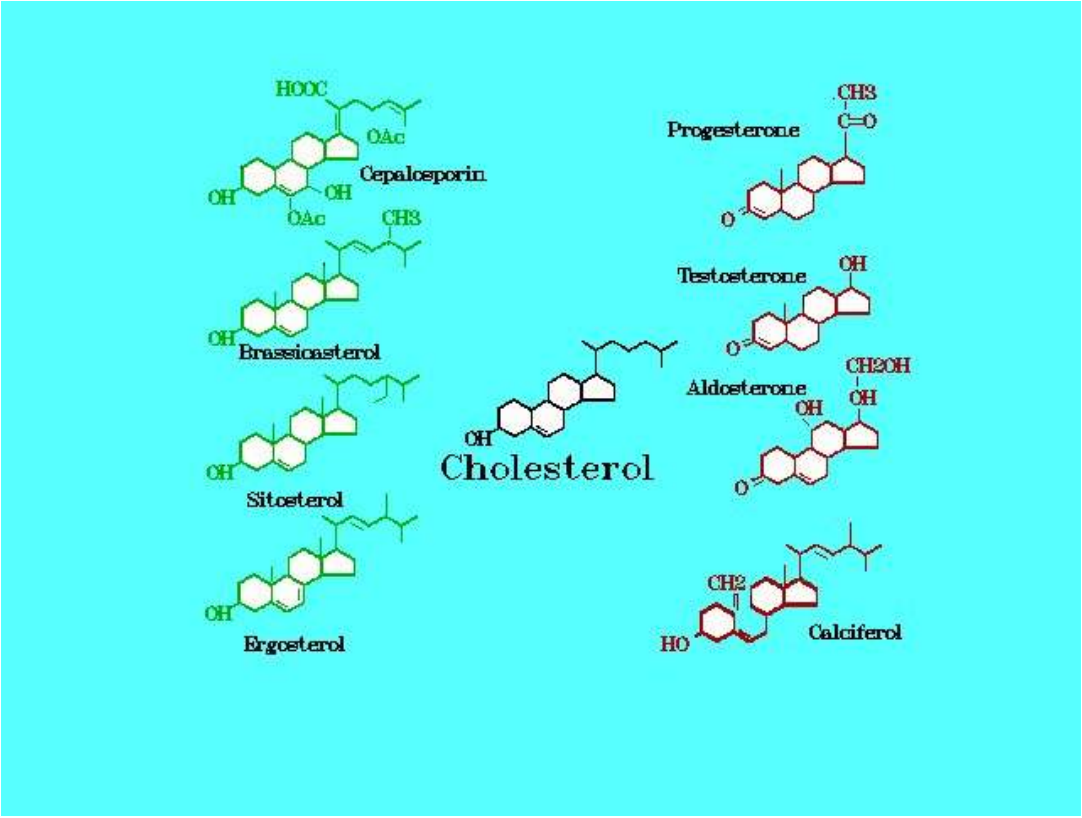
[1] Carney W, and Herzog H. *Microbial Transformation of Steroids*. Academic Press. New York 1967. Library of Congress 68-18661. p.48 "Many microbial genera (Corynebacterium, Mycobacterium, Pseudomonas, and Nocardia) can degrade steroidal substrates to carbon dioxide and water".

Cholesterol is rugged stuff. The inflexible nucleus gets along well with fat so, incorporated into the fatty membrane which acts as the animal cell wall, it becomes a strengthening material:

Humans synthesize 500 mg cholesterol/day[2], further evidence the molecule is essential to normal function. Most omnivores *eat* an additional 500 mg/day[3], which may raise the serum cholesterol above the liver's ability to modify and dump it. It then simply winds up in cells, a serious problem if the cell is on the inside of an artery. It may be that the cell's lysosome (its "stomach") is unable to digest the nucleus[4] which accumulates until the lysosome ruptures and kills the cell which is replaced by an irregular plaque that disturbs the smooth flow of blood. The result is coronary heart disease (CHD), peripheral vascular disease, stroke, and a few oddities so rare as to require *proper* names. All the cholesterol in the plaque comes from the serum cholesterol, not from synthesis within the cell[5]

Cholesterol also applied to the Bureau of Evolution for the hormone franchise. Both plants and animals do a virtuoso performance modifying its carbon side chains to produce a string of related sterols, only a few of which are shown below:



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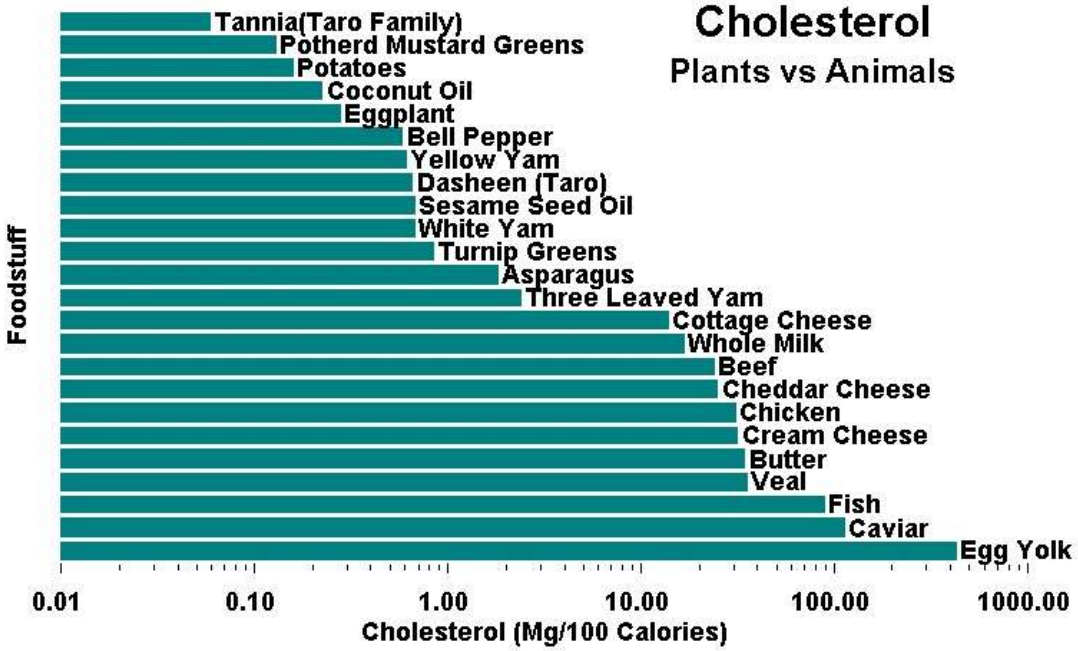
[2] Murray RK, Mayes PA, Granner DK, and Rodwell VW. *Harper's Biochemistry*. Appleton & Lange. Norwalk 1990;ISBN 0-8385-3640-9. p 249.

[3] Robbins SL, Kumar V, and Cotran RS. *Pathologic Basis of Disease*. W.B. Saunders Co. Philadelphia 1989. ISBN 0-7216-2302-6. p 464.

[4] de Duve C. *A Guided Tour of the Living Cell*. Scientific American Books, Inc. 1984. ISBN 0-7167-6002-9. p. 72 "Lysosomal overloading is seen in arteriosclerosis and kidney disease".

[5] Myant NB. *The Biology of Cholesterol and Related Steroids*. William Heinemann Medical Books Ltd. London 1981. p 611.

Plants synthesize small amounts of cholesterol on the pathway that leads to the other plant sterols[6] but the cholesterol content of plants is very much less than that of animals. In fact the cholesterol in plants is generally much less than 1 mg/100 gms and this is below the level of detection by USDA assay methods[7], so the cholesterol content of plant foods is always reported as zero. However, by compiling information from several obscure sources[8,9,10,11,12] the following graph was constructed:



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[6] Goodwin & Mercer p 415.

[7] Weihrauch JL. *Personal communication.* May 3 1990. Nutritionist, United States Department of Agriculture. "In our publications we report cholesterol to the nearest milligram. Most plant products contain cholesterol in amounts less than 1 milligram per 100 grams. At this time we have no plans to report cholesterol values in foods of plant origin...cholesterol in plant foods is insignificant when compared to the amounts in foods of animal origin."

[8] Weihrauch JL, and Gardner JM. *Sterol content of foods of plant origin*. J Am Diet Assoc. July 1978;73(1):39-46.

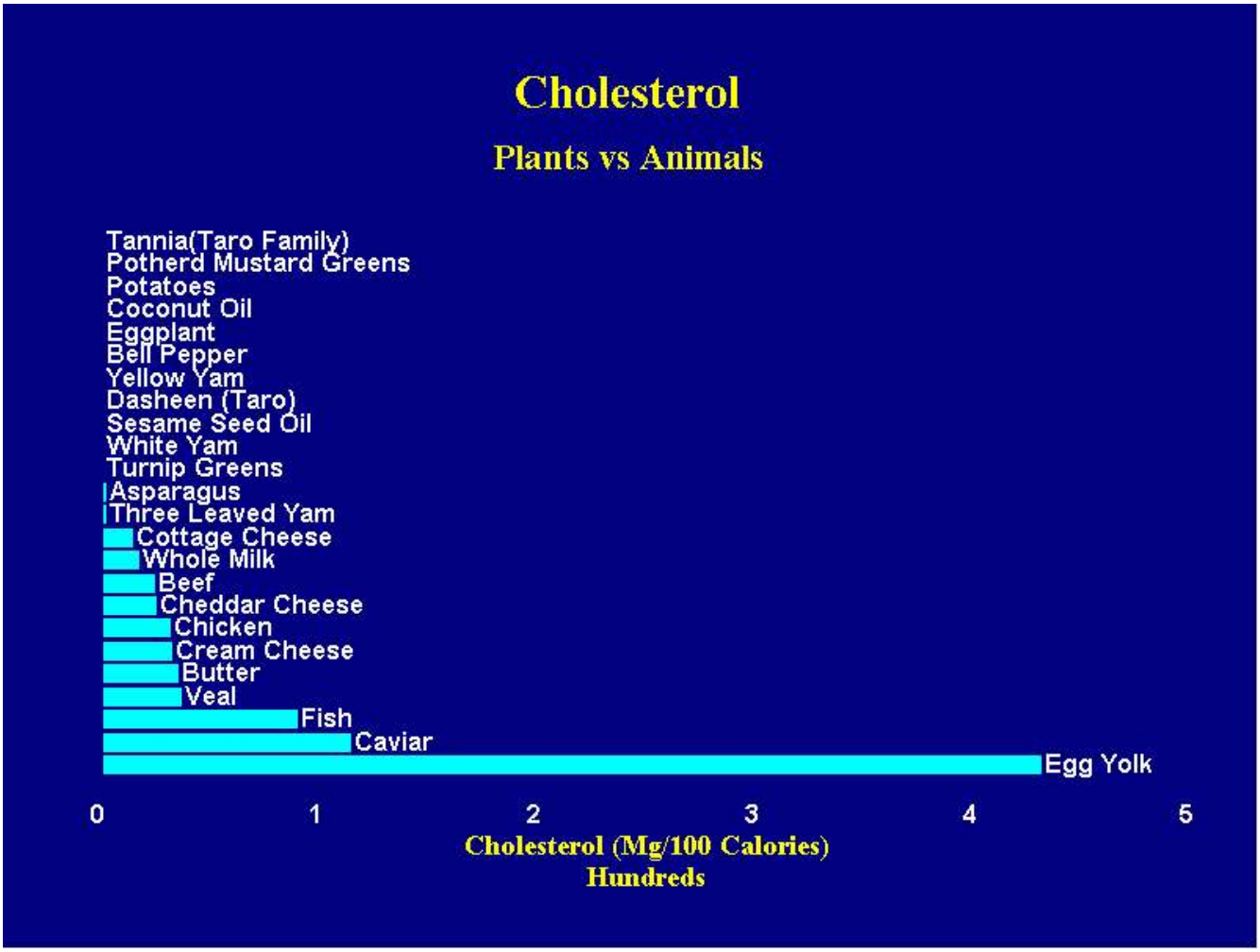
[9] Osagie AU. *Phytosterols in Some Tropical Tubers.* J Agric Food Chem. 1977;25(5):1222-23.

[10] Whitaker BD, and Lusby WR. *Steryl lipid content and composition in bell pepper fruit at three stages of ripening.* J Am Soc for Horticultural Science. July 1989;114(4):648-651.

[11] Oka Y, Kiriyama S, and Yoshida A. *Sterol composition of spices and cholesterol in vegetable foods.* J Japanese Soc Food and Nutr. 1974;27(7):347-355 (Abstract).

[12] Oka Y, Kiriyama S, and Yoshida A. *Sterol composition of vegetables.* J Japanese Soc Food and Nutr. 1973;26(2):121-128 (Abstract).

The cholesterol content of some plants appears formidable until we note the above graph has a logarithmic X-axis. When we redraw it with a normal X-axis the cholesterol content of most plants virtually disappears:



If one consumed nothing but 2400 calories/day of three leaved yams the cholesterol intake would be about 48 mgs. This is no reason to forswear yams although it is notable that yams are such a good sterol source that pharmaceutical companies use them as a starting point in the synthesis of steroid drugs such as cortisone[13] and natural progesterone (which if we're all really lucky may turn out to be the best solution yet for menopausal hormone replacement[14]).

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[13] *Encyclopedia Britannica*. Encyclopedia Britannica, Inc. 1974. Vol. 14, p 193. Vol. 17, p 680.

[14] Lee John R. *Natural Progesterone: The Multiple Roles of a Remarkable Hormone*. BLL Publishing. Sebastapol, CA 1993. ISBN 0-9643737-1-8.

Taking the average of all the known plant sources of cholesterol it's unlikely a 2400 Calorie vegan diet would exceed 10 mg cholesterol per day[15]. By contrast omnivores are trying to cut their intake to 300 mgs/day. The highest reported edible plant cholesterol/Calorie ratio (three leaved yam) is 5.8 times less than the lowest reported animal cholesterol/Calorie ratio (cottage cheese) with the exception of egg white which is very questionably listed as a zero cholesterol food. The vegan diet for practical purposes remains a no cholesterol diet and there is no RDA for cholesterol.

There are 3683 references in Medline to the effects of hypercholesterolemia. The 79 studies on *hypo*cholesterolemia are primarily concerned with strategies to produce the condition; apparently there are no reports of clinical illness resulting solely from inadequate cholesterol intake although problems can surface if cholesterol-lowering drugs are continued after a vegan diet has lowered serum cholesterol below 150 mg%.

A favorite establishment dietary remedy is the substitution of fish and poultry for meat on the theory these foods have less of the bad stuff.

Meat looks bad when it's sorted by cholesterol/weight ratio because it's high in fat which is light, so the meat denominators are small and the ratios large. Meat looks better when it's sorted by cholesterol/Calorie ratio because the fat carries nine Calories per gram so the meat denominators get larger and the ratios get smaller. Vice versa for fish. Animal food aficionados have no easy out, there's 300 mg of cholesterol in 10 ozs (680 Cal) of beef and in 577 Calories (18 ozs) of codfish. Neither sorting method supports much value in eating poultry rather than meat. Eggs and shrimp are no contest. The solution is to quit eating animals, fish included.

There is a genetic condition called familial hypercholesterolemia characterized by serum cholesterol levels in the 1000 mg/dL range and early death from Coronary Heart Disease (CHD). It's said to be unresponsive to diet[16] but this reasoning is flawed. There have been few studies of familial hypercholesterolemia treated with a vegan diet because the researchers can't conceive of such a diet and hence seldom try it[17]. The diets which have proven inadequate in controlling this condition may drop to 200 mg cholesterol/day but this is still a toxic cholesterol load for a species which evolved most of its genes in a sixty million year arboreal primate phase eating only leaves and fruit. The more recent hominids and modern humans have had a relatively short 4 million year exposure to the dietary cholesterol introduced by their omnivorous diet. Whatever cultural adaptations the hominids made in response to the survival value of Calorie-rich animal food it's unlikely that the genes coding for the hypercholesterolemia were ever subjected to Darwinian selection. The burden of high dietary fat and cholesterol does not become lethal until the fourth or fifth decade of life, well beyond reproductive age and beyond the life expectancy of the hominids, estimated at 30 years[18]. Thus no selection process can be expected to weed out the "high-cholesterol genes".

Familial hypercholesterolemia(FH) is cited by MEDLINE in 472 articles. Many of them deal with the reduction of serum cholesterol using drugs. None of them mention the words "vegan" or even "vegetarian". There have been some recent shots fired in the right direction, however. Connor achieved

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[15] Burslem J, Schonfeld G, Howald MA, and Weidman SW. *Plasma apoprotein and lipoprotein levels in vegetarians*. Metabolism. 1978;27:711-719.

[16] Myant (note 5) p 734.

[17] Myant (note 5) p 733.

[18] Scientific American. *Human Nutrition*. W.H Freeman. San Francisco, 1978. p 249.

serum cholesterol reductions of 18-21% in FH patients limited to 100 mg cholesterol/day[19]. More recently a cholesterol *free* diet reduced cholesterol levels in heterozygous FH patients from 323 ± 67 mg/dL to ~ 277 mg/dL[20]. The authors achieved this in a time period "for as long as eleven days", a phrase which suggests either they or their patients could not conceive of such a diet on a permanent basis.

It seems to me the modern pre-occupation with random genetic predisposition is unfruitful. A whole food vegan diet is the proper answer to high serum cholesterol. In addition to an undetectable cholesterol intake, it's also low in fat. Vegan population studies confirm low serum cholesterol and LDL levels, high HDL/LDL ratios, infrequent coronary events, almost non-existent obesity and a ten year increase in longevity[21].

The familial hypercholesterolemics may be sitting under the far end of a bell shaped distribution curve which includes in its middle the CHD patients of Ornish[22] who, when put on no more than a lacto-vegetarian, *near*-vegan diet rapidly lowered their serum cholesterol. Like humans[23], rabbits, and laboratory primates everywhere[24], they also reabsorbed their cholesterol plaques and improved their coronary circulation.

The Ornish study has been ignored by some cardiologists who object that the one year vegetarian regime only increased coronary artery inside diameter by 3%. Curiously they continue to prescribe cholesterol lowering drugs to their patients which accomplishes in an expensive manner what diet does for free.

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[19] Connor WE, Connor SL. *Dietary treatment of familial hypercholesterolemia.* Arteriosclerosis. 1989;9 (1 Suppl):I91-105. ISSN 0276-5047.

[20] Mokuno H, Yamada N, Sugimoto T, et al. *Cholesterol-free diet with a high ratio of polyunsaturated to saturated fatty acids in heterozygous familial hypercholesterolemia: significant lowering effect on plasma cholesterol.* Horm Metab Res. (GERMANY, WEST) 1990; 22 (4): 246-51. ISSN 0018-5043.

[21] Langley G. *Vegan Nutrition, a Survey of Research.* The Vegan Society. Oxford, 1988. ISBN 0-907337-15-5.

[22] Ornish DM, Brown SE, Scherwitz LW, et al. *Can lifestyle changes reverse coronary heart disease?* Lancet 1990; 336:129-133.

[23] Blankenhorn DH et al. *Beneficial effects of combined colestipol-niacin therapy on coronary atherosclerosis and coronary venous bypass grafts.* JAMA 1987;257:3233.

[24] Connor WE, and Connor S. *The Key Role of Nutritional Factors in the Prevention of Coronary Heart Disease.* Preventive Medicine. 1972; 1:49-83 (p 57).

However the flow of any fluid through a pipe is a fourth power function of pipe diameter (Poiseulle's Law)[25].

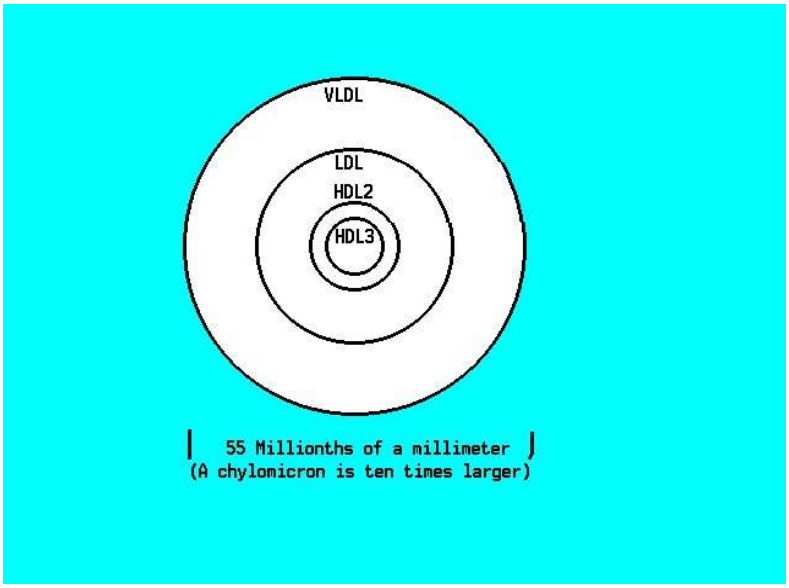
F (Flow)= $\Delta V/\Delta t = (P_1-P_2)(\pi R^4)/8L\eta$ where:
Δ=change
V=volume
t=time
(P_1-P_2)= pressure differential through the length (L) of the pipe
R=radius of the pipe=D(diameter)/2
η=coefficient of viscosity of the fluid

If we hold all variables constant except the diameter and take for a start a coronary lumen diameter of 3 mm we can compare the initial flow ($F_{1)}$ with flow ($F_{2)}$ after a year on the vegetarian diet:

$F_2/F_1=kR_2^4/kR_1^4=(3+.03x3)^4/3^4=(3.09)^4/81=91.17/81=1.13$

So coronary perfusion has been increased 13% although coronary diameter has only improved 3%. This probably explains why the Ornish patients experienced a 91% reduction in the frequency of heart pain. Nor is there any reason to think the resorption of cholesterol plaque would not continue indefinitely on a vegan diet.

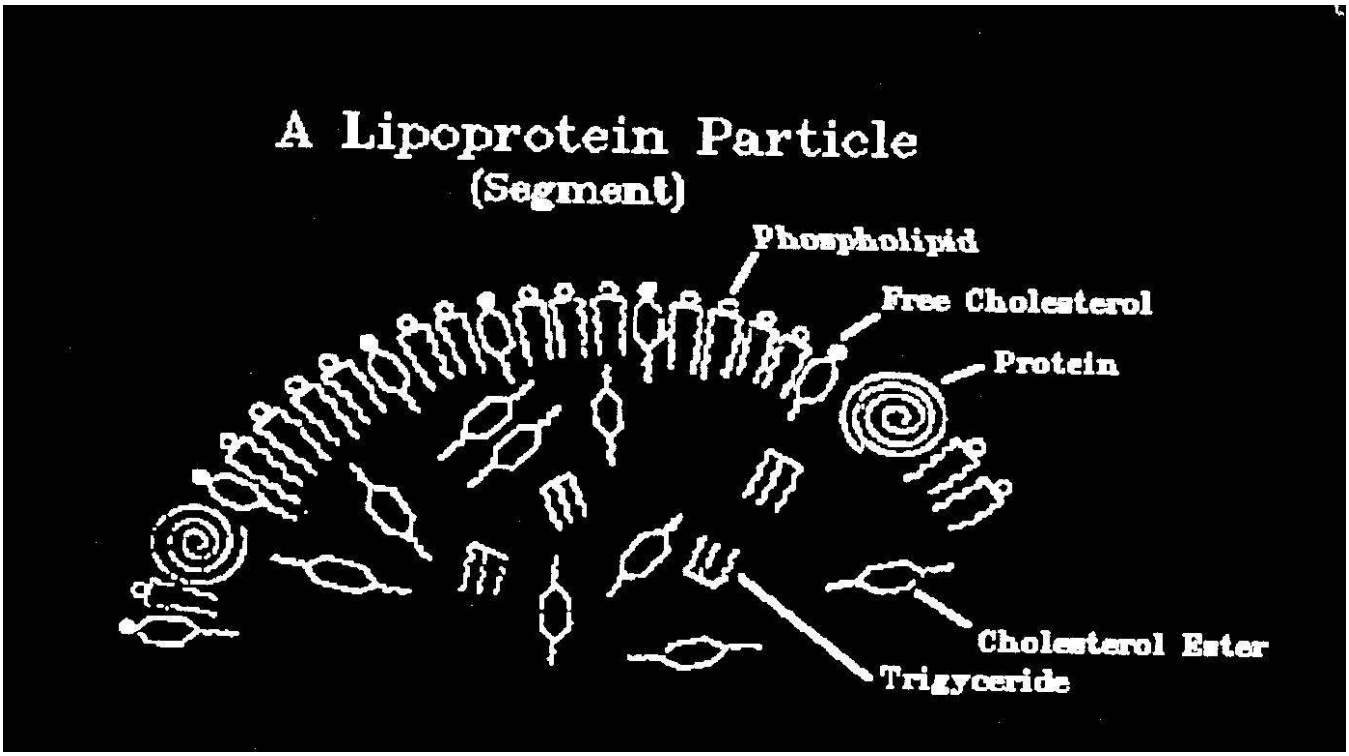
In recent years there has been much talk of lipoproteins, the little bubbles of cholesterol, cholesterol esters, phospholipids and neutral fat which carry the insoluble fats through the bloodstream. The bigger the lipoprotein bubble, the more fat and cholesterol it has and the lower its density relative to water. In a centrifuge the big bubbles wind up on top. Chylomicrons are very large lipoproteins formed during intestinal absorption[26].



[25] Serway R, and Faughn J. *College Physics.* Saunders College Publishing. Philadelphia, 1989. ISBN 0-03-022952-9. p 236.

[26] Ganong WF. *Review of Medical Physiology*. Appleton & Lange. Norwalk 1991. ISBN 08385-8418-7. p 284.

Low density lipoprotein (LDL) is "bad cholesterol", it dumps cholesterol in cells. High density lipoprotein (HDL) is "good cholesterol", it has plenty of room for more cholesterol so it carries cholesterol away for eventual disposal in the bile. The usage is erroneous. Cholesterol is cholesterol and has exactly the same structural formula whether it's taking a ride in HDL or LDL.



Protein synthesis is under genetic control. One can be stuck with genes which code for lots of LDL and with this piece of bad luck in mind, overlook that one has complete control over dietary cholesterol, regardless of genes.

Atherosclerosis and cholesterol metabolism have many facets. Smoking, a sedentary lifestyle, obesity, sugar, low dietary fiber and stress are other risk factors I did not address. The evidence is strong, however, that our distant ancestors were large, arboreal, herbivorous primates who adapted over a 60 million year period to a near-zero cholesterol diet. The more recent hominids have had a relatively short four million year exposure to the dietary cholesterol introduced by the omnivorous diet. Whatever cultural adaptations the hominids made in response to the survival value of Calorie-rich animal food, it's unlikely that the genes coding for the hyperlipidemic states were ever subjected to Darwinian selection. The burden of high dietary fat and cholesterol does not become lethal until the fourth or fifth decade of life, well beyond reproductive age and beyond the life expectancy of the hominids, estimated at 30 years[27] . The various disorders of cholesterol metabolism may not be diseases at all, but simply the result of random genetic variation which would never have been expressed had humans returned to their primate dietary heritage.

[27] Scientific American. *Human Nutrition*. W.H Freeman San Francisco, 1978 p 249

On a vegan diet the lipoproteins are irrelevant. Vegans average serum total cholesterol levels of 150.8 mg/dL[28]. Reference labs currently report any value below 200 mg/dL as "normal" but it should be noted this is only normal in a population dying of CHD at the rate of 978,500 in 1986[29]. In the Framingham Heart Study, CHD vanished at cholesterol levels below 150 mg/dL.[30].

In spite of these considerations a recent science article[31] stated that "extreme dietary change is not warranted for the entire population". Among the reasons cited for this conclusion was that such change would have "severe social and economic consequences". Apparently the authors did not regard 978,500 preventable coronary deaths as a severe consequence.

Meanwhile Americans continue their jetés, arabesques, and fandangos based on the cholesterol two-step. It's a danse macabre.

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[28] Carlson E, Kipps M, Lockie A, and Thomson J. *A comparative evaluation of vegan, vegetarian, and omnivore diets.* J Plant Foods 1985; 6:89-100.

[29] *Science News*. 1989;135 Jan 28.

[30] Castelli W. *Epidemiology of coronary heart disease: the Framingham study.* Am J Med 1984; 76(2A):4.

[31] Brown, M.S., and Goldstein, J.L. *How LDL Receptors Influence Cholesterol and Atherosclerosis.* Scientific American. Nov 1984; 251:5 p 66.