



# **The Junk Science Self-Defense Manual**

How to Recognize, and Protect Yourself  
From, Shady Science!

**By Anthony Colpo**

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The contents of this book are presented for information purposes only and are not intended as medical advice or to replace the advice of a physician or other health care professional. Anyone wishing to embark on any dietary, drug, exercise or lifestyle change for the purpose of preventing or treating a disease or health condition should first consult with, and seek clearance and guidance from, a competent health care professional.

Any individual wishing to apply the information in this book for the purposes of improving their own health should not do so without first reviewing the scientific references cited and consulting with a qualified medical practitioner. All patients need to be treated in an individual manner by their personal medical advisors.

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# Introduction

It is a sad and inescapable fact that most of what passes for health advice today is scientifically untenable nonsense. This includes most of the dietary guidelines promulgated by supposedly 'respectable' health authorities.

Why?

One major factor is the corrupting influence of big business upon the ethical behavior of researchers. In an ideal world, scientists would hold the objective pursuit of facts—not the appeasement of corporate sponsors—as their highest goal. In an ideal world, policymakers would hold public health as a far more important concern than the financial welfare of extremely powerful and wealthy industry groups.

Unfortunately, we do not live in an ideal world. We live in a world where scientific findings are regularly distorted, manipulated, and selectively cited in order to support predetermined agendas.

Over the last five decades, pharmaceutical companies and food conglomerates have exerted an ever-increasing influence over the direction and outcome of scientific research. The heightened involvement of these industries in the scientific arena does not arise from their humanitarian desire to advance the pool of accurate and useful health knowledge; it arises from the desire to increase corporate profits. Recent history is replete with examples demonstrating that attainment of the latter goal often occurs at the expense of the former. What is good for corporations and their shareholders isn't necessarily good for the general welfare; in fact, what is good for the bottom line of food and drug companies is often downright harmful to public health.

By sponsoring and influencing the outcome of research, powerful vested financial interests can use the respectable veneer of science to advance their corporate goals. An unwitting public, not privy to the behind-the-scenes workings of modern science and policy-making, will be none-the-wiser to what is truly occurring. When they read a

sensationalist story claiming that a single high-saturated fat meal can damage arteries, they will uncritically assume what they are reading is scientific fact. When their doctor tells them that lipid-lowering drugs will reduce their heart disease risk by a massive one-third, they rarely ask the doctor how he arrived at this conclusion. When health authorities claim that low-fat diets reduce the risk of heart disease, they automatically assume this to be true.

The sad reality is that most people simply don't bother to critically analyze most of the information presented to them, and that includes information pertaining to health. Most people instead work on the premise that if many authoritative-sounding organizations and individuals are proclaiming the same thing, then it must be true.

Wrong!

In my book *The Great Cholesterol Con*, I thoroughly destroy every single argument that has been advanced in support of the cholesterol theory. I then explain how this unscientific theory came to form a central pillar of modern medical practice. One of the key components in the rise of the cholesterol theory has been the prolific use of junk science.

### **Arm Yourself!**

To become a highly skilled and accomplished fighter typically requires years of regular and strenuous training. However, any fighting instructor worth his salt should be able to teach you simple, effective and easy-to-remember techniques that in a short time frame will greatly increase your ability to defend yourself from physical attack. While such techniques won't transform you into a contender for the UFC heavyweight title, they will make you better able to recognize and defend against physical assailants.

In a similar vein, the purpose of the *Junk Science Self Defense Manual* is not to turn you into a professional researcher (although a great many modern researchers would do well to carefully read the recommendations that follow!). Rather, the purpose of this manual is to alert you to some of the most common shady practices that occur in the name of "science". By learning how to identify these practices

when they occur, you will be far less likely to fall victim to the often-serious consequences that can arise from following scientifically unsound diet and health advice.

Best of health,

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# The Junk Science Self-Defense Techniques

<b>Technique #1: Check the Research Yourself!</b> .....	<b>7</b>
<b>Technique #2: Never Confuse Epidemiological Studies with Controlled Clinical Trials</b> .....	<b>9</b>
<b>Technique #3: Beware the Use of ‘Risk Factors’</b> .....	<b>12</b>
<b>Technique #4: Beware of Inappropriate Extrapolation</b> .....	<b>14</b>
<b>Technique #5: Watch Out For Selective Citation</b> .....	<b>20</b>
<b>Technique #6: Follow the Money!</b> .....	<b>22</b>

## Technique #1: Check the Research Yourself!

This is the single most valuable technique. Alas, it is also the most underused. Few people actually bother to check for the evidence behind health claims for themselves. This makes it easy for those who perpetuate false claims to state that the science supports their recommendations. They know full well that most people won't ever bother to check the research to see if this is in fact true!

When someone makes a health claim, have they cited any research to back this claim? Or have they simply made a statement that you are expected to accept at face value? One of the great downfalls of television and newspaper health stories is that they rarely provide verifiable research citations supporting the claims being made.

And when studies are cited, do not blindly assume that the author has read them and that they do indeed support his/her claims. I've lost count of the number of times I've checked studies that were cited in support of a specific stance, and found they either did not support that stance or even contradicted it!

A striking example of this phenomenon can be found in a joint statement by the American Heart Association and the NIH's National Heart, Lung, and Blood Institute entitled *The Cholesterol Facts*, where one finds the following claim: "*The results of the Framingham study indicate that a 1% reduction...of cholesterol [corresponds to a] 2% reduction in CHD risk*"[1].

Incredibly, one of the papers cited in support of the above statement was a thirty-year follow-up report from Framingham that flatly contradicts any claim that cholesterol reduction is beneficial. This report found that those whose cholesterol levels *decreased* during the study experienced an *increase* in both total and cardiovascular mortality! To quote the Framingham researchers themselves: "*There is a direct association between falling cholesterol levels over the first 14 years and mortality over the following 18 years...*"[2].



So don't be satisfied with the fact that someone has posted a bunch of scientific-looking citations at the end of their article. Check those citations for yourself! Doing so will often paint a very different picture to the one the original author wants you to see!

## **Technique #2: *Never Confuse Epidemiological Studies with Controlled Clinical Trials.***

One of the most regrettable developments in health research has been the tendency to award epidemiological studies the same or even greater status than that given to controlled clinical trials. To understand why this is such a deplorable practice, a quick explanation of both epidemiological and controlled clinical research is in order.

Epidemiological research involves observing the incidence of a particular ailment among a population/s, and then observing what dietary or lifestyle factors are more frequent among those who develop the ailment. The major limitation of epidemiological research is that it is highly prone to what are known as “confounders”. For example, researchers commonly state that Western countries eat higher amounts of saturated fat and have higher rates of heart disease. Indeed they do. But these countries also have higher rates of obesity and diabetes, higher rates of psychosocial stress, lower rates of physical activity, and higher consumption of refined carbohydrates, omega-6 fatty acids, trans fats, and nutrient-depleted packaged foods. Researchers have linked all the aforementioned factors to heart disease. To single out saturated fats, when so many other potential culprits are present, is extremely poor scientific conduct.

This is why randomized, controlled clinical trials (RCTs) are so important. They allow researchers to minimize the influence of confounding factors. RCTs compare groups of subjects that are similar in sex, age and health status. The study participants are randomly assigned to one of two groups. One group receives the studied treatment (be it diet, a drug, vitamins, etc), while the other does not receive the studied treatment and serves as a control group. Ideally, RCTs are 'double-blind', meaning that both researchers and participants are unaware of who is in the treatment group and who is in the control group, a safeguard that helps prevent researcher bias and the possibility of a placebo effect amongst the subjects. [\(A more](#)

detailed explanation of epidemiological and clinical trial research can be found in Chapters 7 and 8 of *The Great Cholesterol Con*).

Because it greatly minimizes the influence of confounding factors, the data from RCTs represents a much higher standard of evidence than that from epidemiological research.

Such tightly controlled research has the rude habit of destroying some of our most ingrained and deeply held beliefs. For example, some epidemiological studies have shown higher rates of saturated fat consumption to be associated with higher rates of heart disease. However, RCTs comparing low- and high-saturated fat diets have completely failed to show any mortality benefit from the former. Health authorities deal with this uncomfortable contradiction simply by ignoring the evidence from RCTs and selectively citing the epidemiological research.

The promotion of 'healthy' whole grain cereal products is another classic example of this atrocious practice. It is epidemiological evidence that has been used to portray whole grain cereals as a 'health food', even though controlled clinical trials have detected no cardiovascular or cancer benefits from whole grain consumption whatsoever.

A reduction in colon cancer, for example, is one frequently cited benefit of whole grain consumption. Indeed, numerous epidemiological studies show that higher levels of whole grain consumption are associated with lower levels of colorectal cancer. However, of the numerous controlled clinical trials that have examined the effect of eating whole grains upon colorectal polyp and cancer incidence, *none* have ever found any benefit whatsoever from consuming whole grains[3].

So why do epidemiological studies repeatedly find lower risk of colorectal cancer in those who eat whole grains? Because whole grain consumers appear to be more health conscious than the average person, and therefore practice numerous other habits that do indeed confer protection against cancer. In the Iowa Women's Health Study, for example, which examined cereal grain intake among over 34,000 women, researchers found that "*higher whole-grain intake*

*was associated with having more education, a lower body mass index and waist-to-hip ratio [and] being a non-smoker, doing more regular physical activity, and using vitamin supplements and hormone replacement therapy.”[4]*

It's not the whole grains that protect these women—it's their healthier lifestyles, which include a higher level of exercise, and a lower prevalence of obesity and smoking. When these factors are controlled for in clinical trials, the utter uselessness of whole grains for preventing cancer is quickly revealed.

Despite this, researchers continue to promote whole grain consumption as a strategy against cancer! Making headlines in 2005 was an epidemiological study of over 60,000 Swedish women that found high consumption of whole grains was associated with a lower risk of colon cancer. The researchers concluded: *“Our findings suggest that high consumption of whole grains may decrease the risk of colon cancer in women.”*[5] This was despite the already known failure of clinical trials to show any such protection!

Don't fall for the epidemiology scam. Always remember the golden rule: *“Association does NOT equal causation”*. As soon as you read that a new study has shown a specific food or diet to confer magnificent health benefits, or to pose a dire threat to your health, ask whether this evidence was from an epidemiological study or a controlled clinical trial. If it was from an epidemiological study, then realize the findings are not proof of anything. They merely represent a statistical association that should not be considered causal until it is confirmed by controlled clinical research.

## Technique #3: Beware the Use of ‘Risk Factors’.

Many ailments can be quickly and readily diagnosed. For example, a quick visual examination will readily detect the presence of bleeding gums or acne. Other diseases present symptoms, such as inflamed joints or swollen glands, which allow a practitioner to readily detect the presence of a particular ailment.

Some ailments, however, cannot be so readily detected. Heart disease, for example, may be present in individuals who look and feel perfectly healthy. For many people, the first ‘symptom’ of heart disease is a heart attack itself.

Because it is so difficult for doctors and researchers to diagnose heart disease in those not exhibiting overt symptoms, modern medicine has embraced the use of ‘risk factors’. These are characteristics that seem to occur more frequently in individuals who fall prey to heart disease than those who do not. Perhaps the most famous risk factor in medical history is blood cholesterol. We have repeatedly been told that high blood cholesterol is a risk factor for heart disease, and doctors routinely test their patients’ cholesterol levels.

The problem with risk factors is that they are statistically--but not necessarily causally--related to the ailment in question. In the case of cholesterol, autopsy studies have revealed that individuals with cholesterol levels as low as 111 mg/dl can possess extremely atherosclerotic arteries, while many individuals with cholesterol levels of 300+ have lived well into old age, free of heart disease (see [Chapter 4 of \*The Great Cholesterol Con\*](#)). Why? Because cholesterol does *not* cause heart disease. It is merely a physiological characteristic that some—but not all—studies have shown to be more frequent in heart disease victims. Having high blood cholesterol does not necessarily mean you have heart disease; it means you have high blood cholesterol. And lowering cholesterol does not mean you will lower your risk of heart disease, as over five decades’ worth of failed dietary cholesterol lowering intervention trials have shown.

So when a newspaper story or research article exclaims that a recent study has shown a certain food or drug "*lowers the risk of heart disease*", read carefully to see if this study measured the actual incidence of heart disease among the subjects, or whether it simply examined changes in statistically related risk factors, such as blood cholesterol.

In October 1999, the U.S. Food and Drug Administration (FDA) authorized the following health claim for foods containing soy protein: "*...foods containing soy protein included in a diet low in saturated fat and cholesterol may reduce the risk of CHD...*" The FDA approved this health claim despite the fact that soy protein has *never* been shown to prevent even a single heart attack. The heart-healthy claim for soy was based almost entirely upon studies showing that soy protein consumption lowers blood cholesterol levels[6]. The FDA chose to ignore decades' worth of clinical trial evidence showing dietary cholesterol lowering to be an abysmal failure in reducing heart disease incidence and mortality.

The FDA claim for soy protein was issued in response to a petition by Protein Technologies International, one of the world's largest soy producers[7]. By ignoring the failure of dietary cholesterol lowering trials, and the lack of evidence that soy reduces actual heart disease incidence, the FDA did little to quell widespread suspicion that it places the interests of industry far above those of public health.

Don't fall prey to the 'risk factor' racket. Changes in blood markers of questionable validity do not automatically equate to a real reduction in disease risk.

## Technique #4: Beware of Inappropriate Extrapolation.

One common research trick is to cite a study that revealed a certain finding, and then claim it actually showed something entirely different.

A textbook case of this phenomenon occurred in August 2006, when headlines around the world screamed: *“One High-Saturated Fat Meal Can Be Bad”*. The accompanying Associated Press article reported on an Australian study that compared two meals identical in every respect except for their fat content. One was rich in saturated fat (from coconut oil), while the other was rich in polyunsaturated fat (from safflower oil, which is rich in omega-6 polyunsaturated fatty acids). A group of fourteen healthy subjects consumed one of the high-fat meals on a single occasion, returning to the lab a month later to consume the other high-fat meal. According to the article, the study showed that eating even a single saturate-rich meal had dire consequences for our arteries. The article quoted the head researcher, Stephen Nicholls, as saying: *“...the take-home, public-health message is this: It's further evidence to support the need to aggressively reduce the amount of saturated fat consumed in the diet.”*

Dr. James O'Keefe, a cardiologist at the Mid America Heart Institute in Kansas City, said the study showed *“a really important concept - when you eat the wrong types of food, inflammation and damage to the vessels happens immediately afterward.”*[8]

In reality, the study showed no such thing.

The researchers did *not* find any *“damage”* in the arteries of the subjects. Indeed, the study methods did not even allow for such a finding to be detected, even if it did occur.

What the study measured was flow-mediated dilation (FMD) before, and then 3 and 6 hours after, the high-fat meals. When researchers measure FMD, a cuff is inflated around the ankle, wrist or forearm for several minutes, occluding the artery to be measured (in this case,

the forearm brachial artery). The cuff is then deflated, and the degree of subsequent dilation in that artery is measured.

In the saturated fat group, the average degree of FMD was 2.2% lower 3 hours after the meal than prior to the meal. After the polyunsaturated fat meal, FMD was only 0.9% lower at 3 hours post meal. This finding was then very loudly trumpeted in the media as proof that saturated fat is harmful.

What rubbish.

Take a close look at the table below, which contains the FMD data from the study[9].

<b>Mean flow-mediated dilation in subjects before and after consuming high fat meals.</b>			
	<b>Immediately before meal</b>	<b>3 hours after meal</b>	<b>6 hours after meal</b>
Polyunsaturated fat meal	5.2%	4.3%	4.8%
Saturated fat meal	6.9%	4.7%	6.2%

You will notice that while FMD did indeed decline to a greater degree at 3 hours compared to baseline in the saturated fat group, it was still higher at every point during the study than in the polyunsaturated fat group! Blood flow was greater in the forearm at every measured point after the saturate-rich meal—hardly supportive of the claim that saturated fat was more ‘damaging’ to the arteries!

Of course, this is not how the results were explained to the public. Instead, the media honed in on the greater percentage reduction in FMD at 3 hours, and cited it as evidence of arterial ‘harm’. But notice that the average baseline (pre-meal) FMD of the study subjects was much higher on the occasion they consumed the saturated fat meal. I’ll explain the possible importance of this in a moment, but first, let’s quickly discuss the concept of ‘statistical significance’.



Researchers regularly calculate the statistical significance of their findings, which refers to the mathematical probability that the findings were simply due to chance. When research findings do not reach statistical significance, then they may be entirely due to chance. And the data from the Australian study clearly shows that the differences in FMD at all time points between the saturated and polyunsaturated meals were *not* statistically significant!

Sometimes research findings fail to meet statistical significance because of limitations in study design, such as a small number of participants. Sometimes, a finding that narrowly misses out on reaching statistical significance would indeed reach statistical significance *if* it were replicated among a much larger group of subjects. But one obviously cannot claim significance for their results until that larger trial is done and significant results are attained.

However, if the researchers and the popular media were going to go ahead and consider the non-significant difference at 3 hours as significant, then for the sake of consistency they should also have considered the higher baseline FMD in the saturated fat group as significant. And this would raise the possibility of what is known as a 'regression to the mean' effect. In a strictly regulated homeostatic environment such as the human body, there may be a narrow range or a threshold for post-meal FMD. If this is the case, then the difference between the two meals may simply be explained by the fact that the higher baseline FMD before the saturate-rich meal necessitated a greater drop to achieve typical FMD levels 3 hours post-meal. In fact, the researchers even raised the possibility of regression to the mean in their study paper. Of course, this potential explanation never surfaced in the sensationalist media articles, which seemed far more concerned with whipping up as much anti-saturate hysteria as possible than with presenting an impartial and reasoned appraisal of the study results.

The bottom line is that all this anti-saturated fat hyperbole stemmed from a study in which:

1. FMD was actually greater after the saturated fat meal!

2. The greater percentage drop in FMD was not statistically significant when the changes after the two meals were compared with each other!

The shady extrapolation from this study did not stop at distorting the FMD data. Before and after the high-fat meals, the researchers also measured the effect of HDL cholesterol on endothelial (arterial) cell expression of intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1). For those of you not familiar with scientific gobbledygook, adhesion molecules are believed to play an important role in the atherosclerotic process by facilitating the components of atherosclerotic plaque to proliferate at the site/s of arterial damage.

Both ICAM-1 and V-CAM-1 were higher at six hours after consumption of the saturate-rich meal, but lower after consumption of the polyunsaturated-rich meal. Again, these findings were vigorously presented as proof that saturated fat exerts harmful inflammatory effects in human arteries.

Just one wee problem—the findings were *not* observed in human arteries! The researchers were *not* observing actual arterial plaque formation or inflammatory activity in real live humans. They were instead observing the effects of HDL cholesterol extracted from humans after eating the test meals on the amount of ICAM-1 and VCAM-1 expressed by umbilical vein endothelial cells in a *petri dish*!

If you have difficulty understanding why these results should not be automatically extrapolated to real live humans, then I suggest you take a moment to grab a small glass dish and stand in front of a mirror. Look at the dish, and then look at yourself. See any difference?

Evidently, the numerous commentators who hyped this study to fever pitch cannot tell the difference between a real live human being and a petri dish. It's anyone's guess as to the long-term relevance of acute reactions observed in a petri dish to plaque formation in human arteries. To claim that these reactions demonstrate that saturated fat is indeed atherosclerotic is to make a massive leap of faith. But that's just what the researchers and many of their peers did.

In the Associated Press article, Dr. Richard Milani, head of preventive cardiology at Ochsner Clinic Foundation in New Orleans, stated: *"...given a choice between something with polyunsaturated fat and saturated fat, please avoid the saturated fat"*.

The absurdity of such advice is nothing short of astounding!

Whether a saturated fat-rich diet causes more heart disease than a polyunsaturated fat-rich diet cannot even begin to be determined by an experiment involving a single meal. The only way to properly test this assertion would be to take a group of volunteers and randomly assign half to eat a saturate-rich diet on a long-term basis, and the other half to consume a polyunsaturate-rich diet for an identical period of time.

Guess what? Numerous such studies, lasting up to eight years, have already been conducted! Since 1965, the results of a dozen randomized clinical trials comparing saturate-rich diets with diets high in omega-6 polyunsaturated fats have been published in the medical literature. If you have read Chapter 8 of *The Great Cholesterol Con*, which carefully describes each and every one of these trials, you will know that *none* of them—not one—has demonstrated any reduction in cardiovascular or overall mortality that can be attributed to saturated fat restriction. In fact, two of these trials found a significantly higher death rate in the groups consuming the polyunsaturated-rich diets. Meanwhile, the longest-running trial that focused on substituting polyunsaturated fats for saturated fats (the Los Angeles Veterans study) showed a significant increase in cancer mortality among the high-polyunsaturate subjects--despite their lower rate of smoking!

So when someone like Dr. Richard Milani recommends polyunsaturated omega-6 fats over saturated fats, the wisest course of action is to ignore the living daylight out of him!

The inferences drawn from the Australian study reflect poorly on the state of modern cardiovascular research. For prominent researchers to enthusiastically embrace the results of a study involving changes after a single meal—changes of extremely questionable relevance to

the development of heart disease in human beings—and completely ignore the far more relevant results of long term RCTs that did indeed examine the actual effect of diet on heart disease incidence and mortality, is an absolute *disgrace*.

The intellectual bankruptcy so prevalent among much of the cardiovascular research fraternity does not just impact upon dietary recommendations. Sloppy science also allows drugs to be recommended to groups of people who actually have no business taking them!

If a clinical trial shows a drug intervention to be effective in reducing morbidity or mortality among a certain group, then great caution should be exercised when extrapolating the results of that trial to other groups. For example, in tightly controlled clinical trials, cholesterol-lowering statin drugs have been shown to lower overall mortality in middle-aged males with existing heart disease and in diabetics. Clinical trials involving women, males free of heart disease, and the elderly have *not* demonstrated any mortality reduction from the use of statin drugs (see Chapters 9 and 23 of *The Great Cholesterol Con*). Yet statin drugs are repeatedly hailed as wonder drugs that should be prescribed to anyone with even moderately high cholesterol. Consequently, they are routinely prescribed to women, males free of heart disease, and the elderly! These folks are being placed at unnecessary risk of drug side effects because their doctors have never bothered to scrupulously examine the mortality data from statin RCTs.

Don't get sucked in by the widespread use of irrelevant extrapolation. Check the research for yourself to see if the inferences being made from a study are factual, or simply an exercise in wishful thinking.

## Technique #5: Watch Out For Selective Citation.

It is easy to support any point of view by citing only supportive studies and ignoring contradictory research. However, good science dictates that one must arrive at a conclusion only after examining all the available evidence—not just that which happens to support one's predetermined conclusions!

A classic example of selective citation is the use of the Japanese as evidence for the alleged benefits of low-fat nutrition. Low-fat advocates continually point out that the Japanese eat, on average, less fat than most Westerners and also suffer much lower rates of heart disease. Indeed, a large study of Japanese emigrants by Dr. Michael Marmot and colleagues showed that after migration to the U.S., their chances of dying from a heart attack rose significantly. But this increased heart disease risk had nothing to do with dietary changes; what the low-fat advocates don't tell you is that neither diet nor serum cholesterol levels were associated with the increased mortality from heart disease amongst the emigrants[10].

They also neglect to mention that in a follow-up study, Marmot found the strongest indicator of risk was the degree to which Japanese emigrants retained their traditional culture. Japanese-Americans who remained faithful to their native cultural traditions experienced an incidence of heart disease as low as that seen back in Japan. In contrast, those that embraced Western culture most extensively were two-and-a-half to five times more likely to suffer from heart disease. What's more, those who adhered to Japanese cultural traditions but ate higher fat American foods, were far better protected than those who adopted the American lifestyle but ate lower fat Japanese fare[11]! As explained in *The Great Cholesterol Con*, psychosocial stress is one of the greatest (and most under-recognized) causes of heart disease known to humankind. The Japanese social structure, which emphasizes co-operation and social support as opposed to one-upmanship and cut-throat competition, may be a major contributor to their lower rate of heart disease.

What the low-fat promoters also fail to share is the fact that among both native and expatriate Japanese, higher saturated fat consumption is associated with lower cardiovascular mortality and greater longevity! Japan's ascent to the top of the longevity ladder has occurred alongside a significant increase in saturated fat consumption. By the year 2000, total fat and animal fat intake in Japan had risen over 250 percent from what it was in 1961, when Greece enjoyed the greatest life expectancy. Stroke incidence and mortality among the Japanese has also declined markedly during this time. Before you ascribe these benefits solely to improved living standards, a follow-up study of over 3,700 Japanese men and women aged 35 to 89 years from 1984 to 2001 found those with the highest intake of animal fat had a sixty-two percent lower risk of ischemic stroke death! A much larger study involving over 40,000 Japanese adults found that, during sixteen years of follow-up, those who ate the most eggs, dairy products, and fish had a twenty-eight percent lower risk of stroke than those who ate the least. Yet another study of almost 5,000 Japanese men and women found that, over a fourteen-year period, those in the highest quartile of saturated fat intake had a seventy percent lower risk of hemorrhagic stroke than those in the lowest quartile!

When the evidence is examined in its entirety, it readily becomes clear that the Japanese enjoy long life and low cardiovascular mortality, not because of their low fat diet, but in spite of it!

**NOTE:** *The Great Cholesterol Con* contains an even more detailed and fully referenced discussion on the Japanese experience and why it does not in any way support the anti-saturated fat theory.

## Technique #6: Follow the Money!

Financial conflicts of interest provide a strong incentive for some researchers and information providers to engage in outright dishonesty, and for others to maintain a state of 'willful ignorance'. As Upton Sinclair once remarked: *"It is difficult to get a man to understand something when his salary depends on his not understanding it."*

Of course, industry sponsorship of research does not automatically signal corrupt and unethical behavior by the researchers involved. Indeed, some entirely ethical and useful data has arisen from industry-sponsored studies. However, the untoward influence of vested financial concerns on the outcome and interpretation of scientific research is a very real and pervasive problem (see [Chapter 12 of \*The Great Cholesterol Con\*](#)).

Financial conflicts of interest can go a long way to explaining why researchers or 'experts' have arrived at a certain stance, even though the science does not support that stance.

The National Cholesterol Education Program (NCEP) is responsible for setting the official blood cholesterol targets that doctors abide by when counseling their patients. Over the years, the upper recommended limits for ideal blood cholesterol levels have gradually sunk lower and lower, allowing doctors to place millions more on cholesterol-lowering medications.

In May 2001 the NCEP revised its guidelines, categorizing the entire population into one of three categories according to CHD risk. Each category was assigned an upper limit of LDL cholesterol. Individuals exceeding their assigned threshold were given three months to achieve their target LDL level, and if their efforts were unsuccessful, the initiation of drug therapy was recommended. The only two side effects listed for statin use in the NCEP guidelines were the relatively benign-sounding terms *"myopathy"* and *"increased liver enzymes"*. This was despite the fact that statins are associated with numerous adverse effects, including rhabdomyolysis (in fact, the statin drug Baycol had to be pulled from the market the previous year after it was

linked to the deaths of 52 people, most of whom died from rhabdomyolysis-induced kidney failure. All statin drugs have demonstrated the ability to produce rhabdomyolysis).

The financial disclosure information at the end of the NCEP article shows that six of the fourteen NCEP committee members had received financial support from multiple pharmaceutical companies. The resultant list read like a *Who's Who* of cholesterol drug manufacturers[12].

In July 2004, the NCEP updated its guidelines yet again, recommending even lower target LDL levels in all but the lowest risk CHD category, again creating millions of new customers for cholesterol-lowering drug manufacturers[13]. Again, the panel was comprised of individuals with conflicting financial ties. This time, all but one of the nine panelists had received grants or consulting or speakers' fees from the manufacturers of some of the most popular statin medications on the market, including Pfizer, Bristol-Myers Squibb, Merck and AstraZeneca[14].

Remember, statins have not been clinically shown to save the lives of anyone except middle-aged males with existing heart disease and diabetics, but nowhere do the NCEP guidelines emphasize this important caveat. And the NCEP's enthusiastic recommendation of low LDL targets was issued despite a complete disconnect between degree of LDL reduction and clinical benefit.

The October 2006 issue of the *Annals of Internal Medicine* featured a sweeping research review by scientists who went looking for the "compelling" evidence claimed by the NCEP for its guidelines. What they found was "...no clinical trial subgroup analyses or valid cohort or case-control analyses suggesting that the degree to which LDL cholesterol responds to a statin independently predicts the degree of cardiovascular risk reduction." [15] In a similar vein, A September 2006 report in the American Medical Association's *Archives of Internal Medicine* featured a pooled analysis of thirteen randomized controlled trials comparing intensive statin therapy with a control treatment (no statins, lower dose statins, or usual care) in patients recently hospitalized for acute coronary syndromes. When the results of these trials were collectively analyzed, at twenty-four months there



was a nineteen percent reduction in overall cardiovascular events. But this risk reduction was independent of LDL cholesterol reduction. As the authors stated clearly in their paper: *"There is no significant evidence that reduction in LDL-C level explains these beneficial effects."*[16]

So why the insistence on specific LDL cholesterol targets by the NCEP panelists? Given the lack of supporting science, it is extremely difficult for even the most forgiving observer to believe that the researcher's intimate financial ties to cholesterol drug manufacturers did not exert a powerful influence.

### **Reverse psychology**

It is also important to realize that conflict of interest concerns are perversely exploited by those who themselves propagate shady health claims. For instance, when a critical commentator attacks the untenable assertion that animal fats cause heart disease, low-fat promoters frequently ignore the evidence that the commentator presents and instead attempt to discredit him/her by claiming that he/she must be sponsored by the meat, dairy or egg industries. The intent is to smear the critical commentator's character, so that people will dismiss him/her out of hand and not even bother to examine the science he/she has cited. Don't fall for such repugnant and blatantly dishonest diversionary tactics.

When someone makes a health claim, check both their personal and organizational financial ties to see if any financial incentives exist for them to make such a claim in the face of weak or non-existent evidence. Similarly, when someone accuses another party of having conflicting financial ties, check to see if this is in fact true.

## **Forewarned is Forearmed**

In the short time that it took you to read this e-book, you have greatly improved your ability to recognize the use of junk science when it occurs. This has been time well spent, for the health consequences stemming from adherence to fallacious diet and health claims can be very serious.

An example is the continued endorsement of low-saturated fat diets by health authorities, doctors and writers, despite the fact that controlled RCTs have repeatedly highlighted the complete failure of low-saturated fat diets to reduce heart disease mortality! For someone at high risk of heart disease, following such scientifically untenable advice could prove fatal. Instead of following a low-saturated fat diet, this person should be focusing on the things that science has shown to really matter—such as optimal omega-3 fat intake, diets comprised of antioxidant-rich whole foods, exercise, stress reduction, and avoidance of smoking, elevated blood sugar, industrially produced trans fatty acids, excessive omega-6 fats, and high bodily iron stores.

Knowledge is power, but only when you put that knowledge to use. Use the information in this e-book to help recognize and protect yourself against shady science.

## About the Author

Anthony Colpo is an independent researcher, physical conditioning specialist, and author of the groundbreaking books *The Fat Loss Bible* and *The Great Cholesterol Con*. Since 1991, he has been helping people from all walks of life get in the best shape of their lives.

Anthony has earned a reputation as an exacting, no-holds-barred commentator with a talent for explaining research findings in a manner readily understandable to the layperson.

Anthony is also the guy that unscrupulous diet 'gurus' and shoddy scientists love to hate. He has a knack for dissecting untenable diet and health claims and exposing, with unrepentant and unassailable logic, the absurdity of such claims.

For more information on Anthony's acclaimed books, visit the following web sites:

***The Fat Loss Bible***

<http://www.thefatlossbible.net/>

***The Great Cholesterol Con***

<http://www.thegreatcholesterolcon.com/>

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