Why the 'Cholesterol Causes Heart Disease' Dogma has Persisted

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It must seem curious to some that the lipid hypothesis is still regarded as gospel by physicians despite overwhelming proof that it is flawed, if not fully fallacious. For example, a scholarly review published in Science several years ago showed that cholesterol levels up to 240 were normal and that women with a cholesterol over 240 had a decreased risk for coronary deaths.

Belief in this lipid doctrine was originally based on experiments in which cholesterol was force fed to herbivorous animals that do not eat meat, and the results are not relevant to humans.

Ancel Keys subsequently demonstrated a straight-line relationship between fat intake and deaths from heart disease in six countries, as well as a close correlation with cholesterol levels. This was so convincing that his views and suggestions became official NIH (National Institutes of Health) policy and a massive campaign to lower cholesterol was launched in 1961.

The problem was that while Keys had 22 countries to choose from, he "cherry picked" only those that had both high fat intake and coronary death rates to support his theories. Had he analyzed the data on all countries, he would have come to a very different conclusion.

For example, Mexico, which had the same fat consumption as Finland, was not included because coronary mortality was seven times higher in Finland. Also omitted were Sweden, Germany, France and Israel, where the higher the saturated fat and cholesterol intake, the lower the incidence of coronary deaths.

This demonization of fats was magnified and perpetuated by the cholesterol cartel of manufacturers of cholesterol lowering products, low fat foods, lipid testing equipment and others who stood to reap huge profits.

It reached its peak with the advent of statins which quickly became and remain the widest used and most profitable drugs ever. Pharmaceutical company efforts were aided and abetted by the FDA, Congress and other regulatory agencies, prestigious academic institutions, organizations and physicians, many of whom were handsomely rewarded for their support and endorsements.

Medical journals as well as the lay media were reluctant to publish anything that promoted a contrary view for fear of losing lucrative advertising revenue. Any opponents were viciously persecuted, as happened with Kilmer McCully, who demonstrated that lowering homocysteine might be more important than lowering cholesterol to prevent heart attacks, and was not only much safer, but considerably less costly than statins.

Like a deep earlobe crease, pot belly, premature vertex baldness and hundreds of other items, an elevated cholesterol is statistically associated with an increased incidence of heart attacks. However association never proves causation.

It would be much more accurate to refer to these as risk markers rather than risk factors, which implies a causal relationship. To get around this, statins are advertised as reducing the risk of heart attacks by implying that lowering cholesterol will result in a corresponding reduction in heart attack rates.

What the ads don't say is that this is relative risk, not absolute risk, which is quite different. Nor do they indicate how many people need to be treated for one person to benefit. For example, your doctor tells you that there is a new statin drug with no side effects and that a study showed that if you take it every day for the next five years your risk of heart attack will be reduced by over one third, which sounds appealing.

The study actually found a 34% reduction in relative risk. What you are not told is that after five years, 2.7% of patients taking this drug had a heart attack compared to 4.1% taking a placebo, which is an absolute risk reduction of only 1.4 percent, which is much less attractive. In addition, seventy-one people would have to take this every day for five years to prevent one person from having a heart attack, but it is not known if that person will be you.

One of the earliest attempts to deceive the public by using relative risk was the NIH sponsored Coronary Primary Prevention Trial, which claimed that in men with high cholesterol, taking cholestyramine (Questran) would lower cholesterol by 28% and result in a 50% reduction in major coronary events after seven years.

Although these goals were not met, it was triumphantly reported that the study showed cholestyramine reduced the risk of coronary events by 19%, and it was recommended for everyone. But 19% was the relative risk based on estimated cholesterol lowering. The actual risk of a coronary event was 1.1%, and for fatal heart attacks, the risk difference was only 0.6%.

Furthermore the study dealt only with a lipid disorder that affects less than one in five hundred, and a very small fraction of the total number of deaths due to heart attacks. There was nothing to suggest that lowering cholesterol would be beneficial for men in other age groups or women of any age.

There was also no indication that cholestyramine would be safe, or even tolerated. Bile acids that are manufactured from cholesterol aid in the digestion of fats, and cholestyramine binds to bile acids so they can no longer perform this function.

Because the drug is indigestible, it and the bile acids are excreted, and the gall bladder has to make more bile by drawing cholesterol from the blood. The study was conducted in 3,800 men with primary hypercholesterolemia, a rare disorder characterized by extremely high blood cholesterol.

It was also limited to those in the 40-60 heart attack prone age group to improve their results. Some men stopped taking the foul tasting 4 to 5 packets of cholestyramine after one or two days, many complained of severe constipation or other gastrointestinal complaints due to the lack of bile acids and most were unable to take the full 24 grams daily, so that relatively few stayed on the required regimen for seven and a half years.

With respect to safety, cholestyramine interferes with the absorption of fat soluble vitamins and numerous common drugs, including Coumadin, digoxin, Inderal, phenobarbital, thiazide diuretics and thyroid medications.

More importantly, little was mentioned about the 21 cases and 8 deaths from gastrointestinal cancer in those taking the drug, compared to 11 cases and just 1 death in the control group.

Small wonder that statins seemed to be a much better option, although their significant side effects have also been swept under the rug. Perhaps the most egregious abuses have come from statin ads, especially on TV. These consistently cite relative risk reduction statistics from a specific demographic group, such as patients who have had a heart attack, and imply that they will prevent heart attacks in men, women and children of any age.

Originally, advertisements were required to state that statins have not been shown to prevent heart attacks, and even though this was in very small type that was difficult to read, drug companies successfully campaigned to have this removed.

Statins do not provide benefits in senior citizens or women of any age. More importantly, it is increasingly clear from JUPITER and other studies that statin benefits are unrelated to their cholesterol lowering effects and are more likely due to anti-inflammatory or antithrombotic effects.

As I pointed out almost a decade ago in the Journal of The American Medical Association, "It might be advisable to find the minimum statin dosage that provides cardioprotection. As with aspirin, this may be much lower than for other indications."

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