The information contained herein is not intended to represent medical diagnosis, treatment or medical advice in any form, as it is general information and cannot be relied upon without consultation with your physician.

The information contained herein is not intended nor is it implied to be a substitute for professional medical advice. In fact, taking full advantage of the Track Your Plaque program will require that you consult with your physician or healthcare provider willing to work with you.

As medical information and your health can change rapidly, we strongly encourage you to discuss all health matters and concerns with your physician before beginning new diagnostic or treatment strategies.
# Table of Contents

**Foreword** to the new *Track Your Plaque Guide*  
**Introduction** Out with the old, in with the new  
**Chapter 1** Taking the guesswork out of heart disease  
**Chapter 2** Your life is worth $112,377  
**Chapter 3** What your doctor didn’t tell you about heart disease  

**Step 1**  
**Chapter 4** Heart disease can be measured  
**Chapter 5** Want to know whether you have heart disease? . . . Know your score!  
**Chapter 6** Can I reduce my heart scan score?  

**Step 2**  
**Chapter 7** “My doctor said my cholesterol was fine . . . So why did I have a heart attack?!”  

**Step 3**  
**Chapter 8** It’s not just about cholesterol: The many causes of plaque  
**Chapter 9** The six *Track Your Plaque* nutrition principles  
**Chapter 10** Vitamin D: Crucial nutrient for the *Track Your Plaque* program
Chapter 11  Is your thyroid to blame?  187
Chapter 12  Omega-3 fatty acids  206
Chapter 13  Control plaque with exercise  222
Chapter 14  Personal Profiles: Stories of real people in the Track Your Plaque program  237
Chapter 15  Putting together your own personal Track Your Plaque program  251
Appendix A  How to arrange lipoprotein testing in your area  256
Index  260
The current design of the Track Your Plaque program, as presented in this Guide, barely resembles the original, as conceived just over 10 years ago.

In the early 1990s, when early CT heart scanners began appearing on the scene, I saw this technology as nothing more than a means of detecting early heart disease. But, once people began returning with heart scan reports in hand, wielding “scores” of 200, 300, 500, etc., signifying the presence of coronary atherosclerotic plaque, it struck me that, if plaque can be precisely measured, it should be something we could change, manipulate . . . reduce?

Thus, the concept of the Track Your Plaque program was born from the simple notion that coronary atherosclerotic plaque, the abnormal material that underlies virtually all heart attacks and the “need” for heart procedures like angioplasty, stents, and bypass surgery, was measurable, trackable, and modifiable.

What I failed to see back then, however, was how quickly the basic concept would evolve. The phenomenon that has accelerated the development of this program at a pace I never imagined in 1995 was the Internet and the burgeoning of the Information Age. In years past, we would have relied on trial and error, long-term outcomes, and draw what we could from the experiences of others. But the emergence of websites, discussion forums, blogs, and other information exchange mechanisms showed us, through thousands of participants all looking to discover solutions to the same questions, what worked, what didn’t; what was accessible, what was not; how these concepts played out not just in my own backyard of Milwaukee, Wisconsin, but worldwide.

I learned that our most vigorous following was in San Francisco, where people and their doctors were more likely to “get’ the Track Your Plaque concepts more readily. Unique insights originate from this group of eclectic participants, coming from varied backgrounds like software engineer, day trader,
pharmacologist, and insurance company CEO. I learned that people on the East coast of the U.S. encountered enormous resistance from doctors who insisted that better solutions for heart disease would not emerge from unique reinterpretations of existing knowledge or from Web-based exchanges that generate new ideas, but from the blue blooded-Universities and the drug industry. Ideas like these were flatly dismissed as offhandedly as just so much alternative-medicine fluff. But it showed us ways to get the program done with as little help from mainstream doctors as possible (a sad reality). I learned that the program could be followed even in faraway parts of the world like India, Singapore, the Arab Emirates, Australia, England, and Poland. In fact, some of the most fascinating and unique lessons have come from these distant followers. I am very grateful for the contributions of all these people, who have compressed 50 years of observation and feedback into a few short years and have helped shape the current program, vastly better than my initial simple concept.

I also learned not to dismiss ideas that, at first, appeared bizarre or improbable. Patients and online discussions should be credited for divining several of the most important current concepts in the Track Your Plaque program. The concept of vitamin D supplementation, for example, for coronary plaque reversal was taught to me by a patient several years ago, a bright mechanical engineer who reasoned that vitamin D, because it was an important player in bone calcium metabolism, might serve a role in normalizing calcium behavior in coronary arteries. My first impulse was to dismiss this as just another nonsensical, useless idea from someone who couldn't possibly understand the intricacies of atherosclerotic disease and the complex metabolism of calcium, although such observations had some basis in scientific studies. But it was hard to ignore the fact that this man had, on his own along with knowledge of the early Track Your Plaque concepts, reduced his heart scan score 300 points in six months, a magnitude and time frame that outperformed anything I had seen achieved (back in 2003). Thus was launched our own investigations in vitamin D’s role in coronary plaque; there has been no looking back since.

The net effect has been that the Track Your Plaque program of today is a very different thing than its original. It is better: smarter, more scientific, more effective. I am reminded of how far the program has come when I discuss these concepts with colleagues of mine, whose eyes rapidly reflect that puzzled look of incomprehension. While it may mean that the Track Your Plaque program has strayed far from the comprehension of mainstream physicians, it also tells me that we have created something very unique.

To be sure, more work needs to be done. While many of the concepts that form the basis for the program lie on a solid foundation of existing science, some unique observations will require additional exploration and validation. The process of user feedback and new observation continues and promises to continue its time- and effort-compressing acceleration towards an effective solution to heart disease for as many people as possible.
The *Track Your Plaque* program that is described in these pages is therefore the sum total of the important lessons learned over more than a 10-year real-world experience that has been conducted at a pace I never dreamed of, an experience that I believe has so dramatically surpassed anything that has come before it that it is bound to draw criticism, accusations, and fear of losing grip on the present-day procedural status quo.

But it works.

William Davis, MD
January, 2011
Introduction

Out with the old, in with the new

Heart disease today is still managed using a paradigm that dates back over 40 years. It's time for a new, more effective approach, one that puts control over heart health back in your own hands.

Old notions in medicine have a peculiar way of lingering.

In 1882, Dr. Robert Koch discovered the tubercle bacillus in tissues of people with “consumption.” By connecting a bacterium with the disease, he usurped the long held notion that tuberculosis was a degenerative disease caused by lack of fresh air. But, for decades after Dr. Koch’s revelation, the “bad air” belief persisted. Surgical collapse of the lung, a painful and barbaric treatment for tuberculosis, persisted well into the 1960s, years after effective antibiotics were discovered in 1947.

The medical community of the 19th century viewed mental illness as the hereditary end-product of nervousness, alcoholism, prostitution and criminal behavior, a bias that remained widespread well into the mid-20th century. Nazi physicians invoked the theory of heritable “mental degeneration” to justify wholesale extermination of schizophrenics. Electro-convulsive therapy (ECT, or “electroshock therapy”) was widely applied to treat schizophrenia, depression, homosexuality, and criminal behavior for over 30 years, gradually abandoned (at least in its original form) after years of abusive application to subdue patients, demonized in the 1975 movie, “One Flew Over the Cuckoo’s Nest,” depicting the author’s real-life experience with ECT.

Long after a theory or practice has been discredited, it can persist, refusing to die. The new and improved may not be adopted into mainstream practice for years, even decades.
Back to the 21st century: What if you realized that, by quirks of human nature and the uneven adoption of health information, your doctor practiced medicine appropriate for 1985? 1975?

While digital information nowadays is transmitted at the speed of light, disseminating as fast as it takes the next juicy tidbit to be “virally” reproduced via social networking websites, it’s the human factor that still operates with the inertia of human behavior. Habits and attitudes slow the adoption of new information in time measured not in seconds, but in years or decades.

A century ago, 20 years were required for the new technology of blood pressure measurement to be adopted after its introduction in the U.S. in 1910, since physicians were long comfortable with the practice of “pulse palpation” (feeling the pulse). (The arcane language of pulse palpation persists to this day, terms like “pulsus parvus et tardus,” the slow rising pulse of a stiff aortic valve; and the "water-hammer" pulse of a leaking aortic valve.)

The discovery of new, health-changing information today in the 21st century disseminates through the ranks of modern healthcare providers at much the same pace as measuring blood pressure did in the early 20th century.

It’s also tempting to paint American medicine as a fiefdom intent on maintaining exclusive rein over health information. Look back over the hierarchical relationship of medicine over nursing in the past century: When blood pressure measurement was adopted on a broad scale in the 1930s, it was practiced only by physicians, since nurses were deemed incapable. (Modern-day nurses should surely have a hearty laugh over this.) Stethoscopes, around even longer than blood pressure cuffs, weren’t permitted to fall into the hands of nurses until the 1960s, since the medical community feared that nurses might command too much control over patient care. Even after nurses were permitted to have their own stethoscopes, great pains were taken to be certain the nurses’ version was readily distinguishable from the “real” tool wielded by physicians; nurses’ stethoscopes were therefore labeled “nurse-oscopes,” or “assistoscopes,” and were required to be smaller and flimsier.

Old and ineffective doesn’t always give way to new and better at once; it is slowed by habit as well as an unwillingness to relinquish control.

Somehow technology marches on. But it does so unevenly, sweeping some along in its first wave, others in its wake, some never at all.

Just as effective antibiotics to cure tuberculosis were available for 20 years while surgeons continued to remove patients’ lungs, so better solutions to heart disease are already available but not yet employed by your neighborhood physician. The primary care physician may have heard about some of the newest means to prevent heart disease, but is too overwhelmed with the day-to-day of
sore throats, diarrhea, and rashes. Cardiologists, intent on inserting the next best stent or defibrillator, have little but passing interest in strategies that might halt or reverse the heart disease that can be “managed,” no matter how imperfectly, with procedural solutions like angioplasty and bypass surgery. We should bear these flawed human tendencies in mind as we explore the world of heart disease prevention.

We need look no farther than the front page of the newspaper to find evidence of the failure of present-day heart disease detection and management. Over the past several years, headlines have carried the likes of Tim Russert, Bill Clinton, Larry King, Dick Cheney, David Letterman, Tommy Lasorda, Ed Bradley, Mike Ditka, Walter Cronkite, Alberto Salazar, all heart disease sufferers. Some, like talk show host David Letterman, survived their brush with heart catastrophe and underwent successful bypass surgery. Others, like marathoners Fiju and Salazar, raised none of the conventional red flags for heart disease. All received standard, “modern” medical care . . . all the way up to their heart attack, bypass surgery, or untimely death.

Like the sphygmanometer (blood pressure) cuffs of 1910, *Track Your Plaque* represents an example of the new. But, unlike the simple practice of taking blood pressure in the early 20th century, *Track Your Plaque* represents an entirely new way to look at coronary heart disease: a new way to measure it, a new way to identify its causes, and a new way to seize control over it, often to the point of achieving reversal of the process. It also puts control over much of this process into your hands and away from hospitals, cardiologists, and heart procedures.

Even though these concepts have been around for a few years, you’ve likely not heard about many of them. Just as physicians were still palpating pulses in 1920 and surgeons removing lungs for TB in 1960, so do present-day physicians still believe that only angioplasty, stents, and bypass surgery are the only options for control over coronary disease, or that cholesterol reduction is the only strategy for prevention.

I could speak of revealing “secrets,” but that’s not true. In *Track Your Plaque*, I simply convey information about heart disease that you were likely unaware existed, strategies that doctors fail to discuss. I assemble them into a “package” that, together, create an enormously empowering unique approach to prevent heart disease and heart attack.

*Track Your Plaque* also challenges the high-tech status quo, practices that occupy exalted places in the enormous cardiovascular healthcare machine that has dominated American healthcare for the past 40 years. I propose that high-tech hospital procedures should join the practice of ECT for homosexuality and insanity—and become yet another relic of the past.
Chapter 1

Taking the Guesswork Out of Heart Disease

Heart disease care today is all high-tech—except when it comes to predicting who will have it. Then it boils down to little more than an educated guess.

Guesswork will give way to better methods to detect it, followed by ways to track and control it. That’s what the Track Your Plaque program is all about.

It took my mother’s death to teach me about heart disease.

Up until 1995, I was consumed with performing the newest, cutting-edge angioplasty procedures, and I wanted to do as many of them as I could. It was an exciting time, ripe with new techniques, new devices, new concepts. Every day brought fresh challenges on how to cut, burn, drill, laser, and stent the coronary arteries of patients with heart attacks, unstable heart symptoms, diseased hearts.

My days typically began in the cardiac catheterization laboratory at 7 am, running late into the evening. In that early era of interventional cardiology, many of the rules had not yet been written. I excitedly followed the lead of angioplasty heroes, like Dr. Geoff Hartzler of Kansas City and Dr. Gary Reuben of the University of Alabama, both at the forefront, extending the leading edge of how far we could go in the Wild West world of coronary “intervention.” The satisfaction and challenges were intoxicating.

In late summer, 1995, I received a phone call telling me that my mother had died—sudden cardiac death at age 62. She’d had an angioplasty several months earlier at a reputable heart center in New Jersey. I had discussed my mother’s case with her cardiologist and had felt satisfied that he seemed fully in touch with her health issues.
It took several weeks for the irony of my mother’s death to sink in. Perhaps I didn’t want to see what my mother’s death could teach me: *What I did professionally as a cardiologist was fundamentally and terribly flawed.*

My mother’s death shattered my long-held conviction that I was delivering the best care to my patients. Years of training and practice, doing the same as thousands of my cardiology colleagues, but it took my mother’s death to help me see how misguided the conventional procedural approach to heart disease truly was.

I began to see that we tackled heart disease as “crisis management”: We waited for a crisis like heart attack to strike, then proceeded to “fix” it. We dealt daily in catastrophe but devoted little thought to why it happened in the first place. Managing crises also meant that many people never survived to take advantage of our help, like my unfortunate mother.

I therefore set out to find answers to several questions: How can coronary heart disease and risk for heart attack be identified *years* before catastrophe strikes? How can risk be reduced or eliminated with greater certainty than provided by crude measures like cholesterol? (My mother had been advised by her doctor that her cholesterol values were “fine.”) How can we measure and *prove* success? More recently, I’ve extended the challenge further: How can we achieve all this *with minimal or no medication* and achieve sufficiently confident results so that heart procedures are *no longer necessary*?

After several years of effort, I believe that a rational, effective, and scientifically-valid program is now available. I call it *Track Your Plaque.*

Once you read about these concepts, I believe that you will agree that the answers to my questions are really maddeningly simple. In fact, you may find that the answers to many of your questions about heart disease have been available all along, but you just didn’t know where to look. You may also be angered by your new appreciation for the misguided beliefs of people around you—perhaps including your doctor—who insist that heart attack is unpredictable, or inevitable, or that the only effective treatments are hospital procedures. Or that prevention of heart disease ends at following a “sensible” low-fat diet, statin cholesterol drugs, and exercise.

It’s all completely untrue.

*“Doctor, will I have a heart attack?”*  
You walk into your family doctor’s office.
Your neighbor, exactly your age, just died from a heart attack—no warning, good health beforehand. In fact, you chatted with him just days before while he took a break from push-mowing his half acre lawn. He looked fine. Understandably shaken, you want to know whether you have heart disease, though you, too, feel fine. You press your doctor for an answer.

How does he/she answer your question? More than likely, it would go something like this:

1) “Do you have chest pains or excessive breathlessness?” he/she begins. You respond that you have no symptoms.

2) An examination follows. Everything is normal, with no physical sign of heart disease.

3) You get an EKG. The doctor declares that you’ve not had a heart attack and your heart rhythm is normal.

4) Your doctor checks your cholesterol values. Your cholesterol values are somewhat high: total cholesterol, 230 mg/dl, and LDL cholesterol, 150 mg/dl.

5) A stress test is performed. It’s completely normal.

You leave the office after receiving advice on cutting the saturated fat in your diet and plans to have cholesterol tests repeated in several months to determine whether a statin cholesterol drug will be necessary. After the entire process, you’d likely be satisfied with your doctor’s thoroughness. You’re convinced that you have no hidden heart disease and won’t fill the cemetery plot next to your neighbor.

But that’s not what decades of clinical science and experience show.

Nothing that your doctor did reliably detects hidden heart disease. You could have extensive silent heart disease or you might have none. You could drop over suddenly watching your favorite reality TV show next week or you might outlive all your neighbors dancing the samba until your 95th birthday. For the great majority of us, none of the tests distinguish these two drastically different fates. True to the formula that has dominated medical practice for centuries, your doctor looked for extremes: a high cholesterol value, symptoms of heart disease, reduction in the heart’s blood flow by stress testing.

Your doctor may even recognize this enormous limitation, but continues to follow a testing menu that has changed little over the last 30 years. Somehow, science failed to trickle down to the mainstream physician, who still accepts as routine that some proportion of his/her patients will “inevitably” have heart attacks or die of heart disease every year despite routine screening efforts.

It boils down to this: The hapless family doctor, internist, or cardiologist looking for telltale signs, red flags, or black and white answers, usually fails to
find any, and instead ends up just hazarding an educated guess based on an antiquated list of risk factors and simple observations. Expecting your doctor to predict whether or not heart attack is in your future from the tests he/she has come to rely on is tantamount to hoping he/she is gifted with supernatural fortune telling abilities.

All too often, they guess wrong.

**Heart disease: A numbers game?**

Your doctor knows it. The drug industry knows it. Cardiologists know it. Now, you know it, too: Cholesterol testing is a deeply flawed approach to heart disease prevention.

There have been efforts to improve on prediction of heart disease by adding other measures to cholesterol testing. The most popular means of incorporating multiple risk measures is the Framingham risk score, a system that arose from observing the residents of Framingham, Massachusetts for evidence of heart disease, starting back in 1948.

The Framingham risk score is a risk-assessment tool that has become the basis for heart disease prediction now used by practicing physicians. The Framingham system determines that:

- 35% of adults in the U.S., or 70 million, are deemed “low-risk.” Low-risk is defined as the absence of standard risk factors for heart disease; low-risk persons have no more than a 1-in-20 chance (5%) of dying from heart disease in the next 10 years (Greenland 2001). The American Heart Association (AHA) advises physicians that no specific effort at risk reduction is necessary.
- 25%, or approximately 50 million, U.S. adults are deemed “high-risk,” based on the presence of 2 or more risk factors. High-risk persons experience 20-30% likelihood of heart attack in the next 10 years (Wilson 1998). People at high-risk are candidates for preventive efforts according to the guidelines set by the Adult Treatment Panel-3 (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults; ATP-III) for cholesterol-reducing statin drug treatment and for “lifestyle-modifying” advice.
- The remaining 40% of the adult population, or 80 million people, are judged “intermediate-risk,” with likelihood of heart attack between 5-20% over the next 10 years (Jacobson 2000). According to ATP-3, this group should receive preventive advice and might be considered for statin drug treatment.
Let’s do some arithmetic. By the above scheme, the low-risk population will experience up to 3,500,000 heart attacks over the next decade, or 350,000 heart attacks per year.

The intermediate-risk population will, without preventive treatment, experience 8,000,000 heart attacks over a 10-year time period, or 800,000 per year (if we take the middle-ground of 10% likelihood of heart attack).

The high-risk population, the group most likely to receive standard advice on diet, exercise, and be prescribed statin cholesterol drugs, will have risk reduced by 35% by preventive efforts over the 10-year period (Smith 1995). This means that heart attacks over 10 years will be reduced from 12,500,000 (once again, taking the middle-ground of 25% likelihood of heart attack) to 8,125,000 by standard prevention efforts, or 812,500 heart attacks per year.

This means that, following the standard approach, applying the widely accepted Framingham risk scoring system and engaging in preventive practices as advocated by the AHA and ATP-III, will permit “only” two million heart attacks this year. (It also assumes that physicians will actually comply with standard advice, including going through the motions of performing the Framingham risk calculation. In reality, they often do not.)

These frightening predictions do indeed play out in real life. The numbers of heart attacks, death from heart attacks, and hospitalizations for unstable heart symptoms, as reported by the AHA (AHA 2008 Update; http://circ.ahajournals.org/cgi/content/full/117/4/e25) correspond to the numbers provided by these predictions.

These numbers are no secret. They are well known facts that have come to be accepted by the medical community. In other words, the standard approach to heart attack prediction makes no mystery of the fact that two million people will succumb to cardiovascular events in the next year. This exercise in prediction is coldly accurate when applied to a large population.

The fundamental problem is that this approach cannot reliably distinguish which individuals will have a heart attack from those who will not. From 100 people chosen at random, for instance, the numbers game played above will not confidently identify who among those 100 will have a heart attack, who will not, who will develop anginal chest pains and end up with stents or bypass surgery, or who will die. We just know that some of them will. Some people at high risk will have a heart attack, some people at intermediate risk will have a heart attack, some people at low risk will have a heart attack. For any specific individual (like you or me), it’s essentially a crap shoot.
“I passed my stress test . . . So why did I have a heart attack?”

If standard risk calculation is so flawed, why not have everyone undergo another sort of screening test, such as a stress test?

Stress testing encounters similar limitations in predicting who will and who will not have a heart attack, especially if applied to a broad population without symptoms of heart disease. Analyses from the Framingham Heart Study, for instance, have shown that, while abnormal stress tests in people without symptoms suggest an increased long-term risk for heart attack, many future heart attacks occur in those with normal stress tests (over 18 years of observation; Balady 2004). The AHA, in fact, classifies stress tests as “Class IIb: Usefulness/efficacy is less well established by evidence/opinion” in people without symptoms of heart disease (Gibbons 2002).

Why don’t stress tests serve to uncover coronary heart disease in people without symptoms?

Stress tests are an effective means to detect areas of poor blood flow to the heart. So, for instance, if there is a 90% blockage in one artery, this can be reliably detected with a stress test. Stress tests usually detect blockages when they meet or exceed 70%—anything less than this is not detected (Lauer 2005). A person with dozens of lesser blockages of, say, 20%, 30%, 40%, or 50% will therefore not be detected by stress tests. For every one person with blockages severe enough to be detected by a stress test, there are dozens, if not hundreds, of people who fail to be detected by a stress test but harbor one or many lesser blockages.

The Uncertainty Factor

In truth, testing for heart disease today is plagued by a substantial uncertainty factor. The uncertainty factor is responsible for the many stories we’ve all heard: the neighbor who passed a stress test on Tuesday but died of a heart attack on Thursday; a grandmother with lifelong high cholesterol who never suffered a stitch of heart disease; the 44-year old slender, active athlete who died suddenly of a heart attack while jogging. None of these situations are predicted by your doctor’s search for extremes.

The practical reality is that 90% of future heart attacks will not be predicted by any of the conventional tests, a nine out of ten chance that the battery of testing you just submitted to failed to identify impending heart attack. The vast majority of doctors follow the sequence of testing described above, not much different than taking a roll of the dice with the odds heavily stacked against you 10 to 1. All too frequently, false reassurances are provided that hidden heart disease is not present, or that there is a cholesterol issue present, or some other fuzzy prediction.
Alternatively, what if a suspicious abnormality is uncovered, such as an abnormally reduced area of blood flow on a stress test? Then comes a visit to the cardiologist, followed by a heart catheterization, an angiogram of the heart performed with insertion of hollow-tubed catheters into the body. While 80-90% of people end up with stent(s) or bypass surgery, the other 10-20% of the time the catheterization reveals no coronary disease. This sequence plays out thousands of times each day in the U.S.

The curious discrepancy is that, while heart procedures like implanting stents and performing bypass surgery are unquestionably good for relief of symptoms like chest pain and breathlessness, they have never been shown to be superior to a program of prevention in people without symptoms. Major heart procedures, regardless of how well they are done, no matter how skilled or experienced the cardiologist or thoracic surgeon, have never been demonstrated to reduce the risk of future heart attack or prolong life in people who begin the process without symptoms.

The dispute is not so much over the value of heart procedures per se, but their value in people without symptoms who are thought to be at risk.

There is even doubt over how beneficial major heart procedures are in people with symptoms of heart disease. In the recently published COURAGE Trial, 1100 participants symptomatic with angina (chest pains) who underwent angioplasty and stent placement were compared with 1100 symptomatic participants who received a basic (what the authors—laughingly—called “optimal”) regimen of medication (statin cholesterol drug, aspirin, blood pressure medication) over a period of five years (Boden 2007). The group undergoing procedures experienced no advantage in terms of heart attack or death, though a proportion of people taking medication only did “crossover” to require a procedure to relieve progressive symptoms. (You will learn, as you hear more about the Track Your Plaque program, how anemic this so-called “optimal” medical therapy truly is.)

Uncertainty on the one hand, certainty addressed with procedures on the other. I think it’s time to change our entire way of thinking.

**Acute cardiovascular care: the $400 billion burden**

American cardiovascular care consumes $400 billion a year.

How much is $400 billion? It equals the amount of money spent during the first three years of the Iraq war. It matches the 2005 U.S. Department of Defense budget, or the national deficit in that same year. It dwarfs the $210 billion cancer care industry. It represents nearly $10 million dollars spent every hour, 24 hours a day, 7 days a week.
Healthcare expenditures absorb 20% of the U.S. economy, equal to that of the entire U.S. manufacturing sector (Center for Healthcare Statistics). Per capita spending on healthcare is $6,280 per year, double that of other modern nations, including England, Finland, Canada, Belgium, Germany and Australia (Organization for Economic Co-operation and Development, 2006).

Add to these staggering costs the revenues generated by thousands of companies that provide the props for the system—manufacturers of stents, defibrillators, operating room supplies for cardiac bypass surgery, drug makers who furnish intravenous medications for medical procedures (often costing hundreds to thousands of dollars per dose) and so on—and you have another mind-boggling sum that also runs well into the hundreds of billions of dollars.

To help us get our arms around some of these unimaginable sums, let’s just suppose that 20–30% of all heart-related procedures in the U.S. were found to be unnecessary. If the total bill for cardiovascular disease in the country is $400 billion, eliminating just 20–30% could save between $80 and $120 billion each year.

That “marginal” savings exceeds the sum the U.S. spends on the domestic war on terror. It’s more than double the combined annual budgets of the FDA, DEA, National Institutes of Health, and FBI. It would put $460 into the pocket of every man, woman, and child in the U.S. every year.

What is this thing we’ve created? Is $400 billion and all the effort really necessary? With Medicare careening towards fiscal crisis and annual health insurance premiums skyrocketing into the five-figure range for a healthy family, how much longer can we afford it?

Procedures and drugs taking aim at heart disease appear to be getting better and better—yet the cardiovascular healthcare system grows bigger, generating more procedures, more expensive pharmaceutical agents, costly new devices. A day doesn’t go by that most of us aren’t assaulted with several TV ads, radio spots, billboards, and news reports of hospital heart care.

From the nurse at the bedside, to hospital systems performing hundreds or thousands of bypass operations per year, on down to the technological infrastructure that supports these services, the cardiovascular healthcare system is immense. In fact, it is unprecedented in human history. Never before has so much manpower and money backed up a system of “health.”

In the inevitable swing of the pendulum of human endeavor, American’s addiction to acute healthcare technology has swung too far towards an unsustainable preference for high-tech, high-cost procedures over preventing the disease in the first place. I offer the Track Your Plaque program as one means to
not only more effectively prevent heart disease, but also help slash the huge burden of healthcare costs.
The dangers of a normal stress test

Coronary disease is the number one cause of death in America, yet most physicians fail to effectively screen a seemingly well person for hidden heart disease. Here is Neal’s story:

Neal, an electrician, was in the midst of a large heart attack that was going to obliterate 50% of his heart muscle. His family physician had performed a stress thallium (a nuclear imaging stress test) one month earlier when Neal had felt perfectly fine. The stress test was normal: no chest pain, no EKG abnormalities, thallium images of coronary blood flow were normal. Neal was advised by his doctor that his heart was in great shape and there was no risk for heart attack in the foreseeable future.

Three weeks later, Neal was lying on a hospital gurney, barely able to talk because of crushing pain in his chest. He answered my questions with few words as possible. He was terrified and bewildered. How could this be happening? We got Neal through this near-death crisis and salvaged most of his heart muscle with an emergency coronary angioplasty and several stents. After he recovered, Neal asked the obvious question: “Why did I have a heart attack? My doctor said my stress test was fine! He said my heart was in perfect shape! Was the stress test wrong?”

The stress test was not wrong. I reviewed the stress test and it was, indeed, completely normal. The problem was that it was the wrong test. Contrary to popular opinion, including that held by many physicians, stress testing is not an effective means of screening people without symptoms for the presence of coronary heart disease. This is such an important issue that I will repeat it: In the great majority of people, stress testing is not an effective method of uncovering hidden heart disease.

Are stress tests worthless?

In truth, stress tests can be useful diagnostic tools, but only when used appropriately. People who go to the hospital with symptoms, particularly chest pain, can benefit by undergoing a stress test to reproduce symptoms. The physician needs to distinguish an impending heart attack from the pain of stomach ulcer, pleurisy (inflammation of the lining of the lungs from previous pneumonia), esophagitis (inflammation of the esophagus), gallstones, etc. If chest pain is provoked by walking on the treadmill during a stress test, this is suspicious for heart disease. The treadmill test (or a pharmacological equivalent) is often combined with a method of imaging blood flow to the heart muscle such as thallium, or methods to image heart muscle strength such as echocardiography (ultrasound). If there is poor blood flow to a specific segment of the heart’s muscle, then a blockage in a coronary artery is likely present and your chest pain may represent warning to a future heart attack.

Using a stress test to detect hidden coronary plaque in someone without symptoms is unlikely to uncover anything, since the majority of people without symptoms have normal blood flow to the heart. The majority of future heart attacks victims are walking around feeling just fine, yet have silent plaque in their coronary arteries. Heart attacks in these people are caused by sudden rupture of a minor plaque, a plaque that may be causing only 20, 30, or 40% blockage, does not block blood flow, and is therefore undetectable by any stress test. Plaque rupture, like a little volcano erupting along the length of the artery, is a process that develops within minutes. Stress testing months, weeks, or days beforehand will not anticipate this event. (We will be discussing the concept of plaque rupture at length later.)

What we really want to know is how much plaque is present in a well-appearing person.

To be truly successful at prevention of heart disease, plaque should be detected early. Wait for symptoms to appear and it might be too late; catastrophe may even be just around the corner. In the Track Your Plaque approach, we do not wait for a stress test to show abnormalities, since only later phases of disease are detectable this way. In Neal’s case, had his coronary plaque been identified before his heart attack, appropriate and powerful preventive action could have been taken. Neal’s near-death episode could have been avoided.
Keeping score on heart disease

If assessment of risk factors is ineffective and allows heart attacks to slip by and stress tests are a failure, then is there a way to uncover early coronary disease in people who feel well?

There is a way to detect early coronary heart disease that can confer something close to fortune-telling abilities to you and your doctor: a heart scan.

A heart scan is an inexpensive and precise test that uncovers silent coronary disease: coronary atherosclerotic “plaque.” (Atherosclerotic plaque is the abnormal material that accumulates in arteries, the focus for stents and other procedures to re-route blood flow around plaque.) Unlike the vagaries of cholesterol testing and Framingham scores, uncovering hidden heart disease becomes a simple matter. But, like blood pressures to the physician of 1910 more comfortable with “pulse palpation,” the wonderfully straightforward approach of scanning for coronary plaque has simply not worked its way into the day-to-day thinking of most practicing physicians.

A heart scan is simply a test performed in one of the newer versions of computed tomography (CT) scan devices able to acquire images of a rapidly-moving heart in constant motion. Imaging the heart, beating at 60 or so times per minute, requires technology that can “freeze” images in a split-second. This engineering feat has only been accomplished in the last few years.

Within just over a decade, CT heart scans have become available to virtually every American near a hospital with a newer-generation CT scan device. It requires all of 30 seconds with radiation exposure equivalent to that of a mammogram or two (the equivalent of 4 to 10 standard chest x-rays). In most states, heart scans are obtainable just by scheduling an appointment: no doctor’s order, no preparation. Cost nationwide averages $200, with cost dropping as hospitals and scan centers compete and try to seize “market share” over the people who have heart disease.

Heart scans provide a “score,” an index of the amount of coronary atherosclerotic plaque present. Just like the score in a basketball game, you can have low scores, intermediate scores, and high scores. A score of zero signifies no plaque; any score greater than zero indicates increasing degrees of atherosclerotic plaque lining the coronary arteries, up to scores in the thousands.

Heart scans are painless and precise. Plaque is detectable—and quantifiable—years, even decades, before symptoms are present, long before it is detectable by stress testing. Plaque can be detected in people with low cholesterol, high cholesterol, diabetics and non-diabetics, people with risk factors or without risk factors, in smokers as well as non-smokers, people with red hair or brown hair, tall people or short people . . . you get the idea.
Heart scans don’t actually measure plaque directly, but measure the calcium content of atherosclerotic plaque. Calcium serves as a gauge for plaque. This important insight was discovered by the Mayo Clinic’s Dr. John Rumberger in 1995, when he and his team demonstrated through autopsy specimens that coronary calcium consistently occupies 20% of total plaque volume in men, women, young, old, people with heart disease and those without. Thus, 2 mm³ of calcium corresponds to 10 mm³ of total plaque (We will explore this phenomenon further.) Subsequent studies have validated this measure and demonstrate that the quantity of coronary plaque can be accurately measured using rapid CT scanners in living, breathing humans.

Plaque is not a risk factor for a disease. It is a measure of the disease itself: coronary atherosclerosis. That’s why it serves so well as a crystal ball for the future, superior to measures of risk.

What is Track Your Plaque?

The Track Your Plaque program answers the questions: If atherosclerotic plaque in coronary arteries can be imaged and measured, can it be tracked over time? Can it be reduced?

You and I can double or triple the quantity of coronary plaque in our arteries in a year’s time while feeling fine, eating a low-fat or low-carbohydrate diet, or following an extreme fitness program. Precisely measure plaque, and you can gauge with confidence whether disease has progressed or not. As coronary plaque grows, the danger of heart attack and death grow with it. Conversely, as coronary plaque shrinks, so do risk for heart attack and death.

Track Your Plaque is not just a new diet that, by eliminating or adding certain foods, boasts of a “cure” for heart disease. Let’s face it: eating more broccoli or less red meat will not “cure” heart disease. Nutrition is important, but no matter how good your diet, it does not cure the genetic causes of heart disease. It is only a part of the final answer.

While the Track Your Plaque program begins by quantifying atherosclerotic plaque with a heart scan, the subsequent steps are what really make the program a success. Once plaque is identified, what can be done to put a stop to it? Surprisingly, many of the answers have been available for years, answers based on hard scientific developments but were never applied in this direction.

Using the framework of what works, what doesn’t in gaining control over heart scan scores, the Track Your Plaque program assembles unique strategies that provide maximum advantage. It is a program that employs nutritional strategies and nutritional supplements targeted to specific causes of coronary plaque with a minimum of medication. While success or failure is ultimately
based on the results of a second heart scan, usually obtained one year after the start of the program, feeling better, having more energy, working and thinking more effectively, all develop almost immediately, within the first few days to weeks. Conventional cholesterol values generally improve to levels far beyond that usually achieved—with little or no medication. Many of you will be interested to know that weight loss of 25, 30, or 40 lbs are typical in the first several months of the Track Your Plaque program. These sorts of results that far exceed the meager gains made through standard approaches is the reason why the Track Your Plaque approach can accomplish what other programs have tried to do but failed.

Track Your Plaque is an information resource on how to apply this new approach. You won’t have to wait for life-threatening symptoms, nor rely on cholesterol numbers. You won’t have to depend on some intuitive feeling that you might have a heart attack. Tracking your plaque tells you whether you have coronary plaque, how much, what the future holds. It provides feedback on the results of your efforts. It is a real world method of managing heart disease risk that anybody can follow.

The Track Your Plaque 3-step program

Track Your Plaque can show you, in three basic steps, how to identify and seize control of heart disease in your life.

In Step 1 of this proven approach, you’ll be shown how to detect hidden coronary heart disease years before danger strikes. The most widely available method to detect silent heart disease is coronary calcium “scoring” obtained through the increasingly available computed tomography (CT) heart scanners. Direct measurement of hidden coronary plaque eliminates the murkiness of “risk factors” like cholesterol testing, and the unacceptable imprecision of stress testing. These new technologies make the identification and precise measurement of coronary plaque a safe, inexpensive, 10-minute process that just about anybody can obtain.

In Step 2, you’ll be shown how to uncover the causes of heart disease. Among the techniques used is the powerful technology of lipoprotein analysis. This exciting technology easily pinpoints the causes of heart disease even when cholesterol testing fails. Time and again, people who’ve survived heart attacks are told that no cause for their heart disease could be found. Yet when lipoproteins are tested, the causes seem almost obvious. Step 2 will also introduce the important concepts of vitamin D and thyroid normalization, two of the newer additions to the Track Your Plaque program. Having greater insight into the causes for heart disease—well before heart attack—arms you with better tools to control coronary plaque.
Once your coronary plaque is scored (Step 1), and its causes pinpointed (Step 2), you can then begin Step 3, applying effective therapies that can, with proper guidance, slow, stop, or reverse coronary plaque and thereby reduce or eliminate the prospect of heart attack or major cardiac procedures in your lifetime. This includes a discussion of the two absolutely crucial nutritional supplements for your plaque-control program, vitamin D and omega-3 fatty acids from fish oil.

*Track Your Plaque* is therefore a three-step program that shows you how to:

1) Detect and *measure* coronary plaque  
2) Identify the *causes* of your coronary plaque  
3) Effectively *treat the causes* in order to arrest or reduce the amount of plaque you have.

If we can now measure coronary plaque, identify its causes, then correct them, the *Track Your Plaque* approach has the potential to crush the current *crisis management* approach of modern cardiovascular care.

**Is *Track Your Plaque* right for you?**

*Track Your Plaque* is primarily designed for people before heart attack and major heart procedures. Part of the reason for this is that the preventive efforts we will discuss can take months to years to take effect. A person who develops progressive or unstable symptoms of heart disease (rapidly progressing to a full heart attack) lacks sufficient time to take full advantage of these principles. As powerful as *Track Your Plaque* treatments are, they do not halt progression to heart attack once this process is near completion. If you are having symptoms of chest or arm pain, or breathlessness, you may not be ready for this program. You may need the testing facilities provided by your doctor or hospital first, before you can safely consider these strategies.

Or, perhaps you’re the kind of person who requires the reassurance of a hospital, people in scrubs and masks, and glitzy marketing. *Track Your Plaque* might not provide you with the kind of support you require. I may be critical of hospital marketing, but I do not underestimate its power to influence people. Some people just need this sort of “legitimizing” presentation of their healthcare, and independent, self-empowering practices may not suit you. If you think that this may apply to you, I’d still encourage you to read on to acquire a healthy dose of skepticism.

On the other hand, the earlier you start your program, the more power you will have over your future. Preventing heart disease is a lot like saving for retirement. If you start at age 35, saving a little at a time will yield a comfortable nest egg. Start at age 60, and you’ve got to scramble to reach the same goal, or
it might not be possible at all. The same holds true for inhibiting progression of coronary plaque.

If you’re a smoker, this book is not for you, at least not until you quit. No program will fully overpower the negative effects of smoking. I’ve seen people who did everything else right yet continue to smoke, and the quantity of coronary plaque doubles in a year’s time. Smoking, even just a few cigarettes a day, so overwhelms all your positive efforts that growth of coronary plaque is inevitable. You’ve simply got to stop smoking to gain control of your plaque.

If you’re looking for a quick answer—a quick diet, nutritional supplement, prescription medicine—that “cures” you of heart disease and then you’re done, you will not find your answer here. People with this attitude often don’t take their risk for heart disease seriously. They don’t participate in the learning process, are unwilling to make lifestyle changes that may be necessary, and will be disappointed.

On the other hand, people who succeed in Track Your Plaque tend to be motivated by learning and searching for self-empowering methods to preserve health. They recognize that health does not come from a single pill or diet, but grows from a combination of healthy habits. They understand that heart disease is a complex process with causes that vary from individual to individual, and that treatment and prevention also need to be individualized. This program appeals to people who are not content following in the footsteps of their parents or other family members with heart disease, don’t accept the inevitability of heart disease, and are willing to invest time and energy to create their own heart disease-free future.

Tracking plaque, while effective and not all that difficult, cannot be distilled down to a single diet or pill. It is a comprehensive program that will require some effort, including a search for local resources in your area to assemble your own program. But the rewards can be great: prevention of a dangerous heart attack and avoidance of major heart procedures, potentially for a lifetime. Once you see the details and experience the results, I believe that you will agree that this is a far more rational way to approach the terribly common specter of heart disease.

Track Your Plaque is a program for life. After you’ve begun your own Track Your Plaque program, going back to old habits and hoping that the initial benefits will hold, will result in failure. Old habits will revive quiescent plaque and reactivate the lipid/lipoprotein and genetic patterns that created plaque in the first place. You’ll be right back where you started. Track Your Plaque is therefore not a one time “quick-fix,” but a lifelong program for health.

We have experienced a confluence of technological development that now makes early heart disease detection and prevention a practical reality. Despite the alarming epidemic of heart disease and its catastrophic consequences, the
concept of early heart disease detection has not yet reached mainstream medical practice. The history of medicine is full of instances in which new technologies and new ways of viewing a disease fail to gain traction in the mainstream medical community for years or decades.

*Track Your Plaque* has made the leap, assembling new technologies into a powerful program of heart disease prevention and reversal.
References:


Chapter 2

Your life is worth $112,377*

*Average cost of coronary bypass surgery
(American Heart Association, 2010)

Modern heart care is dominated by high-cost procedures that generate revenues for hospitals and practitioners. But it doesn't have to be that way.

What has gone wrong with modern health care for the heart?

While understanding of nutrition, nutritional supplements, and tracking of disease has progressed enormously in the past two decades, conventional cardiac care continues to follow a formula that was written somewhere around 1980.

Neglect, ignorance, laziness, profiteering—call it what you will, but the result is little different than doctors of 1955 removing lungs from patients with tuberculosis while effective antibiotics were already available.

Modern cardiac care is a shining example of what high-tech hospitals and industry can accomplish when there is promise of a rich payoff. Most hospitals and cardiologists do a very good job delivering heart catheterizations, angioplasty, stent implantation, coronary bypass surgery and all the other high-tech procedures now possible.

But lost among hospital TV ads and highway billboards for high-tech heart care are the wonderful tools that are already available to all of us, tools that can potentially make the $100 million new local hospital addition for heart care . . . irrelevant.
The hospital revolving door
Ray L. was no newcomer to the hospital.

Ray survived his first heart attack 15 years ago. It ruined a Caribbean vacation and forced his early retirement at age 57. Since then, Ray has endured two heart bypass procedures, carotid surgery (endarterectomy), five heart catheterizations, nine hospitalizations, and has “graduated” from cardiac rehabilitation classes three times. Every time Ray goes to the hospital, he fears he’ll end up with another heart catheterization, if not another bypass.

Most recently, Ray was admitted to evaluate breathlessness experienced while mowing his lawn. A heart catheterization was being planned, no doubt. A review of Ray’s medical history revealed that he consistently receives prompt, expert attention—Ray has never complained about the quality of his hospital care. What is surprising about his record is what is missing: Here was a 72-year old man who’d survived major and sometimes life-threatening events and procedures, yet not one comment had been made in his charts by his physicians about why Ray had such aggressive vascular disease. Not one. The need for more procedures went unquestioned, yet virtually no thought was given to finding out the “why” of his disease. Ray’s disease ended his productive work life, drastically impacted his personal life, and resigned him to a future of hospitals and procedures. Yet no effort had been made to understand the causes behind his disease or to slow the process.

In the world of modern cardiac care, Ray’s story is commonplace. Once you enter the hospital’s cardiovascular system, the revolving door of care begins. Twenty five percent of all bypass surgeries performed in the U.S. are so-called “re-do” procedures, or bypass surgery for people who have undergone a prior bypass operation (ACC/AHA Guideline Update for Coronary Bypass Graft Surgery, 2004). The path for the future is inexorably established with more procedures, more hospitalizations.

Why should we be surprised? If little to no effort is spent to identify the original cause of heart disease, no effort made to correct the causes, why would the disease stop?

What incentive is there to put a stop to heart disease if the payoff for each episode is so generous?

“Coronary disease is a deficiency of polyethylene”
The early 1990s were the Wild West days of coronary balloon angioplasty. We were tantalized by our ability to “open” blockages in coronary arteries by
inflating small balloons (coronary angioplasty), a new and exciting concept at the time.

In 1990, I attended a conference on new techniques in angioplasty. One of the main speakers was a flamboyant cardiologist from Australia, a Crocodile Dundee of cardiology. He joked that, because coronary blockages were so readily opened with balloon dilatation, coronary disease could be viewed as a “deficiency” of 16 mg of polyethylene (referring to the plastic the balloons are made of.)

Even today, this attitude remains the practicing philosophy of many physicians. Though polyethylene plastic has been largely replaced by stainless steel (stents), heart disease is viewed as a procedural disease: Best managed with hospital procedures like angioplasty and bypass surgery—period.

I have had thousands of patients come to me because the only solution offered by their cardiologist was to perform a heart catheterization to determine the “need” for angioplasty, stents, and bypass surgery. If you turn out not to “need” a procedure, you are consigned to “medical therapy,” which means medicines like nitroglycerin, beta-blockers, and statin cholesterol drugs.

Talk to your neighborhood cardiologist and you will find that reversal of heart disease is not even part of his or her vocabulary. The idea is completely foreign because of their exclusive focus on ballooning, stenting, atherectomizing (cutting out) plaque through invasive procedures, or sending you to the operating room for a bypass procedure.

The hospital with the most bypass surgeries wins!

We live in an age when hospitals measure success by the number of coronary bypass surgeries they perform. Incredibly, it is still easier to get a bypass operation than it is to get good information on heart disease prevention. In the city where I live (Milwaukee, Wisconsin), there are billboards on the highways advertising bypass surgery. Selling bypass surgery nowadays is not that different from selling cars or furniture. It is a profit-driven enterprise whose success is measured by the volume of procedures performed.

When I travel, I am amazed by the uniformity with which this message is broadcast by television, newspaper, and magazine ads. In city after city, from New York to San Francisco, there are ads boasting of the strengths of one program or another. Physicians are often featured in “interviews” urging patients to come to their hospital for heart procedures.

Have you seen the TV commercials produced by hospitals? A man stumbles into the emergency room, clutching his chest. He’s having a heart attack. Hospital staff in scrubs and masks pounce on the helpless patient. The
camera pans to the tense eyes of the patient as he bids a tearful goodbye to his wife. The next scene shifts to the patient now beaming, congratulated by family and staff, and being wheeled out of the hospital after his successful coronary bypass operation.

Scenes like this are broadcast every day by hospitals, eager to showcase their high technology and track record of major cardiac procedures. Hundreds of millions of dollars are spent annually to get these messages to you.

That’s how they ensure a continuing stream of revenue from this huge industry called cardiac care.

**Cardiac care is big business**

As a nation, we spend $177 billion on cardiovascular care for coronary artery disease per year (American Heart Association, 2010). Annual hospital revenues for bypass surgery nationwide total in the neighborhood of $45 billion. For most major hospitals, 30% of revenues and 50% of profits are from cardiac care. Heart disease and high-tech heart care to a hospital is like the Accord is to Honda, or Windows is to Microsoft—it’s their big seller, a principal source of revenue.

The average charge for a cardiac bypass operation in the U.S. is $112,377, a stent implantation procedure just under half that figure (American Heart Association 2010). Just 10 bypass operations and your hospital has charged over $1 million; 100 bypass procedures yields over $10 million. There’s no mystery here; it’s just simple math.

There are substantial incentives for doctors to push patients through the cardiovascular procedure machine. There is, of course, the direct incentive provided by generous insurance reimbursement to physicians for performing heart procedures. Every patient can mean thousands of dollars in income.

Less obvious are the indirect incentives provided to physicians who perform the most hospital procedures: access to equipment and staff, being featured in hospital marketing to achieve local notoriety, invitations to participate in decision-making committees, being wined and dined by medical device manufacturers, etc. High-volume practitioners are often paid $10s or 100s of thousands by hospitals for their referrals, often disguised as a fee to perform some administrative or consultative “service.” Such arrangements are more common than you think.

More recently, there has been a nationwide trend to hire physicians as hospital employees and compensate them on the basis of the revenue they produce for the hospital. For procedural physicians like cardiologists, the more procedures they perform, the more they get paid. For primary care physicians,
also employed by the system, sending more of their patients for consultation with cardiologists and other specialists likewise generates larger revenue stream, which often determines their financial compensation.

What results is a widespread, systematic collusion to maintain high-profit hospital business and grow market share.

If you were a hospital administrator, you would be under pressure to sustain and build this engine of growth. Your job would be to capture more and more of this market. You spend more on flashy TV ads, billboards, and radio; try to win the allegiance of busy doctors or, even better, hire doctors as employees, then ensure financial incentives to build procedural revenue; and disparage your competitors.

I have sat through many meetings attended by hospital administrators, cardiology and cardiothoracic surgery colleagues, and outside consultants, all to plan strategies for growth in cardiac care revenues. The questions are always the same: “How do we grow our heart program? How do we increase the number of heart catheterizations, coronary angioplasties, and coronary bypass surgeries in our hospital? How do we beat our competition?”

Hospital staff are the unwitting participants in this deception. When I discuss Track Your Plaque concepts with hospital nurses, technologists, and administrators, they are completely unaware of alternatives to acute hospital care. Many find these ideas personally appealing, but the incentives of the hospital are too hard to resist. After all, they’ve spent years training to do their jobs and salaries are paid by the hospitals providing these services.

Prevent heart disease? That’s just not what hospitals do.

Let’s face it: The days of charitable hospitals devoted to the public good with no interest in profit are long over. The health care industry, cardiovascular care in particular, is a profit-driven enterprise that uses all the tools of the business world—marketing, media relations, cultivating its “customers” (physicians), growing their volume of “product” (patients), and increasing market share.

It doesn’t have to be this way for you. Given the proper tools, you can control your future. As slick as hospital marketing can be, as smooth as the process of bypass surgery is made to look, there are alternatives—powerful alternatives. If you are destined to have a heart attack in your future, the means to identify that potential can be identified today, easily and painlessly. The therapeutic tools that prevent heart attack are also available right now. You just have to know where to look.

Track your Plaque can tell you where.
**Going against the grain**

Following the *Track Your Plaque* principles is not that difficult. But you may have a tough job ahead countering the skepticism of those around you. If you follow the principles in *Track Your Plaque*, you will begin thinking about how heart disease can be identified and measured, arrested, even reversed in a world driven by revenue-generating hospital procedures.

You may encounter skepticism, even opposition, from your doctor when you discuss *Track Your Plaque* concepts. Should you talk to a cardiologist, he/she will more than likely scoff at the idea that coronary plaque can be controlled and regressed.

When I first set out to persuade my colleagues that there was a better way to approach coronary disease, I got the cold shoulder. After all, I was essentially proposing to “turn-off” coronary disease. Coronary procedures like heart catheterizations and angioplasties fill most of the day for a cardiologist. I encountered disbelief that coronary disease could ever be controlled with something less mechanical than a polyethylene balloon or metal stent. Many also pointed out (usually in whispers) that there was simply no money in prevention.

I pled my case to several hospitals in the area and asked them to consider supporting a program to help disseminate the ideas advocated in *Track Your Plaque*. Once again, I encountered glazed eyes and puzzled looks on the part of hospital administrators and medical leadership. The concept of prevention, I learned, is not one likely to be adopted by hospitals. They are, after all, in the business of sickness, not wellness. Most hospitals have programs in “cardiac rehabilitation” that provide counseling and supervised exercise for patients after bypass surgery or heart attacks. That’s as far as it goes.

I continue to hope that hospitals will eventually embrace the idea of a comprehensive approach that addresses heart disease in the decades before procedures are required, but you are unlikely to get this kind of information from them today. If it doesn’t contribute to their bottom line, then it just doesn’t make sense for them.

**A heart attack is the failure of prevention**

There are times when high-tech, invasive care provided in hospitals is necessary. Centers that handle high-volumes of these sorts of patients generally do it well and provide excellent service. Many lives have been saved and understanding of heart disease advanced. These services are often provided by capable, well-trained nurses and physicians, most of whom truly care about their patients and are conscientious about the quality of their work. If you go to the hospital with unstable chest pain or an ongoing heart attack, it would be crazy to
talk about detection of early disease and reversal of plaque at that point. You need the services of a well-equipped, well-staffed hospital—no doubt about it.

But times have changed. It is time to reject the notion of heart disease as something that inevitably results in dangerous events treated with hospital procedures. We should think of every heart attack, every angioplasty, every bypass surgery as a failure to identify these potential catastrophes.

If heart disease requires decades to develop and if methods to detect it are already available, can’t we terminate the process years before trouble starts? Imagine the homes in your neighborhood were destroyed by fires day after day, wouldn’t you demand to know why preventive efforts were not instituted? Isn’t it better to prevent fires than to extinguish a blaze once it’s enveloping the entire house? The same holds true for your heart.

The ideal time to begin thinking about this approach is during the years before catastrophe strikes, while you’re feeling well. Rather than being afraid of heart disease, you should feel empowered to tackle it.

**Heart disease “reversal” is not new**

The concept of reversing, or shrinking, coronary plaque is not new. For years, people have wondered whether plaque can be reduced. Patients often ask, “Don’t you have any Drano® for my arteries?”

Early efforts at plaque regression date back to the 1980’s when the techniques for both measurement of plaque and treatment were primitive and results modest. Back then, clinical trials, such as those conducted by Dr. David Blankenhorn at the University of Southern California, required coronary angiograms (obtained via heart catheterization) to assess the extent of plaque. The treatments used included several medications no longer in use (Blankenhorn 1994). Remarkably, some patients in these early trials did indeed obtain modest regression of their plaque. But these efforts lacked two crucial ingredients: Precise methods to measure plaque and effective methods to control it. (We will discuss why heart catheterization is a flawed measure of plaque later on.)

Results that are now possible are far superior to early efforts because we now have the ability to precisely measure and track plaque, and the “tools” to reduce plaque are more effective—and they’re getting better every day.

**Didn’t Dr. Dean Ornish already write a book about this?**

Since the publication of his bestseller in 1990, “Dr. Dean Ornish’s Program for Reversing Heart Disease,” Dr. Ornish has appeared on television, spoken widely about his experiences to both lay and professional audiences, and has been tireless in discussing his philosophy of how dietary fat causes heart disease.
and how a strict low-fat diet can help control it. Dr. Ornish deserves great credit for popularizing the notion that diet, exercise, and relaxation strategies (specifically meditation and support group interactions) can have positive impact on coronary heart disease. More recently, Dr. Caldwell Esselstyn, an ear, nose, and throat specialist at the Cleveland Clinic, published a book titled *Prevent and Reverse Heart Disease* advocating a similar strict low-fat diet approach.

Drs. Ornish and Esselstyn advocate a program that consists of a vegetarian diet with 8-10% of calories from fat (compared to 40-55% in the average American diet), and permits no caffeine or alcohol. Critics have argued that few people can adhere to such a program for any length of time, let alone a lifetime. But Drs. Ornish and Esselstyn managed to show that, with counseling and supervision, people could indeed stick to it. In his published data, presented as "The Lifestyle Heart Trial" in 1990, Dr. Ornish reported in *The New England Journal of Medicine* how 28 participants managed to reduce the severity of blockage in their coronary arteries. He proved this by having participants, all of whom had advanced coronary disease (at least moderate blockages in two or three coronary arteries) undergo a heart catheterization at the start of the program and then again after five years in the program. The participants reduced the severity of their heart artery blockages from 40.0% to 37.8% (measured by averaging the percent blockage, or "stenosis," along the length of the arteries), compared to a “control” group (people with comparable amounts of coronary disease at the start but not enrolled in Ornish’s program) whose blockages worsened from 42.7% to 46.1% over the same five-year period (Ornish 1998).

What is remarkable about Dr. Ornish’s experience is that the results were achieved with lifestyle changes alone. People eliminated red meats, pizza, greasy fried foods, and sweets, and were counseled on how to eat creatively using only vegetables, fruits and whole grains. No medications were included, nor were supplements used.

Given Dr. Ornish’s public success and notoriety, now echoed by Dr. Esselstyn’s experiences, isn’t that the end of the story?

Not even close. Dr. Ornish is a pioneer in the use of lifestyle modification to reduce heart disease risk. But this approach falls far short of eliminating the dangers of heart disease. Of the 28 participants in the Lifestyle Heart Trial, for example, two participants required bypass surgery, two had heart attacks, eight had angioplasty, two died of heart attack, and 23 were hospitalized over the course of the five year trial. Though this represented about half the number of “events” that occurred in the control group, that’s still an awful lot of trouble. We need to improve on this track record.

In the early years of my practice, I recommended an Ornish-type diet program to many of my patients. Despite its austerity, hundreds of patients enthusiastically followed the guidelines and liked the idea of being in personal
control of their heart disease future. But as I applied more sophisticated measures, I observed that only about 20-30% of my patients demonstrated a truly favorable response. An ultra-strict low-fat diet did indeed lower LDL (“bad”) cholesterol in most (but not all) people, but there were undesirable distortions of numerous other measures, especially reduced HDL cholesterol, increased triglycerides, worsening of small LDL, increased VLDL—“lipoprotein” measures to be discussed in future chapters of *Track Your Plaque*. (My own personal experience on an Ornish-type strict low-fat diet was similar: My HDL dropped to 27 mg/dl, triglycerides skyrocketed to 350 mg/dl, I gained 30 lbs, and I briefly became a type II diabetic—while jogging 5 miles a day!)

When heart scan “scores” were used to track the course of plaque growth, the majority of people’s heart disease worsened. Heart attacks and the need for heart procedures like heart catheterizations, coronary angioplasty, and bypass surgery still occurred despite the diet.

I applaud Dr. Ornish for his efforts, but I believe we’ve come a long way from his crude preliminary experiences. In *Track Your Plaque*, we will apply new technology and new insights. We broaden our choice of therapeutic tools and heighten our chances of success. Not only do I believe that results will be significantly better, but part of this approach is to track progress with precise measurement of the disease.

The tools to identify heart disease and alter its course to prevent cardiac catastrophes are available today. Let’s go on. In chapter 3, we discuss how coronary plaque develops and how we can use this knowledge to easily and readily measure it.
References:


Chapter 3

What your doctor didn’t tell you about heart disease

Why measuring plaque is the most important health test you can get

Plaque is heart disease. That simple realization alone can help make things a lot clearer.

Plaque is not a risk factor for coronary heart disease. It is not a crude index of lifestyle habits, body weight, smoking, or cholesterol. Plaque is the disease of atherosclerosis: atherosclerotic plaque. When we measure plaque, we are measuring the disease itself. When we track its progression (growth) or regression (reversal), we are tracking the course of the disease itself.

The first step of Track Your Plaque is determining whether or not you have plaque in the first place, even in its early stages. We should measure how much disease, or plaque, is present. In other words, we must be able to quantify the disease, not just provide you with a “yes” or “no.”
A one-minute lesson in coronary anatomy

We all have three arteries that provide heart muscle with its own blood flow. These three arteries sit on the surface of the heart (thus their name, “coronary,” meaning crown) with numerous branches that dive into the heart muscle itself. The three arteries are named the right coronary artery, the left anterior descending artery, and the circumflex artery. Think of these arteries as upside-down trees, in that they start with a trunk and branch into successively smaller branches until you reach the twigs.

![Heart with arteries labeled](image)

Front-view of the heart showing the right coronary artery on your left and the left anterior descending in the center. The circumflex artery is seen on your far right. This image is a 3-D reconstruction of a real human heart using a technique called “CT angiography.”

Cutting off the flow of blood at the level of the “trunk” of the artery, that is, in its uppermost portion, is most likely to result in a large quantity of heart muscle damage. Obstructing flow farther and farther downstream along the artery in its “branches” and “twigs” still causes damage but the amount of damage diminishes the farther out you go. A heart attack is exactly that: Obstruction of one coronary artery somewhere along its length. The higher up the artery the obstruction occurs, the larger and potentially more devastating the heart attack.

What is plaque? What are we measuring?

What is this plaque that accumulates and grows over the decades before cardiac catastrophe strikes? Or, as many patients put it, “Exactly what ‘plugs up’ coronary arteries?”

Let’s start at the beginning. As children, our arteries are flexible, thin-walled tubes, free of disease. The walls of the arteries measure only a millimeter
in thickness. As we age, various injurious factors (high blood pressure, nicotine, oxidized cholesterol-containing particles, inflammation, etc.) cause fatty tissue to accumulate in the arterial wall. Fibrous structural material, calcium (just like that in bone), and inflammatory cells also accumulate, all adding to the mix. The gruel-like material that results is called *atherosclerotic plaque*.

Let’s take an even closer look. We’ll pretend that we have a handful of plaque taken from someone’s arteries. We grind it up in a blender and then examine the contents. It certainly wouldn’t be the prettiest mix, but we would have a fairly predictable combination of ingredients. There would be strands of fiber-like material (collagen) that provide strength to the plaque. Various cells would be found in our blender of plaque: muscle cells, fiber-producing cells, as well as inflammatory white blood cells. Cholesterol would be plentiful, since it is both a structural component of plaque, as well as an undesirable by-product that accumulates. Calcium is also present, which is mostly pebble-like and identical to the calcium found in bone. Curiously, calcium consistently occupies 20% of the overall volume of ground up plaque in our blender—an observation that will prove very useful, as you will see.

It’s one thing to blend up plaque that has been removed from someone’s body. The crucial question is whether this mixture can be measured in a living, breathing person without submitting to procedures or tests that invade the body. That’s the tricky part that has plagued doctors for years. For this reason, for the past 40 years doctors resorted to making *statistical predictions* on whether coronary plaque *might* be present based on cholesterol and other risk factors like blood pressure. What they are *not* telling you is whether or not you truly have plaque.

**How can we measure coronary plaque in a living, breathing person?**

Efforts to measure coronary plaque stumbled for years because of the heart’s location within the chest, surrounded by air-filled lungs. (Air obscures some imaging methods like ultrasound.) The heart is also in rapid motion. During each heartbeat, heart structures, including the coronary arteries, travel at a speed of two inches per second. (Part of the motion is rotational, or turning on an axis.) This is simply too fast for most imaging devices. Magnetic resonance imaging (MRI), for instance, is a clever method of passing the body through a magnetic field. However, imaging plaque that lines rapidly moving coronary arteries remains a practical impossibility because of the slow acquisition of images with this technology. On top of these hurdles, the coronary arteries are small, each measuring only about two to four millimeters in diameter. Imagine trying to snap a clear photo of a tiny bird flitting from tree to tree in a dense forest while you stand on the ground 100 feet away.
There is an ingenious way to circumvent limitations in coronary imaging: If we know what plaque is made of, and if plaque components occur in fairly consistent proportions, we can then use one of the components of plaque to measure the total volume of plaque.

Recall from our make-believe-plaque-in-a-blender that calcium comprises 20% of the ingredients in plaque. The measurement of calcium—the easiest component of plaque to image and quantify—can be used as an indirect, but reliable, measure of the quantity of plaque, since it occurs in a predictable proportion. This simple observation has revolutionized coronary plaque detection and quantification and brought it into the mainstream (Rumberger 1995).

The consistent proportion of calcium to total plaque can be used to image coronary arteries with new computed tomography (CT) technologies. Recent developments in this area permit astonishingly rapid, reliable, and inexpensive imaging of hidden coronary plaque. This approach has become accessible, as well, with a suitable device within driving distance for nearly everyone in the U.S. (a somewhat farther distance for people in Canada). For these reasons, new CT imaging technologies are the method of choice for tracking your plaque. In chapter 4, we’ll discuss everything you need to know about coronary calcium scoring on these new devices.

**Heart disease is a disease of youth**

How early in life does plaque get its start? Does plaque first appear at age 40, or 50, or 60?

No. *Coronary plaque first develops in our teens and twenties.* This became clear during the Korean War when young soldiers (average age 22 years old) who died in battle were studied at autopsy. To the surprise of the doctors performing the autopsies, three quarters of these young, seemingly healthy men had atherosclerotic plaque lining their coronary arteries. These men had been engaged in physically demanding battle and were likely not suffering from chest pain or other symptoms (Enos 1986).

This disturbing observation has been confirmed time and again, even with examination of children’s arteries. The message is clear: coronary disease gets its start when we are young and progresses over decades. This doesn't mean, of course, that children or teenagers need bypass surgery, nor does it mean that they will have a heart attack in their twenties. It does mean that the “seeds” of plaque are planted early in life and grow over the ensuing years.
**Track Your Plaque: Hal**

To illustrate the long-term nature of this process, let's invent a hypothetical man we can follow from the beginnings of his coronary plaque as a teenager to his eventual heart attack.

Our make-believe man is named Hal. At age 18, Hal just graduated from high school, healthy and in the best physical condition of his life. His coronary arteries at this point are smooth and open. The only evidence of coronary disease is a mild thickening of the internal lining of all three arteries. Hal’s life is filled with pizza and fast foods, hectic days and nights with little sleep, but he feels great as he prepares to go to college.

Hal finishes college, lands a job, marries and has children. Hal endures the pressures of work and family, but leads a relatively sedentary life. He rarely exercises except for playing with his kids or an occasional round of golf, and gains about 20 pounds. At age 30, Hal tips the scale at 202 pounds, the most he’s ever weighed, although he wouldn’t regard himself as obese. Outside of work stress, Hal feels pretty good.

If we were to examine Hal’s coronary arteries, we’d find that he already has a thick lining of plaque throughout the entire arterial tree. In some areas, the plaque is as thick as two or three millimeters, even though the opening of the artery (or “lumen”) is also about three millimeters. In fact, if we compare the external diameter of Hal’s artery at age 30 to that at age 18 (i.e., looking at the artery from its exterior), his arteries at age 30 are substantially larger. Hal’s arteries have adapted to the accumulation of plaque by enlarging (a process called the Glagov phenomenon, or adaptive remodeling; Glagov 1987). This process permits plaque to grow without impinging on the path for blood flow. It means that, if Hal were to undergo a heart catheterization in his hospital, he would be told that he has no coronary disease. His arteries would be given a clean bill of health and appear completely “clean,” because the internal diameter was still normal and the thick, extensive plaque in the artery wall was not detected. In reality, Hal’s arteries are loaded with plaque, just disguised by the process of remodeling.

(Does the enlargement process protect Hal from heart attack? No, not at all. In fact, Hal’s remodeled, enlarged arteries are more likely to cause heart attack. Blood flow is maintained, but the probability of sudden closure of his arteries has increased. We will discuss why further on.)

Fast-forward to Hal at 40 years old. His kids are in their teens and Hal has advanced in his career. Although Hal has gained another 15 pounds—he now weighs 217—he still feels that he’s in perfect health. After all, he feels fine, his energy is good, he doesn’t smoke, drinks only occasionally, and he still manages to fit in a round of golf now and then and walk the neighborhood with his wife. Now Hal’s coronary arteries are lined top to bottom with a thick layer of plaque.
Why are “minor” plaques dangerous?

Any coronary plaque has the potential to rupture. However, plaques more likely to rupture have several features:

1) “Pools” of semi-liquid fat, or lipid, with the consistency of toothpaste
2) Inflammatory cells
3) A thin-walled covering between the lipid "pool" and bloodstream

A plaque that is likely to rupture. Note the thin rim of tissue acting as a barrier between the lipid pool and blood (called a “cap”). The white unlabeled areas represent collections of calcium that can be used to measure total plaque volume. The severity of blockage has little to do with the likelihood of a plaque rupturing.

Upon rupture, plaque contents are exposed to blood flowing past and are triggers of blood clot formation. The growing blood clot can completely occlude the artery and cut off blood flow, creating a heart attack, or myocardial infarction.

People accumulate not just one or two plaques but dozens, and they may cover nearly the entire length of the lining of each artery. Each and every plaque has some potential for rupture. So having many plaques poses substantial risk, since a single rupture can cause heart attack, even if it begins as only a 20% blockage.

The process of enlargement has now been exhausted and any further accumulation of plaque begins to grow inward. At this point, if Hal had a heart catheterization, he would be told that he did indeed have some plaque, although the full extent of his plaque would be underestimated. He’d be told, “Your blockages are minor, so you don’t need any stents or bypass surgery.”
The truth is that minor blockages are only the tip of the iceberg of extensive plaque lining the arteries. Still, Hal has no chest pain, no difficulty breathing, and is completely unaware of his coronary plaque.

Hal’s doctor is concerned about Hal’s mildly elevated LDL cholesterol of 144 mg/dl and the fact that Hal’s father suffered a fatal heart attack at age 59. He therefore suggested that Hal be screened for hidden heart disease by undergoing a stress test with radioactive imaging of his heart, a stress thallium. It’s completely normal. Despite Hal’s neglect of vigorous exercise, he exceeds the level expected for a man his age, experiences no abnormal symptoms, shows no EKG abnormalities, and coronary blood flow is normal by the radioactive thallium images.

So Hal, now age 50, goes about his life, feeling just fine, yet harboring a growing burden of plaque lining his arteries. One day, Hal wakes up early with a vague ache in his chest. One of Hal’s numerous coronary plaques has ruptured, or eroded its thin covering of tissue, exposing the plaque’s internal contents of fat and cholesterol to the blood flowing past. The exposed material is a powerful instigator of blood clot formation. Within minutes, the clot grows and cuts off blood flow in the coronary artery.

Hal breathes deeply, moves around, hoping that this muscle or stomachache will just go away. Over the next few minutes, it intensifies, becoming excruciating, the worst pain he’s ever experienced. Hal’s wife knows right away what’s happening. Hal is having a heart attack.

Hal’s story is, of course, fictitious. But stories just like Hal’s are an everyday reality. Little or no warning, no symptoms in the weeks or months leading to heart attack, often dying suddenly—from a process in the coronary arteries that had been present since teenage years.
**Hal's arteries as a child.** No plaque is present. The artery is thin-walled and flexible.

**Hal's arteries at age 18.** Plaque has begun to grow. Plaque (dark gray) contains cholesterol, supportive tissue, and inflammatory cells. Calcium (white) has started to appear, also.

**As Hal enters his 30's,** plaque grows. The diameter of the artery increases to accommodate the growing plaque ("remodeling"). More calcium (white) accumulates, occupying 20% of plaque area.

**Hal, age 36.** Plaque has grown larger with more calcium, as well as scattered collections of fatty material (not shown). The arteries can no longer enlarge to accommodate growing plaque and the internal diameter gets narrower.
Hal, age 40. Plaque is extensive and lines the entire length of his arteries. There is active inflammation and lots of fat ready to "erupt". At this point, Hal's stress test is normal and a heart catheterization would show "mild blockages".

Hal just before his heart attack at age 50. Inflammation and fatty tissue make the plaque contents unstable. One day, these materials digest their way to the surface and become exposed to blood. A blood clot forms and blood flow is cut off. Hal suffers a heart attack. Had Hal undergone a stress test in the days or weeks before his heart attack, it may or may not have been abnormal.

Hal's coronary plaque from age 18 to 50 as it progressed from minor plaque accumulation to extensive fat- and calcium-containing plaque tissue. Remember that the three coronary arteries are each several inches long, and this process, represented in cross-section, develops along the entire length of the arteries.
Your stress test is normal—but you have heart disease!

Let’s consider this for a moment. Hal had advanced coronary atherosclerosis with plaque lining the entire length of his heart’s arteries. Yet his stress test was normal. (Although Hal’s story is fictional, this situation is common.)

How can that be? Don’t doctors commonly use stress testing to screen people for heart disease?

Unfortunately, physicians in practice do indeed use stress testing in various forms to screen people without symptoms for heart disease, despite the fact that the American Heart Association, or AHA, has issued policy statements discouraging this practice. The AHA recognizes that the majority of people with silent heart disease will not be identified by stress testing—the test will be absolutely normal. The AHA has also recognized that many people without heart disease will have abnormal stress tests, so-called “false-positives,” that lead to unnecessary additional testing, such as heart catheterization (American Heart Association, Exercise Testing in Asymptomatic Adults, 2005).

The most persuasive argument against stress testing in people without symptoms is the observation made as long ago as the 1980s that, of every 100 people who have a stress test, about 3 will be abnormal (Fowler-Brown 2004). The majority of future heart attacks and deaths will occur among the 90 people whose stress tests are normal.

A recent DeBakey Heart Center study, for example, showed that having extensive coronary plaque with normal stress test (nuclear) carried 355% increased risk (relative risk 3.55) for heart attack and death from heart attack over seven years of observation (Chang 2009).

If you have undergone a stress test and were told that your heart was in great shape and there’s nothing to worry about, you now know that this could be absolute nonsense.

Stress testing is, however, an effective method your doctor can use to understand symptoms. If you report an ache in your chest to your doctor, an EKG and a stress test can help understand whether the chest discomfort is an impending heart attack or hiatal hernia (when the upper stomach slides up and down through the diaphragm), esophagitis (inflammation of the esophagus), gallstones, etc., since a wide variety of disorders can cause similar symptoms.

Stress testing is based on the principle that, when plaque accumulates sufficient to obstruct blood flow through the artery (generally occluding the diameter of the artery by 70% or more), several things happen. A person may experience symptoms like chest pain or breathlessness, the EKG may show abnormalities and, if the heart is imaged using radioactive materials or ultrasound...
(echocardiogram) during exercise, evidence for poor blood flow can be detected (reduced uptake of radioactive material on a nuclear image or reduced contraction of the left ventricle on echocardiography). But unless there is 70% or more blockage with reduced blood flow, a stress test will be normal. Recall from Hal’s story that extensive plaque can accumulate in the artery walls without blocking blood flow because of the process of remodeling, or enlargement of the artery.

Should everyone considered to be at risk for heart attack undergo heart catheterization to detect silent coronary plaque? (Don’t laugh—this argument has actually had some proponents.) The reality is that catheterization greatly underestimates the amount of plaque in the wall of the arteries because of issues like remodeling, as described in Hal’s arteries. Having performed several thousand heart catheterizations myself, I can tell you that, when x-ray dye is injected directly into the heart arteries, we fill the artery lumen (like the inside of a water pipe) and observe its contours. While this approach can show your cardiologist where the most severe areas of narrowing are located, it reveals nothing about the condition of the artery walls. In fact, the cardiologist often cannot tell whether your arteries are severely thickened, lined with atherosclerotic plaque, or whether they are entirely normal. This test is also invasive, requiring catheters to be inserted into the leg or arm and navigated to the heart. Catheterization costs around $27,000, even if done without admitting you to the hospital. Cost, potential danger, and imprecision make heart catheterizations an undesirable choice for screening purposes.

Who cares if you have silent plaque?

Many people ask, “If you have plaque in your coronary arteries that can’t be detected by stress testing, so what? Are there any dangers from ‘silent plaque’”? You bet there are. In fact, study after study through the late 1980s and 1990s demonstrated that, much to the surprise of many cardiologists, the majority (more than 70%) of heart attacks originate from “mild” blockages of 20–50%. These plaques don’t block blood flow and don’t cause symptoms. They are not ballooned, stented, or bypassed. Yet mild plaques pose the greatest risk and are undetectable by stress testing (Little 1990).

A technology called intracoronary ultrasound has helped make sense of the seeming disconnect between quantity of plaque in the artery wall and the severity of blockage. Intracoronary ultrasound is the closest thing to actually examining arteries as if they were right in the palm of your hand. In this procedure, an ultrasound probe is passed into a patient’s coronary artery and highly detailed cross-sectional images obtained. Ultrasound studies have shown that the do with the amount of plaque present in the wall, and vice versa. (See
Often, portions of the artery that appear normal or minimally diseased by catheterization are actually loaded with plaque by ultrasound.

It is, therefore, the amount of plaque in the wall of the artery, not a reduction in the internal diameter, that determines risk for heart attack. Whether a specific portion of an artery has a 10%, 30%, 50%, 70%, or even 90% blockage has very little to do with the heart-attack potential of this blockage. (An exception to this is when you are just on the verge of a heart attack and the artery is about to close. In this situation, a severe blockage may be properly judged to be the active source of trouble. This is more likely to be the case for people, for instance, in the emergency room with accelerating chest pain symptoms.) It is, however, the 90% blockage that is “fixed” (by ballooning or stenting) or bypassed, as it can be a source of symptoms. All the other less severe blockages—of which there may be dozens—are not fixed but continue to pose substantial risk for heart attack because of potential for rupture.
Cardiac catheterization does not reveal all the plaque
Images made at catheterization do not necessarily reveal the true amount of plaque in the artery wall. Cross-section A is from an area of mild blockage on the catheterization view but, when examined by intracoronary ultrasound ("A," at right) is really filled with thick plaque covering an arc of over 180°. The area occupied by plaque, in fact, exceeds the path for blood to flow (smaller circle). Cross-section B is also from an area of mild blockage and intracoronary ultrasound does indeed show only a mild arc of plaque.
**Just having plaque is bad enough!**

I hope you now see that having *any* quantity of plaque in your coronary arteries is sufficient reason to be concerned that heart attack might be in your future. Keep in mind that, once plaque is established, it grows—and it can grow rapidly. We used to believe that plaque growth was a slow phenomenon requiring years to get worse. While plaque does indeed require decades to reach the point at which heart attack or symptoms develop, newer methods show that plaque growth, when tracked precisely, can be rapid. In some instances, the volume of plaque in your arteries can *double* in a year.

Step 1 of *Track Your Plaque* is to measure coronary plaque. We therefore need a tool to do so reliably, easily, painlessly, at little or no risk, and inexpensively. The best tool to provide this service today are CT heart scans that yield a coronary calcium score. We discuss this further in the next chapter.

**Summary**

The conventional approach to diagnosis of coronary disease with measures like cholesterol and stress testing leaves the vast majority of people with silent heart disease undetected and unsuspecting, despite the fact that approximately half of all American adults over 35 harbor hidden coronary plaque.

Plaque does not appear out of the blue on the eve of heart attack, but begins early in life and accumulates over decades before heart attack.

Because of technological limitations, direct and precise measurement of coronary plaque is not yet possible in living, breathing people. However, calcium in plaque occurs in a consistent proportion of 20% of total plaque volume; 2 mm$^3$ of calcium thereby signals 10 mm$^3$ of total plaque (20%). Calcium is readily and accurately imaged using the newest generation of rapid CT scanners.
References:
American College of Cardiology/American Heart Association 2002 Guideline Update for Exercise Testing.


Step 1
Chapter 4

Heart disease can be measured

If you have a “yardstick” for coronary plaque, you can detect it, measure it, and track it.

Take a hypothetical woman, Joan, with LDL cholesterol of 143 mg/dl. Joan is 46-years old, a non-smoker, 15 lbs. overweight, with a family history of heart attack in her father at age 60. She feels perfectly fine and exercises moderately.

Based on this information, can you tell me if Joan has heart disease or not?

I certainly can’t, and neither can your doctor. This is the dilemma doctors face every day, trying to decide who will have heart attack in future based on cholesterol values and other risk factors. For all we know, Joan may have extensive heart disease and is at potential risk for dying within the next year . . . or she may have no disease at all. Trying to extract this information out of cholesterol values is a useless exercise. LDL cholesterol, in particular, i.e. the amount of cholesterol contained in the low-density lipoprotein fraction of fat-carrying particles in the blood, is the usual starting point in determining heart disease risk. LDL cholesterol is used by most doctors as the pivotal gauge of risk, a practice that is deeply flawed despite generating $27 billion per year in revenues for the drug industry.

If you have hidden heart disease, is it still early in the process? Do you already have extensive plaque, and the burning fuse on this bomb is getting shorter until your heart attack next week or next month? Or perhaps your heart attack is far in the future, say in 10 or 20 years, allowing you plenty of time to develop a preventive program. Or maybe you have absolutely no plaque, and you will never suffer a heart attack.
Obviously, these are crucial distinctions. They are distinctions we cannot make by looking at cholesterol or blood pressure, or by how you feel, your weight, even a stress test. You may feel fine. You haven’t experienced chest pain, breathlessness, etc. You may have “great” cholesterol values, perhaps you’ve never smoked, and maybe you passed a stress test. Yet you can still have plaque lurking silently in your heart’s arteries, and it may be minimal or it may be extensive.

**My doctor said my cholesterol was fine . . . So why did I have a heart attack?**

If we polled 100 people who suffered heart attacks, what do you think the average LDL cholesterol would be? You’d expect it to be high, wouldn’t you?

The average LDL cholesterol in this group would be 134 mg/dl. The majority of LDL cholesterol values, in fact, would range from 80–180 mg/dl (National Health and Nutrition Survey III, 1988-1994). Compare this to the average LDL for the healthy U.S. population: 130 mg/dl. This means that there is substantial overlap in LDL cholesterols among people destined for a heart attack and those who will never have one. Does having LDL cholesterol greater than 130 mg/dl mean you are about to die of a heart attack? Does having an LDL less than 130 mg/dl mean that you will never have a heart attack? The answer to both questions is no, of course.

Half of all heart attacks occur in people with LDL cholesterols below 134 mg/dl, half occur in people with LDL cholesterols above 134 mg/dl. The national guidelines for cholesterol treatment (National Cholesterol Education Program Adult Treatment Panel-III guidelines, 2001) don’t even suggest cholesterol-reducing treatment for LDL at this level in the general population.

How can this be? If high cholesterol causes heart disease, how can you have coronary disease and heart attacks even with low cholesterols? How can we possibly identify people at risk for heart attack from this confusion?

Relying on cholesterol values to identify hidden heart disease is no better than tossing a coin. If we decided to focus only on people with LDL cholesterols greater than 134 mg/dl, for example, then we’ll miss half of all heart attacks. The same is true for other common measures, like HDL cholesterol or the total cholesterol/HDL ratio. None of these values can distinguish, with good confidence, who will or will not have a heart attack.

This is such a crucial point in the *Track Your Plaque* line of reasoning that is bears repeating for emphasis: **No cholesterol value, high or low, can determine whether an individual does or does not have coronary disease.**
Should we treat you to prevent a future heart attack—heads or tails?

Cholesterol does have its usefulness. Cholesterol levels are indeed statistically related in a graded fashion to risk of heart disease, i.e., the higher the cholesterol, the greater the likelihood of heart attack over time. The Multiple Risk Factor Intervention Trial, or MRFIT (“Mister Fit”), demonstrated this principle. Among 12,000 participants, the MRFIT trial showed that the higher the total cholesterol, the higher the risk for heart attack, with likelihood of heart attack in the people with the highest 25% of cholesterol three times that in the lowest 25%. About 40% of all heart attacks, however, still occurred in people with low total cholesterols below 240 mg/dl. There are also many more people with low or middle-range cholesterols than there are people with high cholesterols. As a result, there are just as many people with heart attacks who start with low cholesterol as there are with high cholesterols. So the higher the cholesterol, the higher the statistical risk of heart attack, but a significant number of heart attacks still occur at low or normal cholesterol values (MRFIT 1976).

The widely publicized Framingham trial also illustrated this phenomenon. Thousands of residents of Framingham, Massachusetts were studied over a period of 20 years to discern what measures might predict heart attack. The Framingham researchers found that cholesterol values of people who developed heart attacks overlapped with 80% of the people without heart disease. In other words, four out of five people fell into this large middle range of cholesterols, whether or not they developed heart disease. Extremely low total cholesterols of <150 mg/dl had low risk (though not zero risk) for heart attack; extremely high cholesterols >300 mg/dl had higher risk for heart attack (three-fold higher). The overwhelming majority of people fell in between these extremes, and the greatest number of heart attacks was suffered by these people (Kannel 1979).

Using cholesterol testing to identify people with heart disease is like trying to predict who is going to die in an accident on a high-speed freeway. If the likelihood of dying in an automobile accident on the highway tripled if you drove faster than 65 mph, does this help you decide who is going to die and who is not going to die in a highway accident? It does, but only in a vague statistical sense. These broad measures of risk help to analyze the behavior of large numbers of people. When you try to apply these rules to a specific individual, they utterly fall apart. Does driving 68 miles per hour mean you’re going to die in a car accident? Does driving 62 miles per hour mean you will never die? Of course not. These observations are only statistically predictive.

So why not assume that everybody with LDL cholesterol above 100 mg/dl has coronary disease and just initiate treatment? Or above 90? Or even 50?

The problem is that, the lower the LDL cut-point you use for treatment, the more people—tens of millions!—will be treated who will never have a heart attack (though it would serve the drug industry quite well.) That could represent
decades of cholesterol-reducing treatment for millions of people at substantial cost, not to mention risk of side-effects, without benefit. In addition, there are many other causes of heart disease besides LDL cholesterol. Instituting treatment for LDL cholesterol does not eliminate heart attack or other catastrophes—not even close.

The best risk factor of all is . . .

Is there a risk factor that, when present, predicts a high probability of future heart attack in a specific individual? A risk factor that can distinguish with great confidence who will or will not have a cardiac catastrophe in future? A risk factor that tells us whether you have extensive coronary plaque or no coronary plaque?

Yes: the presence of coronary plaque.

The person at highest risk for heart attack (from a “ruptured” plaque) is the person who already has plaque. This is true regardless of whether LDL cholesterol is 92 or 192. This is because **coronary plaque is a measure of the disease itself.**

If you think about this for a moment, it makes perfect sense. **The risk for rupture of a plaque occurs when there is plaque already present to rupture.** If you don’t have any plaque, you can’t rupture it. So it’s not so much whether cholesterol is high or low, it’s whether you have the necessary plaque to permit rupture.

A hint of this phenomenon was suggested as long ago as the 1970s and 80s in the Coronary Artery Surgery Study (CASS), when people who were told they had mild coronary disease (blockages of <50%) still suffered substantial risk of future heart attack and death, similar to people with severe degrees of blockage (Davis 1995). (Although the obvious lesson here is that any quantity of plaque carries potential danger, the practical solution at the time was to submit more people to bypass surgery.) Once again, **just having plaque** was a powerful predictor of future danger. Even today, people with mild blockages (<50%) are often wrongly told, “You only have mild blockage. There’s nothing to fix. You don’t need a bypass and your risk for heart attack is low.” Unfortunately, physicians equate the lack of need for bypass (or stents) with mild and therefore inconsequential coronary disease. This is simply wrong. People with even mild plaque have substantial risk for heart attack, not very different from people with severe blockages.

To illustrate the superiority of plaque measurement to cholesterol, let’s compare the likelihood of heart attack in people with high cholesterols (say, total cholesterols above 300 mg/dl—really high) to people with high coronary calcium scores (and therefore with extensive plaque). High cholesterol (>300 mg/dl)
increases risk of heart attack three-fold (compared to people with low cholesterol). High calcium scores increase risk as much as 20-fold (when compared to zero scores). In other words, having coronary plaque identified is a powerful predictor of future heart attack, far superior to cholesterol. In fact, very high scores (>1000) can predict a 20% per year risk of heart attack or death even when no symptoms are present and stress test is normal. No cholesterol measure has this kind of power to predict the future.

Having no detectable plaque, on the other hand, can tell us confidently who is not at risk for heart attack. Dr. Paolo Raggi of Tulane University has shown that likelihood of heart attack in people with a heart scan score of zero is nearly zero (Taylor 2007). If you don’t have coronary plaque, it is highly unlikely you will have a heart attack. (There are rare exceptions—people who take cocaine or amphetamines, for instance, can experience heart attacks due to severe coronary artery spasm, even without prior plaque build-up.) In one of Dr. Raggi’s studies that included over 600 people, one heart attack occurred in the group with zero coronary calcium score, while over 99% of the heart attacks occurred in people with measurable plaque (with abnormal heart scan scores). Dr. Raggi also showed that risk was graded: The higher the score, the greater the likelihood of heart attack (Raggi 2000). No other risk factor comes close to this kind of discriminatory value.

In short, the most powerful indicator of future risk of heart attack (or needing bypass surgery, angioplasty, or stents) is the quantity of coronary plaque present.

This approach turns the whole world of cholesterol testing topsy-turvy. We discard the idea of using cholesterol values as the first step to tell us whether or not you should be seriously considered as being at risk for heart disease. If you’ve been told either that you don’t have coronary disease based on low cholesterol, or if you’ve been told you likely have coronary disease based on high cholesterol, it’s time to start all over again. Let’s go straight to a measure of the disease itself: coronary plaque.

We therefore need an accurate means of quantifying all coronary plaque, both visible and hidden. We need to do so not just at one or two spots along the arteries, but along the entire length of all three coronary arteries, from top to bottom. The more extensive the plaque, the higher the risk for heart attack—even in the absence of severe blockage. The greater the quantity of plaque that lines your coronaries, the more opportunity there is for rupture that results in heart attack. Detecting and measuring coronary plaque is the crucial first step of the Track Your Plaque 3-step program.
The plumber’s dilemma

When you think about it, it’s not an easy thing to do: precisely measure the amount of plaque lining three coronary arteries along their six-inch lengths, all while the heart is beating, concealed within the chest, in between breaths.

Imagine asking a plumber to assess the water pipes in your home to tell you just how much rust is lining them. Obviously, he can easily tell you that the pipe in the bathroom sink is leaking or that the kitchen faucet delivers water. But could he assess the internal condition of the entire length of the pipes in the house? Not so easy.

What if another plumber came along and told you, “58% of the pipes in your home have an average of 1.6 mm of circumferential rust lining the internal surface, giving you a total of 7,890 cubic mm of rust. This is more severe than 90% of all other homes.” In other words, this plumber precisely quantified the amount of rust present throughout the entire water pipe system. You now have a much better picture of the “health” of your plumbing. This is analogous to what we are trying to achieve in the coronary arteries. We would like to measure the health of the entire tree of arteries.

As discussed in chapter 3, many physicians still regard heart catheterization as the gold standard to diagnose coronary disease, despite the fact that numerous studies have definitively shown that blockages seen at catheterization greatly underestimate the true extent of plaque. More detailed (though still invasive) examination of the artery walls can be made with intracoronary ultrasound, which provides images in cross-section by passing a high-frequency sound emitting catheter along the length of each artery. Surprisingly, ultrasound reveals that the most extensive plaque frequently occurs in areas along the length of the artery with no significant blockage. Severe blockages are just the tip of the iceberg of a process that is far more extensive. The artery wall, in fact, can be loaded with abnormal plaque, yet the internal diameter appears normal. This would be like the first plumber who was only able to identify the sites of leaks—helpful information, but it tells you little about the overall condition of the pipes. You might even be misled to believe that, because you had no obvious leaks, your pipes were entirely free of rust.

Can plaque be measured in a living person?

Let’s re-visit our hypothetical friend, Hal (chapter 3), before his heart attack at age 50.

You will recall that, at age 40, Hal already had a thick layer of plaque lining the length of all three coronary arteries from top to bottom. You’ll also recall that Hal underwent a stress test (a stress thallium to assess the volume of coronary blood flow) that was normal. Had he undergone a heart catheterization as well
(needlessly, by the way), he would likely have been told “Hal, your arteries are in
great shape. There are only a few minor blockages but nothing that needs to be
fixed.” That assessment is only partly right. Hal does not need any procedures to
“fix” severe blockages, but he still has extensive plaque lining the entire coronary
tree. Then how could we measure the volume of plaque lining Hal’s arteries?

With present technology, it is impossible to directly measure the volume of
plaque in the arteries of a living human being. Intracoronary ultrasound provides
a pretty good approximation, but it is invasive, expensive, and carries real risk of
complications (like tearing the artery).

Can we quantify volume of plaque easily, non-invasively, precisely, and
inexpensively in a living person? Recall our discussion of the various
components that make up atherosclerotic plaque. What if one of the components
of plaque could be measured precisely? What if this component was present in a
consistent proportion in everyone’s plaque? We could then measure the single
component and use this indirect measure to calculate the total volume of plaque.
That is the basic principle behind scoring coronary calcium, and the approach
that might have saved Hal’s life had he known the extent of plaque he had
hidden within.

The birth of calcium scoring

While at the Mayo Clinic, Dr. John Rumberger examined the hearts of
people who had died (both from heart disease and other causes) and studied the
amount of calcium and the total amount of plaque in their coronary arteries. He
discovered that, regardless of age or sex, calcium comprised 20% of the volume
of atherosclerotic plaque. For example, if there were 2 cubic millimeters of
calcium, there would be $2 \times 5 = 10$ cubic millimeters of total plaque volume. Dr.
Rumberger concluded that, if calcium could be measured, plaque volume could
be easily calculated. But measuring calcium and plaque in arteries removed from
the body was not possible in walking, talking people (Rumberger 1995; Sangiorgi
1998). The next step was to reproduce these observations in living people.

Dr. Rumberger possessed the unique insights of a dual background, a
doctorate in electrical engineering, as well as medical training as a physician and
cardiologist. Along with his study of calcium in human coronary arteries, Dr.
Rumberger was also investigating applications of the new (at the time) electron-
beam CT scanner invented several years earlier in the San Francisco area by
physicist Dr. Douglas Boyd. Dr. Boyd had developed a CT scanner that obtained
images at extremely rapid speed, requiring a small fraction of the time required
by conventional CT scanners. CT scanners in the 1980’s required a full 1 to 2
seconds to obtain each cross-sectional image of the body. This may seem fast
until you realize how much motion goes on inside the living human body.
The applications of this “ultra-fast” CT scanner, which he subsequently called “electron-beam tomography,” or EBT, were not evident at first. Among the first useful applications was in young children, who, as any parent knows, are unable to lie still for even a few seconds.

Dr. Boyd envisioned applications in the heart. The slow scan time of other CT devices rendered them useless for imaging the heart. The human heart beats, on average, 70 beats per minute. There are also multiple phases of motion in the heart during each heartbeat (atrial systole and diastole; ventricular systole and diastole). Even if you hold your breath (to eliminate motion of the nearby lungs), you have motion in the coronary arteries of two inches per second. If you had a camera and tried to obtain still-frame images of the heart with a shutter speed of a full second (1000 milliseconds), you would record a picture that traced (back and forth and rotational) movement of up to two inches in that second. On conventional CT scanners, the heart appears as a meaningless blur, where no detail can be discerned. If you instead had a camera—or scanner—that could obtain images in fractions of a second, you could eliminate blur-producing movement.

Dr. Rumberger proposed that the unique rapid scanning feature of Dr. Boyd’s EBT device could be used to quantify the calcium of coronary arteries. Its split-second scanning time could essentially freeze the motion of a beating heart, exposing even tiny collections of calcium in coronary artery plaque that are commonly a millimeter or so in diameter. When tested in living humans, the EBT scanner, in fact, provided wonderfully detailed images of calcium in the arteries despite the heart’s rapid motion. Because precise quantification of calcium could provide an indirect measure of the total plaque volume in a person’s coronary arteries, Dr. Rumberger proposed that coronary calcium could provide a “yardstick” of coronary plaque.

Dr. Rumberger initially encountered skepticism from physicians, many of whom were already aware that calcium was a component of atherosclerotic coronary plaque. This wasn’t entirely new information. After all, for years, cardiologists had known that patients with the most severe coronary disease (generally elderly patients who came to the hospital with heart attacks or had
undergone multiple heart procedures) commonly had calcium visible in their coronary arteries. Calcium was visualized during heart catheterization as dense white spots or large streaks. In some patients, there was so much calcium that it could be seen even on a simple chest x-ray. Calcium in the coronary arteries was commonly believed to accompany only the most advanced stages of coronary disease.

Dr. Rumberger proved that, if a sensitive and precise tool like the EBT scanner were used, calcium could be detected and quantified even in the early phases of coronary disease, years or decades before it was detectable by other techniques like heart catheterization (Rumberger 1996). The more advanced the coronary disease, the more extensive the coronary atherosclerotic plaque, and the more calcium would be measured. (Subsequent studies showed that the relationship is linear.) Since those early observations, Dr. Rumberger and others have shown that even young people can have calcium that can be detected and scored when other techniques fail to show any calcium or plaque whatsoever.

Confused beginnings

Like any new concept, the history of calcium scoring has had its share of confusion and misperceptions.

In the early years of heart scanning, many people were scanned and told that they had coronary calcium present. A heart catheterization or stress test would then be performed, and the patient advised, “Your arteries only have mild plaque and your stress test is normal. The heart scan must be wrong!”

The calcium score was not wrong, it just identified plaque at an earlier stage, years before a stress test became abnormal, and years before severe blockage was present. Doctors often failed to recognize that the mere presence of plaque was a significant risk for heart attack, regardless of whether severe blockage was present or not.

Just as many surgeons of the 1950s clung to the idea that lung removal was the only certain treatment for tuberculosis, so many critics still cling to the notion that calcium represents advanced disease, not recognizing that improved methods of imaging can detect calcium and therefore coronary plaque during earlier phases of the process. They argue that the deposition of calcium is a phenomenon of aging and has no value in predicting heart attack. They also argue that calcium is a marker for “hard,” stable plaque, not of the “soft,” more unstable plaque.

But a considerable quantity of evidence now supports the concept of calcium scoring. Literally thousands of scientific and clinical studies have been performed. Virtually every study has persuasively shown that the coronary calcium score is the single most powerful predictor of heart attack available. We
should view coronary calcium in much the same way as Dr. Rumberger originally viewed it: as an index of total plaque: “hard,” “soft,” and all other components combined. Calcium is simply the component of plaque we can measure, an indirect yardstick to gauge how much total atherosclerotic plaque is present in all three coronary arteries, top to bottom.

Dr. Boyd’s original ultra-fast EBT scanner has since been largely replaced by another generation of CT scanners using somewhat different technology, the so-called multidetector CT scanners, or MDCT. MDCT scanners, more than EBT, have proven to be multiple-use devices that suit facilities like hospitals and have therefore become more widely available.

Because of scanning speed, the EBT and MDCT devices are the preferred methods to obtain a coronary calcium score. The entire process requires several minutes, the scan itself requires 10-40 seconds. With current technology, no other method can identify silent plaque so easily, safely, and reliably. In chapter 5, we will discuss the nitty-gritty of calcium scoring in more detail.

Other methods to detect early heart disease:

Though imaging technology is advanced rapidly, many methods are not yet ready for mainstream use. There are several alternatives, however, to coronary calcium scoring using EBT or MDCT scanners that are already available. Let’s discuss these alternatives and their strengths and weaknesses.

Carotid Ultrasound

Atherosclerosis is a body-wide disease that affects all arteries of the body. That means plaque can develop simultaneously in the heart (coronary arteries), the brain (carotid arteries and cerebral circulation), the abdomen (abdominal aorta and mesenteric arteries), legs (iliac and femoral arteries), etc. Plaque develops in these parts of the body in parallel to the heart, though to varying degrees.

Most people with atherosclerotic disease tend to show evidence of disease in their heart first, i.e., they have a heart attack, or develop angina, or undergo a cardiac procedure. The other arteries of the body tend to develop plaque more slowly. This is partly due to the larger diameter of other arteries. Compare their relative sizes: coronary arteries generally measure 3–4 mm in diameter; carotid arteries measure 5–8 mm; femoral (thigh) arteries measure 6–9 mm, sometimes larger. Less plaque accumulation is, therefore, required in the smaller heart arteries before trouble begins. Any plaque that ruptures is more likely to lead to trouble (i.e., heart attack) in smaller arteries than in larger arteries.

Nonetheless, because there is a parallel tendency for various arteries to develop plaque, some physicians have proposed that other arteries be measured
in place of the heart. The imaging technique usually used is ultrasound, since it is easy, painless, and can be somewhat quantitative.

In ultrasound (like that used for intracoronary ultrasound), images are generated by a high-frequency sound-emitting crystal. The data is processed by a computer and converted into images. The best-studied technique involves ultrasound imaging of the carotid arteries, in which the device is applied gently to the neck and the carotid arteries (right and left) are examined. Using this technique, a measure called carotid intimal-medial thickness (CIMT), or the thickness of the internal lining of the arteries, is obtained. Note that CIMT is a measurement of the lining of the artery, not of carotid plaque itself. There have also been studies examining other arteries of the body (particularly the abdominal aorta, iliac, and femoral arteries), but they correlate less well to heart disease than the carotids.

Among the most experienced in this technique is Dr. Howard Hodis of the University of Southern California. His extensive experience does indeed suggest that measuring CIMT can predict heart attack risk, is correlated to a moderate degree with the extent of coronary plaque, and can be used to track the course of disease, i.e., progression or regression (Hodis 1996). CIMT measurement has been quite popular in research settings to examine the efficacy of various therapies.

Ultrasound is safe, since no radiation is involved. Devices capable of obtaining quality images are also very widely available. Most hospitals and even many cardiologists’ offices will have at least one if not several ultrasound units. Carotid ultrasound is already routinely performed to look for large plaques that pose risk for stroke.

So why isn’t carotid ultrasound performed more widely for identification of early heart disease?

There are several reasons. One reason is that the relationship of coronary disease to carotid IMT is not perfectly parallel. Coronary and carotid arteries respond somewhat differently to various influences and so develop plaque at different rates. Carotid IMT, for instance, is very sensitive to blood pressure effects; coronary plaque less so. The correlation of carotid IMT to coronary plaque is around 60 to 70%, meaning that a certain carotid IMT measurement will be around 60–70% accurate in predicting the extent of coronary plaque (Folsom 2008). It would be like buying a used car and trying to gauge the accuracy of the odometer mileage by looking at the wear on the rubber of the gas pedal—you can make relatively crude predictions, but it’s not very accurate.

Despite wide availability of ultrasound devices and the relative ease of obtaining this measure, the vast majority of facilities do not offer CIMT. CIMT measurement requires special software that most facilities do not have.
Insurance also does not pay for CIMT. Ultrasound facilities, however, can measure carotid plaque, though in a non-quantitative way. Anyone who has undergone a conventional carotid ultrasound has likely been provided the frustratingly imprecise results, e.g., “10-49% plaque in the right internal carotid artery.” Conventional carotid ultrasound is neither quantitative along the length of the artery and therefore cannot provide plaque volume measurement, nor is it precise in quantifying cross-sectional plaque.

Another weakness of carotid ultrasound is the difficulty in using this measure to track plaque in a specific individual. Over a year or two, the change in CIMT can be tenths or hundredths of a millimeter. In practical life in most ultrasound laboratories, this sort of precision is simply not obtainable. Likewise, because measures of carotid plaque are so crude, precise measures of small changes are not possible with present-day technology.

Patients and physicians also struggle to understand this indirect measure. How can scanning a neck artery predict heart attack? That, combined with its technical limitations, make this a less attractive choice for use in a prevention program. If heart scanning EBT or MDCT scans are not available in your area, and a conversation with your physician suggests that someone with expertise with the ultrasound approach is available, only then might carotid ultrasound be a viable alternative for you.

Ankle-Brachial Index

The ankle-brachial index, or ABI, is mentioned just for information. It is not a desirable method for heart disease detection because of serious shortcomings. Nonetheless, some physicians recommend this technique to identify people at risk for heart attack.

Just as plaque accumulates in coronary arteries, it can also grow in leg arteries. If sufficiently advanced, blockages reduce blood flow to the legs, even without symptoms. (Blockages in the leg arteries can cause a cramping in the calves, thighs, or buttocks with walking, called claudication.) In contrast, the arteries to the arm rarely develop blockages. Some physicians have therefore proposed that, if you compare the blood pressure (an indirect measure of the volume of blood flow) in the legs to that in the arms, it can tell whether you have plaque in the leg arteries. Because atherosclerosis is a body wide disease, if there is plaque in your leg arteries, you likely also have it in your heart’s coronary arteries. The ankle-brachial index is simply the ratio of systolic blood pressure in the legs divided by the systolic blood pressure in the arms. If this ratio is <0.9, this indicates that blood flow to the legs is diminished.

The advantage of this test is that it can be done virtually anywhere a blood pressure device is available. Several studies have confirmed that people with low ABI’s do indeed have higher risk for heart attack.
There are several critical disadvantages to this test. For one, it is a crude measure of blood flow to the legs, not the heart. The ABI also tends to be a measure of very advanced vascular disease. Blood flow is affected only when there is a substantial quantity of plaque lining the entire arterial tree of the legs and severe blockage is present. Just as with stress testing, the great majority of people who have silent heart disease will have normal ABI's, not showing any abnormality until heart disease is far progressed. Measuring ABI is therefore not an effective method to detect early heart disease. Even in people in advanced heart disease who've had heart attacks or bypass surgery, only about a third will have an abnormal ABI (Doobay 2005).

ABI is a method of last resort. Choose this approach only if you live in Timbuktu and no better method is available to you and your doctor.

**Magnetic Resonance Imaging (MRI)**

Various tissues in the body respond differently when placed in a magnetic field. This principle can be applied in an ingenious imaging method called magnetic resonance imaging, or MRI. MRI is used in mainstream medical practice to image the chest, abdomen, brain, joints, and other organs, with exquisite detail.

One fascinating aspect of MRI is its capacity to characterize tissue, such as soft and fatty vs. hard and fibrous. Dr. Valentin Fuster and colleagues at the Mt. Sinai School of Medicine have used MRI to image the aorta (the large artery that emerges from the top of the heart and supplies the major branch arteries to the rest of the body). When aortic plaque is imaged before and after cholesterol-lowering therapy (statin drugs), plaque volume shrinks within a year’s time. Fatty tissue is replaced by denser fibrous tissue, suggesting plaque stabilization with diminished tendency to rupture (Corti 2005).

The drawback to MRI is that the current devices are too slow to reliably image rapidly moving coronary arteries. Preliminary efforts have yielded snapshots of portions of the arteries, but imaging the entire coronary tree remains elusive. MRI promises to be an exciting technique to screen people for hidden coronary plaque, but right now it’s just too unreliable and not good enough to track your plaque.

In chapter 5, we will discuss everything you need to know about CT technologies that provide coronary plaque measurement in the here-and-now.

**Summary**

While cholesterol values and other risk factors can, at best, reveal potential risk for heart disease, measuring coronary plaque provides an index of the disease itself, coronary atherosclerotic plaque.
Imaging technologies are advancing at breakneck speed. The days of invasive procedures to diagnose heart disease are going the way of exploratory abdominal surgery and eight-track tapes. The newest CT scanning technologies offer the best balance of precision, ease, cost, and availability for detection of coronary plaque. The relationship of plaque to likelihood of heart attack is graded: No coronary plaque means extremely low-likelihood of heart attack; lots of plaque means high-likelihood of heart attack.

The perennial problem for imaging the heart has been its rapid motion. The most recent CT scanners are “ultra-fast” and able to provide crystal-clear still-frame images that allow precise quantification of coronary plaque.
References:


Chapter 5

Want to know whether you have heart disease?

Know your score!

Like a game of golf or baseball, you can’t play the game without knowing the score. The same idea applies to coronary plaque.

We finally get down to the “nitty-gritty” of tracking your plaque in this chapter. To see what it’s like getting a coronary calcium score, let’s accompany Anna through her heart scan experience.

A “walk-through” a heart scan

Anna has lived much of her adult life in fear of heart disease. Her father died at 59 of a heart attack, her mother had urgent bypass surgery at age 62. Other relatives in Anna’s close extended family suffered heart attacks or died of heart disease, all between the ages of 40 and 60. Anna just turned 45 and suspected that her clock was ticking. She was also devoted to her husband and six year-old daughter and wished to remain alive and healthy for their sake.

Anna heard about the Track Your Plaque program and asked whether the program was right for her, given her family history. I told her that she was a perfect candidate, and that a heart scan should begin the process.

Anna called the scanning center and made an appointment. Once at the center, she filled out the basic demographic information on the standard forms (I often tell patients that the paperwork takes longer than the scan!), then was
escorted by the technologist, back to the scanning area. While remaining in her clothes, the technologist applied EKG leads to Anna’s chest and was instructed to lie down on the scanning table. The technologist, now at the control console behind a glass panel, asked Anna to hold her breath while a several-second preliminary scan was performed to identify landmarks in Anna’s chest. Anna was then instructed to hold her breath again, this time for 30 seconds, while Anna’s heart was scanned. Anna watched as the device circled her during the scanning process. A minute later, Anna hopped off the table and the technologist invited Anna to have a seat at the workstation while they reviewed images of her heart.

The technologist pointed out some of the basic anatomical features on Anna’s images, like her lungs, spine, the aorta, as well as heart structures. She also showed Anna the small, dense spots colored white within the coronary arteries of the heart, which Anna observed were present on about 5 of the 30 cross-sectional images. The technologist advised Anna that, as the technologist performing the scan, she was unable to provide Anna with her final “score.” This would be done later when Anna’s scans were reviewed in detail by one of the center’s cardiologists.

Later that evening, I reviewed Anna’s scans. Using the computer software that helps calculate the amount of coronary calcium present, I obtained a “score” of 63. I called Anna at home the next day. I told Anna that a score of 63 in a 45-year old woman was not a high score, but it was a high score for a woman in her age group. It confirmed that coronary disease was established and measurable. Based on her score, Anna’s near-term risk heart attack was 4.0% per year, a figure that, surprisingly, provided Anna some comfort, since she had assumed that it was much higher. But I also pointed out that a 4.0% annual risk represented (approximately) 33% likelihood over the next 10 years, at which time Anna would be a relatively young 55 years old. I also pointed out to Anna that her score of 63 would increase at a rate of 30% per year. If we do the math, Anna’s score would climb predictably: 63, 82, 106, 138, 179, 233, etc. If we permitted Anna’s score to increase (as it undoubtedly would without appropriate action), the likelihood of heart attack would escalate.

My message: Let’s take this score seriously and apply a program of prevention. So I advised Anna of the next step in the program.
For Anna, getting a score was easy and painless, and despite her high score for a young woman, actually provided an understandable number on which she could “hang” her fears: It made it more real, but also made it seem more manageable. It also solidified Anna’s commitment to beginning a more serious effort at prevention.

One example scan from the 30-40 total images from a heart scan. The coronary calcium in the left anterior descending artery and its diagonal branch are circled in white. This is “scored” (using area and density) and the score of each image added to those of all other images to obtain a total calcium score. The scores can be broken down into component arteries to obtain a score specific to each coronary artery.
### Who should be scanned?

How do you decide whether you should have a heart scan in the first place?

Age is the best guide to determine who should have a heart scan. Many authorities recommend that men over age 40, women over age 50 be scanned and therefore have their heart disease identified and "scored."

Beyond age, there are no criteria to reliably help decide who should and who shouldn’t be scanned. For example, if you use high LDL cholesterol (e.g., >130 mg/dl) to identify whom to scan, half the people with heart disease will be missed and will not be scanned. Likewise, if you use a low HDL(<40 mg/dl) to decide who to scan, you will again miss about half the people with heart disease (Hecht 2001). Similar patterns apply for any other screening parameter you can devise.

Think about this: Can we screen you for the presence of heart disease by screening you with another test first? You may begin to see the absurdity in this approach. A study by Drs. Arad and Guerci at the St. Francis Medical Center in New York, for instance, showed that 67%—two-thirds—of people classified by the broadly-used Framingham risk scoring system were misclassified: people labeled low-risk were actually high-risk; people labeled high-risk were actually low-risk (Arad 2005). Cholesterol values and conventional risk factors cannot be reliably used to decide whether or not to have a scan. We therefore resort to using age as a starting point.

If there is some high-risk measure in your life—parent with heart attack before age 55; diabetes, total cholesterol >300 mg/dl, smoking—then you might consider being scanned 5 years earlier (35 for men, 45 for women.) This approach provides the best balance between identifying the greatest number of people with important scores while not subjecting others to unnecessary scans.

### What exactly is a calcium “score”?

Anna’s score was 63. This might be a great golf score, but what exactly does this “score” represent in the heart?

A heart scan consists of a series of overlapping cross-sectional images of the heart from top to bottom. (The scans overlap in order to not miss any segment of the heart.) Just as I reviewed Anna’s scans, a cardiologist (sometimes a radiologist) will review each “slice” and select what he/she believes represents calcified plaque in the coronary arteries. This is a simple, straightforward process, since calcium is easy to distinguish from the surrounding “soft” heart tissues. A computer decides whether the selected area meets specific density and size criteria. We multiply the area (in square millimeters) by the density of the plaque and this yields a “score” for this specific plaque. We score all the plaques in every image slice and add all the scores up. This yields a total score, the one reported to the patient (the number 63 reported to Anna).

You will sometimes hear the total score called an “Agatston” score, named after Dr. Arthur Agatston from Miami (also of South Beach Diet fame), who first developed this method of adding up and comparing scores from different people.
Dr. Agatston’s method is now one of the standard calculations performed on all heart scans.

Remember: The higher your score, the more plaque lines your coronary arteries.

**Does the calcium score predict heart attack?**

No test, no matter how good, can serve perfectly as a “crystal ball.” But your heart scan score can do a pretty darn good job of predicting your future.

In most heart scan centers, your total calcium score is compared with a database of over 40,000 other people who’ve also been scanned, based on the enormous experience provided by Dr. George Kondos at the University of Illinois–Chicago and others. The database allows your score to be compared with that of other people your age and sex (Hoff 2001). The scan center reports this as your “percentile rank.” The percentile rank is the best predictor of heart attack risk, even better than your score alone. For example, Anna’s score of 63 provided a percentile rank of 90%. This means that 90% of all women her age had lower scores, 10% of women her age had higher scores. The 90th percentile also tells us that Anna’s risk for heart attack is 4.0% per year (see table).

The percentile rank provides insight into the aggressiveness of plaque growth. Dr. Paolo Raggi of Tulane University in New Orleans, Louisiana, has extensively researched this issue. Dr. Raggi observed that the same score in different age groups and sexes predict different risks for heart attack. For instance, a score of 125 in a 48 year-old man represents a small to moderate amount of plaque. It reflects moderately aggressive plaque growth and moderate risk for heart attack. The identical score of 125 in a 65 year-old man represents the exact same amount of plaque that is not as active since it took 65 years to accumulate, rather than 48, and poses only small risk for heart attack. The same score of 125 in a 48 year-old woman, however, represents the same amount of plaque but reflects aggressive plaque growth and high risk for heart attack. The same heart scan score can therefore predict very different futures, depending on age and sex (Raggi 2001).
How much blockage do I have?

Though your score does not tell you whether there is a “severe” blockage, the higher your score the more likely a severe blockage is present (sufficient to be detected by a stress test and/or result in symptoms like chest pain or breathlessness). Here’s a breakdown:

<table>
<thead>
<tr>
<th>Score</th>
<th>Amount of plaque present</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None – severe blockage highly unlikely</td>
</tr>
<tr>
<td>1-10</td>
<td>Minimal plaque; severe blockage highly unlikely (&lt;1%)</td>
</tr>
<tr>
<td>11-100</td>
<td>Mild quantity of plaque; very low likelihood of severe blockage (2-3%)</td>
</tr>
<tr>
<td>101-400</td>
<td>Moderate quantity of plaque; 11-15% likelihood of severe blockage</td>
</tr>
<tr>
<td>401-1000</td>
<td>Extensive plaque with 20% or more likelihood of severe blockage</td>
</tr>
<tr>
<td>&gt;1000</td>
<td>Very extensive plaque with 30% or more likelihood of severe blockage</td>
</tr>
</tbody>
</table>

(Berman 2004; Anand 2004; Ramakrishna 2006)

At a score of 200, for example, there is an 11-15% chance that a “severe” blockage is present.

A specific calcium score cannot, however, be equated to a specific percent blockage, as plaque accumulates in various patterns. It does not necessarily all accumulate at one spot and thereby create a single “severe” blockage. The calcium score can only be associated with the statistical likelihood of a severe blockage. Recall that we are also interested in more than “severe” blockages—remember that any amount of plaque, even in the absence of severe blockage, can result in heart attack because of plaque “rupture.” The more plaque you have, the greater the likelihood of plaque rupture.

To predict your heart attack risk, i.e., the likelihood of a plaque “rupture,” we use the “percentile rank” (see below).

Percentile rank and annual risk for heart attack

Heart scans are generally reported as both a calcium (or Agatston) score and a percentile rank. The percentile rank is the best predictor for the future risk of heart attack.

<table>
<thead>
<tr>
<th>Percentile Rank</th>
<th>Annual Heart Attack risk (% per year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;25%</td>
<td>&lt; 1%</td>
</tr>
<tr>
<td>25-50%</td>
<td>1-2 %</td>
</tr>
<tr>
<td>50-75%</td>
<td>2%</td>
</tr>
<tr>
<td>75-90%</td>
<td>3%</td>
</tr>
<tr>
<td>&gt;90%</td>
<td>&gt;4%</td>
</tr>
</tbody>
</table>

The annual heart attack risk applies only when no effort at heart disease prevention has been instituted. (After reading Track Your Plaque, of course, your risk will be much lower!) The most recent analyses have also demonstrated that such predictions are true regardless of race, including Caucasians, African Americans, Hispanics, and Asians. Also, note that heart scan scores of >400 should be regarded as high-risk, regardless of percentile rank.

(Raggi 2001; Budoff 2008; Budoff 2009; Elkeles 2008; Detrano 2008)
Heart scans: Does the device make a difference?

In the early years of heart scanning (the 1990s), there was just one choice of device fast enough to perform a heart scan: electron-beam tomography, or EBT (also called “Ultra-Fast CT”). This ingenious device focused x-rays by applying a magnetic field, thereby requiring little motion of either device or the human body being scanned.

Drawbacks: Hefty cost (nearly $2 million) and limited usefulness for applications outside the heart. This meant that the device was largely a device for heart scanning. Though it could perform other imaging tests, such as virtual colonoscopy, EBT was first and foremost a heart scanner. Scan centers appeared nationwide, but struggled financially since they could offer little beyond simple, relatively low-cost heart scans.

A competing technology called spiral CT came along, in which an x-ray-generating gantry rotated around the patient. However, spiral CT initially proved inadequate for heart scan purposes: too slow, generating blurred images at high radiation cost. Engineers improved on spiral technology by adding multiple detector rings, i.e., detectors of the x-ray beam projected through the scanned area. First came 4-detector, then 16-detectors, then 64-detectors. As of the writing of this edition of Track Your Plaque, there are several 256-detector devices nationwide. This technology, called multi-detector CT (MDCT), has largely supplanted EBT, though some original EBT scanners are still in operation.

With multi-detector technology, more detector rings mean faster image acquisition at lower radiation cost. Imaging quality has also improved, since faster imaging means less heart motion.

Nowadays, 64-slice devices—a “64-slice CT”—generates image quality nearly on a par with the original EBT devices. Because multi-detector technology provides a more general purpose scanner (e.g., brain, pelvis, joints), it has emerged as the most popular device for heart scanning, especially in hospitals.

This means that either EBT or the multi-detector 64-slice (or greater) scanners are your best choices for high-quality heart scans.

Do I need bypass surgery?
People often ask, “If I have a high score, do I need bypass surgery?”

Provided you have no symptoms of heart disease, such as chest pain or breathlessness, just having plaque in your coronary arteries—no matter how high the score—does not necessarily mean you need to have bypass surgery or other major procedures. There are additional evaluations that need to be performed to help you and your doctor make this determination (discussed later). In the absence of symptoms, plaque should rarely—perhaps never—be the sole
indication for a major procedure. In other words, just having plaque measured by a calcium score does not necessarily mean it needs to be bypassed or stented.

For most people, a simple stress test (usually performed with some method of imaging the heart muscle, such as echocardiography or nuclear imaging) is a useful method to assess coronary blood flow through the arteries that contain plaque, as well as assess the safety of exercise, blood pressure responses, etc. We’ll discuss this in more detail later.

That said, it is true that the higher your score, the more likely it is that your coronary plaque is so extensive and blocks blood flow that a major procedure may be necessary for safety. Approximately 30% of all people with scores exceeding 1000 (very high!) require additional testing beyond a stress test that may include hospital procedures. But this decision can almost never be made on the basis of your heart scan score alone.

**Keeping score on coronary plaque**

Just as you keep score during a baseball game, you can do the same with your coronary plaque. Let me tell you about Stephen R.’s experience.

Stephen, a 57-year old businessman, had a heart scan two years earlier at age 55, mostly because his wife “made him do it.” Stephen’s score: 227, in the 75th percentile for men his age. When Stephen told his doctor about the scan and showed him the report, the doctor looked at it for a moment, then handed it back to him and said, “I don’t know what the heck this means. This isn’t even a real test. We’ll do a stress test.”

Stephen’s stress test was normal and the doctor declared the heart scan useless and a waste of money. He told Stephen to go about his life and return in a year.

But Stephen’s wife, Laura, suspected there was more to it. She had asked numerous questions during her own heart scan and recognized that there was valuable information in the heart scan score. She had a hard time persuading Stephen to share her views at first, given his doctor’s negative comments. But a year later, Laura managed to convince him to undergo another heart scan. His score: 363—a 60% increase. This really grabbed Stephen’s attention. He didn’t bother telling his doctor but came to my office instead. When I told him that his increasing score put him a very high risk for heart attack despite the normal stress test, he agreed to participate in the program without hesitation in the hopes of averting catastrophe.

If, like Stephen, nothing is done about the causes of your plaque, your score can increase 30%, 40% or more per year. Your score can be used not only as a “yardstick” for the initial measurement of coronary plaque, but it can also be
used to track its increase or decrease over time. People who experience this rate of increase launch themselves into a very high-risk group for future heart attack—as much as 20-fold higher than people with unchanging scores (Raggi 2004).

People who, instead, succeed in arresting their score don’t have zero-percent risk of heart attack (nobody has a zero-risk), but risk is very low. Scores that fall below the starting value provide even lower and lower risk of heart attack. Achieving a stable or decreasing heart scan score can therefore be an important goal to sharply reduce risk of heart attack.

When considering a repeat scan, you should do so no sooner than a year after the first. Even better, your second scan should be timed approximately one year after the lipid/lipoprotein abnormalities causing your plaque are corrected. (We will discuss this further in chapter 8.) Correction of the lipid/lipoprotein patterns that caused your heart disease can require several months. Repeating your heart scan a year after lipids/lipoproteins are corrected will give you an idea of what impact you’ve had on your score as a result of your corrected patterns. In our center, experience has shown that most people require a full year or more to halt an increasing score, or achieve a decrease. This is discussed further in the next chapter.

What if you’ve had a heart procedure?
Some unique approaches are required if you’ve had coronary stent(s) or bypass surgery.

Coronary stents are a fine tubular mesh constructed of stainless steel or similar material. One problem area in heart scanning is that metal looks just like calcium when visualized by any CT scan device. Even though the structure of a stent can generally be made out, it cannot be reliably distinguished from calcium covered by the stent(s) that are immediately adjacent. For this reason, if you have one or more stents in a single coronary artery, you can still obtain a score, but it will not include the stented artery. The total score will therefore underestimate the total extent of coronary plaque. But your score can still be used to track your score on future scans when the same two arteries are scored. So a single artery with stent(s) still permits you to obtain a score and apply it to your Track Your Plaque program.

The real problem is people with multiple arteries (two or three) that are stented. The stented arteries can no longer be excluded without sacrificing crucial information. We are presently researching methods to circumvent this limitation, but at present, the presence of multiple arteries with stents is a real stumbling block. People with this problem can still obtain great benefit from many of the other concepts in Track Your Plaque, but the scoring process is one step that is impossible with present technology.
The other problem area is people who have had bypass surgery. When the surgeon places a bypass graft, your own coronary arteries are distorted by the act of manual handling. In addition, bypass grafts themselves often obscure the images of your own arteries, making the scoring process difficult or impossible. Having bypass grafts might not entirely eliminate the possibility of obtaining a usable calcium score, but less than half the people with prior bypass surgery can be scored. Usually the only way to find out if you can get a score is to try, provided the center you use has an interpreting physician willing to devote the effort. As with people who've had stents and are unscorable, people with bypass surgery can still obtain great benefit from the other aspects of Track Your Plaque concepts, minus the scoring component.

Calcium Score “Red Flags”
Are there calcium scores that can be “red flags” for potential danger in the near-future?

There definitely are, and these “red flags” are generally any score in the 90th percentile or greater. However, even if the scanning center you choose does not report a percentile rank, here are some general guidelines for identifying “red flags”:

• If you are younger than 50 years old, a score >100
• If you are 55 years old or older, a score >400
• Any score >1000—This is the worst, most dangerous of all.

These are scores that may present dangers even in the next few months.

Should you have a heart scan score that fit any of the above “red flag” criteria, prompt preventive action on your part is warranted. Discuss your score with your healthcare provider so that you can talk about the need for stress testing and additional testing, if necessary.

Radiation exposure: Achille’s heel of heart scanning?

In years past, nobody paid much attention to radiation exposure during testing for medical purposes. But recent studies have raised concerns over the long-term dangers of radiation exposure. The issue has becoming increasingly important as CT scanning has become as commonplace as drawing blood. Recent estimates show that 10% of the U.S. population undergoes one or more CT scans in some form every year, totaling 75 million annually (Smith-Bindman 2010).

Radiation exposure is typically measured in units called milliSieverts (mSv). A standard (two-view) chest x-ray exposes you to 0.15 mSv, a relatively minor amount, while exposure to natural radiation from the atmosphere exposes you to 1-2.4 mSv per year (Huda 2007; Ron 2003). While a CT scan of the brain
exposes you to 2 mSv of radiation, an abdominal-pelvic CT scan can expose you to as much as 31 mSv (Brenner 2007; Smith-Bindman 2009). Radiation experts have expressed concern that unrestrained use of CT scans and other radiation-based diagnostic testing may increase an individual’s lifetime risk of cancer. This is especially a concern for people younger than 20 years old, who have many more years to express any cancer risk (Brenner 2007).

Heart scans of the sort we are discussing for tracking your plaque expose you to 0.4-0.6 mSv if performed on an EBT device, 0.6-2.0 mSv if performed on a 64-slice MDCT device (Nakazato 2009; Stolzmann 2009). A CT heart scan to generate a coronary calcium score should not be confused with an increasingly popular test called “CT coronary angiography,” or CTA. While performed on the same EBT and MDCT devices as heart scans, CTAs are angiograms that require placement of an IV, administration of X-ray dye, and require many times greater radiation to generate images, with estimates putting radiation exposure at 8-14 mSv (Schmermund 2008), sufficient to potentially cause a radiation-related cancer in 1 in 270 females, 1 in 600 males if the scan is performed at age 40 (Smith-Bindman 2009).

Unavoidably, there is therefore a modest radiation exposure from a heart scan to obtain a coronary calcium score. Because all of us are over age 20, the health value of this important information at the price of modest radiation exposure likely outweighs the very small incremental risk.

What does a heart scan cost? How about insurance coverage?

As the number of scanners capable of performing coronary calcium scoring grows, the price of getting a scan has plummeted. In many cities, you can get a heart scan for $200 or less. Though this may seem a lot at first, compare this to the cost of a very similar (often lower quality) CT scan of the chest performed in a hospital. Price? Around $4000! Heart scans are a bargain.

Just because a test provides valuable information doesn’t mean your insurance will cover it. Insurance coverage for EBT or MDCT scans is variable, depending on the state you live in and other factors generally beyond your control. You may encounter a wide range of responses from your insurance company from outright silly comments like “We don’t cover experimental procedures” (it’s not experimental, of course), or you may be fully covered. It helps to ask ahead of time. It also helps to have a doctor’s order for a scan, since this is sometimes a factor in your insurer’s decision. (Some states require a physician’s order to obtain a scan, while other states permit you to undergo a scan without a physician’s order; just ask the scanning center you’re planning to use.) Some employers have special arrangements for their employees to receive a scan at a discount. Even if your company does not have such a program, many employers who’ve suffered the loss of an employee or borne the extraordinary
health insurance expense of employees with heart disease will be receptive to making some kind of contribution.

Many people ask, “If heart scanning prevents heart attacks and expensive heart procedures, why won’t health insurers cover it?” First of all, from an insurance perspective, prevention of disease can be very expensive. You may need to treat 20, 30, maybe 50 people to prevent a single heart attack over a two or three-year timeline. Even if prevention of the disease costs only a tenth of the cost of “catastrophic care,” e.g., bypass surgery, the greater number of people treated to prevent the process erodes any savings. Secondly, many people change insurance companies every two to three years. Why would company X spend money to prevent a heart attack and expensive procedures 10 years from now when you will likely be insured by company Y by that time? It makes no financial sense from the perspective of your insurance company to prevent your heart attack—even if we knew for certain it would occur in, say, 7 or 8 years.

Even if insurance does not cover the cost of a heart scan, it is money well spent for the priceless information it can provide. For the cost of a new DVD player or dinner for four, you can obtain high-tech insight into your heart health that just might save your life.

Summary

Getting your coronary calcium score with an EBT or MDCT scanner is Step 1 in your personal Track Your Plaque program. These technologies offer a safe, convenient, and inexpensive method of quantifying plaque in the coronary arteries. The calcium “score” provided by these tests provides you with an indirect, though extremely reliable, gauge of the plaque present in your coronary arteries. The higher the score, the greater the quantity of plaque in your coronary arteries, and the higher the risk for heart attack and other "cardiac catastrophes.”

The coronary calcium score can be used as a “yardstick” for plaque and can be tracked for its increase, stabilization, or decrease.
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Chapter 6

Can I reduce my heart scan score?

*If we can accurately measure coronary plaque, we should aim to reduce it.*

Like a weed in your garden, coronary plaque can grow rapidly. With growth unchecked, plaque scores increase, on average, at a rate of 30–35% per year (Janowitz 1991; Maher 1999; Budoff 2000; Raggi 2005). A starting score of 100 will become 130, 169, 220, 286, 371, . . . kaboom!!

Heart attack doesn’t necessarily occur once a specific heart scan value is exceeded, but the *likelihood* of heart attack escalates along with the score. It’s like building a house of cards: the more cards you stack, the shakier the structure, until you add that final card and the whole thing collapses. Just as each additional card takes you one step closer to a final unstable point, *growing* plaque is *unstable* plaque; the more plaque that accumulates, the less stable the structure, the more likely a rupture develops.

If you know your heart scan score, your future is at a crossroads. One path leads to life with a score that doesn’t increase (or decreases) versus another path with your score increasing the expected 30-35% per year. How different is your future between the two paths? Even in the ensuing two years, an increasing score means your heart attack risk rockets *20-fold*. It means you’re getting closer and closer to that day when catastrophe strikes. In contrast, a stable or decreasing score means high likelihood of remaining *free* of heart attack and major heart procedures.

Can you reduce your score? Most people can, given the proper tools, adherence to the program, and sufficient time.
What does it mean to reduce your score?

When a heart scan score is held stable or is reduced, this is evidence that, not only is plaque no longer growing, it is being inactivated. (Plaque activity, i.e., plaque inflammation, enzyme production, metabolic activity, etc., cannot be directly measured in a live human, so we need to rely on indirect methods.) A reduction in heart scan score means that you have reabsorbed fatty tissue in the plaque, shrunk plaque size, turned off inflammatory processes and enzymes, and extracted some of the calcium. When plaque is inactivated, it is far less likely to rupture and cause heart attack.

You and I can live happily with plaque. We just don’t want plaque that is growing and potentially rupture-prone. A stable or reduced heart scan score can be viewed as indirect evidence of plaque inactivation. Inactive plaque is far less likely to rupture, to cause heart attack and other catastrophes.

How do I know if my score has increased or decreased?

How do you know what plaque is doing—shrinking or growing? Simple: get another scan.

Many people ask: Doesn’t having a “perfect” cholesterol value with treatment guarantee a reduction in score? Unfortunately, it does not. How about perfect lifestyle—strict adherence to diet, vigorous exercise, adequate sleep, etc? This won’t guarantee that plaque shrinks, either. Cholesterol values, even lipoproteins (discussed in chapter 8) are only starting points to identify potential tools to shrink your plaque. The only way to measure results in a specific individual is to re-measure the quantity of plaque present: get another scan. Reducing cholesterol, eating healthy, etc. are indeed helpful and enhance the likelihood of stopping your score, but no specific measure guarantees it.

In fact, there is nothing that truly tells you what your score is doing except... another score. Tracking your plaque is therefore a two-scan experience. There is no way to accurately and reliably predict what your score has done without looking at the score again.

The Track Your Plaque 5 Stages of Reversal

I break the course of tracking plaque down into five distinct phases, what I call “The Track Your Plaque 5 Stages of Reversal”:

Stage 1 Deceleration: Slowing plaque growth to ≤30% per year

It’s an unambitious goal, but a modest effort can slow growth to below the “natural” rate of 30% annual growth. This is the rate of growth experienced by many people who take statin cholesterol drugs (Lipitor®, Zocor®, etc.) as sole
strategy for combating heart disease.

Slowing growth to less than 30% per year is regarded as an unsatisfactory result in the Track Your Plaque program, one that can be improved substantially. While this represents an improvement over natural or accelerated plaque growth, substantial risk for heart attack persists with this level of growth.

**Stage 2 Deceleration:** Slowing plaque growth to ≤20% per year

A modest reduction in heart attack risk occurs when growth of plaque is slowed to 20% or less per year, but remains above 10%. Stage 2, like Stage 1, is also a typical result for someone who does nothing but adds a statin cholesterol drug and follows a conventional (e.g., American Heart Association low-fat) nutritional program.

While existing data suggests that achieving Stage 2 Deceleration modestly reduces the risk of heart attack and the likelihood of heart procedures, there’s plenty of room for improvement.

**Stage 3 Deceleration:** Slowing plaque growth to ≤10% per year

Now we’re starting to have a real impact. When plaque grows at 10% or less per year, it hasn’t stopped, but has slowed considerably from its “natural” rate of growth. Plaque growth rates of 10% or less per year are associated with a substantial reduction in heart attack. Achieving this rate of growth should prompt the question, “What could I do just a little bit better?”

**Stage 4 Zero Growth:** Zero percent plaque growth per year

This means that plaque growth has halted. Even though plaque has not reversed, zero percent plaque growth is associated with dramatic reduction in risk for heart attack (Raggi 2005). This is probably due to the fact that, while calcium has not changed, the active elements in plaque, like inflammatory cells and fatty material, have been reabsorbed, resulting in reduced potential for plaque rupture. In my experience and in published experience, the likelihood of a heart attack is virtually zero at this stage.

**Stage 5 Reversal:** Reducing your heart scan score

This is the “holy grail,” the goal we seek. It’s the prize that has tantalized the hopeful who’ve been misled into dead ends like low-fat diets, chelation, and other blind alleys. When achieved in the Track Your Plaque program, it is truly an enormous personal success that I would equate with graduating college, getting married, or being cured of cancer.

Reducing your heart scan score signifies that coronary atherosclerotic plaque has reversed—it is smaller in volume. All the components of plaque have diminished, including inflammatory cells, fatty tissue, and calcium. It also means that plaque has been essentially inactivated, its potential for rupture virtually shut down. It also means that your risk for heart attack is zero. In other words, in all
practicality, heart disease risk has been eliminated. It also means that, although plaque is still present, the fatty portion of plaque has been replaced by solid structural tissues that allow plaque to exist quietly without inflammation and without activity that triggers rupture. A decreasing heart scan score provides powerful indirect evidence that plaque has become stable and inactive.

In my experience, the majority of people who adhere to Track Your Plaque can slow or completely stop the otherwise inevitable increase in score, though the time required to do so may vary. In the first year, if all the proper steps are taken, a very realistic goal is to achieve an increase in score of no more than 10% (Stage 3 Deceleration). The existing data suggest that a score increase of <10% represents low-risk, and heart attack becomes less and less likely as your plaque is inactivated.

A zero-percent increase or decrease in score is more commonly encountered after two years on this program. Obtaining a reduction of score with present treatments is therefore a one to two-year long process for most participants.

It is important to point out that the lower your starting score, the more easily it is reduced. Scores of 200 or less have a greater chance of being reduced in the first year than scores >200. In our program, 70% of people with starting scores of 200 succeed in the first year. This drops to 30% success in the first year if your score is >200, 50% by end of year 2. The message here is clear: the earlier you start to Track Your Plaque, the more control you will have over your heart’s future. Nonetheless, if you start with a higher score, don’t give up hope. You may have to work harder and be patient, since this process requires at least two years for most people to enjoy substantial score-reducing or slowing effects.

Certain groups of people can anticipate greater difficulty in controlling their score. People with established diabetes will encounter more of a struggle. Unfortunately, if you’ve already been diagnosed with diabetes, reducing your score is less likely. The Track Your Plaque principles still do represent the most powerful prevention program you can follow, but it is more likely that you simply “decelerate” your plaque growth with these efforts, rather than achieve score reduction as long as you remain diabetic. (However, we will discuss how diabetics can supercharge their plaque control effort using our unique Track Your Plaque nutrition principles that reduce the diabetic tendency, many times reverse it.

People with the metabolic syndrome who have a combination of low HDL, high triglycerides, high blood pressure, blood sugar levels >110 mg/dl, and are overweight, will also struggle to control plaque. The metabolic syndrome generally precedes the onset of full-blown diabetes but has a similar, though lesser, impact on plaque. The most powerful tool for control of plaque growth for
many people like this is weight-loss achieved through the strategies discussed later in our *Track Your Plaque* Nutritional Principles. It is possible to control plaque with uncorrected metabolic syndrome in the picture, but it can be considerably more difficult.

Once score stabilization (zero change) or reduction is achieved, the need for any future scans to detect additional change is really an individual decision. Since the score has started to drop, the most important goal has been achieved. It is worth considering another scan, however, if there is some significant change in your program. For instance, significant weight gain, reversal of diabetes, or a prolonged period of treatment interruption are among reasons for repeating a scan despite initial control of the score.

The *Track Your Plaque* Study

In 2008, along with nutrition scientist, Dr. Susie Rockway, and statistician, Dr. Mary Kwasny, both of Rush University Medical Center, we published a portion of the *Track Your Plaque* experience (Davis 2008).

In this group of 45 participants, within 18 months 20 participants achieved a reduction in heart scan score of 14.5% (mean), while 22 participants experienced zero change in score. Of the 45 participants, only 3 experienced an increase in score. One participant, a 52-year old woman, achieved an incredible 64% reduction in heart scan score, among our best outcomes to date.

In other words, 42 of 45 participants, or 93%, for all practical purposes eliminated risk for heart disease by halting or reducing their heart scan scores.

Why such a small number of participants? Actually, prior to publication of this study, we had enrolled several hundred people in the program. But once vitamin D was added to the *Track Your Plaque* program, we began to witness faster and larger reductions in heart scan scores in a greater proportion of participants. So this small study included only the modest number of participants who had been taking vitamin D for the duration of the study, but did not report the several hundred people who had participated “pre-vitamin D.” (There’s much more on the crucial role of vitamin D in plaque control later in the book.)

Since publication of the study, we have improved the *Track Your Plaque* program even further by adding new strategies that have potential to achieve even greater reductions in heart scan score in more people. More on that to come.
What if my score doesn’t stop going up?

What if your score fails to stabilize and continues to increase, even after two years of effort? Does this mean heart attack is inevitable? Should you just throw up your hands and schedule your hospitalization?

No, absolutely not. But it is worth taking this increase very seriously. In the absence of symptoms, you and your doctor may need to repeat lipoprotein analysis to be certain you are achieving the desired endpoints. If you are not at the recommended endpoints, changes in your program will be necessary to achieve or maybe even exceed endpoints. You should also re-examine lifestyle changes of diet and exercise (detailed in the next several chapters). A lax approach to diet and exercise are common reasons for imperfect control of plaque.

If you’ve started with conventional lipid analysis rather than lipoproteins, you and your doctor should consider obtaining the more comprehensive and powerful lipoprotein analysis to identify hidden deficiencies in your program. Usually, your score will fall in line with some additional tweaking of your program.

Let’s go on to Step 2 of Track Your Plaque, in which we focus on identifying the causes of coronary plaque.

Summary

Unchecked, coronary plaque grows at the alarming rate of 30–35% per year.

Being able to measure plaque precisely through coronary calcium scoring provides a means of tracking and controlling the growth of plaque. Tracking the heart scan score provides powerful feedback on your ability to halt or even shrink plaque. This opens a whole new age of potential coronary plaque control and reversal.
References:


Step 2
Chapter 7

“My doctor said my cholesterol was fine . . .
So why did I have a heart attack?”

Cholesterol values are a crude risk measure, not a measure of the disease itself. High, low, or in between, no cholesterol measurement, calculation, or other manipulation can serve to identify coronary plaque.

Rick, a successful real-estate developer, has religiously had his cholesterol values monitored every six months since age 38. The numbers fluctuated within a narrow range with LDL cholesterols that never exceeded 95 mg/dl. “Excellent, as always,” his family doctor declared.

But a heart attack struck Rick down without warning at age 54, leaving him breathless and fatigued, as well as anxious for his future. It caused him to demand, “My doctor said my cholesterol was fine. So why did I have a heart attack?”

And Rick deserves an answer.

The answer is that there are many ways to develop heart disease. The list of potential causes of coronary plaque is long. In fact, most people with coronary disease have five, six, or more reasons to have plaque. High cholesterol might be just one cause on this list, and many patients with extensive coronary plaque have low cholesterol values. Yet most of the time your doctor makes a myopic attempt to decide whether you have heart disease by looking only at your cholesterol.

Measuring plaque brings the picture into sharp focus. If you have an abnormal heart scan score (any score above 0), you know that you have
coronary plaque. The higher the score, the greater the quantity of hidden plaque. The greater the quantity of hidden plaque, the greater your future heart attack risk. We don’t need “risk factor” assessment to tell us this. Measuring plaque is therefore Step 1 of the Track Your Plaque program.

We now proceed to Step 2 of the Track Your Plaque program: recognizing that there are causes of plaque beyond cholesterol testing.

Who needs cholesterol?

“Now wait a minute,” you might say. “If we use calcium scoring to detect coronary plaque, can we just throw cholesterol away?”

In a way, that’s right. Once your heart scan score shows that you have coronary plaque, we no longer need cholesterol to make this initial decision. It doesn’t matter whether your cholesterol is 150 mg/dl or 350 mg/dl. If you have coronary plaque, you have coronary artery disease and potential for heart attack in future—period. In Track Your Plaque, we don’t need risk factors to tell us who will or who won’t have a heart attack. We also won’t rely on the calculations intended to improve on single measures, like the “Framingham risk calculator” or the cholesterol/HDL ratio. If you have a calcium score >0 (or if you’ve had coronary disease identified by some other means like heart attack, angioplasty, stent(s), or bypass surgery) then and only then can we begin to discuss how plaque got started in the first place. (The only time we use various risk factors to identify future risk of heart attack is when your heart scan score is zero. In other words, perhaps you are 28 years old and your heart attack is still 30 years away. You have no measurable coronary plaque, but risk factor assessment can still be used as a crude means of anticipating your long-term statistical risk for heart disease.)

Should we completely discard cholesterol measures like total cholesterol, LDL cholesterol, and HDL cholesterol? No. Despite their shortcomings, these simple measures still have their usefulness. If we try to establish “cause and effect” issues leading to heart attack, then cholesterol testing helps identify some of the “causes,” even though they do not necessarily identify the “effect” (coronary plaque).

Risk factors are therefore primarily useful as tools for treatment. LDL cholesterol is less a measure of risk, but a more effective tool for management. Even though your LDL cholesterol doesn’t tell us whether you do or do not have coronary plaque, reducing it can nonetheless help control the plaque. But it doesn’t end there. If treatment of coronary disease were as easy as treating your LDL cholesterol, lowering LDL cholesterol would cure heart disease. It does not.

Treating LDL cholesterol does not cure heart disease because there are many other ways to develop heart disease besides LDL cholesterol. Let’s put
aside for a moment obvious causes like smoking, hypertension, and diabetes, and focus on causes that are hidden. Sometimes other causes can be suggested by the conventional cholesterol panel, such as low HDL cholesterol. Other times, the causes are not at all evident in the cholesterol panel and can only be uncovered through additional testing.

I’ll use Lisa’s story to illustrate.

Lisa is a 43-year old wife and mother of two teenage children. She works as a radiology technologist at a local hospital. Lisa has lived with growing concern over her future because her father suffered a fatal heart attack without warning at age 59. More recently, Lisa’s older brother, age 45, had a second heart attack. Thankfully, he survived and had a stent placed in one of his coronary arteries. Lisa was highly motivated to not suffer the same fate and avoid heart procedures. She therefore had a heart scan. Her score: 449, in the worst 1% of all women her age (99th percentile). In fact, Lisa had the highest score I’ve ever seen in a woman less than 50 years old.

Lisa has an upbeat, pleasant personality. She is a non-smoker, thin and physically active. At the start of the program, her LDL (“bad”) cholesterol was 68 mg/dl, HDL (“good”) cholesterol a very wonderful 74 mg/dl—all without treatment.

After a more thorough analysis, Lisa proved to have an inherited abnormality called “lipoprotein(a),” or Lp(a). This, in fact, was the only abnormality in an otherwise perfect lipoprotein profile. Given her high heart scan score, Lisa’s risk for heart attack over the next year was 4.5%, with virtually 100% chance of heart attack or unstable symptoms sufficient to require bypass surgery or stents over a longer timeline. (Incidentally, Lisa also underwent a stress test, which was entirely normal. Had we relied on a stress test to tell us whether Lisa was at risk, we would have completely missed the boat.)

A heart scan succeeding in revealing the true extent of Lisa’s heart disease, while standard risk factors failed miserably. Though most people like Lisa have a half-dozen reasons for plaque, Lisa was an exception in this regard, having only a single compelling lipoprotein reason. (There are causes for coronary plaque beyond lipoproteins, such as vitamin D deficiency, that we will also be discussing later.) Many of the hidden causes are simply not revealed by standard cholesterol testing. Identifying the cause for Lisa’s plaque allowed us to develop an effective program to stop or reduce coronary plaque.

Let me tell you about Bob.

Bob is a 49-year old mason who owns a small construction company with his brother, Dave. Bob is slender and muscular due to the demands of his work, laying brick and cinder blocks much of his day. Bob smoked for about 14 years, but quit in his early 40s. His family doctor had identified LDL cholesterols in the
200 mg/dl range and Bob had been taking the cholesterol drug, simvastatin, for about three years. I had been seen Bob’s brother, Dave, who I met after a heart scan showing a moderate quantity of coronary plaque. So Bob wanted to learn more about the process used in Track Your Plaque. Both brothers were motivated to avoid their dad’s fate, a bypass operation in his early 60s that scarred him with an infected chest incision, chronic leg edema (swelling), and a faulty memory.

So Bob underwent a heart scan. Sure enough, his score: 939—very high and in the 99th percentile compared to other men his age. (Annual risk for heart attack 4.5%.) Bob’s score, in fact, outstripped his older brother’s score by a wide margin. Bob complained of some aches and pains in his chest and shoulders that he suspected were due to the muscular strain of his work, but I had Bob undergo a stress test anyway to help understand the meaning of his symptoms. The stress test was normal, suggesting that Bob’s symptoms were unlikely to be from coronary disease.

But Bob had extensive plaque. Where did it come from? Was his prior high LDL cholesterol and prior smoking sufficient explanation? Would reducing LDL with simvastatin and not smoking be enough to keep his heart scan score from climbing higher?

A more thorough assessment of Bob uncovered eight additional causes for his high score, involving abnormalities of several lipoproteins, as well as several non-lipoprotein measures. Based on these results, I added several treatments to Bob’s simvastatin, including counseling on specific changes in his diet to help counteract the effects of some of his lipoprotein patterns. One year later, another heart scan score showed a 12% increase in score, representing a marked “deceleration” over the expected increase.

Bob’s high score was the result of multiple causes, some genetic, some lifestyle-related. A high score like Bob’s in a relatively young person, even in a former smoker, is nearly always caused by multiple factors.

**Why does coronary plaque develop in the first place?**

Let’s talk for a moment about the causes of coronary artery disease, the various ways plaque can accumulate in coronary arteries and lead to heart attack. This may help you understand why lipoproteins and other measures beyond cholesterol more accurately identify causes of coronary plaque and heart attack.

The development of atherosclerotic plaque is a complex, multi-faceted process that begins in childhood. It is not entirely clear what the initial inciting event might be, but it probably involves injury to the thin-walled inner lining of the artery. Injury can take many forms: high blood pressure, nicotine, high blood
sugar, deposition of abnormal lipoprotein particles, etc. Cholesterol has been a focus because it is found in abundance within plaques (in various forms such as crystalline and esterified, as well as larger blobs with a toothpaste-like consistency, called “lipid pools”) and it can be used as a crude index to characterize lipoproteins in the bloodstream. This led to the over-simplified assumption that high levels of cholesterol in the diet and in the blood caused cholesterol to be deposited within the artery wall, forming plaque. As we will discuss later, it is not high dietary or blood cholesterol level that encourages deposition in plaques, but the type of particle that delivers the cholesterol and exerts other effects. This is the rationale for studying “lipoproteins,” or the blood protein particles that carry cholesterol, triglycerides, phospholipids, and proteins.

Plaques are hotbeds of internal activity. They produce proteins that attract inflammatory white blood cells (called monocytes and macrophages) that infiltrate the plaque, much as they would any wound. Inflammatory cells produce digestive enzymes that weaken plaque structure. The enzymes inadvertently degrade the covering “cap” of the plaque that separates plaque contents from blood. The cap is then weakened and can “rupture,” exposing plaque contents to blood. This is a highly blood clot-promoting situation and is the cause for heart attack in most cases. Plaque inflammation is a hot area for research. One way to indirectly assess the contribution of inflammation to plaque activity is the measurement of “C-reactive protein” in the blood. (More on that later.)

As plaque grows, small bits of calcium are formed, sometimes as little “pebbles” and sometimes as larger “arcs.” This, of course, is the basis for calcium measurement to indirectly measure plaque volume. The larger the plaque grows, the more calcium accumulates (occupying 20% of total plaque volume), the higher your “score” (Rumberger 1995). The reason why calcium is deposited in the first place, however, is not clear.

Circulating HDL (“good”) cholesterol particles in the blood are responsible for a process called “reverse cholesterol transport,” in which HDL particles extract cholesterol from plaque for disposal in the liver. HDL particles are therefore crucial participants in the process of plaque “regression.” For Track Your Plaque, HDL becomes a very important tool. In fact, HDL is one of the most important parameters to measure and correct.

Because blood clot formation is required to suffer a heart attack, people who are prone to blood clotting are at higher risk for heart attack once a plaque ruptures. It is the basis for the reduction in heart attacks provided by aspirin, omega-3 fatty acids from fish oil, and other agents that inhibit the clotting of blood platelets.

To date, there are approximately 300 different known risk factors that contribute to plaque formation and blood clotting. It becomes impractical to try and “fix” each and every one of these risk factors. Fortunately, many therapies
that we will be using tend to improve multiple causes. One great example is fish oil that provides omega-3 fatty acids. Fish oil is a simple, inexpensive supplement that reduces triglycerides, reduces the clotting protein fibrinogen, has anti-inflammatory activity, reduces the "stickiness" of inflammatory cells to the plaque wall, reduces postprandial (after-meal) lipoprotein levels, and can reduce the risk of death from coronary disease by stabilizing the heart's potential for unstable rhythms. One treatment, multiple benefits.

**Lipids and Lipoproteins**

When your doctor says your cholesterol is high, what exactly does that mean?

Cholesterol is an *oil-based* substance; blood (actually plasma, the clear fluid that remains when red blood cells are removed) is a *water-based* liquid. Cholesterol does not float freely in your blood. If it did, it would separate just like oil and vinegar. This, of course, does not occur in your blood stream. Cholesterol travels in the blood as a “passenger” on a family of protein particles, called “lipoproteins” (meaning lipid-carrying proteins). This allows cholesterol to be soluble in blood. It also permits proteins to steer the lipoprotein particle to target the liver, plaque, or other places in the body. Proteins also determine to what degree its passenger lipid will interact and be deposited into plaque. In other words, it is the protein component of the particle that determines the behavior of the lipoprotein particle. The cholesterol component just goes along for the ride.

When your cholesterol is tested in your doctor’s office or hospital, the amount of cholesterol present is measured; the protein component is ignored. It would be like letting salad dressing separate and then measuring how much oil you have at the top. But this approach assumes that all cholesterol is the same and ignores the critical fact that behavior of a cholesterol particle depends on its protein partner. When you are told that your LDL cholesterol is 150 mg/dl, this means that the amount of cholesterol in the LDL (low-density lipoprotein) fraction is 150 mg/dl (150 milligrams in a deciliter, or 100 milliliters, of blood). The problem is that the low-density lipoproteins in this fraction are actually composed of a *varied mixture of particle types* that differ in their potential for causing heart disease. You simply cannot judge this from knowing that the LDL cholesterol is 150 mg/dl.

In other words, LDL cholesterol is really just a *convenience of measurement*, a crude and indirect means to gauge the behavior of LDL particles.

Likewise, when you’re told your HDL is 45 mg/dl, this really means that the cholesterol in the HDL fraction of blood is 45 mg/dl. Like LDL, HDL is also a heterogeneous mixture of particles with varying behavior not revealed by HDL cholesterol testing.
Unlike cholesterol values, analyzing lipoproteins in your blood provides insight into what particles are present to trigger the processes that cause plaque. In the past, measuring lipoproteins was a cumbersome process that was available only in research laboratories. But over the last 20 years, testing technology has advanced considerably and several methods to measure lipoproteins are now widely available.

Lipoproteins change the whole language of cholesterol and factors that cause coronary plaque. The most commonly used methods used to measure lipoproteins are gel electrophoresis, ultracentrifugation or vertical auto-profile (VAP), and nuclear magnetic resonance (NMR) spectroscopy.

Drs. Ronald Krauss and Robert Superko of the University of California-San Francisco are among the two leading physician-researchers who have explored and popularized the use of electrophoresis to study lipoproteins. In this technique, blood (plasma) is placed in a gel subjected to an electrical field. Lipoproteins migrate through the gel in patterns that depend on size and electrical charge of the particles. The amount of lipoproteins within various classes is then measured. This method is well-researched and supported by thousands of scientific publications.

The vertical auto-profile (VAP) is a technique that uses a modified version of the traditional method of spinning plasma at very high speeds ("ultracentrifugation") to separate blood particles by density, a technique developed by Dr. Jere Segrest at the University of Alabama.

A newer technique is NMR spectroscopy. Using a device similar to that used to image the body (MRI), a magnetic field is applied to blood (plasma). Biochemist Dr. James Otvos of LipoScience, Inc. developed the background science to understand how a magnetic field reveals the kinds and amounts of lipoproteins present. NMR is quick (performed in minutes), not dependent on the skill of the technician (who simply inserts the blood sample into the device), and is relatively inexpensive. Even after using this technology for several years, I am still in awe at the genius of this elegant technique.

Each testing method has its pluses and minuses, but all reveal aspects of lipoproteins that are simply not evident through cholesterol testing.

Can I “get by” with just lipids?

Ideally, you begin your program with a full “map” of all plaque causes, including one of the advanced lipoprotein tests listed above. This will allow you to develop the most effective plaque-control program right from the start.
If you are unable or unwilling to have your lipoproteins tested, what sort of success can you expect by relying on lipids alone? This varies, depending on whether you do or don't have multiple causes of plaque not revealed by lipids (such as lipoprotein (a), small LDL, etc., all discussed in the next chapter). It is reasonable, however, to begin with a therapeutic program based on cholesterol testing results. You and your doctor can then tailor your treatment based on the Track Your Plaque concept of a repeat heart scan score. In another words, if your score stops increasing or goes down based only on cholesterol management, you’ve succeeded. But if your score goes up, particularly >20% per year, you should re-consider having lipoproteins performed to design a better treatment program. The goal remains the same: Keep your score from increasing.

Another reason to consider lipoprotein testing: It is your best chance of gaining control over plaque without medication. This is because lipoprotein testing, specifically testing for small LDL particles, can predict your response to specific changes in diet. Simple cholesterol testing can do this in only the crudest manner.

The concept of lipoproteins represents a major advance in understanding the causes behind atherosclerotic coronary disease. As powerful as lipoproteins are, do they tell you whether or not you have coronary plaque present? No, we still rely on Step 1 of Track Your Plaque, your heart scan score, to tell us this. Lipoproteins represent an important component of Step 2 of Track Your Plaque and provide insight into the causes of heart disease that are commonly not revealed by standard lipid testing. They provide powerful tools to gain control of your coronary plaque.

**Vitamin D: Superhuman heart nutrient**

If there is one vital nutrient for heart health, it's vitamin D—hands down.

For a variety of reasons that we will discuss, vitamin D deficiency is the rule, not the exception. The majority of people leading modern lives will, at least part of the year if not year round, be deficient. Vitamin D enters the heart health picture at a number of places, including improved lipid patterns (striking increases in HDL, reduction in triglycerides), improved insulin responses, reduced blood sugar and potential for diabetes, anti-inflammatory effects, and direct effects on plaque tissue.

I added vitamin D to the Track Your Plaque program in late 2004. Because the results were so incredible—increasing the number of people who halted or reversed their heart scan scores—vitamin D quickly became one of the crucial components of the Track Your Plaque program. Not only did more people stop or drop their heart scan scores by larger margins than ever before, they also
experienced increased energy, better coordination and mental clarity, and improved bone health (arthritis and osteoporosis).

Because vitamin D is so important to the *Track Your Plaque* program and your overall health, I have devoted an entire chapter to it.

**The Thyroid-Plaque Connection**

Like vitamin D, thyroid normalization is a recent addition to the *Track Your Plaque* program.

Over the past few years, large clinical studies documenting the important relationship between thyroid and coronary disease have emerged: Even the most *marginal* low thyroid (hypothyroidism)—even without symptoms—can double or triple heart attack risk and plaque growth. In *Track Your Plaque*, we aim for *perfect* thyroid function.

In addition to feeling more energetic with clearer thinking and better mood, perfect thyroid function also yields more rapid weight loss, improved cholesterol patterns (especially reduced LDL cholesterol), and reduced blood pressure. Chapter 11 is devoted to a detailed conversation on how thyroid normalization fits into your plaque control program.

Put it all together and you get a program that has potential to return control over coronary plaque and heart disease to you. It might involve cholesterol reducing drugs, it might not. It involves several counterintuitive changes in diet. It might involve easier-to-achieve-than-you-think weight loss. It will definitely involve several critical nutritional supplements. Hopefully, it includes the willing participation and eagerness to learn by your doctor; it might not. But it all adds up to an effort that puts far greater control over heart disease risk than you ever thought possible.

In chapter 8, I elaborate further on the components important to Step 2 and also proceed to Step 3 of the *Track Your Plaque* program and discuss the individual lipid and lipoprotein risk factors that can be measured and become tools for treatment for your plaque control program.
References:
Step 3
Chapter 8

It’s not just about cholesterol:
The many causes of plaque

*Plaque usually requires five, six, seven, or more reasons to develop. Identifying as many causes as possible is an important step in gaining control over plaque.*

Step 2 of the *Track Your Plaque* approach is to pinpoint *all the causes* of your coronary plaque. Twenty years from now, we may learn that only 75% of all causes of plaque have been identified today, and that several more have yet to be discovered. *Track Your Plaque* represents a collection of all that is *currently* known about coronary plaque, its causes, and strategies to stop or reduce it. Because knowledge is likely to change in the coming years, think of the *Track Your Plaque* approach as a work in progress—but the most advanced and effective for its time.

We start with lipid and lipoprotein testing. Under no circumstance do these tests tell us whether or not plaque is present, but tell us how plaque might get its start and how it might continue to grow. We then use treatment of lipid and/or lipoprotein abnormalities as the hammers and chisels to chip away at your coronary plaque, which is Step 3 of *Track Your Plaque*.

Your treatment program will depend on the type and severity of the abnormalities identified through lipid and/or lipoprotein testing. This chapter serves as a starting place to appreciate the potential abnormalities that can be identified, as well as beginning a discussion about treatment. Step 3, correction of most lipid/lipoprotein abnormalities, is best undertaken with the involvement of
your physician or healthcare professional, so I would encourage you to discuss your treatment program with that person.

We'll begin with the measures included in the standard lipid panel: LDL cholesterol, HDL cholesterol, total cholesterol, and triglycerides. We'll then further explore the concept of lipoprotein testing for those of you interested in a more precise and powerful approach.

**LDL cholesterol**

Despite its deficiencies in identifying people with heart disease, LDL cholesterol (i.e., cholesterol in low-density lipoproteins) is still a useful tool for treatment. Advantages: widely-available and recognized by most people and doctors. Disadvantages: low reliability, even occasionally grossly misleading results. LDL cholesterol is also often perceived as the *sole* source of coronary risk, which is far from the truth.

Recall that LDL cholesterol is not measured, but *calculated* from the other three values in the standard cholesterol panel. The equation used for the calculation, the Friedewald equation, while simple, is built on some assumptions that, like many assumptions, sometimes contain truth, often do not. The Friedewald calculated LDL is therefore a crude tool, but occasionally useful. (We will be discussing superior methods to quantify LDL particles that do not involve making assumptions or calculations.)

Nearly everyone already has had at least one LDL cholesterol value. What is a desirable level of LDL if you have an abnormal heart scan “score”? Most authorities suggest that LDL cholesterol should be reduced to <100 mg/dl if you have a coronary calcium score >100 or percentile rank of 75% or greater identified by heart scanning. This is the same LDL target that applies just as if you had coronary disease identified via a history of heart attack, angioplasty, stent, or bypass surgery (Grundy 2004). In other words, your doctor might consider scores of >100 or percentile rank of 75% or greater as “coronary risk equivalents,” predicting the same risk for future heart attack as a history of previous heart attack.

In *Track Your Plaque*, we try even harder. The LDL level that heightens the likelihood of plaque shrinkage is <70 mg/dl. The majority of our participants who’ve succeeded in the program manage to keep LDL in the 60-70 mg/dl range. Reducing LDL cholesterol to this level does not guarantee that plaque will not grow, but it makes it far less likely.

Dietary strategies that reduce LDL cholesterol include avoiding hydrogenated fats, increasing intake of complex fibers such as those from raw nuts and flaxseed, and—surprisingly—elimination of wheat products. Elimination of all breads (whole grain and white), pasta, noodles, bagels, pretzels, crackers,
all breakfast cereals, pizza, and muffins yields unexpectedly powerful LDL reductions. (Wheat elimination is an especially incredible means to reduce LDL if the particles are small. Much more on this coming in chapter 9.) Raw nuts, such as almonds, pecans, and walnuts; pectin sources like citrus peels; and ground flaxseed are particularly effective fiber sources that can reduce LDL 10% or more. Ground flaxseed is a versatile fiber source that packs a substantial LDL-lowering punch when you add 2-3 tbsp per day to unsweetened yogurt, protein drinks, etc. or used as a hot cereal itself.

Statin drugs are effective for reducing LDL cholesterol. Statin drugs are, for many people, not a requirement to control your heart scan score, though they can make life easier getting your LDL to goal if you start with a LDL of >150 mg/dl. Reductions in LDL of 30-40% are easily achieved, with maximum effect of around 60%. Ezetimibe (Zetia®) is a non-statin prescription agent that reduces LDL around 18% used alone, or can be used to essentially triple or quadruple the potency of a statin drug. However, while ezetimibe undoubtedly helps reduce LDL, its effect on reducing atherosclerotic plaque has been questioned (Taylor 2009).

Red yeast rice is a nutritional supplement that can also help reduce LDL, but it is essentially a weak statin drug. It reduces LDL cholesterol similar to statin drugs, though rarely reducing it more than 30-40 mg/dl. Red yeast rice shares all the side-effects of the statin drugs, though perhaps to a lesser degree. It also suffers from tremendous variation from brand to brand. We have had good results with the Cholestin® brand.

Our experiences with purported LDL cholesterol reducing supplements like policosanol, pantetheine, and gugulipid have been disappointing. They are therefore not recommended for use in the Track Your Plaque program.

**HDL cholesterol**

HDL (i.e., cholesterol in the HDL fraction) represents a protective class of lipoproteins responsible for a process called “reverse cholesterol transport,” scavenging cholesterol from plaque. HDL is critically important for regression of plaque.

Low HDL cholesterol (below 40 mg/dl) is among the most common causes for heart disease. Of every 100 people with heart disease, at least 50 have a low HDL. It is the cause of heart disease in many people who’ve been told that either they have no reason for heart disease, or they have a cause that is untreatable. Both statements are untrue.

Low HDL is the tip of the iceberg for several other closely related abnormalities. People who have low HDL nearly always have a smorgasbord of small LDL particles, high triglycerides, and abnormal triglyceride-rich lipoproteins
called VLDL (very low-density lipoproteins). HDL is very sensitive to body weight. As you gain weight, HDL plummets, while HDL increases with weight loss. Low HDL can also be a marker of potential for hypertension (high blood pressure), glucose intolerance (“pre-diabetes”) and diabetes. (This combination is often called the metabolic syndrome.)

In Track Your Plaque, we aim to increase HDL to 60 mg/dl or greater. This appears to be the minimum level necessary to gain control of your score.

How do you increase HDL? Diet can help a great deal. But a standard low-fat diet does not raise HDL, and super low-fat diets reduce HDL, increase the number of small LDL particles, increase triglycerides and VLDL particles—all of which heighten risk for heart disease. Low-fat diets are, in fact, quite destructive influences on HDL and associated lipids and lipoproteins. People with low HDL or any of the other associated abnormalities do better with specific modifications in diet that we will discuss in more detail in the chapter 9, such as using foods rich in monounsaturated fatty acids (like raw nuts and olive oil), eliminating foods like wheat products that reduce HDL, and increasing protein and fat intake.

Vitamin D increases HDL, often spectacularly. The HDL-increasing effect is slow, however, often taking 1-2 years to develop. This and other benefits of vitamin D are so tremendous that there is an entire chapter devoted just to this important vitamin/hormone. (Yes, “vitamin” D is really a potent hormone.)

Exercise increases HDL. Going from a sedentary lifestyle to a moderately active lifestyle (e.g., walking 30 minutes per day 5 days a week) typically increases total HDL two to five milligrams/dl. Serious exercisers, e.g., triathletes and long-distance runners, can increase HDL 10 mg/dl or more. This effect lasts about two weeks after exercise stops before your HDL drifts back down. Exercise therefore needs to be consistent and long-term in order to prop up your HDL.

Omega-3 fatty acids (fish oil) can increase HDL modestly, but are more effective in shifting HDL to larger particle size. The treatments to raise HDL are identical to those that are used to treat the small LDL particle size (see below).

**Triglycerides**

Triglycerides are one of the four measures included in every standard lipid panel. But triglycerides can be a very tough customer to figure out: When they’re high, they may or may not cause coronary disease; when they’re low, they may or may not cause coronary disease. How do we make sense out of this jumble?

For the most part, we are interested in triglyceride levels of up to 400 mg/dl, as this is the range that tends to contribute to growth of coronary plaque. When triglycerides are greater than 400, your doctor will need to consider whether several other conditions might be to blame, such as unknown or poorly-
controlled diabetes, an underactive thyroid, kidney disease, or certain genetic disorders (e.g., familial hypertriglyceridemia). Levels this high may or may not contribute to heart disease risk (which can be decided when your doctor looks at your LDL particle number or apoprotein B, discussed below). In the rare instances when triglycerides >1000 mg/dl, the pancreas (in your abdomen) can suffer damage. Levels this high need to be urgently addressed by your doctor.

Triglyceride levels of 60-400 mg/dl are common and contribute significantly to plaque growth. Triglycerides are among the most neglected measures. What makes triglycerides bad? First of all, triglycerides are present in virtually all lipoprotein particles to various degrees, such as VLDL and small LDL. The higher the triglyceride level (up to 400 mg/dl), the greater the quantity of VLDL and small LDL, all of which are potent causes of plaque growth. Only when triglycerides are <60 mg/dl can you confidently predict that hidden triglyceride-containing particles are absent.

Diet is enormously important for gaining control over triglycerides. The conventional approach to reducing triglycerides is to reduce fat intake. Low-fat diets may work initially and reduce triglycerides modestly. However, triglycerides eventually increase on a low-fat diet, often substantially (Pleke 2000). We therefore do not follow a low-fat restriction to reduce triglycerides.

We start by reducing carbohydrates, especially those made with wheat flour, cornstarch, oats, and sugars. Replacing carbohydrate calories (from wheat flour- and cornstarch-containing products like breads, bagels, pasta, cakes, breakfast cereals, pancakes, waffles, pretzels, and crackers; corn chips, tacos, tortillas, and foods thickened with cornstarch; potatoes; white and brown rice; oatmeal and oat bran; candy and soft drinks) with protein and healthy fats like the monounsaturates from raw nuts, olive and canola oils, and the omega-3's from fish, will all help reduce triglycerides.

Reduction of carbohydrate intake is far more effective to reduce triglycerides, even though fats are composed of triglycerides, because the human liver has an enormous capacity to manufacture triglycerides from carbohydrates. Your liver’s capacity to make triglycerides and flood the blood with them far exceeds your ability to ingest fats and increase triglycerides. This is due to the process of **de novo lipogenesis**, the manufacture of triglycerides from sugars (Lee 2008).

Avoiding processed foods containing high-fructose corn syrup is crucial, as this ubiquitous additive (in everything from low-fat salad dressings to beer) increases triglycerides significantly. Other fructose sources to avoid include sucrose (table sugar, which is 50% fructose), maple syrup, honey, and agave syrup. Even excessive consumption of fruit, especially modern fruit that has been cultivated for size and sugar content, can lead to increased triglycerides; for this reason, in the **Track Your Plaque** diet program (chapter 9), we limit fruit
consumption to two small servings per day (a serving being what fits in the palm of your hand).

Fish oil supplements (but not flaxseed oil) can be used as specific treatment that dramatically reduces triglycerides. This approach can reduce triglycerides several hundred, even several thousand, mg/dl. Everyone is advised to take a fish oil supplement to provide a minimum of 1800 mg of omega-3 fatty acids, EPA and DHA (obtained by adding up the EPA and DHA content per capsule specified on the product label) with a target triglyceride level of <60 mg/dl. The dose may need to be increased long-term to reach this goal; rarely is more than 3000-3600 mg EPA and DHA per day necessary; higher doses exceeding 1800 mg per day should be undertaken with the assistance of your healthcare provider.

Prescription agents that reduce triglycerides include niacin (doses >500 mg, which should be monitored by your physician), the “fibrates” like gemfibrozil (Lopid®) and fenofibrate (Tricor®); and the statin agents, especially atorvastatin (Lipitor®) and rosuvastatin (Crestor®). However, following the above approach, drugs are rarely, if ever, needed to gain control over triglycerides.

### Jack and Walter: Same LDL cholesterol, very different fates

Jack has an LDL cholesterol of 124 mg/dl. “Not bad!” his doctor says. Jack’s friend Walter also has an LDL of 124 mg/dl. Jack has a major heart attack at age 53. Walter never has a heart attack.

How can two men with identical LDL cholesterol have such different fates?

Walter’s lipoprotein analysis showed normal LDL particle size and a low number of LDL particles. Jack’s analysis, on the other hand, showed abnormally small LDL particles and a high number of LDL particles—small particles and too many of them. (This can be measured as high LDL particle number or high apoprotein B, along with small LDL.)

Despite the very same LDL cholesterol, these two men had extremely different risks for heart disease, with Jack’s very high risk conferred by a high number of small LDL particles.

In the Track Your Plaque program, LDL cholesterol is regarded as a crude indicator, certainly not the only indicator. Better measures, like LDL particle number and apoprotein B, are available that should be considered.

### Total cholesterol

Total cholesterol is mentioned only for completeness—it is a measure that we do not use in Track Your Plaque, as it is a combination of 3 measures:

\[
\text{Total cholesterol} = \text{LDL} + \text{HDL} + \text{triglycerides}/5
\]

In other words, by simple arithmetic, if LDL or triglycerides go up, your cholesterol also goes up. But if your HDL goes up—a positive change—cholesterol again goes up. Low total cholesterol due to low HDL, in fact, is a frequent reason people are often judged to be low-risk for heart disease when they are actually high-risk. Total cholesterol is a combination of these other measures and is therefore too sloppy a measure for us to use. My personal view
is that total cholesterol should be removed from the four measures on a standard cholesterol panel, as it provides misleading information as often as it provides true information.

Let’s move on to discussing various lipoprotein measures, the blood tests beyond the four values in a standard cholesterol panel.

**LDL particle number**

Remember our discussion about how your doctor measures cholesterol? He/she essentially lets the oil (cholesterol) separate and measures how much there is to crudely gauge the quantity of a specific lipoprotein fraction present. Thus, cholesterol in the low-density fraction of lipoproteins yields “LDL cholesterol.” Cholesterol is therefore a convenience of measurement, a crude “dipstick” for lipoproteins. But this flawed approach neglects the fact that cholesterol really occurs as a passenger on lipoprotein particles, i.e, cholesterol is a passive component of a larger blood particle. Lipoprotein particles, even

<table>
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<th>The Track Your Plaque Rule of 60</th>
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<td>As easy rule-of-thumb that we use in Track Your Plaque that has survived the test of time is the Track Your Plaque Rule of 60.</td>
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From the perspective of basic lipids (LDL, HDL, and triglycerides), we aim to achieve 60:60:60, or LDL cholesterol of no more than 60 mg/dl, HDL cholesterol 60 mg/dl or greater, triglycerides of 60 mg/dl or less. Among the arguments in favor of these target values include:

- LDL cholesterol of 60 mg/dl has been associated with the greatest reduction in heart attack and coronary plaque reversal (Ballantyne 2008)
- HDL cholesterol of 60 mg/dl or greater ensures that a healthy proportion of truly protective large HDL is present. HDL at this level also ensures sufficient antioxidative effect from HDL protective functions (Freedman 2004).
- Triglycerides of 60 mg/dl or less maximizes the likelihood that plaque-forming triglyceride-rich lipoproteins have been minimized or eliminated (Freedman 2004).

While there is more to the Track Your Plaque program than achieving 60:60:60, this easy-to-remember “rule” can tip the scales heavily in your favor in achieving control over coronary plaque.

within a single class, i.e., LDL or HDL, can also vary substantially in size and behavior, regardless of cholesterol content.

One way to improve on this crude practice is to actually count the number of lipoprotein particles present in a given volume, say, per milliliter of blood.

Think of it this way: The more particles there are in your blood, the more likely they will enter the wall of the artery and contribute to plaque formation.
Some call this a “gradient-driven” process. You might recall from high school science class that when a concentration gradient is present, it will work towards achieving equilibrium to eliminate the concentration difference. The same principle applies when a higher concentration of LDL particles is in blood compared to the artery wall: The more numerous LDL particles in the blood infiltrate the artery wall in an effort to balance the concentration gradient.

LDL particle number is one of the most powerful tools available. It can be measured directly as “LDL particle number” (by the NMR method) or apoprotein B, which is more widely available (one of the measures available through the Berkeley HeartLab as well as many local laboratories throughout the U.S.). The Quebec Cardiovascular Study is among the studies demonstrating how LDL particle number (in this instance, measured as apoprotein B) was the best lipid/lipoprotein predictor of heart attack (St-Pierre 2005). In this study, heart attack still occurred even when LDL cholesterol was low but particle number was high. In other words, LDL particle number proved a better indicator of heart attack potential, even when LDL cholesterol was favorable (below the average range).

You cannot predict LDL particle number by just looking at LDL cholesterol. In other words, you can have a high LDL particle number with low LDL cholesterol, a low LDL particle number with low LDL cholesterol, etc. LDL particle number or apoprotein B needs to be specifically measured. How can LDL cholesterol be low when the particle number is high? The amount of cholesterol contained per particle varies widely. If your LDL particles have less cholesterol in each particle but there are many of them, the measured LDL cholesterol can

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**Low LDL cholesterol and high LDL particle number**

A very common situation is to have low or “normal” LDL cholesterol but a high LDL particle number. This situation can lead to plaque growth even though LDL cholesterol is not high.

A good example is Tim R. Tim is a 34-year old investment analyst. Although his parents (in their 60’s) didn’t have heart problems, his maternal uncles both had heart attacks in their 50’s. Tim underwent a heart scan. His score: 30—a low absolute score, but very high for his young age (75-95th percentile). A future of heart disease is inevitable unless we take action.

Tim’s lipoprotein profile (NMR) showed:

- LDL particle number: 2254 nm/l
- LDL cholesterol: 124 mg/dl
- HDL cholesterol: 31 mg/dl
- Triglycerides: 360 mg/dl

(Tim also displayed a complete shift of LDL particles to the undesirable small fraction.)

One of the principal causes of Tim’s coronary plaque was high LDL particle number even though LDL cholesterol was favorable (below the average range).

(See box.)
remain low—but your heart disease risk will be high (because of the high particle number and the resultant “concentration gradient.”)

There are many people with established heart disease who’ve been told their LDL cholesterol was fine and no cause for their heart disease could be identified. Many people like this have excessively high LDL particle numbers or apoprotein B.

You can still have a heart attack while being treated for high LDL cholesterol with a statin agent (Zocor®/simvastatin, Lipitor®, Pravachol®/pravastatin, Lescol®/fluvastatin, Mevacor®/lovastatin, Crestor®) if your LDL particle number is high. In other words, despite the appearance of a “good” response to a cholesterol-lowering agent, heart attack still occurs because the number of LDL particles is still excessive.

Reducing hydrogenated fats in your diet, cholesterol-reducing statin drugs, and several dietary strategies like raw nuts and flaxseed all reduce LDL particle number. If LDL particle number consists of a substantial quantity of small LDL particles (below), then elimination of wheat, cornstarch, oats, and sugars (see chapter 9) yields vigorous reductions in LDL particle number and apoprotein B. These are the same treatments that reduce LDL cholesterol. (See above.)

If you rely only on LDL cholesterol and neglect to measure LDL particle number or apoprotein B, you will be groping in the dark: You won’t know if treatment is required nor will you know if treatment is sufficient. At some point, you simply need to measure LDL particle number or apoprotein B to know. For participants in Track Your Plaque, our targets for treatment that enhance likelihood of stopping plaque growth are an LDL particle number of <700 nmol/L or an apoprotein B of 70 mg/dl or less.

Small LDL particles

Just like people, LDL particles come in a variety of sizes: some big, some small, some in-between. Small LDL is a crucial factor to identify when you are interested in understanding how plaque got its start and how you can seize control over it. Small LDL, in fact, is the number one most common abnormality identified in people with coronary plaque. People with coronary plaque very commonly have 50% or more small LDL particles. More than any other measure, small LDL particles are the basis for many of the strategies in the Track Your Plaque diet approach, as well as many of the other components of the program.

Children are most likely to have healthy, large LDL particles. Small particles become more common as we age. Small LDL can be an inherited trait unmasked by unhealthy lifestyles and excessive body weight. When the genetic trait is strong, it can even occur in slender people with healthy lifestyles. Small LDL particles can double or triple the likelihood of developing coronary plaque.
and heart attack. It is among the most common reasons for heart disease, being found in >70% of all people who eventually develop heart attack. It often occurs alongside low HDL, though not always. Some call small LDL “LDL pattern B,” as distinguished from “LDL pattern A” with predominant large LDL particles (though the precise cutoffs of percentage small vs. large in types A vs. B are debated).

Like LDL particle number, you can’t judge LDL size by just looking at LDL cholesterol. In other words, even if LDL cholesterol is low, particle size can be small—but it can still be responsible for building coronary plaque. Small LDL usually begins to appear when triglycerides are >70 or HDL <60. But even high HDL or low triglycerides do not tell you with absolute confidence whether or not small LDL is present. Like LDL particle number or apoprotein B, LDL size needs to be directly measured.

Small LDL particles are a powerful cause for plaque growth. Smaller particles more readily penetrate artery walls and deposit material into plaques. Small LDLs are also more adherent to the material that resides in plaque. Small LDLs are more prone to oxidation that leads to inflammatory responses. Because they are poorly recognized by the liver LDL receptor, they also linger in the blood longer than larger particles, providing more opportunity to do damage (Julius 2007). Small LDL particles are generally triggered by carbohydrates in the diet; they are also unusually prone to the process of glucose-modification, or glycation, which makes them even more susceptible to oxidation (Younis 2008). In the Track Your Plaque approach, we aim to keep small LDL particles at no more than 30% of total LDL.

While small LDL is a bad thing to have, it is usually easy to correct. The small LDL pattern is exceptionally weight-sensitive. Weight loss is therefore a powerful method of increasing LDL size and shifting LDL particles from small to large. The amount of weight loss required to correct LDL size varies widely. Some people can convert back to a large LDL size just by losing 10 pounds. Others may require more, depending on starting weight and genetic factors. Exercise also has a modest, though transient, effect, increasing LDL size.

If there is one dietary maneuver that can reduce the number of small LDL particles, it’s the elimination or dramatic reduction of wheat products. Yes, I know: You’ve been told to eat plenty of “healthy whole grains.” Nothing could be further from the truth. Wheat products have come to dominate the diet of many Americans: breakfast cereal with whole wheat toast for breakfast; a sandwich with whole wheat bread for lunch; whole wheat pasta for dinner; pretzels and whole grain crackers for snacks. A diet dominated by wheat, regardless of being whole grain or not, causes the small LDL pattern to explode, not to mention increases in visceral (abdominal) fat, blood sugar, pre-diabetes and diabetes, inflammatory responses, and triglycerides (Krauss 1995; Wood 2006). Ironically, “official” advice from the USDA, the American Heart Association, the American Diabetes Association, and the American Dietetic Association all advise generous
inclusion of “healthy whole grains” made from wheat flour. This is a recipe for disaster. In short, wheat is among the most destructive food ingredients you can eat.

To reduce or eliminate the small LDL pattern, as well as achieve dramatic reductions in LDL cholesterol, wheat elimination is a profoundly effective strategy if small LDL particles are present.

After wheat elimination, reducing exposure to other carbohydrates can further reduce small LDL particles. This means reducing or eliminating foods made with cornstarch (cornmeal, tacos, tortillas, chips, gravies, breakfast cereals, processed foods with added cornstarch), oatmeal and oat bran, sugars, and rice, beans, and potatoes.

Can you be at ideal weight, be vigorously active, even eliminate wheat and still have small LDL particles? Yes, unfortunately. While most people can eliminate the small LDL pattern, others can only minimize it. In other words, small LDL can be a genetic characteristic that is worsened by excess weight and inactivity, minimized with weight loss and diet. The pattern can persist at low levels in some people even when everything appears perfect. (Because we are not yet able to identify the gene that underlies formation of small LDL, we can only presumptively gauge whether a genetic tendency is present: If you achieve ideal weight and eliminate the foods that trigger small LDL, yet small LDL particles persist, then small LDL is likely to be genetically determined.)

That’s when we turn to treatments beyond diet. Niacin (vitamin B3) is an important strategy to correct LDL size. Doses of 1000-1500 mg are effective. Niacin is best taken under the supervision of your healthcare provider who has experience in dealing with the peculiar effects of niacin (most of them harmless), like feeling hot and itchy. Unfortunately, many patients and physicians are scared by these effects and give up. The best forms of niacin available are over-the-counter Sloniacin® and Enduracin® (available in pharmacies) and prescription Niaspan®. (I prefer Sloniacin® and Enduracin®, since they are 95% less costly than the prescription form.) These preparations release niacin over several hours, minimizing both liver toxicity and the hot flush. These “extended-release” niacin preparations should not be confused with the “slow-release” forms of niacin (yes, the Sloniacin® name is confusing—it is not slow release, but “extended-release”) that release niacin over much longer periods; these preparations have been associated with liver toxicity. Health food stores also sell “immediate-release” niacin, which, as the name suggests, releases niacin immediately, usually causing an intolerable hot flush; and “no-flush” or “flush-free” niacin, a form of niacin called inositol hexaniacinate, a version that, in my experience, doesn’t work at all—a scam product that you should avoid.

We start niacin at 500 mg per day, usually at dinner or a major meal, for the first 4 weeks, then increase to 1000 mg. If a further dose increase is desired,
this can be achieved in another 4 weeks. The slow increase in dose helps minimize the “hot flush” that can follow niacin about 30-60 minutes after taking the tablet(s). When the hot flush occurs, drink 2 glasses (8-10 oz) of water; this is usually sufficient to eliminate the flush. Please be advised that niacin is best taken under medical supervision. Liver side effects are rare with the Sloniacin®, Enduracin®, and Niaspan® preparations, but it is nonetheless wise to have your liver tested periodically by your doctor when you’re taking any form of niacin (usually every 6 months).

Some of the other components of the Track Your Plaque program exert modest effects on increasing LDL particle size. This is true of omega-3 fatty acids from fish oil, vitamin D, and achieving ideal thyroid function (to be discussed).

Should you have small LDL particles identified as one of the causes of your heart disease, set yourself on a course of action: Increase physical activity, eliminate the food triggers of the small LDL pattern (especially wheat, cornstarch, and sugars), weight loss when appropriate, supplements like fish oil and vitamin D, and consider niacin to increase the particle size and “turn off” this potent abnormality.

Just as you often cannot tell whether you have small LDL particles at the start of your program, you will likewise not be able to gauge whether it’s been eliminated until you measure it again. Another test of LDL particle size will eventually be necessary to assess treatment effects.

Large HDL

Like LDL, HDL is really a collection of different particles. We call the entire family “total HDL.” The truly effective HDL that participates in “reverse cholesterol transport” (shrinking plaque) is “large” HDL, sometimes also known as “HDL2b.” In other words, not all HDL is beneficial—only the large HDL particles are truly helpful. The smaller fractions can comprise a major portion of total HDL, and a seemingly favorable HDL of, say, 50 mg/dl can conceal a lack of effective large HDL particles.

Unlike LDL, you can make some preliminary judgments about HDL size based on total HDL. Above a total HDL of 60 mg/dl, large HDL is pretty confidently in a good range. Below 40 mg/dl and you can be confident that deficiency of large HDL is present. Between a total HDL of 40 and 60 is an indeterminate range, and you may or may not have a lack of large HDL. This is where measuring HDL size is most helpful.

In Track Your Plaque, we try to keep your large HDL at least 15 nmol/L by NMR, or 30% HDL2b by electropheresis (Berkeley HeartLab), or 30% HDL2 by VAP, aiming for a total HDL of 60 mg/dl or greater. (The differences derive from different definitions of HDL size.) You can increase the proportion of large HDL
by using the same treatment strategies as those used to correct the small LDL abnormality (above). A lack of large HDL is a relatively easy abnormality to treat, but you just have to recognize whether it’s present and whether it is fully corrected with your treatment efforts.

**Very low-density lipoprotein (VLDL)**

VLDL particles are lipoproteins produced by the liver, larger and richer in triglycerides than LDL particles. Triglycerides and VLDL particles are so closely tied together that, while it’s helpful to understand how and why VLDL particles participate in the processes that create plaque, control over VLDL is really the same as control over triglycerides.

VLDL loves to share its plentiful triglycerides with other particles, i.e., VLDL particles interact with other particles in the bloodstream. This is the process that leads to the formation of small LDL and small HDL particles. High triglycerides are not required to have excessive VLDL: you can have an excessive quantity of VLDL even with triglycerides in the "normal" range of 150 mg/dl or less.

The dangers of triglyceride-containing particles are therefore not fully evident from the triglyceride level on a lipid panel. Though you’re probably safe if triglycerides are <100 mg/dl, enough VLDL can occasionally be present even at this level to contribute triglycerides to other particles and produce small LDL and HDL patterns. For this reason, and to eliminate the contribution of triglycerides and VLDL to the size distortions of other particles, we aim for a triglyceride level of 60 mg/dl or less; this eliminates the contribution of VLDL to abnormal lipoproteins.

The treatment for excessive VLDL is therefore the same as for high triglycerides (above). Omega-3 fatty acids from fish oil are an especially effective way to reduce VLDL and triglycerides, with significant reductions beginning at 1800 mg/day of EPA and DHA (combined).

**Lipoprotein (a)**

Lipoprotein (a), or Lp(a) (read “LP little a”), is a powerful cause of plaque. Lp(a) is potentially aggressive and can be responsible for heart attacks at a young age, not uncommonly women in their 50s and men in their 40s. About 17% of people with heart disease have increased Lp (a) (Emerging Risk Factors Collaboration 2009; Jones 2007). It is also one of the greatest challenges to treat and perhaps the one we struggle with the most.

Lp (a) is actually an LDL particle with an additional protein attached called “apoprotein a.” Lp(a) magnifies the dangers of all other abnormalities, especially LDL particles. Lp(a) is unusual in that it has a dual dangerous effect: It
encourages atherosclerotic plaque growth and also triggers blood clot formation when plaque ruptures. Lp(a) is also a potent cause of “endothelial dysfunction,” or abnormal constriction of arteries. When Lp(a) causes endothelial dysfunction, arterial injury results that contributes to further plaque growth. High blood pressure is common with Lp(a) and is probably related to abnormal constriction of arteries throughout the body. People with Lp(a), especially females, can often have significant high blood pressure in their late 50’s or 60’s. This, in turn, causes even more damage to the artery wall and encourages plaque growth.

Lp(a) is a genetic trait that you inherit from either mom or dad, and that you pass on to each of your children with 50-70% likelihood. For this reason, if you have Lp(a), you should consider advising your grown children to be tested, particularly if they are over 30 years old.

Treatment for Lp(a) is controversial. Most experts agree that, at the very least, LDL cholesterol should be reduced to a level no higher than 80 mg/dl. There are several other possibilities for reducing Lp(a), but they all suffer from some unpredictability—while one treatment might work great for one person, the same treatment might not work at all in another. There is, unavoidably given current knowledge, some hit-and-miss with Lp(a) management.

In the *Track Your Plaque* program we usually begin to attack Lp(a) with high-doses of omega-3 fatty acids from fish oil in the range of 6000 mg EPA and DHA per day (*not* the dose of fish oil, but of the combined EPA + DHA contents), based on the observations made in the Lugalawa Study, in which Lp(a) levels were 48% lower in high consumers of fish (Marcovina 1999).

High-concentrations of EPA and DHA per capsule will be required at this dose to avoid having to take a lot of capsules. You should use a fish oil preparation that contains *at least* 600 mg total EPA and DHA per capsule; even that concentration means 10 capsules per day. You can find capsules with EPA + DHA concentrations of 750 mg, 850 mg, even 900 mg per capsule that can make this approach easier to achieve. Liquid fish oil (usually lemon- or orange-flavored) is also available in health food stores. Despite the high-doses, we have had very little difficulty with this strategy; nonetheless, you should work with your healthcare provider when using this approach.

High-dose fish oil has worked for 60% of our patients, with full effect requiring a year or more. Even if no response develops after a year, we usually try to maintain the high dose, since it has been shown to increase the size of apoprotein(a), a feature that makes Lp(a) less harmful (Winnicki 2010).

Niacin, in addition to increasing HDL and reducing small LDL, is also helpful to reduce Lp(a) (Scanu 2008). While, in past, we used high-doses of niacin of 2000-4000 mg per day, we now rarely exceed 2000 mg per day due to potential side-effects, such as liver toxicity and insulin resistance (higher blood
sugars) at these higher doses. We now try to gain control over Lp(a) using a combination of other strategies, such as high-dose fish oil, while using doses of niacin of 1000-2000 mg per day. Niacin at doses exceeding 500 mg per day should only be taken under supervision of a healthcare provider because of the greater likelihood of side-effects. The forms of niacin that are effective and safest are Sloniacin®, Enduracin®, and prescription Niaspan®.

Lp(a) is exceptionally sensitive to thyroid status, particularly the level of the thyroid hormone, T3 (Dullaart 1995; de Bruin 1993). Thus, achieving ideal thyroid status that includes ideal levels of the T3 hormone is a very helpful strategy. In the Track Your Plaque approach, this means: thyroid-stimulating hormone (TSH) of 1.0 mIU/L or less; free T3 and free T4 in the upper half of the “reference range”; and iodine supplementation. (We will discuss this at length in chapter 11.)

The adrenal gland hormone, dehydroepiandrosterone, or DHEA, is a helpful aid for reducing Lp(a) (Kostner 2005). While the effect is modest, rarely reducing Lp(a) more than 30-40 nmol/L or 5-10 mg/dl, it is a relatively benign nutritional supplement that is widely available. Because DHEA is a hormone present at high levels up to our late 30s, only people 40 years old or older should consider 1) having a DHEA level drawn, then 2) considering a low-dose DHEA supplement. We usually start at 10 mg per day and increase to 50-100 mg over time. If blood levels are used to guide dose, the dose can be increased to achieve a DHEA-S (the most common DHEA measurement) blood level of 250 µg/dl or the upper range of the “reference range” quoted by your laboratory.

While high-doses of DHEA (e.g., 300 mg, 1000 mg, 2000 or even 3000 mg per day) were used in past for purported anti-aging effects, these doses also generated undesirable effects, such as aggression, hypersexuality, and masculinizing effects in females. At the modest doses we use in Track Your Plaque of 10-100 mg per day, these effects are rarely experienced. The only side-effects we see on occasion at these modest doses are excessive assertiveness or short-temperedness. If you experience these effects, a reduction in dose is in order.

In females, estrogen preparations may reduce Lp(a), generally around 10-25%. Of course, estrogen use has been recognized as having both positive and negative effects. In the HERS trial of post-menopausal estrogen (estrogens from horses as Premarin®), while most women obtained no cardiovascular benefit, participants with Lp(a) experienced 15-22% reduction in heart attack risk (Shlipak 2000). This is a conversation to have with your family doctor or gynecologist and preferably the conversation includes discussion of bioidentical hormones, i.e., human hormones, rather than non-human synthetics. For men, testosterone can reduce Lp(a) approximately 25% (Zmunda 1996). (Both estrogen preparations and testosterone are prescription agents.)
There are several nutritional supplements and foods that contribute to reducing Lp(a), including raw almonds, flaxseed, coconut oil, and coenzyme Q10, though the magnitude of Lp(a) reduction is small. Given the difficulty in lowering Lp(a), it’s worth considering these adjuncts if you have this abnormality.

One thing to keep in mind with Lp(a): It is slow to respond. It means that, for example, if you add niacin or DHEA, to wait at least 3-6 months before reassessing your Lp(a) level. Too soon, and you may be misled into thinking that it’s not working. With high-dose fish oil, a minimum of six months is required to even begin to observe an effect, with full effect developing over one year or longer.

The Track Your Plaque approach to Lp (a) is similar to all other abnormalities: Treat, then adjust treatment to arrest the growth of plaque, i.e., until your heart scan score no longer increases, or a reduction in score is achieved. In this way, you and your doctor can add or subtract agents or intensify your program, depending on whether you've gained control of your plaque.

C-reactive protein

C-reactive protein (CRP) is one of a family of markers that we can use to measure the body’s inflammatory state. If inflammatory white blood cells are active in some part of the body—joints, lung, colon, etc., as well as plaque in coronary arteries—CRP (produced by the liver) will be increased. Inflammation increases the likelihood that plaque “ruptures,” or experiences breakdown of the surface covering, exposing underlying plaque contents and provoking blood clot formation and heart attack. An elevated CRP suggests that inflammation may be ongoing in your coronary plaque.

You can immediately see a difficulty here: If there is inflammation anywhere in the body, CRP levels will be increased. For this reason, measures of inflammation are best applied in the well, asymptomatic person who does not have some obvious source of inflammation like arthritis or colitis, recent surgery, recent infection, viral illness, etc. In these situations, high CRPs are meaningless from a heart standpoint. In apparently well people, however, modest elevations of CRP can suggest hidden inflammation in the plaque of coronary arteries. For this reason, a newer method of measuring CRP, called “high-sensitivity” CRP, has been developed that can detect subtle elevations that can signify increased coronary plaque inflammation. While levels >10 mg/L nearly always represent inflammation outside of the heart and should not be used to prognosticate coronary risk, lower levels (usually <4 mg/L) can be used to gauge coronary plaque inflammation.

Dr. Paul Ridker of Harvard University is a noted authority on CRP. He has convincingly shown that high CRP levels, and therefore greater levels of plaque inflammation, pose up to 3-fold higher risk of heart attack, even when LDL
cholesterol is low. When both CRP and LDL cholesterol are high, then risk is even greater. When an elevated CRP occurs in the company of small LDL particle size, an especially high risk for heart attack is present, increased as much as 6 to 7-fold (St-Pierre 2003).

An interesting study from UCLA explored heart attack risk posed by the combination of CRP and coronary calcium score. Participants in this study who had both low CRP and low coronary calcium scores (<4) had a very low risk for heart attack and death. Participants at the opposite extreme with a high CRP (highest quartile) and high calcium scores (>142) had 7.5-fold greater risk. Interestingly, participants with low calcium scores (<4) but high CRPs had only a minimal increase in heart attack risk (Park 2002). This makes sense: How can you rupture plaque if you have little or no plaque to inflame and rupture?

How to treat high CRP? Here’s the difficulty. Virtually of the research demonstrating the benefits of reducing CRP have involved statin cholesterol-reducing agents. Most research suggests that statin agents reduce CRP by about 30% within a few months of starting treatment. Ezetimibe (Zetia®) also reduces CRP by about 10-20%. But there are other strategies that reduce CRP to an even greater degree than statin agents: vitamin D (Timms 2002); elimination of wheat and cornstarch; weight loss. (Because these strategies are inexpensive, like vitamin D, or virtually free like elimination of wheat and cornstarch, there are no deep-pocketed corporations to support the research enjoyed by statin agents; it’s no wonder you don’t hear about them.) CRP reductions of 70% or more are typical with these simple strategies. Statins drugs are not required to reduce CRP.

Aspirin and omega-3 fatty acids, because they have anti-inflammatory properties, also reduce CRP modestly. A diet rich in fiber, particularly ground flaxseed, raw nuts, and vegetables (not wheat!) can have a significant CRP-reducing effect. Weight loss, in particular, causes CRP to plummet (Puglisi 2008).

From a practical viewpoint, if you have an elevated CRP, you should be regarded as being at higher risk for heart attack, even in the absence of other lipid or lipoprotein abnormalities. Efforts to reduce CRP should be considered, such as changes in diet (to be discussed further), vitamin D (also more to come), omega-3 fatty acids from fish oil, raw nuts and other healthy non-wheat fiber sources, weight loss and exercise. The only time you might not have to take an elevated CRP level seriously from a heart standpoint is if your coronary calcium score is 0 or very low (<10).

**Goodbye lipid panel?**

If lipoproteins are superior, do we even need to bother with plain old lipids (LDL cholesterol, HDL cholesterol, triglycerides, and total cholesterol)?
In Step 2 of *Track Your Plaque*, a full lipoprotein analysis is the preferred method to *diagnose* how coronary plaque was caused. Lipoprotein analysis is also the best way to determine whether, after treatments have been introduced, initial abnormalities have been fully corrected. But simple lipids (not lipoproteins) are still useful to *monitor the effects of treatment* along the way, even if your abnormalities were not fully evident on initial lipid testing.

Let me explain. While Steps 1 and 2 of *Track Your Plaque* can be completed in a matter of days, Step 3 or correction of your lipoprotein disorders often requires several months or longer, along with time required to make adjustments of the dose or type of treatment.

The easiest and least expensive way to monitor response to treatment is to perform simple lipid measures. In other words, lipids can provide *feedback* along the way before you reach your final destination of full correction of lipoprotein abnormalities. For instance, a common situation is a person with a low total HDL (e.g., <40 mg/dl), high triglycerides (e.g., >150 mg/dl), and an abundance of small LDL particles. This person eliminates wheat, cornstarch, and sugars; loses 15 pounds, begins omega-3 fatty acids from fish oil, corrects vitamin D deficiency, and adds niacin. After three months, a lipid panel is checked to assess whether there has been the expected change in HDL or triglycerides. Lipid panels are readily available in just about any clinic or hospital laboratory. In this example, if the lipid panel shows an increase in HDL cholesterol and a drop in triglycerides, then we can be confident that the quantity of small LDL particles has also decreased, as these three parameters all respond together and to a similar degree. Perhaps the HDL has increased to 48 mg/dl and the triglycerides are 117 mg/dl. Better, but not yet to our goals of an HDL of 60 mg/dl and triglycerides 60 mg/dl, and so the niacin dose is increased by 500 mg, perhaps more fish oil is added, then another lipid profile is scheduled in three months to assess the results again. In this way, we can use readily available and inexpensive lipid panels to obtain feedback on whether desired effects are being achieved.

Similarly, LDL cholesterol on the standard lipid panel responds *nearly* the same as LDL particle number and apoprotein B. In this way, we can easily and readily gauge whether the chosen treatments are yielding the benefits we desire just by doing a simple lipid panel. Other such patterns that can be used to gauge the effects of treatment are shown in the box.

In *Track Your Plaque*, we aim to achieve LDL cholesterol of 60 mg/dl, HDL of 60 mg/dl or greater, and a triglyceride level of 60 mg/dl, all values +/- 10 mg/dl. Once lipids are where you and your doctor would like them, then the full lipoprotein panel can be checked again. This approach simplifies the work required, since full lipoprotein analysis is required only in the beginning and to
assess whether you’ve achieved your lipoprotein goals. It also cuts down on the need for the more expensive lipoprotein testing.

<table>
<thead>
<tr>
<th>If your abnormality is ______</th>
<th>You can use ______ to assess treatment</th>
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</thead>
<tbody>
<tr>
<td>Small LDL</td>
<td>HDL, triglycerides</td>
</tr>
<tr>
<td>Low large HDL</td>
<td>HDL (total)</td>
</tr>
<tr>
<td>VLDL</td>
<td>Triglycerides</td>
</tr>
<tr>
<td>LDL particle number, apoprotein B</td>
<td>LDL cholesterol</td>
</tr>
</tbody>
</table>

**Lipoproteins: How to get them in your neighborhood**

If you’ve chosen to begin using a standard lipid panel, then you may need to discuss your lipid goals with your healthcare provider, since the values required to arrest plaque growth differ from those offered by standard guidelines (the National Cholesterol Education Program Adult Treatment Panel). At the very least, if your calcium score is >100 or 75th percentile or greater, then you might have to persuade your physician to treat your lipids as if you had coronary disease (i.e., you should be regarded as having a “coronary risk equivalent”). This provides you with maximum protection at a stage when your plaque is most controllable.

If you’ve decided to pursue the lipoprotein route, you will need to work with a healthcare provider who has invested the time and effort to be educated on these methods and knows how to use them in clinical practice. Thankfully, more and more physicians are recognizing the deficiencies of conventional lipids and have turned to lipoprotein testing for better answers. You will therefore need to locate a healthcare provider near you to help obtain testing and then provide interpretation. An emerging specialty called “lipidology” is making lipid and lipoprotein specialists more available in urban areas. A listing of lipidologists is maintained by the National Lipid Association at [http://www.learnyourlipids.com/resources.php](http://www.learnyourlipids.com/resources.php). However, be aware that lipidologists, as well as the National Lipid Association, are very drug-centered and are generally not involved in dispensing nutritional advice. The physician you choose should also offer recommendations regarding potential therapies for your lipoprotein abnormalities. He/she may also help monitor treatment with periodic lipid tests and decide when a lipoprotein re-evaluation is in order.

A listing of resources to help you obtain these tests in your area will be found in Appendix A. If you would like to help your own doctor become better informed on lipoprotein testing, you might begin by referring him/her to these...
resources. Lipoprotein testing companies provide professional education and recognize that an understanding of lipoproteins does not occur overnight. They will also help your doctor educate his/her staff or the laboratory’s staff on how to prepare your blood specimen before submitting it for lipoprotein testing.

Lipoprotein testing is fairly well covered by health insurance, certainly better than in years past. The initial panel that we obtain on *Track Your Plaque* patients (using NMR spectroscopy, LipoScience) costs a total of around $300 (around $90 for basic NMR; $200 for the additional tests). The Berkeley HeartLab service is approximately twice that. Atherotech’s VAP test is approximately $100-230 (depending on the lab). Your insurer may not be familiar with lipoprotein testing and occasionally requires justification from the ordering physician. Most insurance companies will cover most or all the cost when appropriate rationale is provided. Even if you are among the few whose insurance will not cover the cost, you can usually persuade them to contribute at least the cost of a conventional lipid panel (ranging from $10 to $100) towards the lipoprotein costs.

Another option is to just obtain the tests yourself. Services like Direct Labs ([www.directlabs.com](http://www.directlabs.com)), for instance, provide NMR and VAP lipoprotein testing for less than what you will pay through a conventional laboratory. (The blood sample is obtained through a local blood draw site, then sent to Liposcience or Atherotech.) Many current *Track Your Plaque* followers have used this route successfully, then obtained feedback through our online *Track Your Plaque* Forum. A chain of retail stores called Any Lab Test Now® ([www.anylabtestnow.com](http://www.anylabtestnow.com)) also provides VAP lipoprotein testing at low-cost.

**Can I get by just measuring lipids?**

If you are unable or unwilling to get lipoprotein testing, what sort of success can you have relying on simple lipids alone?

This varies, depending on whether or not there are multiple hidden causes for your coronary plaque. If you choose to begin with the lipid-only route, it is reasonable to correct the lipid abnormalities, along with the other basic components of the *Track Your Plaque* program that we will discuss (vitamin D, thyroid measures, and blood sugar), then repeat your heart scan score (approximately a year later) and assess the results. If your score is unchanged or lower, you’ve succeeded using lipids alone. But if the rate of increase in score is >10%, you may have hidden abnormalities that should be uncovered by lipoprotein testing.

A more serious effort to get these tests done will often be rewarded with greater success in achieving control of your plaque.
Track Your Plaque: Suggested lipoprotein panel

This is a list of the components of the basic lipoprotein panel we’ve used successfully for tracking and controlling plaque. If you and your doctor choose to use NMR (LipoScience, Inc.), the panel will automatically include:

- LDL particle number
- LDL (calculated), HDL, triglycerides, total cholesterol
- LDL, HDL, and VLDL particle size profiles

Lipoprotein(a) is *not* included and will need to specified.

If you go through Berkeley HeartLabs, Inc., the panel should include:

- LDL gradient gel electrophoresis (LDL GGE)
- HDL gradient gel electrophoresis (HDL GGE)
- LDL, HDL, triglycerides, total cholesterol
- Apoprotein B
- Lipoprotein (a)
- Apoprotein E
- Fibrinogen
- High-sensitivity c-reactive protein

The Vertical Auto-Profile (VAP) from Atherotech includes:

- “Real” LDL cholesterol
- Lipoprotein(a)
- Intermediate-density lipoprotein
- LDL, HDL VLDL subclass analysis
- Apoprotein B (calculated) and apoprotein A1 (calculated)

The type of lipoprotein testing is less important than the confidence of the interpretation you receive and/or the information you extract on your own from these valuables insights into causes of heart disease.

If you are going to obtain *all* lab tests relevant to the *Track Your Plaque* program, including some measures we haven’t yet discussed, here’s a list of the other important tests we use:

- 25-hydroxy vitamin D
- TSH, free T3, free T4 (thyroid measures)
- Fasting glucose, Hemoglobin A1c
- High-sensitivity c-reactive protein (if not included in your lipoprotein analysis)
It may be a somewhat long list, but remember: You are trying to obtain every possible advantage in gaining control over coronary plaque, something that just taking a statin drug and tossing a coin in the fountain will \textit{not} do.

**How far do we go?**

To what degree do lipoproteins need to be corrected?

This can vary depending on your specific pattern of lipoproteins. Ideally, endpoints for treatment are best determined by your healthcare provider, who can use your heart scan score as a feedback tool to decide whether your lipoproteins have been treated adequately.

If your score continues to increase at a rate of 10% per year or more, then your lipoprotein endpoints may require intensification. An increase in heart scan score of <10% suggests that modest improvement in your program may be beneficial, e.g., an additional 10% reduction in LDL particle number or apoprotein B, or LDL cholesterol, or a 10% further increase in HDL. If your score has stopped increasing (0% annual change) or, even better, has decreased, then you know that your lipoproteins have been adequately corrected.

To give you and your doctor some reference points for thinking about how to treat your lipoproteins, here are the endpoints that we’ve used successfully in \textit{Track Your Plaque}:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Treatment Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL cholesterol</td>
<td>50 - 70 mg/dl</td>
</tr>
<tr>
<td>LDL particle number</td>
<td>&lt;700 nm/l</td>
</tr>
<tr>
<td>Small LDL</td>
<td></td>
</tr>
<tr>
<td>NMR</td>
<td>&lt;300 nmol/L</td>
</tr>
<tr>
<td>Electropheresis</td>
<td>&lt;15% of total LDL</td>
</tr>
<tr>
<td>VAP</td>
<td>&lt;30% of “real” LDL</td>
</tr>
<tr>
<td>Apoprotein B</td>
<td>&lt;70 mg/dl</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>≥ 60 mg/dl</td>
</tr>
<tr>
<td>Large HDL</td>
<td></td>
</tr>
<tr>
<td>NMR</td>
<td>≥15 nmol/L</td>
</tr>
<tr>
<td>Electropheresis</td>
<td>≥30% HDL 2b</td>
</tr>
<tr>
<td>VAP</td>
<td>≥30% HDL 2</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>≤60 mg/dl</td>
</tr>
<tr>
<td>Lipoprotein (a)</td>
<td>&lt;30 mg/dl or &lt;60 nmol/L</td>
</tr>
<tr>
<td>C-reactive protein</td>
<td>&lt;1.0 mg/L</td>
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</tbody>
</table>
Once again, these are only suggested targets. If your heart scan score continues to increase despite achieving these endpoints, you and your doctor should consider treating beyond these suggested goals.

Summary

Step 2 of Track Your Plaque is to identify all causes of your coronary plaque. Lipoprotein testing is the most accurate and comprehensive method available to reveal the full extent of abnormalities creating plaque in your coronary arteries. Alternatively, conventional lipids can be used, but should then be treated intensively, particularly if your heart scan score is >100 or your score is at the 75th percentile or higher.

Lipoprotein testing is also the most reliable method for your doctor to determine whether the proper goals have been achieved, e.g., reducing LDL particle number sufficiently. To simplify the process along the way, however, readily obtainable lipid panels of LDL, HDL, triglycerides, and total cholesterol, available just about anywhere, can easily and inexpensively guide you and your doctor.

Treatment of your lipid/lipoprotein disorders is Step 3 of your Track Your Plaque program. Treatment is specific for each lipoprotein pattern and your treatment program needs to be individualized to your results.
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Chapter 9

The Six Track Your Plaque Nutrition Principles

and the

New Track Your Plaque Diet

Metabolic insights obtained through lipoprotein testing help guide our unique nutrition program. It means that better control can be obtained through diet: better lipoprotein control, better weight control, better control over coronary plaque.

People have experimented with just about every kind of diet imaginable: Low-fat, ultra low-fat, high-fat, low carbohydrate, high carbohydrate, high protein, low protein, vegetarian, Mediterranean, Oriental, rice, grapefruit, Atkin’s, “Zone,” and on and on . . .

Is there one diet that’s right and all the rest are wrong?

Probably not. In fact, every diet fad has taught us something new. The American obsession with diet has amounted to a nationwide experiment in the value—or danger—of various nutritional manipulations. To add to the confusion, because people vary genetically, they respond to the same diet in different ways. A diet, for instance, that drops one person’s LDL cholesterol 30 mg/dl could cause someone else to increase LDL 30 mg/dl.

The diet advocated by the American Heart Association is a modest fat-restricted program designed to reduce LDL cholesterol by 10%. Surely we can do better than that. Likewise, the USDA Food Pyramid advocates a diet that, in my experience, causes obesity, pre-diabetes and diabetes, fatigue, abdominal
symptoms like cramping and diarrhea, and fuels plaque growth. “Official” diets tend to be guilty of a one-size-fits-all and a people-really-can’t-stick-to-diets-anyway mentality. They also are the product of outside commercial interests that influence the message.

For further proof of the misguided nature of conventional diet advice, you need only take a look at the American Heart Association “Heart-Check Mark” Program stamp of approval on boxes of Cocoa Puffs®, Count Chocula®, and Berry Kix® to understand that marketing and financial motivations lie behind much diet advice, rather than nutritional wisdom.

How about ultra low-fat diets often advocated by those who claim it “reverses” heart disease? Many years ago, I prescribed the Ornish diet. This program advocates 10% of calories or less from fat, along with fruits, vegetables, and whole grains. In my experience, people following this diet showed two varying responses: A few initially did well—reducing LDL cholesterol, losing weight, and apparently slowing their heart disease. Others enjoyed little or none of these benefits. This much larger second group lost weight at first, then gained weight (especially fat around the waist), followed by reductions in HDL cholesterol, increased triglycerides, and increased blood sugar to the near-diabetic or diabetic range. When lipoproteins were examined, there was marked increase in small LDL particles. These are all changes that encourage growth of coronary plaque. Needless to say, I abandoned the ultra low-fat approach.

Can we construct a diet that benefits from these lessons? Is it possible to obtain the benefits we desire yet maintain flexibility and not force you to adhere to strict menus and formulas? Can we use diet effectively in light of lipoprotein analysis and its impact on calcium scoring?

The Track Your Plaque nutrition principles help correct the causes of atherosclerotic plaque as identified by lipid or lipoprotein testing. Our approach maximizes the health benefits of diet, minimizes, often eliminates, the need for other treatments including statin drugs, and helps you achieve other health goals, including reduction in waist size and weight loss, often dramatic.

Not another diet!

The Track Your Plaque nutrition program is not a precisely structured program with percent calories from fat, carbohydrate, etc. dictated to you. Instead, it educates you about important principles that create diet habits that help you succeed in reducing heart disease risk. This approach is therefore flexible, permitting modification to suit personal tastes and varying lipid/lipoprotein patterns.

The New Track Your Plaque Diet includes animal products, though it can be adapted to a vegetarian lifestyle, as well. The traditional argument against
animal products has been saturated fat. Let’s put aside the saturated fat question for a moment. Beyond saturated fat, several questions emerge:

• If humans were meant to be vegetarian, why do omega-3 fatty acids (mostly from wild game and fish) yield such substantial health benefits, including dramatic reduction in sudden death from heart disease?
• Why would vitamin K2 (from meats and milk, as well as fermented foods like natto and cheese), obtainable in only the tiniest amounts on a vegetarian diet, provide such significant benefits on bone and cardiovascular health (Geleijnse 2004)?
• Why would vitamin B12 (from meats) be necessary to maintain a normal blood count, prevent anemia, and lead to profound neurologic dysfunction when deficient?

Omega-3 fatty acids and vitamins K2 and B12 cannot be obtained in satisfactory quantities from a pure vegetarian diet. The consequences of deficiency are not measured in decades, but as short as a few years. The conclusion is unavoidable: Evolutionarily, humans are meant to consume at least some foods from animal sources.

It pains me to say this, since I’ve always favored a vegetarian lifestyle, mostly because of philosophical concerns, as well as worries about the safety of factory farm-raised livestock and inhumane practices. But, stepping back and objectively examining what nutritional approach appears to stack the odds in favor of optimal health, I believe that only one conclusion is possible: Humans are omnivorous, meant to consume some quantity of animal products.

Let’s take this argument a step further: If humans were meant to consume the kill of Stone Age hunters, what role is there for cultivated grains? Grains, of course, had little to no role in the diet of hunter-gatherers, who were nomadic by necessity, never staying put long enough to till a field and plant seeds.

Vocal proponent of this Paleolithic diet concept, Dr. Loren Cordain, and author of the book, *The Paleo Diet*, has pointed out that the profile of human disease (judging by analysis of bones and teeth of primitive humans, examination of last meals from stomach contents, and other such piecing-together-of-the-puzzle) shifted dramatically 8,500 years ago (a mere second on the evolutionary time scale)—just at the time when humans learned to gather and cultivate wheat. While hunter-gatherer predecessors died of trauma and infection, grain-consuming humans began to develop cancer, diabetes, and heart disease, diseases that were previously rare (Cordain 1999; Cordain 2005).

Fast-forward to the 21st century and the “healthy whole grain” craze has seized everyone from the USDA and AHA, to Kellogg® and General Mills, all the way down to the grain-consuming obese pre-diabetics or diabetics on Main Street, U.S.A. If we average the wheat flour intake of every infant, child, and
adult in the U.S., the annual per capita consumption is 133 lbs per year (USDA/Economic Research Service), providing 20% of all calories consumed worldwide. Since no infant or child eats 133 lbs of wheat per year, it means that many adults consume far more. Because of the properties of wheat, including its unique ability to provoke formation of small LDL particles, wheat elimination has become a major point of focus of the New Track Your Plaque Diet.

We will therefore focus on principles of healthy eating and not dictate what proportion of this or that component of diet we should consume. While it may seem more lax than other approaches, it has achieved extraordinary results to date, yielding effects like weight loss and correction of lipoprotein abnormalities. We construct a diet based on what is evolutionarily appropriate for Homo sapiens, a diet that includes animal products and minimizes or eliminates grains. Following an approach like this, issues like percent calories from fat or proteins just seem to take care of themselves.

The new thinking on diet

*If the members of the American medical establishment were to have a collective find-yourself-standing-naked-in-Times-Square-type nightmare, this might be it. They spend 30 years ridiculing Robert Atkins, author of the phenomenally-best-selling "Dr. Atkins' Diet Revolution" and "Dr. Atkins' New Diet Revolution," accusing the Manhattan doctor of quackery and fraud, only to discover that the unrepentant Atkins was right all along. Or maybe it's this: they find that their very own dietary recommendations—eat less fat and more carbohydrates—are the cause of the rampaging epidemic of obesity in America.*

Gary Taubes
*What If It's All a Big Fat Lie?

Since the original diet principles advocated in the Track Your Plaque program were released in 2004, a number of new developments have surfaced. While some new concepts are simply passing fads that should not impact our thinking, there are also some lessons to learn. All of these have been incorporated into the New Track Your Plaque Diet.

First of all, I believe that we can all agree that:

- Hydrogenated, or “trans” fats, synthetic fatty acids created by food manufacturers to replace saturated fats, are a failed experiment that has resulted in more heart disease (via its LDL-increasing, HDL-decreasing effects), high blood pressure, and cancer. I believe that we can agree that hydrogenated oils should be entirely removed from our diets. Any product with a label disclosing hydrogenated or partially-hydrogenated oils should be avoided.
• Polyunsaturated fats represent yet another failed experiment. The “official”
dietary advice of the 1960s and 70s was dominated by recommendations to
reduce saturated fat and replace them with polyunsaturates like corn oil,
safflower and sunflower. While this reduces LDL and total cholesterol, it has
more recently been shown to increase inflammatory responses.
Polyunsaturates are rich in the fatty acid, linoleic acid (not to be confused
with the healthy linolenic), a precursor to inflammatory prostaglandins like
arachadonic acid (Lahoz 1997). Polyunsaturates are subject to oxidation and
thereby more atherogenic (plaque-causing), both at time of ingestion, as well
as in the body (Reaven 1991). Polyunsaturated fatty acids are also readily
incorporated into coronary plaque, more so than saturated fats.
Polyunsaturated oils should therefore play a minor role in diet. (Their
ubiquity, even in natural foods, makes them unavoidable to a degree.)
• Commercially-produced meat today is not the same as that eaten by our
ancestors, particularly if compared to wild game caught by our hunter-
gatherer predecessors. Factory farm-raised chicken, beef, pork, and even
fish are modified by confinement in small spaces, being fed corn or other
industrial meal, growth hormone, long-term exposure to antibiotics, among
other “modern” strategies used to increase yield. The accelerated maturation
and grain and corn feed yields an obese animal at time of its kill. (Cattle, for
instance, go from birth to slaughter in as little as 18 months, compared to the
usual four years.) The end product that arrives on your dinner table has
greater polyunsaturated fatty acid content, along with reduced omega-3 fatty
acids, not to mention antibiotics and hormones (Tollefson 2000; O’Keefe
2010).

There are other new developments that we should incorporate into our nutritional
thinking:

• Total fat composition of the diet is not important—In past, we often
obsessed over what percentage of calories fats should comprise in the diet:
10%, 20%, 30% etc. The focus on saturated fat as a cholesterol-increasing
fat fraction led us to initially believe that elimination of saturated fat along with
a reduction in total fat would reduce risk for heart attack—it does not.
Reductions in saturated fat and across-the-board reductions in total fat do
not result in reduced risk for cardiovascular events (Siri-Tarino 2010). Total
fat intake is not an important aspect of health at all, although it can become
an adverse factor if purposefully restricted—a phenomenon directly opposite
to what we had previously believed.

• Saturated fat is not bad—The low-carbohydrate craze has uncovered an
unexpected phenomenon: When excessive carbohydrates are removed, the
undesirable effects of saturated fat are reduced, perhaps eliminated. While
saturated fat sources do indeed increase (large) LDL cholesterol (but also
increase total HDL, large HDL or HDL2b, and reduces lipoprotein(a)), the
association with increased risk of heart disease is weak or non-existent. But combine saturated fats with excessive carbohydrates and the combination causes the dreaded small LDL particles to skyrocket. So, saturated fat is not the culprit. It is soaring small LDL triggered by carbohydrates, only worsened by saturated fats.

• “Healthy whole grains” are not—This phenomenon has become a particular bugaboo for the Track Your Plaque program. In past, it was a real struggle to help people successfully lose weight and improve patterns associated with overweight and obesity, like low HDL, high triglycerides, small LDL, increased blood sugar and blood pressure. It began with advice to eliminate wheat flour-containing products—all breads, bagels, muffins, pretzels, crackers, breakfast cereals, pasta, etc.—simply to reduce blood sugar, since wheat raises blood sugar higher than table sugar. Lo and behold, people began to lose weight, often precipitously. Blood sugar dropped, blood pressure dropped, triglycerides dropped, HDL and small LDL corrected, appetites shrunk dramatically. Some diabetics became non-diabetics. Unexpectedly, many people suffering chronic bowel problems, like cramping and diarrhea, experienced dramatic relief, rashes resolved, some arthritis improved or disappeared, asthma improved sufficient for some people to toss their inhalers, rashes improved or disappeared, mood and energy improved. The unavoidable conclusion was that grains, particularly wheat, were the causes of an entire panel of undesirable health effects; elimination reversed them. There has since been no turning back. Of course, this information goes against the “grain” of conventional advice from the AHA and USDA. But the strategy of reducing, even eliminating, wheat from the diet to correct many common lipoprotein abnormalities is among the powerful strategies in diet and health that we will consider at greater length.

The New Track Your Plaque diet incorporates the collective wisdom gained through this grand nationwide nutritional experiment. But we also apply our unique perspective on applying healthy eating and nutrition in a program of coronary plaque control and reversal using lipoprotein patterns to light the way. Lipoprotein patterns provide invaluable insight into metabolic responses that abbreviate our path to success and yield superior results faster.

Will I lose weight?
The Track Your Plaque nutrition principles promote weight loss. Weight loss can, in fact, be dramatic.

Starting with an average American diet, many people lose an incredible pound per day for the first 10 days—similar to starvation—slowing to about 5-10 lbs every 30 days thereafter. Losses of 20-30 lbs. in the first 3 months are therefore typical, followed by a more gradual downward trend over the ensuing year. Losing excess weight significantly impacts lipid and lipoprotein patterns,
reduce diabetic tendencies, and make you feel better, all of which contribute to control over plaque. What makes the rate of weight loss incredible is that we do not limit calories.

People who begin the process substantially overweight usually experience as much as 50-70 lbs weight loss over the first year. (The pace and total quantity of weight depend on how overweight you are to begin with, as well as other factors, such as adherence to diet, amount of physical activity and exercise, and genetic factors.)

There is a hidden “secret” built into the Track Your Plaque nutrition program: We eliminate foods that trigger appetite. Four food ingredients are powerful stimulants of appetite: wheat flour, cornstarch, high-fructose corn syrup, and sucrose (table sugar). The first step in the Track Your Plaque nutrition program is to eliminate these ubiquitous components of processed foods. Usually within a few weeks, appetite plummets. Remove these four common processed food ingredients and appetite will be driven by the need for sustenance, but not abnormal impulses to eat . . . and eat and eat.

Because appetite is dictated by need, rather than impulse, the average calorie intake on the New Track Your Plaque diet is reduced 350-400 calories per day—without hunger, without cravings.

In fact, a common conversation we have around Track Your Plaque is “Am I too skinny?” I have to frequently remind people that, because most of their friends and neighbors are overweight or obese, people who are at their normal weight tend to stick out in a crowd. For the great majority of people, the Track Your Plaque approach achieves ideal weight.

**Can medication make up for a bad diet?**

Just how important is diet?

Some people ask, “What if I follow the program—I’ll take the medicines and supplements and exercise—but I really don’t want to give up my pancakes and waffles, bagels, and chips. Can I still reduce my score?”

People who fail to adopt the principles discussed here and continue to follow unhealthy eating habits do not enjoy the kind of control over coronary plaque as people who pay attention to diet. For one thing, eating an average American diet virtually ensures excessive weight gain. When you ingest large quantities of refined carbohydrates from processed foods, many lipid/lipoprotein abnormalities—particularly HDL, small LDL particles, and VLDL—are more difficult to control, necessitating more medication and ultimately causing growth of coronary plaque.
There are also many aspects of diet that cannot be controlled by any medication or supplement. For instance, the after-meal flood of lipoproteins in the blood that occurs during the first few hours after eating is largely influenced by the content of diet. The foods you choose are crucial determinants of whether you will or will not shower your arteries with globules of plaque-causing lipoproteins for the 6 or so hours after a meal. Fat content of the diet is only a minor determinant of the composition and magnitude of after-meal lipoproteins. Carbohydrate content of the diet is the principal determinant; the higher the carbohydrate content of the diet—such as the inclusion of “healthy whole grains”—the worse the after-meal flood of lipoproteins (Volek 2009).

In fact, without the diet changes discussed here, the only medications that are capable of reversing many of the metabolic derangements triggered by diet are diabetes medications. Given a choice of diet versus taking diabetes medications, I believe the choice is easy.

How do I know my diet is working?
Lipids and lipoproteins can be used to gauge success with diet. For instance, let’s say you have low HDL, excessive small LDL particles, and high triglyceride level identified at the start of your program. You make changes in diet, increase physical activity, and lose 20 pounds. You also begin vitamin D and omega-3 fatty acids from fish oil, along with some other treatments to correct your lipoprotein patterns. Will that be enough?

This can be decided by re-assessing lipids or lipoproteins to see whether your abnormalities have been corrected. (Track Your Plaque lipid and lipoprotein suggested endpoints are listed in chapter 8.) But don’t commit the mistake that some people make. They succeed in following all the strategies, then declare “I’ve stopped all my supplements since I’m doing so well!” Unfortunately, just feeling good or losing 20 pounds are not reliable indications of whether your genetic lipid/lipoprotein patterns have been corrected. That decision can only be made by repeating lipid or lipoprotein testing and, ultimately, by the effects on your heart scan score.

Let’s now discuss the six Track Your Plaque principles of healthy eating.

Diet Principle #1:
Correct metabolic responses with elimination of wheat, cornstarch, oats, and sugars; limited dairy

Eliminate:
• All wheat products
• All cornstarch and oat products
• Fruit drinks, fruit juices, and soft drinks
• Candies and other sugary snacks

Limited dairy:

• No more than 1-2 servings per day milk, cottage cheese, yogurt; 2-4 oz cheese

Over the last several years, there has been an explosion in the prevalence of the collection of abnormalities labeled “metabolic syndrome.” You can recognize the metabolic syndrome by its characteristic features: protuberant abdomen, higher blood sugar, low HDL cholesterol, high triglycerides, high blood pressure, small LDL particles and abnormal measures of hidden inflammatory patterns, e.g., C-reactive protein.

The Adult Treatment Panel-3 (ATP-3), the national guidelines for cholesterol management, arbitrarily define metabolic syndrome as having any three of the following: HDL 40 mg/dl or less in men, 50 mg/dl or less in women; triglycerides 150 mg/dl or more; BP 130/85 or greater; waist size 35 inches or more in women, 40 inches or greater in males; fasting blood glucose 110 mg/dl or greater. However, this definition tends to identify only the most advanced cases.

Excessive insulin release, followed by resistance to insulin’s action, underlie the nutritional responses that trigger these phenomena. The most sensitive measure of the process underlying the metabolic syndrome is small LDL particles (particularly if measured by the NMR method; Liposcience). More than any other measure, small LDL fluctuates with the ebb and flow of insulin sensitivity and carbohydrate intake.

Small LDL has therefore leapt to number one spot on the list of most common abnormalities identified through lipoprotein testing. It also occupies number one spot as most frequent cause of coronary plaque. Over 90% of participants in the Track Your Plaque program begin with a substantial quantity of small LDL.

Small LDL and the features of the metabolic syndrome and excessive weight are all powerfully corrected by eliminating the foods that created them in the first place.
Wheat, wheat everywhere

Any food that triggers rapid release of blood sugar also triggers formation of small LDL particles. The list of most flagrant culprits that trigger small LDL formation include (in this order):

- Wheat products—breads, bagels, muffins, pastas, cakes, cookies, pancakes, waffles, pretzels, crackers, breakfast cereals
- Cornstarch—tacos, tortillas, corn chips, cornbreads, sauces, gravies, breakfast cereals
- Gluten-free foods—Despite having no wheat, foods made of dried cornstarch, potato starch, rice starch, and tapioca starch, the four starches usually used to replace wheat gluten, can act similarly to wheat and trigger blood sugar and small LDL particles.
- Fruit juices, soft drinks—No, fruit juices are not that good for you. (While they contain some healthy components, the sugar load is simply too great for the majority of people.)
- Snack foods—potato chips, rice cakes, popcorn, candies, pies
- Rice—white and brown
- Potatoes—especially white and red
- Beans—red, black, pinto, kidney, etc.

In fact, foods made with wheat increase blood sugar faster and to a higher level than even table sugar, a Milky Way® bar, or Snickers® bar (i.e., wheat has a higher glycemic index). In particular, wheat has come to dominate the diet of most Americans; it’s not uncommon for people to eat wheat products four, five, or more times per day.

Cornstarch is often used to thicken sauces and gravies. Cornmeal, such as that in tacos, tortillas, and chips, can be nearly as bad as cornstarch.

Lesser culprits on the list of small LDL triggers are rice (white and brown) and potatoes (white and red). Rice and potatoes are less offensive, mostly because they do not occupy the same dominant role in diet that wheat and cornstarch-based foods play. However, if permitted to occupy a frequent role with larger portion sizes (e.g., greater than ½ cup per serving), then it will exert small LDL- and blood sugar-provoking effects.

If wheat, cornstarch, snacks, and fruit drinks are the most potent triggers of insulin, small LDL, and the entire constellation of abnormalities of the metabolic syndrome, then we can reverse this entire situation by . . . eliminating them. Because wheat products have become so dominant in the average American diet, just eliminating wheat fixes about 90% of the problem.

Eliminating the causes of the problem leads to an extraordinary panel of benefits:
• Weight loss—Rapid, often profound, weight loss ensues. (The magnitude and rapidity of weight loss response depends on genetic factors, as well as the starting condition of diet and how overweight you are.) 20-30 lbs lost—effortlessly—within the first 3 months is a typical response. Most of the lost weight will be from visceral fat, fat within the abdominal cavity that is responsible for insulin resistance (expressed on the surface as “love handles”).
• Reduction in small LDL—Because small LDL particles are such a sensitive index of carbohydrate intake and weight, they respond promptly and dramatically.
• Increase in HDL cholesterol—a process that develops over many months to years
• Reduction in triglycerides—Reduction of up to several hundred mg/dl are common within several months.
• Enhanced sensitivity to insulin
• Reduction in blood sugar and HbA1c
• Reduction in blood pressure—An effect that requires 3-6 months
• Reduction in inflammatory measures such as c-reactive protein

Subjective improvements also occur: increased mental clarity, increased energy, improved sleep, more stable moods.

Curiously, a common criticism of this approach is the statement: “But we need wheat (or grains) in our diet!”

Entirely untrue. Wheat and grains are not necessary components of the diet for long, healthy life. Less than 5% of the time humans have spent on Earth have involved consumption of wheat and other grains, the other 95% spent consuming the non-grain foods available to hunter-gatherer cultures. Beyond the fact that a host of abnormal metabolic patterns shift towards normal with elimination, wheat and grains provide no beneficial component of diet that cannot be obtained through other foods, provided calories are replaced by real foods like vegetables, raw nuts, and meats, and not junk foods. The quantity of fiber, for instance, lost with elimination of wheat can be readily matched or exceeded by eating raw nuts, vegetables, and fruit. B vitamins like thiamine, folic acid, and riboflavin are easily replaced by those from nuts, meats, and vegetables.

Wheat addiction, wheat withdrawal

There is an important group of people, about 30% of the population, who experience something that can only be described as wheat addiction and withdrawal.

People afflicted with this odd condition crave wheat products and eat pretzels, crackers, bread, etc., every day. Cravings occur in approximately two-hour cycles, consistent with the sugar and insulin rollercoaster that results from
wheat products. Missing a snack or meal causes distress: shakiness, nervousness, mental “fog,” headache, fatigue, and intense cravings.

They can also experience a syndrome of “wheat withdrawal” consisting of fatigue, mental fogginess, and diminished exercise capacity that usually lasts two to five days, sometimes longer, when the flow of wheat products ceases. The effect is most prominent when going “cold turkey.”

Wheat withdrawal is a benign phenomenon, but it can be emotionally challenging. While I’ve seen it happen in many hundreds of people, I have never seen any genuinely adverse effect beyond the psychological and emotional struggles.

But beware: If you’ve had a wheat addiction, followed by withdrawal, be careful of the temptation that wheat can exert over your impulses. I’ve seen people with this tendency successfully go through withdrawal and remove wheat from their diet until a single cracker, pretzel, or cookie indulgence opens a floodgate of sugar and wheat cravings. The initial 30 lbs lost is rapidly regained. There is no realistic way to keep this from happening except to be aware of the phenomenon. People with this health issue need to be vigilant and not let a single indulgence trigger uncontrollable impulses.

Why does wheat withdrawal occur? It is likely that years of high-carbohydrate eating cause metabolism to become reliant on a constant supply of readily-metabolized sugars. Removing sugar sources forces the body to convert to mobilizing fatty acids instead, a process which may not be immediate but requires several days to kick in. Wheat has also been found to trigger formation of “exorphins,” similar to the “runners’ high” ascribed to endorphins (Zioudrou 1979). Remove the modest euphoric effects of wheat exorphins and a withdrawal syndrome can be provoked. (Yes: Wheat exerts an opiate-like effect. Interestingly, the effects of wheat-derived exorphins can be blocked with opiate-blocking drugs like naloxone and naltrexone. A drug company, in fact, is planning to release a medication to exploit this effect for weight loss. Of course, a more rational solution is to eliminate the addiction-provoking food, rather than block the addiction with a drug.)

There are two ways to deal with this effect. One is to taper wheat gradually over a week. However, be warned: Some people are so addicted to wheat that they find even this tapering process to be overwhelming, and sometimes going “cold turkey” is necessary to break the addiction, just as the alcoholic has to suffer through his/her withdrawal.

Two: Select a time to begin the process when you don’t need to be at your top performance, e.g., a week off from work or a long weekend. Perhaps it won’t be the most action-packed weekend, but it will allow you to return to work and life in far better shape.
Wheat-Free Testimonials
Here are some testimonials we’ve received via The Heart Scan Blog on the effects of removing wheat from the diet:

Barbara W said:

It’s true! We’ve done it. My husband and I stopped eating all grains and sugar in February. At this point, we really don’t miss them any more. It was a huge change, but it’s worth the effort. I’ve lost over 20 pounds (10 to go) and my husband has lost 45 pounds (20 to go). On top of it, our body shapes have changed drastically. It is really amazing. I’ve got my waist back (and a whole wardrobe of clothes—I’m thrilled.

I’m also very happy to be eating foods that I always loved like eggs, avocados, and meats—without feeling guilty that they’re not good for me.

With the extremely hot weather this week in our area, we thought we’d “treat” ourselves to small ice cream cones. To our surprise, it wasn’t that much of a treat. Didn’t even taste as good as we’d anticipated. I know I would have been much more satisfied with a snack of smoked salmon with fresh dill, capers, chopped onion and drizzled with lemon juice.

Aside from weight changes, we both feel so much better in general—feel much more alert and move around with much greater flexibility, sleep well, never have any indigestion. We’re really enjoying this. It’s like feeling younger.

It’s not a diet for us. This will be the way we eat from now on. Actually, we think our food has become more interesting and varied since giving up all the “white stuff.” I guess we felt compelled to get a little more creative.

Eating out (or at other peoples’ places) has probably been the hardest part of this adjustment. But now we’re getting pretty comfortable saying what we won’t eat. I’m starting to enjoy the reactions it produces.

Anonymous said:

My life changed when I cut not only all wheat, but all grains from my diet.

For the first time in my life, I was no longer hungry—no hunger pangs between meals; no overwhelming desire to snack. Now I eat at mealtimes without even thinking about food in between.

I’ve dropped 70 pounds, effortlessly, come off high blood pressure meds and control my blood sugar without medication.

I don’t know whether it was just the elimination of grain, especially wheat, or whether it was a combination of grain elimination along with a number of other changes, but I do know that mere reduction of grain consumption still left me hungry. It wasn’t until I eliminated it that the overwhelming reduction in appetite kicked in.

As a former wheat-addicted vegetarian, who thought she was eating healthily according to all the expert advice out there at the time, I can only shake my head at how mistaken I was.
Add non-wheat grain(s)

There is one (yes, only one) non-wheat grain that does not trigger the unhealthy effects of wheat and actually provides benefit: ground flaxseed.

Flaxseed does not have the addictive potential of wheat. Ground flaxseed has virtually no digestible carbohydrates, consisting purely of fiber, protein, and oils (including the “omega-3” linolenic acid). Ground flaxseed can be used as a hot cereal (heated, for instance, with almond, coconut, or soy milk with added walnuts and blueberries), or added to foods like cottage cheese, chili, “breading” for chicken and fish.

Other non-wheat grains, such as wild rice, rye, barley, quinoa, sorghum, and bulghur, tend to yield responses that are intermediate. While not as undesirable as wheat, they do take exert an adverse metabolic toll. Therefore, these grains are best used after the withdrawal process (above) is over, metabolic goals and weight loss have been achieved, and a loosening of diet is permissible. However, if you are among those with a powerful potential for wheat addiction and/or exaggerated diabetic potential and small LDL particles, these foods should remain off your list of safe foods. Also, if you have celiac disease or positive celiac markers (antigliadin, transglutaminase, or endomysial antibodies), you should meticulously avoid rye, oats, and barley, as well as wheat, since they all contain gluten.

The role of dairy products

While cheese is safe for any pattern and, in fact, provides additional vitamin K2, other dairy products are conditional. The suggested limitation of 2 to 4 oz per day has nothing to do with the fat content of cheese; it relates to the potential for cheese to pose a substantial acid challenge (with implications for bone health) and the high content of Advanced Glycation End-products (see below).

Milk, cottage cheese, and yogurt are sources of only a modest carbohydrate load, but they have the unique capacity to trigger insulin excessively (as much as 3-fold increased area under the insulin response curve) and contribute to distortions of insulin metabolism. i.e., they are “insulinotrophic” (Liljeberg 2001). I therefore advise including dairy products outside of cheese in only small quantities, e.g., 1-2 servings per day. However, if you are among those with extreme carbohydrate sensitivity, avoidance may be necessary. (Contrary to popular advice, sufficient dietary calcium can come from non-dairy sources, such as green vegetables, especially if normalization of vitamin D levels is achieved.)

Substitutes for dairy include unsweetened almond milk, coconut milk and coconut water, and full-fat soy products such as soy milk (unsweetened).
Diet Principle #2:
Don’t limit fats, but choose the right fats

Enjoy these healthy oils in unlimited quantities:

- Olive
- Flaxseed
- Avocado
- Coconut

Include fish at least once per week and supplement omega-3 fatty acids (dose depending on lipoprotein patterns; minimum 1800 mg EPA + DHA per day).

No restriction on saturated fat intake though intake can be adjusted to achieve Track Your Plaque LDL targets

Avoid hydrogenated and polyunsaturated oils; avoid excessive heating of oils, especially deep-frying

We’ve learned plenty of new lessons on gaining metabolic control to improve lipoprotein patterns, improvements that yield control over weight and correct the abnormal metabolic fallout of past dietary mistakes. One of the most important new lessons is that fats have come to play a prominent role in the New Track Your Plaque Diet.

For many years, all fats were demonized based on the fact that any fat, good or bad, contains 9 calories per gram, compared to 4 calories per gram of carbohydrate or protein. It logically led to the belief that caloric density accounted for weight gain: the more fat that was included in diet, the more weight was gained. The logical corollary was that foods with reduced caloric density—read “carbohydrates”—would help lose weight.

As we now know, the opposite happens. Although it seems counterintuitive, calorie-dense fats help lose weight and manage it more effectively. The essential feature of food that triggers appetite vs. induces satiety is driven by insulin response, not calorie density. Carbohydrates, of course, trigger insulin galore. A meal that includes whole grain bread, whole wheat
crackers, or whole wheat pasta, for instance, causes a large volume of sugar to be released into the blood. The pancreas responds by releasing a large quantity of insulin to hasten the entry of sugar into the body’s cells, which is then stored as energy. Gorge on carbohydrate-rich foods, such as fruits, roots, and berries—or, in our modern society, breads, pasta, and snacks—and your body stores the energy in preparation for extended caloric deprivation. Of course, the period of deprivation never comes in a modern world of plenty. Instead, it is followed by continued and virtually unlimited access to even more carbohydrate calories, allowing more and more storage.

The results are evident all around us in the overweight and obese in our society.

The currently popular nutritional mantra of “eat more healthy whole grains” is therefore much of the explanation behind the national epidemic of obesity and diabetes. It also underlies the enormous explosion in small LDL and related patterns.

In contrast to carbohydrates, fats are the least insulin-provoking, followed by protein as a close second. Less insulin provocation means less storage as fat. It also means that appetite is not recurrently triggered. Instead, satiety ensues. Coincident with the recent re-examination of the role of fat composition of the diet that has led to its “pardon,” it’s time to talk about adding back fats—plenty of fats—to our diet.

But, while fats all share the same capacity to generate satiety, fail to trigger insulin responses, the various fatty acids that comprise fats do indeed differ in other ways.

The fatty acid line-up

Hydrogenated fat is the one fat that we can all agree everyone should avoid. Hydrogenation is the process used to solidify liquid vegetable oils by adding hydrogen groups in an unnatural “trans” (opposite sides of the carbon backbone) configuration (“trans fats”). Common examples are vegetable shortening and margarines (though recently, some manufacturers are using non-hydrogenated canola and other vegetable oils).

Manufacturers love to use hydrogenated oils in food processing. You’ll find them in baked cookies and cakes, pies, pastries, snack chips, frozen foods, salad dressings and mayonnaise, and many convenience foods. Hydrogenated “trans” fats not only raise LDL cholesterol, but reduce HDL cholesterol, trigger increase of Lp(a), and divert fatty acids towards inflammation-increasing pathways. To see whether a product contains hydrogenated fats, refer to the label of ingredients. If “hydrogenated” oil or “partially hydrogenated” oil is listed on the label, avoid it. (The FDA now mandates that labels specify the inclusion of trans fats.)
Another group of oils that we would like to minimize are **polyunsaturated oils**. Polyunsaturates are rich in *linoleic acid*, a principal member of the omega-6 family. While polyunsaturates were widely advocated in the 1980s and 1990s because they modestly reduce LDL cholesterol, it has become increasingly clear that linoleic acid-rich oils trigger an inflammatory cascade, yielding increases in such inflammatory mediators as thromboxane (Calder 2009). Corn, sunflower, soybean, grapeseed and cottonseed oils contain abundant omega-6 fatty acids. These oils are best used sparingly, if at all. There are better forms of oils to use. Also beware that many processed foods include polyunsaturated oils, in addition to hydrogenated trans fats.

**Monounsaturated fatty acids** (lacking one hydrogen) are getting more attention due to the success of the so-called Mediterranean diet in reducing risk of heart attack. The Lyon Heart Study examined the health benefits of a Mediterranean diet rich in olive oil (70% monounsaturated oils), vegetables, and fish, similar to that eaten along the Mediterranean coast in Europe. People following this diet (as compared to an American-like diet) suffered 40% fewer heart attacks (de Lorgeril 1999). While monounsaturated oils reduce LDL cholesterol with approximately the same effectiveness as polyunsaturates, monounsaturates do not trigger abnormal inflammatory responses. The best monounsaturated-rich oil is olive oil (70% monounsaturates). Extra-virgin olive oil (often green and cloudy, particularly when cool) also contains high quantities of polyphenols, which may further add to health benefits.

Canola oil, another oil rich in monounsaturates (60%), has been the topic of controversy. The intensive purification process required that involves high-temperature heating and hexane extraction has been shown to increase the relative content of *trans* fats (O'Keefe 1994) and exert a variety of unhealthy effects in laboratory animals (Ohara 2006). While the genuine scientific data are not as damning as many critics of canola make them out to be, sufficient doubt has been raised that canola is not recommended as part of the New *Track Your Plaque* diet.

Raw nuts are another excellent source of monounsaturates. People often shun nuts because of their high fat content. But much of the fat is monounsaturated. Raw nuts are filling, requiring hours to digest. In fact, eating a ¼ cup or more of raw almonds or walnuts every day can reduce total and LDL cholesterol by 20 mg/dl due to the monounsaturated oils and fibers. As long as they are raw, nuts do not cause weight gain.

Because the four healthy oils exert virtually no ill-effect, but reduce LDL, induce satiety, and smooth insulin-blood sugar excursions, the New *Track Your Plaque* Diet includes plentiful oils. Abundant oils can be included with every meal, including breakfast—*especially* breakfast. An oil compatible with each food can be chosen, e.g., a tablespoon or two of olive oil with scrambled eggs, a
teaspoon or two of flaxseed oil mixed with ground flaxseed. The added oils help smooth insulin and blood sugar responses. Because appetite is suppressed, weight loss is also accelerated.

**Omega-3 fatty acids**, docosahexaenoic acid, or DHA, and eicosapentaenoic acid, or EPA, are in a class by themselves. While biochemically they are polyunsaturates, their unique long-chain structure and biological properties set them apart from other polyunsaturates. Omega-3s are used both as treatment and preventive nutritional strategy.

Fish are the source for omega-3 fatty acids: cod, halibut, trout, menhaden, salmon, mackerel, tuna, and sardines. Eating two servings of fish per month is enough to yield a sharp drop in risk of dying of heart attack. However, fish oil supplements ensure higher intakes of omega-3's for additional cardiovascular benefit. Because the quantity of omega-3s required for maximal protective effect is greater than can be obtained with eating fish occasionally, we use specific supplementation in the *Track Your Plaque* program.

Omega-3 fatty acids are the component of diet in fish-eating cultures responsible for reducing heart attack dramatically. Japanese women have four-fold higher blood levels of EPA and DHA and only 20% of the cardiovascular risk of American women; Japanese men also have four-fold higher levels with 40% of the risk of American men (Iso 1989).

Omega-3's reduce blood pressure, reduce triglycerides, increase HDL and make LDL particles bigger, in addition to reducing risk for heart attack and death. There is evidence that omega-3's can reverse atherosclerosis. When people who've suffered heart attacks eat a diet rich in omega-3's or take fish oil supplements, the risk of dying of heart attack is cut by 35-45% (Marchioli 2001). Evidence suggests that omega-3's have cancer-preventing effects, inhibit Alzheimer's dementia, and help alleviate depression.

Omega-3 fatty acids can also be used to treat specific lipoprotein abnormalities. Triglycerides can be reduced up to 50%; lipoprotein(a) can also be reduced. Higher doses are needed for these purposes. Fish oil also reduces fibrinogen, a blood clotting protein.

Flaxseed, either as the whole seed or as flaxseed oil, is also a source of omega-3's. However, the omega-3's in flaxseed don't occur as DHA or EPA, but as linolenic acid. While linolenic acid is beneficial in its own right, it cannot take the place of the omega-3 fatty acids from fish oil. The conversion of linolenic acid to the active DHA/EPA is inefficient. The quantity of EPA and DHA yielded from flaxseed oil is small, less than 1 part DHA/EPA for every 10 parts linolenic acid taken. Flaxseed oil does not yield the same benefits, particularly those on lipoproteins, as that provided by fish oil. Fish oil is therefore the preferred source of omega-3's.
Linolenic acid nonetheless has been associated with reduction in cardiovascular events and is suspected to be at least one of the important components of both the Mediterranean diet and Japanese diets (Geleijnse 2010). Linolenic acid provides the building blocks for anti-inflammatory eicosanoid responses, in contrast to linoleic acid (omega-6) that increases inflammatory responses. Flaxseed oil is the most plentiful source of linolenic acid (51%). Walnuts, hazelnuts, and ground flaxseed are also healthy sources of linolenic acid.
The importance of Advanced Glycation End-products (AGEs)

Just as someone can be wrongly accused and convicted for a crime he didn’t commit, so AGEs may be the real culprit behind many, if not all, of the adverse health effects blamed on saturated fat.

AGEs are an emerging group of compounds (and poorly named, since not all AGEs involve glycation) that have been shown to be responsible for many of the processes leading to coronary plaque formation, as well as diabetes, high blood pressure, and cancer. AGEs also serve as the basis for an actively-pursued theory of aging, the “AGE theory of aging.”

AGEs are formed via two routes:

• **Endogenous AGEs**—Endogenous AGEs form in the body due to the reaction of blood glucose with various proteins. The higher the blood glucose, the more AGEs are formed, with some AGE formation even occurring with normal blood glucose levels. Endogenous AGEs are responsible for cataracts (AGEs involving lens proteins), kidney disease, atherosclerosis, skin aging, and many of the other manifestations of both aging and disease. AGEs also explain why diabetics experience the collection of phenomena that could be viewed as accelerated aging: earlier onset of cataracts, kidney disease, atherosclerosis, skin aging, etc. The Track Your Plaque strategy of limiting foods that increase blood sugar, especially wheat, will lead to reduced formation of AGEs.

• **Exogenous AGEs**—Exogenous AGEs are formed outside the body (“exogenous”) in the foods we consume. Our exposure therefore depends on whether or not we eat foods that contain AGEs. AGEs are primarily formed from chemical reactions between fats or proteins and carbohydrates, especially when high-temperature (greater than 350° F) cooking for prolonged periods are involved. This means that meats that are broiled, fried, or barbecued are the principal source of exogenous AGEs, while the same meats cooked at lower temperature for shorter periods of time have far lower AGE content (Vlassara 2002). Cured meats, like hot dogs, sausage, and bacon, also contain high levels of AGEs, as do foods cooked with butter or margarine.

Butter and margarine by themselves are also problem sources of AGEs, The AGE content, for instance, of one pat of butter (5 grams), has the same AGE content as nearly a pound of roast beef (264,873). For comparison, the AGE content of a hard-boiled egg is 573 and grilled broccoli is 2,260 (Goldberg 2004).

Saturated fats have taken the blame for the adverse effects that arise from consumption of large quantities of animal products, when it may have been AGEs that were the real cause all along. So the New Track Your Plaque Diet is, in effect, a low-AGE diet. We therefore eliminate carbohydrates like wheat, oats, cornstarch, and sugars, a strategy that dramatically reduces endogenous AGE formation. We also avoid roasting, frying, or barbecuing meats, and use baking, sautéing, boiling, and steaming whenever possible. We use only modest quantities of butter and avoid margarine. Eat foods raw or minimally cooked whenever possible.

The AGE conversation remains preliminary, but it has potential to clarify many uncertainties and inconsistencies that have existed in nutrition.
We now come to the thorny issue of saturated fats. Saturated fats have been the subject of scorn over the past 50 years, originating with early observations that cultures with greater intakes of saturated fat experienced higher risk for heart attack. Public health advice to cut all fats, along with saturated, then led to replacement of fat calories with carbohydrate calories, spawning the national movement towards incorporating abundant “healthy whole grains,” as well as the initial push for hydrogenated and polyunsaturated oils to replaced saturated. As many of us now know, this shift in caloric composition has tragically backfired, and has played a major role in creating the current epidemic of diabetes and obesity.

More recent re-analyses of the original data indicting saturated fat have called these interpretations in question. Specifically:

- Many of the original observations were made using total cholesterol, rather than LDL and HDL. While saturated fat undoubtedly increases total cholesterol, about half the effect is due to increase in HDL.
- While saturated fats increase total and LDL cholesterol, they increase the large LDL fraction, while carbohydrates increase small LDL. Small LDL is five-fold more atherogenic (plaque-causing) than large (Lamarche 1999).
- Not all saturated fats are the same, though they have been demonized as a group. The various fatty acids (lauric, myristic, stearic, palmitic, butyric and others) comprising saturated fats differ in their effects on LDL, HDL, coagulation, etc. In particular, lauric and stearic acids are essentially neutral, exerting virtually no lipoprotein effects (Mensink 1997).
- Foods containing saturated fats consist of a range of different food choices, from egg yolks to red meats to cured meats (sausage, hot dogs, bacon) that differ in oxidized fat and oxidized cholesterol content, AGE content, and other factors. Grouping this varied mix of foods because of saturated fat is misleading.

Saturated fats have been criticized unfairly and do not need to be feared. However, there are still some lingering concerns with saturated fats:

- Saturated fats occur principally in animal products. In fact, saturated fats may be little more than a surrogate for meat and animal product intake. Data comparing vegetarians to omnivorous populations have shown that vegetarians enjoy longer life with fewer cardiovascular events (Sabaté 2003). Higher intakes of animal products, whether from the saturated fat component or some other component (such as AGEs, iron-containing proteins, or nitrogenous by-products) have been associated with increased risk for prostate, colon, and possibly breast cancers; increased likelihood of diabetes; greater likelihood of osteoporosis,
and—paradoxically—loss of lean muscle mass (as compared to low-fat or high unsaturated fat diets) during a weight loss effort.

- Foods rich in saturated fats (though not necessarily due to saturated fat itself) increase arterial constriction, i.e., “endothelial dysfunction” compared to foods containing monounsaturates or polyunsaturates. This may contribute to arterial injury of the sort that encourages plaque growth.
- High intakes of animal products (AGEs?) paradoxically block insulin responses, i.e., create a diabetic-like pattern.
- High intakes of animal products increase blood pressure.
- Animal fats are the repository for pesticide residues and hormones in factory farm-raised livestock. (This tends not to be an issue in grass fed, organic livestock, nor in wild game.)

Effects of saturated fat are very difficult to separate from that of animal products. Unfortunately, nutritional studies to date have not conclusively separated saturated fat from its principally animal sources, nor have they separated the effect of saturated fats from other potential problem sources like AGEs.

Vocal critics of standard “cut saturated fat” advice point out that the clinical studies examining adverse effects all failed to reduce carbohydrate intake to low levels. They argue that much of the adverse health effect of saturated fats only becomes an issue in the presence of carbohydrates and that the effects of saturated fats have not been examined in isolation to truly gain an understanding of their real potential for good or bad. While such studies have been limited, the Track Your Plaque lipoprotein experience is consistent with this stand.

Nutrition is a work in progress, with new evidence continually causing re-examination of the old. For the present, however, our stand on animal products—and saturated fat—from the perspective of the New Track Your Plaque Diet is:

- We do not restrict saturated fats. The only time to consider cutting back on saturated fat is if LDL values (including apoprotein B and LDL particle number) are above your target values.
- Because AGEs accompany animal products that are cooked at high temperature (350° F or greater), cooked for prolonged periods (e.g., roasting), deep-fried, barbecued, or cured (see box), animal products are best eaten rare or at least not well-done; boiled, baked, steamed or sautéed; and not eaten in large quantities.
- Cured meats that contain sodium nitrite, including bacon, sausage, and hot dogs, should be minimized because of their carcinogenic potential confirmed across multiple epidemiologic studies. Cured meats are also rich in AGEs.
• Saturated fat sources that can be included: red meats, pork, chicken, and turkey, (all preferably grass fed or organic), or wild game; whole eggs; cheese, provided it is real cultured cheese.
• Use butter lightly due to its unusually high AGE content.

Diet Principle #3:

Unlimited vegetables, some fruits

Limited potatoes, rices, starchy beans

No more than 2 servings fruit per day, preferably berries

Vegetables are easy: Follow the rule of eat vegetables, vegetables, and more vegetables.

Vegetables should serve as your primary source of fiber, phytonutrients (including flavonoids, polyphenols, and carotenoids, the brightly colored substances that confer anti-cancer and heart disease-protecting effects), vitamins, and minerals, but make only a minor contribution to carbohydrates and calorie load. Vegetables are the closest we come to a perfect food group.

Vegetables are also a substantial source of potassium and exert an alkalinizing (acid-neutralizing) effect, benefits that are principally important for bone health, but may also provide protection from cancer and heart disease (Lanham-New 2008).

Focus on vegetables over and above all other food sources. Take advantage of the vast variety and versatility of vegetables as the foundation of your nutrition program: eggplant; peppers; alliums like onions, garlic, scallions, shallots; cucumbers; zucchini; leafy plants like red leaf, Boston, Romaine lettuces and spinach; sprouts; cabbage; roots like radishes and carrots; celery; mushrooms like shiitake and portabella; herbs like basil, oregano, mints, coriander.

Nearly all plant-based food sources are beneficial. Among the rare exceptions are white and red potatoes, starchy beans, and processed rices (white and brown); while potatoes and rice have some healthy components (e.g., vitamin C and potassium), these vegetables release blood sugar similar to table sugar. These vegetables should therefore be minimized, though not necessarily eliminated (since they generally do not yield the same addictive potential as wheat.) “Vegetables” like French fries and fried rice should virtually never be consumed.
Vegetables are best eaten fresh, as they are most nutrient dense. Frozen vegetables are a good second choice if fresh is unavailable. Canned vegetables (and canned food in general) should be avoided, not just because of reduced nutrient density, but also because of bisphenol A released from the lining, a substance associated with 40% increase in heart disease when ingested habitually (Lang 2008).

Phytonutrients from vegetables, including flavonoids, polyphenols, and carotenoids, such as quercetin, luteolin, catechins, anthocyanidins, and literally thousands of others (more than 4000 at last count) are widely believed to be the factors responsible for the marked reduction in cancer, high blood pressure, and heart disease in populations that include greater quantities of vegetables and fruits in their diet. The means by which these benefits are accomplished is an area of active research, with findings pointing towards anti-oxidant effects (e.g., peroxynitrite scavenging); endothelial health through enhanced nitric oxide synthesis; improved insulin responses; anti-inflammatory effects, including reduced expression of adhesion molecules, abnormal growth factors, and thromboxane; blood-thinning effects, including reduced activation of clot-forming platelets; decreased LDL cholesterol and increased HDL cholesterol; reduced blood pressure (Mulvihill 2010; Willcox 2009).

Vegetables should be eaten first and in unlimited quantities. Seconds, thirds—as long as it's healthy vegetables, eat them in unrestricted quantities and fill up on them before you move on to anything else.

Fruits, like vegetables, are rich in phytonutrients and therefore share in providing many of the same beneficial effects. However, fruits are far richer in sugar. Some, like bananas, have as much sugar as processed foods like cookies, candy, or breakfast cereals. Despite the benefits of fruits, eating too much (which happens quite often as people shift from less processed foods to whole foods) can still trigger unhealthy patterns, like higher blood sugar, excessive insulin spikes, and inflammation.

It is therefore advised fruit be limited to no more than two servings per day, with a serving fitting flatly in the palm of your hand. Favor berries of all varieties (blueberries, raspberries, strawberries, cranberries, blackberries), citrus, melons, apples, peaches, pears, and kiwis. Because of high sugar load (>20 grams per 4 oz serving), we should minimize bananas, grapes, mango, and any dried fruit (raisins, dried apricots, figs).

**Diet Principle #4:**
**Unlimited raw nuts and seeds**

**Unlimited:**
Nuts are number two in the hierarchy of healthy foods, after vegetables. Like vegetables, nuts are rich in vitamins and minerals (especially magnesium), fibers, healthy oils (monounsaturates), and are wonderfully filling. People who eat nuts have been consistently shown to experience fewer heart attacks (as much as 50% reduction), have lower LDL cholesterol and blood pressure, and reduced incidence of diabetes (Fraser 1992; Jenkins 2002). A quarter-cup or more of nuts per day can reduce LDL cholesterol 20 mg/dl or more (Kendall 2010).

Nuts contain polyunsaturated fats, which makes them less than perfect. But nuts are, on balance, healthy because of their overall composition. Including abundant nuts in the diet should not overload your diet with polyunsaturates, provided you are not adding other polyunsaturates like corn, sunflower, safflower, grapeseed, or other vegetable oils.

Because heating or roasting changes the structure of oils (oxidation, acrylamide formation that has been linked to cancer, and AGE formation) and make any sugars more available, nuts are best eaten raw. Raw nuts are often sold bulk or in bags; they do not need to be shelled (unless you prefer them that way). They are sometimes labeled “raw,” or simply lack any further description such as “roasted.”

Dry roasted is second best, but be sure that your nuts are dry roasted with no other processing introduced, such as roasting in hydrogenated cottonseed oil, wheat flour, cornstarch, maltodextrin, salt, sugars, etc., all common with roasted nuts. Processed nuts, often known as party mixes, honey roasted, mixed nuts, beer nuts, etc. should be avoided; these contain unhealthy ingredients and do not provide the benefits of raw nuts. In fact, these processed versions of nuts are the reason why nuts acquired a bad reputation in past: They increase LDL cholesterol, reduce HDL, increase resistance to insulin, and make us fat. Avoid
The best nuts are those with a fibrous coating and include:

- Almonds
- Walnuts
- Pecans
- Pistachios
- Filberts
- Brazil
- Hazelnuts

Raw cashews, though not as fiber-rich and a bit more carbohydrate-rich, are still a great snack choice. Mix raw cashews with some of the fiber-coated nuts listed above.

Peanuts are not nuts, but legumes, and cannot be eaten raw; dry roasted is preferable, provided they are truly just dry roasted with no other added ingredients. Check the list of ingredients; it should read “dry roasted nuts”—period. No cornstarch, sucrose, maltodextrin, etc.

In the world of seeds, healthy choices include:

- Pumpkin seeds (pepitas)
- Sunflower seeds
- Pine nuts
- Sesame seeds

Like nuts, seeds are best consumed raw. Pumpkin seeds in particular provide a substantial quantity of magnesium of approximately 160 mg per quarter cup; sunflower seeds likewise provide plenty of magnesium, approximately 120 mg per quarter cup.

Both raw nuts and seeds can be consumed in unlimited quantities. Contrary to popular perception, as long as they are raw and unprocessed, they will not cause weight gain.
Diet Principle #5: Unlimited healthy oils

Unlimited:

- Flaxseed oil
- Extra-virgin olive oil
- Avocado oil
- Coconut oil

Four healthy (non-marine) oils are advocated in the New *Track Your Plaque* Diet:

- Flaxseed—Richest in linolenic acid (51%), the so-called non-fish source of “omega-3” (though it will *not* substitute for fish oil).
- Olive—Rich in the monounsaturated, oleic acid (70%), and polyphenols.
- Avocado—Like avocados, avocado oil is rich in monounsaturates (70%).
- Coconut oil—Although coconut oil was regarded as a “no no” in past due to high saturated fat content, over 50% of the fatty acids contained are the neutral saturated fat, lauric acid, that yields little to no effect on such factors as LDL cholesterol. It’s also versatile and delicious.

All five oils are low in the omega-6 linoleic acid and are neutral with respect to inflammatory patterns or may exert modest anti-inflammatory effects (Zhao 2004). (Omega-6 linoleic acid activates thromboxane and other inflammatory patterns.)

These oils can be consumed *ad libitum*; add as much as you want to salads and other foods. A useful strategy using these four oils is to include some in every meal, especially breakfast. Not only does this induce satiety (fullness and diminished cravings), but insulin and sugar fluctuations are reduced. This leads to better weight control and weight loss, particularly when combined with the strategy of reducing or eliminating wheat, cornstarch, oats, and sugar products.

Of course, oils should never be heated to high temperature (e.g., deep-frying), as it triggers formation of unhealthy oxidative byproducts and AGEs. Minimal (e.g., lightly sautéed) or no heating is preferable whenever possible, especially with olive and flaxseed oils, as linolenic acid and polyphenols degrade rapidly with heat. Oils are therefore best added towards the conclusion of cooking, e.g., olive oil added to scrambled eggs just after scrambling, olive oil brushed on asparagus or chicken just before removing from the oven or grill.
Diet Principle #6: Foods Should Be Unprocessed

Unprocessed foods are whole foods:

Whole, unprocessed foods are not dried (like oatmeal and instant mashed potatoes), not powdered (instant soups, sauces), and not a “mix” (pancake and cake mixes, macaroni and cheese). They do not require reconstitution—adding water and heating, or some similar process. Whole, unprocessed foods are not modified by hydrogenation, desiccation, are not sweetened and don't contain artificial flavorings or colorings.

Unprocessed foods tend to look like they occur naturally. You may have to remove an outer shell (nuts) or skin (oranges, avocados) but they remain essentially intact. Of course, you may need to cut whole foods into smaller pieces, but the basic structure remains the same. Unprocessed foods are generally fresh.

When food is left whole, it retains more of its original naturally occurring nutrients. It is also digested more slowly, causing a natural slow, gradual rise in blood sugar or none at all. Diabetics who switch to a diet of unprocessed foods commonly witness dramatic drops in blood sugar, often sufficient to reduce their requirements for medication or insulin.

Unprocessed foods are colorful foods. Look at the wonderfully deep colors of plums, eggplant, oranges, tomatoes, and spinach. Colorful foods are rich in flavonoids, naturally-occurring substances that lower LDL cholesterol and raise HDL, lower blood pressure, block abnormal clotting by platelets, block the adhesion of inflammatory blood cells to plaque, and reduce risk of heart attack.

Processing destroys good food!

Processing is not simply heating or drying. Processing often destroys heat-sensitive phytonutrients, oxidizes oils, and increases glycemic index.

Processing frequently involves the addition of undesirable additives to improve taste, consistency, or extend shelf-life—hydrogenated oils, food colorings, sweeteners like high-fructose corn syrup and sugar, thickeners like cornstarch, and synthetics. They make food look prettier, last longer, and maintain texture and consistency during storage, but do health little or no good. High-fructose corn syrup is a sweetener that kids love and is found in everything from fruit drinks to spaghetti sauce that increases triglycerides and contributes to undesirable lipoprotein patterns like small LDL and VLDL, and even increases the likelihood of diabetes.
Processed foods are a major culprit behind the national epidemic of metabolic syndrome. Processed foods are all around us. Shelf after shelf, aisle after aisle of eye-catching, colorful, enticing processed foods. Not one or two kinds of cookies or cupcakes to choose from, but hundreds. The temptations are tremendous.

Many people struggle when forced to part with the glitz and glamour of processed foods. Marketing people who create these ads are very clever. They know that advertising can make you feel good about eating certain foods. They want you to feel proud to feed your family a “healthy” dish, sexy if you drink a certain drink (think Coca Cola®), successful if you can whip up a dinner of convenience foods in five minutes.

You'll get none of this reinforcement when you restrict yourself to the world of unprocessed foods. Unprocessed foods are not glamorous. They don't have fancy labels or packaging. You might even have to buy them “bulk.” Yet it's the unprocessed, unrefined foods that are powerful tools for health. Whole, unprocessed foods are more filling, take longer to digest, and keep you satisfied longer.
Putting the program to work
Have you ever noticed how often you eat the same foods?

For most of us, 20 or so different foods comprise 90% of our meals over the course of a week. We then repeat the same 20 foods, week after week. You may modify preparation and mix combinations, but tend to rely on a small
number of food choices. Creating new nutrition habits really means just selecting a handful of healthy new foods and re-organizing meals around these new choices.

There’s no need to develop hundreds or thousands of new gourmet recipes in order to follow the New Track Your Plaque Diet program. Simply choose 10–20 basic dishes you enjoy to provide the basis for your broader diet.

Use unique toppings and condiments—mustards (hot, brown, horseradish, Dijon), horseradish, salsa, pico de gallo, tapenades, pestos, wasabi sauces. Whenever choosing oil-based condiments, try to choose those made with olive oil. Avoid non-fat/low-fat salad dressings made with high-fructose corn syrup.

Do your grocery shopping in the outer aisles of the store, where you find produce and dairy products. You’ll avoid the temptations of the processed, wheat, cornstarch, and sugar foods in the center aisles. If you’re just starting to re-design your diet, you may spend a lot of time reading labels and experimenting with new foods and methods of preparation. But commit yourself to a few months of effort and you'll find that eating healthy will become second nature.

Be guided by the idea that you are returning to eating and preparing real food— not food defined as “healthy” by processed food manufacturers. Not low-fat, microwaveable, just-add-water, “heart healthy” foods, as defined by silly criteria.

Real vegetables, raw nuts, vegetables, meats, eggs, cheese, and fruit. Real food.
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Chapter 10

Vitamin D: Crucial nutrient for the Track Your Plaque program

The addition of vitamin D supplementation to the program has supercharged the Track Your Plaque approach, allowing better, faster, and more vigorous results than ever before.

Since we added vitamin D supplementation to the list of Track Your Plaque strategies in 2007, there has been no turning back. It made control over the factors that cause plaque easier. People feel stronger, mentally clear, and more energetic. Vitamin D has substantially increased the number of people who have been able to stop or reduce heart scan scores.

In fact, the recognition of vitamin D’s powerful health effects is one of the most important health discoveries of the last 100 years, one we apply to substantial advantage in the Track Your Plaque program.

Vitamin D deficiency: Coronary risk factor

Look up “coronary risk factor” in any standard medical textbook and you’ll find a list that includes high cholesterol, diabetes, smoking, sedentary lifestyle, obesity, and hypertension.

How about vitamin D deficiency? Not even listed. You may find vitamin D in the list of causes of childhood rickets and osteoporosis. But a coronary risk factor? You’ll not find it.
The case is building for vitamin D as a cause for coronary heart disease. Many of the beneficial effects of restoring vitamin D to normal levels—such as anti-inflammatory, anti-hypertensive, and anti-diabetic effects—are the very same that reduce heart disease risk. Vitamin D experts argue that vitamin D receptors are involved with some 200 genes that influence cardiovascular health (Holick 2010).

An early hint that deficiency of vitamin D might underlie heart disease came from a New Zealand study of 179 people presenting with heart attacks. Heart attack sufferers proved to have lower vitamin D blood levels (25-OH vitamin D3) compared to people without heart attacks (Scragg 1990). In a nationwide database of 259,891 heart attacks in the U.S. maintained by the National Registry of Myocardial Infarction, heart attacks surged by 53% during sun-deprived winter months compared to summer (Spencer 1998). Further corroboration came from observations in semi-tropical or tropical climates, where intense sun shines year-round and seasonal variation in heart attack rates does not occur (Ku 1998).

If vitamin D is indeed related to heart disease risk, then we would also expect to see fewer heart attacks in sunny climates and more heart attacks in cooler climates. Indeed, Dr. David Grimes and colleagues at the Blackburn Royal Infirmary in England demonstrated a consistent worldwide relationship between sunlight exposure and heart disease: the farther north in latitude, the more heart attacks are observed (Grimes 1996).

Osteoporosis and heart disease are known to share a curious close relationship. For years, it’s been known that women with osteoporosis are more likely to have coronary heart disease (Kuller 2000). 2500 postmenopausal women with osteoporosis were observed to have four-fold greater risk of heart attack (Tanko 2003). A University of Illinois study showed that coronary calcium score in women with osteoporosis averaged 222, while women with normal bone density had average coronary calcium scores of 42 (Barengolts 1998). In other words, more calcium (and thereby plaque) in coronary arteries is associated with less calcium in bones; vitamin D deficiency may explain the apparent relationship of these phenomena, since calcium controls the fate of calcium.

Vitamin D deficiency extends its effects to vascular disease beyond the coronary arteries. A recent study determined that, the greater the vitamin D deficiency, the more carotid atherosclerotic plaque was present by carotid ultrasound (Targher 2006). Similarly, peripheral arterial disease of the pelvis and legs is twice as common in people with vitamin D deficiency (Melamed 2008).

Kidney disease may serve as a model for studying the benefits of vitamin D replacement on reducing risk of heart attack. Inadequate vitamin D is likely part of the reason for the high incidence of cardiovascular disease in people with abnormal kidney function. People with little or no kidney function have disturbed
control over vitamin D activity, since the kidney is required to produce active 1,25-(OH)_2 vitamin D from 25-OH vitamin D. In the majority of people with kidney disease, deficiency of 25-OH vitamin D co-exists with failure to produce 1,25-(OH)_2 vitamin D. In one study of people with severe kidney disease, 92% of 273 participants had marked deficiency of 25-OH vitamin D, with over half having levels as low as 15 ng/ml (Taskapan 2006).

As we would predict, in a Japanese study of 240 people with kidney failure on dialysis, participants who received treatment to correct vitamin D deficiency resulted in 70% (relative) reduction in death from heart disease (Shoji 2004).

The emerging science argue strongly in favor of vitamin D as an important risk for cardiovascular disease with the potential to safely, inexpensively, and dramatically reduce risk.

Vitamin D: Deficiency is the rule

“The basis for adult vitamin D recommendations [was] arbitrary. Thirty-six years ago, an expert committee on vitamin D could provide only anecdotal support for what it referred to as ‘the hypothesis of a small requirement’ for vitamin D in adults and it recommended one-half the infant dose, just to ensure that adults obtain some from the diet.”

Dr Reinhold Vieth
University of Toronto

Few adults living a modern life have a healthy level of vitamin D in their bodies. Deficiency is the rule with the majority of people deficient, not uncommonly to severe degrees.

Human skin is rich with latent pre-vitamin D converted to its active form by sunlight. Humans are meant to obtain vitamin D through sunlight exposure. “Vitamin” D is not really a vitamin, as it is not contained to any significant degree in food, nor was it meant to be obtained through oral intake. Over the centuries, people have migrated to colder, sun-deprived climates, work indoors in offices or factories, travel by car, exercise in gyms, and wear clothes that cover all but 5% of body surface area. Oral intake of vitamin D has only become necessary as humans developed lifestyles involving less and less sun exposure. Add to this the sun phobia fostered by dermatologists advising us that sun exposure causes skin cancer, and most of us are terribly sun-deprived and thereby leave the pre-vitamin D in the skin unactivated.

In the 1960s, it was known that administration of 200 units vitamin D per day was just enough to prevent osteomalacia, a form of bone softening. It was also clear that children who failed to receive at least 300–400 units per day, usually supplemented as cod liver oil, developed “bow legs,” or rickets, due to abnormal bone maturation in the legs. In 1963, when the Institute of Medicine drafted the original Recommended Daily Allowance (RDA) for vitamin D, there
were literally no scientific data available to help determine requirements for health in children or adults, i.e., not just prevention of osteomalacia and rickets. The vitamin D dose recommended was purely—and admittedly—fabrication.

Further investigation using blood levels of vitamin D have uncovered the extraordinary prevalence of deficiency. A study of 1500 women receiving treatment for osteoporosis showed that 52% of participants were deficient (defined as <30 ng/ml 25-OH vitamin D); 18% were severely deficient (<20 ng/ml 25-OH vitamin D) (Holick 2005). The likelihood of vitamin D deficiency ranges from a low of 36% in healthy adults 18–29 years old, to 57% of a general adult population in the U.S., to as much as 100% in the elderly (Holick 2006). North of 37 degrees latitude (approximately a line drawn horizontally connecting Norfolk, Virginia, to San Francisco, California), sunlight is insufficient to trigger vitamin D conversion in the winter months, even if sufficient skin surface area is exposed (Webb 1988). Living in a southern climate is no guarantee that deficiency won’t occur. A study conducted in Miami, Florida showed that 40% of 222 adults were deficient in winter, with little improvement in summer (Levis 2005).

Several groups of people are at especially high risk for vitamin D deficiency: the elderly, who convert 4 times less vitamin D with sunlight exposure compared to 20-year olds; obese; and African-Americans and other dark-skinned races due to high melanin skin content (Vieth 1999; Wortsmann 2000).

The more recent response from the Institute of Medicine (IOM) to studies demonstrating widespread deficiency has been to increase the Recommended Daily Allowance (RDA) for vitamin D intake in adults to 600 units per day and the Tolerable Upper Intake Level (UL) to 4000 units per day. Oddly, the most recent opinion offered by the IOM based their recommendations on achieving a 25-hydroxy vitamin D level of 20 ng/ml, a level they felt sufficient to avoid osteomalacia and rickets (http://www.iom.edu/Reports/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D.aspx).

Unfortunately, obtaining the RDA virtually guarantees vitamin D deficiency in most adults. We can wait for the Institute of Medicine to catch up to science . . . or we can just go ahead and correct our deficiency and obtain all the health benefits of vitamin D restoration.

There are few dietary sources of vitamin D. An 8 oz glass of milk contains 100 units (though not always—inconsistency reigns) (Holick 1992). Other dairy products, like cheese and yogurt, usually have little or no vitamin D added. Oily fish like salmon, mackerel, and sardines naturally contain 200–360 units vitamin D per 3½ ounce serving (and may be part of the reason Inuits had less heart disease when following their traditional diet). Many breakfast cereals contain 40–100 units per serving.
Despite this, more than 50% of people fail to obtain even the modest RDA for vitamin D from diet every day. Strict vegetarians and lactose intolerant individuals are especially likely to be substantially vitamin D deficient (Zadshir 2005).

More recent vitamin D research, however, has uncovered the fact that exposure to just 10–20 minutes of sunlight yields the blood-level equivalent of 20,000 units of vitamin D taken orally (Vieth 1999). Why would the naturally intended source of vitamin D yield levels far beyond that specified by the RDA?

A study conducted during the winter months in Omaha, Nebraska in healthy adult men showed that the participants utilized 3000–5000 units of vitamin D per day to maintain a steady vitamin D blood level. The researchers concluded that the current RDA is inadequate to meet these requirements (Heaney 2003).

In the Track Your Plaque experience, deficiency is even more common than suggested by these studies. Most studies now define vitamin D deficiency as levels below 30 ng/ml. However, the ideal level of vitamin D (25-OH vitamin D) is likely higher.

**Vitamin D deficiency: A lot more than osteoporosis**

What are the potential consequences of vitamin D deficiency? Here’s where it gets interesting.

Among the wide-ranging effects that have been recently identified are:

- **Blood pressure regulation**—People deficient in vitamin D are much more likely to have high blood pressure. Restoration of vitamin D reduces blood pressure (Pfeiffer 2001). A Swedish study demonstrated a 10–20 mmHg reduction in blood pressure in men with pre-diabetes when vitamin D was supplemented (Lind 2005). Vitamin D likely exerts this effect by blocking the blood pressure hormone renin (Li 203).

- **Anti-cancer effects**—The cancer preventing effects of vitamin D appear to be quite potent, judging by experimental and animal preparations and in limited human experience. The human experience in prostate and colorectal cancer suggests that the experimental benefits extrapolate to humans, with improved cancer remission and survival with higher vitamin D levels (Vieth 1999).

- **Anti-inflammatory effects**—Several recent studies suggest potent effects on reducing inflammation, as measured by inflammatory markers C-reactive protein (CRP) and matrix metalloproteinase (MMP). Reductions in CRP of 60% or more have been documented. (Compare this to the 30% reduction of CRP by statin cholesterol drugs.) In a British study of 171 human subjects, long-term vitamin D administration yielded an unprecedented 68% reduction.
in MMP levels (Timms 2002). No other strategy has achieved this magnitude of reduction of an inflammatory measure with the exception of weight loss.

• Anti-diabetic effects—Several studies have shown that vitamin D administration reduces blood sugar and increases sensitivity to insulin. Improvement in insulin sensitivity triggers a cascade of benefits, including beneficial effects on lipoproteins (reduced triglycerides, increased HDL) (Zitterman 2006).

• Osteoporosis prevention—Vitamin D is sorely neglected in this area. Women are prescribed expensive prescription drugs costing hundreds of dollars per month yet have uncorrected vitamin D deficiency. Vitamin D replacement to healthy levels increases bone density more effectively than calcium supplementation (Cranney 2007). Intestinal absorption of calcium is doubled when sufficient vitamin D is present (Holick 2006).

• Prevention of stress fractures—Even young men with an average age of 19 years were shown to experience more stress fractures when blood levels of 25-OH vitamin D3 were low (Ruohola 2006).

• Prevention of multiple sclerosis—Several studies have related vitamin D deficiency to increased likelihood of developing this debilitating neurologic disorder (Hayes 1997). Vitamin D treatment trials are underway.

The list of organs and health conditions that are susceptible to vitamin D’s effects is growing rapidly. In fact, it appears that few, if any, organ systems are not affected by vitamin D deficiency and improved with restoration. Restoration of vitamin D is among the most important steps you can take to gain overall health.

How much Vitamin D is enough?

Personally, I take 5,000 units in the late fall, winter, and early spring, and then I vary doses the rest of the time depending on sun exposure. I also have my 25(OH)D level checked twice a year, once in the early spring and again in the early fall. My 10-year-old daughter takes 2,000 units a day in the winter months and my three-year-old takes 1,000 units a day in the winter.

Dr. John Cannell
The Vitamin D Council
www.vitamindcouncil.com

If you’re from the northern U.S. (states like Massachusetts, New York, Pennsylvania, Wisconsin, Michigan, the Dakotas, etc.), Canada, or northern Europe, there’s a high likelihood that you’re deficient in vitamin D. If you’re like most Americans, you get sun sporadically in summer (weekends) and virtually none from September to April. Dark-skinned races are at even greater risk of vitamin D deficiency, since melanin pigment in skin acts as a natural sunscreen. Dark-skinned individuals require five times longer sun exposure to obtain the
same amount of vitamin D as a fair-skinned person. African-Americans, for this reason, are among the most vitamin D deficient of all.

The best way to know your vitamin D status is to measure the blood level of 25-OH vitamin D. (25-OH vitamin D should not be confused with 1,25-(OH)$_2$ vitamin D, the related measure that is useless for gauging your vitamin D status, a common tripping point for physicians. 1,25-(OH)$_2$ vitamin D is the kidney product of 25-OH vitamin D, a better reflection of kidney function than vitamin D status.) The minimum level of 25-OH-vitamin D required for health is controversial. However, most authorities have argued that a rock-bottom minimum 25-OH-vitamin D3 level of 30 ng/ml, or 75 nmol/l, is a level at which phenomena associated with deficiency begin to correct (Zitterman 2003). However, vitamin D authority Dr. Reinhold Vieth of the University of Toronto and others have argued that a blood level of 40 ng/ml (100 nmol/l) should be achieved (Vieth 1999). Vitamin D toxicity generally does not develop unless vitamin D blood levels exceed 150 ng/ml (200 nmol/l) (Holick 2006; Vieth 1999).

Despite the powerful and compelling observations made in epidemiologic studies, we still lack large prospective, placebo vs. vitamin D studies that explore the levels of 25-OH vitamin D that correlate with all measures of health, i.e., drug-like studies. (This is part of the reason for the IOM’s reluctance to recommend higher vitamin D intakes.) Nonetheless, in the Track Your Plaque experience, we’ve aimed for 60-70 ng/ml with favorable results: Phenomena of deficiency reliably recede and, in several thousand experiences, we have never observed any evidence of toxicity (e.g., increased calcium blood levels).

Ten minutes of sun exposure in midday, wearing shorts and t-shirt to expose skin surface area, during the summer will provide most Caucasians younger than 30 years old plentiful vitamin D. This limited time minimizes the risk of skin cancer. Any longer than this and you should apply a sunscreen (which blocks sunlight as well as vitamin D activation in the skin.)

However, if you are older than 30 years old, or if sun exposure is sporadic, supplementation is important to obtain the full benefit of vitamin D’s panel of biologic effects. Cholecalciferol, or vitamin D3, is the preferred form, not ergocalciferol, or vitamin D2, since D3 is 70% better absorbed than D2, more effectively increases blood levels of 25-OH vitamin D, and has both longer duration of action and persistence in tissues (Trang 1998). D2 is the plant-sourced form and the form found in invertebrates; D3 the human form. There is no reason to prefer the plant form over the human form. The prescription form of vitamin D is D2 and, for this reason, is the form often (wrongly) prescribed by doctors.

In the Track Your Plaque experience, the individual dose required for vitamin D to achieve our target of 25-OH-vitamin D of 60-70 ng/ml varies widely. The majority of men and women require 6000 units per day year round to
achieve this level. Occasionally, someone will require far less; many require more. 25-OH vitamin D levels should be monitored, with a blood level assessed no sooner than six weeks after initiation or change in dose, since that time period is required for the full effect of the dose change to stabilize (Viet 1999). At least for the first few years, we assess 25-OH vitamin D levels twice per year, ideally during summer and again in winter, in case you are among the few who maintain the ability to activate vitamin D in the skin from sun exposure.

Oil-based gelcaps of cholecalciferol (vitamin D3) are absorbed more consistently than tablets; drops are also well absorbed. Vitamin D now commonly comes in 1000, 2000, 4000, and 5000 unit gelcaps, making higher doses easier to manage.

Anyone with kidney disease, cancer, glandular disorders, sarcoidosis, or a history of high calcium blood levels should only take vitamin D with medical supervision.

**Vitamin D and heart disease**

Vitamin D has assumed a crucial role in the *Track Your Plaque* program. Let’s review the experiences that led to putting vitamin D at the top of our list of important strategies for plaque control.

Several recent analyses have demonstrated increased likelihood of diabetes, hypertension, myocardial infarction (heart attack), sudden cardiac death, and cancers with lower levels of 25-OH vitamin D (Anderson 2010; Autier 2007; Steinvil 2010). Mortality from cardiovascular disease, for instance, in people with 25-OH vitamin D levels <30 mg/dl is doubled over a period of 7 years.

In the NHANES (1988-1994) survey of 16,603 Americans, those with 25-OH vitamin D levels of 20 ng/ml or less had greater likelihood of coronary disease than participants with levels 20 ng/ml or greater; even after factoring out hypertension, LDL cholesterol, diabetes, smoking, high BMI and other risk factors, low vitamin D levels increased (relative) risk for coronary disease by 20% (Kendrick 2009). The 2001-2004 NHANES survey showed that 74% of participants had 25-OH vitamin D levels below 30 ng/ml; likelihood of coronary disease increased with lower 25-OH vitamin D categories, with 5.3%, 6.7%, and 7.3% coronary heart disease with 25-OH vitamin D levels of 30 ng/ml or greater, 20-30 ng/ml, and less than 20 ng/ml, respectively. Similarly, for heart failure, values were 1.5%, 2.4%, and 3.2%; for stroke 2.5%, 2.0%, and 3.2%; and for peripheral vascular disease 3.6%, 5.0%, and 7.7% (Kim 2008).

In the 18,225-participant Health Professionals Follow-up Study, men with 25-OH vitamin D levels of 15 ng/ml or less had 2.42-fold greater likelihood of heart disease over 10 years compared to males with 25-hydroxy vitamin D levels of 30 ng/ml or greater (Giovannucci 2008). In the 1739-participant Framingham
Offspring Study, 25-OH vitamin D levels of 15 ng/ml or less were associated with 50% greater likelihood of a cardiovascular event over five years (Wang 2008). Of 3299 patients who underwent coronary angiography for various indications, those with 25-OH vitamin D levels of 10 ng/ml or less had five-fold greater likelihood of sudden cardiac death or death from heart failure over 7.7 years compared to those with levels of 30 ng/ml or greater (Pilz 2008).

Several insights into how vitamin D might reduce coronary risk and influence coronary plaque are emerging:

• Vitamin D enhances insulin responsiveness and reduces hemoglobin A1c (Borissova 2003). Recall that poor sensitivity to insulin underlies multiple lipoprotein abnormalities, including formation of small LDL particles.
• Inflammatory cell activity and oxidized LDL particle uptake by plaque are suppressed (Oh 2009).
• Having adequate levels of vitamin D reduces hyperparathyroidism, i.e., excessive activation of the parathyroid glands that leads to higher levels of parathyroid hormone, higher blood levels of calcium, greater resistance to insulin, and greater inflammatory responses (Lee 2008).
• Vitamin D improves endothelial responses by 50% in vitamin D deficient subjects and reduces measures of oxidative phenomena by 18% (Tarcin 2009). Part of the explanation underlying improved endothelial responsiveness may be reversal of the effects of advanced glycation end-products (AGEs) that block nitric oxide synthase activity (Talmor 2008).
• Vitamin D reduces the activation of the blood pressure-increasing hormone, renin; administration of vitamin D reduces systolic and diastolic blood pressure (Li 2002).
• Higher 25-OH vitamin D levels are associated with reduced levels of asymmetric dimethylarginine, a compound that has been associated with endothelial dysfunction and cardiovascular disease. Asymmetric dimethylarginine blood levels also vary with season: lowest in summer and autumn, highest in winter and spring, likely corresponding to seasonal sunlight exposure and vitamin D (Ngo 2010). (Incidentally, observations such as this are part of the reason why we removed L-arginine used previously in the Track Your Plaque program, since many of the benefits of this amino acid can be provided by restoration of vitamin D.)

Unfortunately, the vitamin D experience does not yet include a large prospective clinical trial in which doses of vitamin D sufficient to achieve desirable levels have been used, with participants observed over several years. For instance, the Women’s Health Initiative Trial, in which only 400 units vitamin D was administered (along with 1000 mg calcium); not surprisingly, no effect on coronary calcium scores or cardiovascular mortality was observed with this low dose (Manson 2010; LaCroix 2009).
In the *Track Your Plaque* experience to date, adding vitamin D to our list of suggested strategies, we have witnessed more people obtain slowing, stopping, or reversal of coronary plaque and to a larger degree than achieved prior to the introduction of vitamin D. With the addition of vitamin D, we have seen reductions in coronary calcium (heart scan) scores of 20%, 40%, 60% or more, magnitudes of reduction that substantially exceed our experience prior to adding vitamin D.
Vitamin D: Vitamin or Hormone?

Vitamins are crucial participants in the body's reactions and are generally obtained from food. Vitamin C, for example, comes from citrus fruits and vegetables. Vitamin K1 comes from green vegetables. The B vitamins are found in meats, dairy products, and grains.

How about vitamin D? What foods contain vitamin D? It's a short list:

<table>
<thead>
<tr>
<th>Food</th>
<th>International Units (IU) vitamin D per serving</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cod liver oil, 1 Tablespoon</td>
<td>360</td>
</tr>
<tr>
<td>Salmon, cooked, 3½ ounces</td>
<td>360</td>
</tr>
<tr>
<td>Mackerel, cooked, 3½ ounces</td>
<td>345</td>
</tr>
<tr>
<td>Tuna fish, canned in oil, 3 ounces</td>
<td>200</td>
</tr>
<tr>
<td>Sardines, canned in oil, drained, 1¼ ounces</td>
<td>250</td>
</tr>
<tr>
<td>Egg, 1 whole (vitamin D is found in egg yolk)</td>
<td>20</td>
</tr>
<tr>
<td>Liver, beef, cooked, 3½ ounces</td>
<td>15</td>
</tr>
</tbody>
</table>

Most vitamin D is contained in food because it is “fortified,” or added during manufacture:

- Milk, nonfat, reduced fat, and whole, 1 cup: 98
- Margarine, fortified, 1 Tablespoon: 60
- Cheese, Swiss, 1 ounce: 12

Ready-to-eat cereals fortified with 10% of the DV for vitamin D, ¾ cup to 1 cup servings (servings vary according to the brand): 40

(Modified from the Office of Dietary Supplements, National Institutes of Health)

You'll note that the only naturally-occurring food sources of vitamin D are fish, egg yolks, and liver. All other vitamin D-containing foods like cereal, milk, and other dairy products have vitamin D only because it is added.

Personally, it takes 8000 units of vitamin D per day to bring my blood level to a desirable 60-70 ng/ml. To obtain this from eating salmon, I would have to eat 78 ounces, or nearly 5 pounds of salmon—every day. Or, I could eat 40 cans of tuna fish. If I didn't want to eat loads of fish every day, I could drink 80 glasses of milk every day. After I recovered from the diarrhea, my vitamin D might be adequate, provided the milk indeed contained the amount stated on the label (which it often does not when scrutinized by the FDA).

If vitamin D is a vitamin, how are humans supposed to get sufficient quantities? I don't know anybody who can eat 5 lbs of salmon nor drink 80 glasses of milk per day. But don't vitamins come from food?
The problem is that vitamin D is not really a vitamin, it's a hormone.

If your thyroid hormone level was low, you'd gain 20, 30, or more pounds in weight, you'd lose your hair, become constipated, develop blood clots, become incapacitated by fatigue, and eventually die of coronary disease and heart failure. In other words, you'd suffer profound changes in health. Likewise, if thyroid hormone levels are corrected by taking thyroid hormone, you'd experience profound correction of these phenomena.

That's what I'm seeing with vitamin D: restoration of this hormone to normal blood levels (25-OH- vitamin D3 60-70 ng/ml) yields profound changes in health, including coronary health.

If there's one thing that I've come across lately that packs extraordinary potential to help us in reducing heart scan scores, it's the vitamin—sorry, the hormone—cholecalciferol, or D3.

**Vitamin D and HDL**

I'm seeing more and more of it and I am convinced that there is a relationship: significant boosts in HDL cholesterol from vitamin D supplementation.

This remains an underappreciated phenomenon. There have been several observers over the last two decades who have noted seasonal fluctuations in total cholesterol: cholesterol goes up in fall and winter, down in spring and summer; year in, year out. This phenomenon was unexplained but makes perfect sense if you factor in vitamin D fluctuations from sun exposure.

But little has been made of HDL’s relationship with vitamin D. (Data are limited to correlations of vitamin D levels to apo A1, for instance, in South Asian people.) But I am convinced that I am seeing it. Replace vitamin D to a blood level of 60-70 ng/ml and HDL goes up.

Say starting HDL is 36 mg/dl. You take niacin, 1000 mg; eliminate wheat like breakfast cereals, breads, cookies, and bagels; reduce overall carbohydrate intake; exercise or work in your yard four days a week; add 4 oz of red wine and 2 oz of dark chocolate a day. You shed 15 lbs towards your ideal weight. After 6 months, HDL: 45 mg/dl. Better but hardly great.

Add vitamin D at a dose of, say, 6000 units per day (oil-based gelcap, of course!), and re-check HDL six to twelve months later: 65 mg/dl would be typical.

I've seen it happen over and over. It doesn't occur in everybody but occurs with such frequency that it's hard to ignore or attribute to something else. What I'm not clear about is whether this effect only occurs in the presence of other strategies we use to raise HDL, a “facilitating” effect, or whether this is an independent benefit of HDL that would occur regardless of whatever else you do. Time will help clarify.
We are tracking our experience to see if it holds up, how, and to what degree on a more formal basis. Until then, a rising HDL is yet another reason—among many—to be absolutely certain your 25-OH-vitamin D3 level is at 60-70 ng/ml.

Is Vitamin D toxic?

"My primary care doctor said to stop the vitamin D because it's toxic. So I stopped it and I just take a multivitamin. He said that a multivitamin and two glasses of milk a day was all I needed."

So proclaimed Eleanor to me. This happens around once every week in my office, perpetrated by doctors frightened of vitamin D.

So I reminded Eleanor that, before starting vitamin D supplementation, her blood level of 25-OH-vitamin D3 had been 17 ng/ml—severe deficiency.

On 4000 units per day (oil-based gelcap), her blood level had been 37 ng/ml—still deficient, below the desirable range of 60-70 ng/ml. Even by the lax standards of most physicians, she’s just above the "normal" threshold of 30 ng/ml. But 4000 units was the dose Eleanor's doctor had declared "toxic."

When exactly does toxicity develop? There's not full agreement on this, but Dr. Michael Holick of Boston University, among the most experienced and insightful authorities on vitamin D, states that toxicity is more likely when blood levels exceed 150 ng/ml (Shinchuk 2007).

In other words, Eleanor and her doctor should not be concerned with toxicity, but with the persistent levels of deficiency she is suffering.

Some authorities call the behavior of vitamin D "biphasic": Deficiency is toxic, while excessive levels are also toxic. We're really just trying to achieve a middle ground, a "just right," level of vitamin D that is above deficiency but below toxicity.

In reality, deficiency is exceptionally common. Toxicity, on the other hand, is exceedingly rare. It can develop when someone inadvertently or unknowingly takes high doses without monitoring blood levels of 25-OH vitamin D. If the question of toxicity comes up, you and/or your healthcare provider can monitor blood calcium levels. (Provided vitamin D blood levels have been restored to our targets, I do not recommend that anyone take calcium supplements, certainly not take a dose above 500 mg per day. Occasionally, someone will develop high blood calcium levels from calcium supplementation.)

So, it's not toxicity that is the overwhelmingly common worry, but deficiency, severe and sustained.
What role calcium?

Conventional advice to prevent or treat osteoporosis and osteopenia, both representing “demineralization” or lack of bone calcium, is to supplement calcium, usually at a dose of 1200 mg or more per day. However, there are data specifically examining the cardiovascular consequences of such advice with some disturbing observations of increased risk. A recent meta-analysis, i.e., a reanalysis of data combined from a collection of smaller studies, suggests that calcium supplementation of 500 mg or more per day increased (relative) risk for heart attack by 27% (Bolland 2010).

On top of this, supplementing vitamin D doubles intestinal absorption of calcium (Seamans 2009). Potential for adverse effects from calcium may therefore be increased with restoration of the far more important vitamin D. Indeed, abnormally high levels of calcium have been observed on occasion with higher doses of calcium supplementation (even in the absence of vitamin D).

The notion that calcium supplementation may increase cardiovascular risk flies in the face of years of advice that calcium helps control osteoporosis. However, until we have clarification of this issue, for our plaque-control purposes I recommending that we take no more than 500 mg calcium per day. In fact, I believe that we could make a case for taking no calcium at all beyond that obtained through a healthy diet, provided vitamin D is restored.

Incidentally, there is more to bone health than even vitamin D and calcium; other nutrients play important roles in maintaining bone strength and architecture, such as vitamin K2, magnesium, and potassium, and non-nutritional issues like acid-base balance of the diet and muscle mass (bone density tends to follow muscle mass). By limiting calcium intake to a dose that does not contribute to cardiovascular risk, normalizing calcium absorption with vitamin D restoration, and taking steps to maintain bone health is an overall superior way to prevent or correct osteoporosis, rather than just taking a high dose of calcium.

Summary

The world of vitamin D is rapidly evolving. Compelling and substantial evidence suggest that most people, particularly those living in northern climates or having lifestyles with limited sun exposure, are substantially deficient.

Replenishing vitamin D can reduce blood pressure, reduce blood glucose and lessen resistance to insulin, and dampen inflammation. Growing evidence is adding support to the idea that vitamin D deficiency contributes to coronary risk, replacement of vitamin D reduces risk. The vitamin D from dairy products, foods, and sun exposure fails to provide sufficient quantities for the majority of Americans.
When supplementing vitamin D:

1) Vitamin D should be **vitamin D3 or cholecalciferol**, **not** D2 or ergocalciferol.

2) Vitamin D3 supplements should be *oil-based* capsules, gelcaps, or drops, **not** tablets. Tablets are poorly or erratically absorbed.

3) It is important to periodically monitor blood levels of 25-OH vitamin D levels, e.g., every 6 months, especially in the first several years of supplementation, as needs can change over time. We aim to achieve a level of 60-70 ng/ml, a level that requires 6000 units per day of vitamin D3 in the majority of adults with dose adjusted over time according to levels of 25-OH vitamin D.
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Chapter 11

Is your thyroid to blame?

A subtle or mild degree of hypothyroidism is a surprisingly common contributor to coronary plaque. Knowing when thyroid status enters the picture can be a crucial part of your long-term plaque control program.

When it comes to seizing control over coronary plaque, conventional notions of thyroid health may need to be thrown out the window.

Marginal thyroid dysfunction is common and can be an important contributor to distortions of LDL cholesterol, Lp(a), and body weight. Correction of even subtle degrees of thyroid dysfunction is important to gain full control over coronary plaque growth. It also can make you feel happier, more energetic, and thinner.

The thyroid gland modulates metabolism, fine-tuning the function of virtually every tissue, from lowly cells at the base of the fingers making fingernails, to the neurons in your brain guiding memory and thought, to the cells lining your arteries.

Hypothyroidism, or deficiency of active thyroid hormones, can wreak devastating effects on health. It’s been known for decades that, when severe, signs of hypothyroidism are obvious and advanced degrees of atherosclerosis and heart failure develop. However, more recently, it has come to be recognized that even mild degrees of hypothyroidism can also contribute to heart disease. Mild hypothyroidism is also proving to be far more common than previously suspected. Because it is less dramatic, it can go undetected longer, doing damage slowly over many years, including allowing growth of coronary plaque. Subtle degrees of hypothyroidism can also be trickier to diagnose. Add to this the
debate among the medical community over the boundary between "normal" and abnormally "low" thyroid function, not to mention the widespread tendency to treat only laboratory values while ignoring the patient.

It is *Track Your Plaque*’s mission to help identify every possible advantage for stopping or reversing plaque growth. To that end, normal—no, perfect—thyroid function may be key.

**Thyroid disease: Emerging from the dark ages**

For many years, the condition of hypothyroidism (*hypo*-, underactive) proved a mystery, doctors standing by helplessly while the patient became weak, bloated and swollen, lost hair, with a fatal end. While the grotesque degenerative changes that struck sufferers fascinated physicians through the ages, insight into its causes didn’t emerge until the late 19th century.

An 1878 description of a woman in life, followed by the autopsy descriptions after her death, paint a picture of the profound nature of the disease:

“. . . there was a marked slowness of perception, and a marked slowness of response of muscles to voluntary or reflex nerve-impulse. She stated that she could not act or think quickly, that her thoughts would only come slowly; there where any operation, such as dressing, took her half an hour formerly, it now took her two hours . . . She felt always tired, so that her life was utterly wretched.”

After her death, the woman’s autopsy showed that “the arteries were everywhere thickened, the larger ones atheromatous . . . Overgrowths appear to have led to the obliteration of arteries . . .”

The woman was only 54 years old.

The author, Dr. William Ord, was so struck with the edema that was evident in every organ that he dubbed the condition “myxedema.” Despite the detailed observations of the woman in both life and death, Dr. Ord ascribed her condition to “. . . the evil result of a restricted indoor life, [without] the benefits of out-door exercise, of bracing air, of the exposure in journeyings by sea.” In other words, she didn’t get enough fresh air.

Subsequent research pinpointed the cause of the mysterious myxedema as deficient thyroid function. Physiologist Moritz Schiff later demonstrated that thyroid extract taken from the thyroid glands of animals and administered orally to humans could successfully correct myxedema.

While we might chuckle today at the misguided notions of the 19th century, controversy over diagnosis and management of thyroid disorders persists even today. The unfortunate woman in Dr. Ord’s description would be readily
diagnosed today, but a hotly contested question remains in discerning the boundary between normal and low.

For us, the debate is not just academic: New data suggest that not only are such factors as physical stamina impacted, but there are implications for lipids, lipoproteins, and coronary plaque.

The thyroid gland: A primer

To help navigate thyroid issues relevant to control over plaque, let's review some basic thyroid physiology.

The thyroid is a three inch-wide, butterfly-shaped gland located in the front of the neck. Though bridging across the trachea (airway) and located just beneath the surface of the skin, you should not be able to feel a normal thyroid. Enlarged thyroids can be felt, however.

The thyroid gland produces hormones that regulate body metabolism:

**T4** (thyroxine)

T4 accounts for roughly 80% of the hormones produced by the thyroid gland. T4 is a “prohormone,” or precursor hormone, converted to the physically active hormone, T3, via the action of deiodinase enzymes in body tissues that remove one iodine atom. T4, with an elimination half-life of about 7 days (i.e., 7 days to drop to half its level), acts as a “reservoir” for supplying the much shorter-lived T3 to the body.

**T3** (triiodothyronine)

T3 is the “real” thyroid hormone that controls metabolic rate at the cellular level by mediating the rate of oxygen consumption of virtually every tissue in the body. The thyroid produces only small amounts of T3 directly (roughly 15-20% of bodily requirements), relying mostly on conversion of T4 to T3 in various organs, mostly the liver and kidney. T3 is a particularly powerful hormone, such that the body requires only picogram (trillionths of a gram) quantities per deciliter of blood to function properly.

The conversion of T4 to T3 can go awry: the deiodination process also creates what is known as “reverse” T3 (rT3), structurally similar to T3 except for the location at which T4 loses an iodine atom. rT3 is indistinguishable from T3 on most diagnostic tests. rT3 also binds to the same tissue receptor sites as T3 but is inactive and therefore blocks the action of “real” T3. It is thought that the body regulates T3 and rT3 production as a means of increasing and decreasing metabolism in response to conditions such as sickness, stress, and scarcity of food. (A controversial condition known as Wilson’s Syndrome, in which a disproportionate amount of rT3 is produced due to overproduction of cortisol by
the adrenal glands during excessive stress, is currently being debated as the cause of certain forms of hypothyroidism.)

**T2** (diodothyronine), **T1** (moniodothyronine)

The thyroid produces only trace amounts of T2 and T1 with additional production occurring from the further activity of deiodinase enzymes on T3. Little is known about the effects or purpose of T2 and T1. Recent research suggests they may be necessary for conditions in which rapid energy utilization is required, such as cold exposure or overfeeding and may also be involved in reducing weight via regulating effects on fat storage.

The thyroid gland is under the control of the pituitary gland (deep within the brain). The pituitary produces thyroid-stimulating hormone, TSH, to stimulate thyroid production of thyroid hormones, T4 and T3. If tissue levels of T4 and T3 are low, the pituitary responds by increasing TSH. The TSH level is therefore the mostly commonly used clinical blood test used to diagnose hypothyroidism.

After release into the blood, T3 and T4 are transported through the body by attaching to the blood protein, thyroxine-binding globulin (TBG). T3 and T4 are only active when disassociated from TBG and in their “free” form. Reflecting their impressive potency, less than 1% of all T3 and T4 are present in their free state, but it is the “free” form, the biologically active form, that we are most interested in.

**Hypothyroidism basics**

Disorders of the thyroid can be broken down into two general categories: hypothyroidism (underactive thyroid) and hyperthyroidism (overactive thyroid). By a wide margin, hypothyroidism is the more insidious that often evades diagnosis, unrecognized for years while contributing to growth of coronary plaque. Hyperthyroidism, while important, is far less common and carries less implication for our plaque-control efforts and will therefore not be the focus of our discussion.

Hypothyroidism is a condition in which the thyroid produces inadequate amounts of thyroid hormones, T4 and T3.

Overt hypothyroidism nowadays tends to be recognized before it reaches the extreme stage of Dr. Ord’s day, manifesting itself by high TSH levels and usually accompanied by one or more symptoms including:

- Substantial drop in energy; fatigue; need for more sleep
- Feeling cold when other people feel warm; reduced or absent sweating
- Dry, itchy skin
- Dry, coarse, brittle hair; hair loss or thinning
- Loss of appetite with weight gain (5-20 pounds) and difficulty losing weight
- Short-term memory impairment, slowed thinking
• Feelings of pins and needles in the hands and feet (paresthesias)
• New constipation
• Puffiness around eyes, hands, ankles, and feet due to fluid retention
• Carpal tunnel syndrome
• Heavier and/or more frequent menstrual periods, worse cramps, worse premenstrual symptoms
• Depression, sadness or indifference
• Goiter (swelling in the front of the neck, caused by enlargement of the thyroid)
• Abnormally slow heart rate
• Higher diastolic blood pressure (bottom value)
• Iron deficiency anemia, low ferritin (an iron storage protein)

Diagnosing hypothyroidism can be difficult if based purely on symptoms, as symptoms are not specific to hypothyroidism. Symptoms may also be vague. Levels of thyroid hormones, free T3 and T4, along with TSH, are therefore used to confirm the diagnosis.

Diagnosis can be especially troublesome in lesser degrees of underactive thyroid function. The frequency of hypothyroidism increases with age and, though estimates vary due to differing cut-points for TSH, most estimates cite a range of 2-4% early in life to as high as 15-20% later in life, with greater prevalence in females (Andersen 2002).

TSH remains the primary method of confirming that symptoms may be attributable to hypothyroidism. Unfortunately, there is extensive disagreement about what constitutes a “normal” TSH level.

The standard (though disputed) TSH ranges (in mIU/L) from the American Thyroid Association are listed below.

<table>
<thead>
<tr>
<th>Range</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0 – 0.4</td>
<td>Hyperthyroidism</td>
</tr>
<tr>
<td>0.4 – 2.5</td>
<td>Normal Range</td>
</tr>
<tr>
<td>2.5 – 4.0</td>
<td>At Risk: (repeat TSH test at least once a year)</td>
</tr>
<tr>
<td>4.0 – 10.0</td>
<td>Sub-clinical (mild) hypothyroidism</td>
</tr>
<tr>
<td>Above 10.0</td>
<td>Hypothyroidism</td>
</tr>
</tbody>
</table>

Other commonly used laboratory tests to gauge thyroid function include:

**Free T4 (fT4)** and **free T3 (fT3)** are the preferred tests that measure T4 and T3 unbound to protein and therefore active, excluding the 99% of hormone bound to proteins. Reference ranges for free T4 and T3 are supplied by the test lab. As with TSH, what is “normal” for the standard population may not be normal for every individual and there can be considerable differences in the T4 or T3 levels ideal for each person.
There are a number of other thyroid tests, such as T4 uptake and T3 uptake, that have been replaced by direct measurement of free T4 and free T3 and should therefore \textit{not} be used.

A common inflammatory condition of the thyroid, called Hashimoto’s thyroiditis, can be responsible for impairing thyroid function over time. Thyroid inflammation can be responsible for both hypo- and hyperthyroidism and can be identified by antibodies against thyroid components:

\textbf{Thyroid Peroxidase Antibody (TPO Ab)}

Thyroid peroxidase (TPO) is a thyroid enzyme responsible for manufacturing thyroid hormones. Approximately 90\% of sufferers of the inflammatory thyroid disorder, Hashimoto’s thyroiditis, will test positive for elevated TPO antibodies (Carlé 2006).

\textbf{Thyroglobulin Antibody (TG Ab)}

Another common autoimmune condition results in the body attacking thyroglobulin. Approximately 60\% of Hashimoto’s thyroiditis sufferers will test positive for elevated thyroglobulin antibodies. Testing positive for \textit{both} TPO and TG antibodies increases the likelihood of Hashimoto’s thyroiditis to 95\% (Carlé 2006).
Iodine: Essential trace mineral for thyroid health

Iodine is essential for health. It is an important trace mineral to maintain or regain control over thyroid health and thereby coronary plaque.

Just as deficiency in vitamin C will lead to teeth falling out, open sores over the body, and eventually death, so will iodine deficiency lead to debilitating disease. Simply meeting the Recommended Daily Allowance (RDA) of 150 mcg per day will keep goiter (enlarged thyroid from lack of iodine) from developing and maintain thyroid hormone production at a “normal” level for the majority of people. Because Americans increasingly avoid use of iodized salt, more people are developing iodine deficiency.

A century ago, Americans were plagued with goiters, with as many as a third of the inhabitants of some parts of the country (e.g., the Great Lakes region, AKA the “goiter belt,” and much of the Midwest) affected. The connection between goiter and deficiency of iodine wasn’t made until a family doctor in Cleveland, Ohio, conducted an experiment on schoolgirls in 1916. Dr. David Marine administered what we now recognize as a very large dose of iodine: 170-340 mg (170,000 – 340,000 micrograms, mcg) sodium iodide per day to 900 girls. Virtually none of the girls receiving iodine supplementation developed goiters, compared to 22% of the control group not receiving iodine (Zimmerman 2008).

This led to the introduction of iodized salt in 1924 with FDA advice to use more salt. Fast forward to the 21st century and many health-conscious people proudly declare their assiduous avoidance of salt, especially iodized table salt. Others have turned to alternative preparations of sodium chloride, such as sea salt (very little iodine content), Kosher salt (no iodine), and potassium chloride-based salt substitutes (no iodine). As a result, iodine deficiency and goiters are making a comeback.

If iodine is unavailable to the thyroid gland, production of thyroid hormones, T3 and T4 (the “3” and “4” referring to the number of iodine atoms per molecule of T3 and T4), begins to suffer, production drops, and hypothyroidism ensues. Iodine deficiency over time leads to a thyroid gland that enlarges, or “hypertrophies,” an attempt to overcompensate by growing larger and better able to extract the little iodine available from the body. It is not necessary to have a goiter for thyroid dysfunction to develop.

Athletes and persons engaged in heavy physical effort lose considerably more iodine than other people in their perspiration, increasing need for iodine (Smyth 2005; Mao 2001). Vegetarians also have substantially greater likelihood of iodine deficiency than carnivorous people (Remer 1999).

What is not clear is just how much iodine we all need for optimal health. Does alleviating goiter also mean that thyroid function is optimized? Goiter may simply represent the grossly visible manifestation of hypothyroidism. Is there an intake of iodine that can further improve thyroid function, even after goiter is reversed or suppressed?

To further complicate the situation, what is the quantity of iodine required in the presence of now ubiquitous environmental blockers of thyroid function, such as bromine (pesticides like polychlorinated biphenyls (PCBs), flame retardants like polybrominated diphenyl ethers, brominated pool water); bisphenol A (from polycarbonate plastics, plastics with recycling code 7, microwaveable plastic, and the resin lining of canned foods); perchlorates (fertilizers, explosives in fireworks)? All of these substances have been shown to block thyroid action; having sufficient iodine helps prevent these chemicals from entering thyroid tissue (Blount 2006; Schmutzler 2007). How should this factor into our decisions on dose of iodine?

Simply adhering to the RDA of 150 mcg per day for adults or thereabouts is likely just enough for most people. Note that many multivitamins or multi-minerals contain the RDA for iodine. Obtaining iodine through the use of iodized salt is both unreliable and unhealthy, since salt increases blood pressure in the susceptible, causes fluid retention, and can accelerate osteoporosis. Iodine in salt is also inconsistent, poorly absorbed, and volatile (evaporating from the container within weeks of opening).
Hypothyroidism: Lipids, lipoproteins, and other factors

Hypothyroidism of all degrees has been linked to distortions in lipid and lipoprotein patterns. Recognizing this connection can provide a unique and effective means of gaining better control over these measures.

**LDL Cholesterol**

Hypothyroidism increases LDL cholesterol and all related measures. The higher the TSH, the higher the LDL cholesterol, as well as apoprotein B and LDL particle number (NMR).

Although the blunted metabolic state of an underactive thyroid results in reduced LDL cholesterol production, cellular uptake of LDL is also reduced to a greater degree, resulting in net accumulation of LDL. Correction of hypothyroidism therefore reduces LDL. This relationship has been confirmed in a

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If there is any indication of hypothyroidism, then strong consideration should be given to an increase in iodine intake to the 500-1000 mcg (microgram, not milligram) per day range, preferably from an iodine supplement such as kelp tablets, a form that approximates the natural, ocean-derived source. Iodine is also available from health food stores as potassium iodide drops, capsules, and tablets. Also, note that there may be a mild increase in TSH for several months after iodine is initiated, only to drift back down over time (Clarke 2003).

Thyroid testing can suggest iodine deficiency by the pattern of a low free T4, e.g., at or below the "reference range," along with a slightly-higher–than-optimal TSH of 2.5 mIU/L or greater. This is usually corrected after 3-6 months of iodine replacement if iodine deficiency is the cause, especially if any thyroid enlargement is present.

Keep in mind that, if hypothyroidism or goiter is present, iodine intake may need to be individualized by your healthcare provider. Rarely, someone with hypothyroidism or goiter will develop abnormal thyroid responses to iodine. This occurs because of iodine deficiency present before correction distorts thyroid function; adding iodine can actually worsen the situation temporarily. Iodine replacement may therefore be best undertaken alongside monitoring of thyroid function by you and your health care provider. Alternatively, some people have success by increasing the dose of iodine gradually, e.g., starting at the RDA of 150 mcg per day, building up by 50-100 mcg increments gradually over 6 months until the desired dose, e.g., 500 mcg per day, is achieved.

Unfortunately, dose-escalating studies for iodine that track thyroid function have not been thoroughly explored. If the RDA for iodine of 150 mcg yields some improvement of thyroid measures, would 300 mcg, 600, 900, even 10,000 mcg further improve thyroid function or other aspects of health? Sadly, sufficient study has not yet been done to answer these questions.

Should we take a lesson from the Japanese, who, through their dietary reliance on seaweed (e.g., kelp, kombu, nori, etc.) and abundant seafood, have iodine intakes 30- to 100-fold higher (5,280-13,800 mcg per day) without excessive thyroid disease and reduced incidence of fibrocystic breast disease and breast cancer (Patrick 2008)? This is also unclear, though it suggests that much higher intakes of iodine are, for the majority, safe. It may even be preferable.
number of studies in thousands of subjects (Meier 2001; Iqbal 2006; Canaris 2000). The magnitude of reduction of LDL cholesterol, apoprotein B, and LDL particle number is inversely proportional to TSH, with a larger reduction in those with the highest TSH levels (more severe hypothyroidism). In persons with mild hypothyroidism, LDL and apoprotein B reductions of 20 mg/dl are typical (Monzani 2004).

The HUNT Study likely closes the door for good on this argument. This large Norwegian study examined the thyroid hormone levels of 35,000 people. Thyroid hormone levels were then correlated with various parameters. As in previous studies, a clear relationship between higher TSH levels (i.e., hypothyroidism) and higher LDL levels was observed. However, the HUNT Study was unique not only for the extraordinary size of the group studied, but because the investigators extended the analysis to TSH levels ordinarily considered to be within the normal range: TSH levels as low as 1.0 mIU marked the start of a gradual increase in LDL (Asvold 2007).

What about small vs. large LDL? Here the data are not so clear, with conflicting observations. However, the best information emerges from the one study that used NMR lipoprotein analysis, which suggested that the large, less atherogenic (plaque-causing) fraction doubled with lower thyroid function (higher TSH). This suggests that, while LDL cholesterol increases with diminishing thyroid and rising TSH, it is the less harmful large fraction (Pearce 2008). However, in the Track Your Plaque experience, the effect is more likely to be variable, often depending on weight effects, diet, and other factors.

Higher TSH levels are also associated with greater levels of oxidized LDL. Oxidized LDL is more likely to contribute to coronary plaque by triggering LDL particle ingestion by macrophage inflammatory cells. Correction of hypothyroidism reversed the excessively oxidized state (Duntas 2002).

HDL Cholesterol
In the HUNT Study, as TSH increased above 1.0 mIU/L, LDL increased, but HDL decreased. However, the effect is quite small, amounting to a difference of no more than 2 mg/dl. This is similar to the findings of other studies.

Triglycerides
As you might expect, along with increases in LDL, hypothyroidism is accompanied by increased triglycerides; the higher the TSH, the higher the triglycerides. The degree of increase parallels the increase in LDL, though is slightly less (usually 10-20 mg/dl in mild hypothyroidism) (Asvold 2007). Correction of hypothyroidism reduces triglycerides.

Homocysteine
While increasing levels of homocysteine are unquestionably associated with increasing levels of cardiovascular risk, several randomized clinical trials
have failed to show reduction of risk with reduction of homocysteine using a combination of B vitamins (folic acid, B6, B12). Nonetheless, the observation that hypothyroidism is associated with higher homocysteine levels and that correction of hypothyroidism leads to reduction of homocysteine (-44%) raises some interesting questions about the thyroid’s role underlying homocysteine as a predictor of cardiovascular events (Hussein 1999).

Could homocysteine serve, at least to some degree, as a surrogate measure of low thyroid function? While some authorities are ready to dismiss homocysteine as a dead end, significant questions remain that may, in time, yield important insights into just what role homocysteine should play in our coronary plaque control efforts.

Until then, a high homocysteine (>10 μmol/L) should at least cause you to consider an underactive thyroid gland as a contributor.

**Lipoprotein(a)**

Though the effect is variable (as with all other aspects of Lp(a)), many people with Lp(a) show substantial reductions in Lp(a) with thyroid correction. Thyroid correction is one of the most important strategies we have for reduction of Lp(a).

Clinical studies suffer from variation in the method of Lp(a) measurement and the substantial genetic variation in Lp(a) size (Kung 1995; Becerra 1999; Tzotzas 2000; Yildirimkaya 1996). Another factor is time: Lp(a), for unclear reasons, requires at least 3-4 months or longer to respond to any intervention; reassessment before this time period yields misleading results (Kung 1995).

Correction of T3, in particular, may be especially important in people with Lp(a). A small Dutch study in patients after thyroid removal demonstrated 50 mg/dl reduction of Lp(a) with T3 supplementation (Dullart 1995). Whether adding T3 to T4 treatment adds to Lp(a)-reducing effect has not been systematically examined, but has proven helpful in many cases in the Track Your Plaque experience. This means using both T4 and T3 thyroid preparations or using combination preparations, such as Armour® thyroid, to correct even subtle degrees of thyroid dysfunction to gain control over Lp(a).

**Endothelial dysfunction**

Thyroid hormones, especially T3, enhance the action of nitric oxide in the arterial wall, which causes a reduction in arterial tone and blood pressure. This effect is similar to that of L-arginine. People with mild hypothyroidism have impaired artery dilating responses, corresponding to a rise in diastolic blood pressure, an effect which reverses when hypothyroidism is treated (Taddei 2003).
Heart rate and parasympathetic tone

Beat-to-beat variation in the interval from one heartbeat to the next is a desirable feature, a condition of heightened parasympathetic tone (opposite of sympathetic “fight-or-flight”). Increased variability is seen in healthy people, high levels of fitness, and people who meditate. People with hypothyroidism display diminished beat-to-beat variability, reflecting impaired parasympathetic tone, a potential marker for poor health and adverse outcomes (Kahaly 2000).

Do-it-yourself-at-home thyroid test: Basal body temperature

The regulation of body temperature—thermoregulation—is a reflection of the body’s capacity to adapt to the external environment and maintain body temperature within a narrow range. Deviations from the narrow range of body temperature can suggest disruption of internal control. If it weren’t for our ability to regulate body temperature, we’d have to lie in the sun like snakes and crocodiles to control body temperature.

Should thermoregulation go haywire due to external influences that overwhelm it, trouble results. Anyone who has experienced a fever of 104º F or hypothermia from cold exposure knows that just a few degrees in either direction is distinctly unpleasant, even life-threatening.

What is normal body temperature? Recent analyses, as well as a comprehensive review of temperature data from studies from 1935 to the present, suggest that normal oral temperature ranges from 96.3º F/35.7º C to 99.9º/37.7º C (Sund-Levander 2002; McGann 1993). This differs from the 98.6º F/37.0º C often quoted as normal, a relic of 19th century observations on human temperatures.

Body temperature also undergoes a predictable circadian rhythm, with highest temperature of the daily 24-hour cycle at around 8 p.m., lowest at around 4 a.m. It is the temperature low that is most reflective of thyroid status.

There is no question that the thyroid gland and thyroid hormones, T3 and T4, play a crucial role in temperature regulation. A principal sign of hypothyroidism (high TSH, low thyroid hormone levels) is low body temperature, while hyperthyroidism (low TSH, high thyroid hormone levels) is associated with increased body temperatures.

When can low temperature be attributed to hypothyroidism and not other causes? There are frustratingly few data that explore this relationship. While there is no dispute that low temperatures can accompany hypothyroidism, it is not clear how reliable an indicator it is, since the relationship has not been studied in a formal manner. In anecdotal experience in many clinical practices, however, low body temperature is commonly used to identify hypothyroidism.

We have used first-upon-arising oral temperatures in the Track Your Plaque program and they can indeed be useful. At a practical level, any first a.m. temperature (immediately upon arising) consistently less than 97.3º F or 36.0º C is suggestive of hypothyroidism; the lower the temperature, the more likely it represents hypothyroidism. A temperature of 94.7º F, for instance, is more strongly suggestive of hypothyroidism than a temperature of 97.1º F.
Followers of the experience of Dr. Broda Barnes adhere to his original belief that axillary (armpit) temperatures are the preferred method to assess body temperature. However, of the various ways to measure body temperature, axillary is the least reliable and the one most prone to inaccuracy. More so than other methods, axillary temperatures are subject to external ambient temperature, amount of clothing worn, sweating, whether right or left arm is used (since there is variation of up to 2.0º F degrees from right to left), the amount of cutaneous (skin) dilation or constriction of blood vessels. Axillary temperatures track internal (rectal) temperature poorly, with wide variation in the day-to-day and minute-to-minute fluctuations of temperature, and especially marked divergence from rectal temperature in morning (temperature nadir) and evening (temperature peak) hours, with as much as 1.8-2.7º F/1.0-1.5º C variation within several minutes (Cattaneo 2000; Kelly 2006). Axillary temperatures are therefore too variable and unreliable for use in assessing thermoregulation and thyroid status and should not be used to assess thyroid status.

Despite its uncertainties, temperature is still a useful tool to identify hypothyroidism, particularly when laboratory values like TSH, free T3, and free T4 are borderline or equivocal, or when symptoms are unusual or atypical. Low temperature may suggest low thyroid status even when all other measures, including TSH, are normal, but this is on more shaky ground scientifically. Temperature can also be useful to follow trends over time to gauge the adequacy of thyroid replacement.

There are several lessons to keep in mind if we are to use temperature to assess the body’s capacity for thermoregulation and thyroid status:

- Normal immediately-upon-arising oral temperature is 97.3º F or 36.0º C.
- Oral temperatures are best used as a gauge of thyroid status alongside symptom assessment and thyroid laboratory evaluation that includes TSH, free T3, and free T4.
- Oral temperatures only should be used (unless internal temperatures are available). Oral temperatures track most truly to internal temperatures. Axillary temperatures do not track with internal temperatures and are subject to unacceptable variation and should not be used.
- Oral temperatures should be assessed immediately upon awakening without drinking water or other liquids or eating any food; alcoholic beverages should be avoided the evening prior. Temperature should not be assessed during calorie restriction, fasting, or a period of substantial sleep deprivation.
- The temperature nadir (bottom) usually occurs between 3 am and 6 am. If oral temperature is taken upon awakening at, say, 8 a.m., it will be higher than the true nadir. Waking temperatures later than 6 a.m. can be adjusted to the “6 a.m. equivalent” by subtracting 0.18º F/0.1º C for every hour after 6 a.m.
- Menstruating females should take oral temperatures during the first 7 days after beginning menstrual bleeding during the follicular phase, the phase that does not show the exaggerated increase in temperature triggered by ovulation or progesterone.

Temperature represents a simple means to gauge metabolic rate and thereby thyroid status. It is another measure like blood pressure or blood sugar, a simple assessment you can perform on your own and track over time.
Hypothyroidism and coronary plaque

Not only have overt and mild hypothyroidism been associated with worsening of factors in the coronary risk profile, but they are also associated with increased risk for heart attack. Risk develops with TSH values even commonly regarded as “normal.”

The Rotterdam Heart Study showed that, among 1149 women participants, those with TSH >4.0 mIU/L showed a 70% greater likelihood of aortic atherosclerosis and more than double the likelihood of heart attack (Hak 2000). (Widely quoted “normal” or “reference ranges” for TSH are usually less than 4.5 or 5.5 mIU/L.)

Individual studies showed widely varying magnitude of increased risk with hypothyroidism (as high as 80% in a 2005 study Walsh 2005). An analysis of 10 combined studies (a “meta-analysis”) examining a total of 14,500 participants showed that mild hypothyroidism was associated with 20% increase in risk for heart attack (Ochs 2008). A small study using coronary angiography showed that 5 of 7 people with “adequate” thyroid replacement (150 mcg levothyroxine) showed no progression of coronary disease, compared to a group “inadequately” treated (<100 mcg levothyroxine) all of whom showed progression (Perk 1997).

The HUNT Study extended these observations by analyzing risk for fatal cardiovascular events over eight years along the entire spectrum of TSH levels, even down into the “normal” reference range <3.5 mIU/L. By breaking their groups down by TSH levels, the group with TSH 1.5-2.4 mIU/L showed a 41% increase in events; the group with TSH 2.5-3.5 mIU/L showed a 69% increase in events, when groups were compared to the group with TSH 0.50-1.4 mIU/L. The relationship was stronger in women than in men (Asvold 2008).

Unfortunately, clinical studies demonstrating the effects of thyroid correction on cardiovascular risk are limited.

In a large anecdotal experience in 1,110 patients treated with thyroid hormones (mostly Armour® thyroid) over an average of 5.6 years, four heart attacks were observed, compared to the “expected” number of 72 heart attacks in a demographically similar group of people not taking thyroid (i.e., not a true control group) (Barnes 1973)

A study dating back to 1971 suggested reduced risk with thyroid replacement. 347 patients with coronary or peripheral arterial disease, nearly all of whom started without hypothyroidism, were given a combination thyroid preparation equivalent to Armour® thyroid, starting at ½ grain (30 mg) and increasing as tolerated to 2-3 grains (120-180 mg) and observed over 5 years. Total cholesterol was reduced by 22% and mortality reduced by 44% when compared to the expected death rate (judged by actuarial tables, not a true control group) (Wren 1971).
A small study of 45 people with mild hypothyroidism (median TSH 6.31 mIU/L) compared to 32 controls (median TSH 1.19 mIU/L) demonstrated that those with hypothyroidism began with higher LDL cholesterol levels (+25 mg/dl), equivalent HDL levels, higher triglycerides (+14 mg/dl), higher Lp(a) (3 mg/dl), and greater carotid intimal-medial thickness (CIMT; +19%), a measure of atherosclerosis in the carotid artery. After 6 months of treatment with T4 (only) to achieve TSH of 1.32 mIU/L, the participants with starting hypothyroidism showed 11% regression of CIMT. In addition, LDL was reduced by 20 mg/dl; Apo B by 9 mg/dl; triglycerides by 6 mg/dl; Lp(a) by 2.3 mg/dl (Monzani 2004).

At the very least, maintaining ideal thyroid hormone status shifts lipids and lipoproteins in the right direction. In many people, the reduction in LDL cholesterol/apoprotein B/LDL particle number is sufficient to achieve Track Your Plaque targets without statin drugs. Because thyroid replacement also typically causes weight loss or makes weight loss occur more readily, weight-sensitive measures, such as small LDL and HDL, can improve substantially, as well.

The Track Your Plaque approach to hypothyroidism

In many people, subtle degrees of hypothyroidism with TSH values within the “normal” range can act as a coronary risk factor and drive plaque growth. Correction of hypothyroidism is also accompanied by many positive side-benefits: people feel more energetic; they are happier; weight loss is accelerated; LDL cholesterol, triglycerides, and Lp(a) values are reduced; and more people enjoy better control over coronary plaque.

The first step in achieving correction of thyroid function is to ensure adequate iodine intake (see box). This is especially important for people who avoid use of iodized salt, exercise or work in hot environments, in people with the low free T4/high TSH pattern, and people with any degree of thyroid enlargement.

The conventional approach to thyroid management, as practiced by most primary care physicians and endocrinologists, is not ideal for coronary health. Given experiences like the HUNT Study, we aim to keep TSH 1.5 mIU/L or less, along with free T3 and free T4 levels in the upper half of the reference range; this has maximized lipid and lipoprotein benefits, generally maximizes a sense of well-being, and has proven practical and effective from a plaque-control viewpoint.

It is also necessary to look beyond thyroid testing measures to gauge the adequacy of thyroid function. TSH serves as an unreliable index of thyroid function in some people and can be falsely low or “normal” even when hypothyroidism is truly present. Recent data appear to support this argument, with one recent study, for instance, showing that, as we age, the increase in TSH
with low thyroid function is 75% less compared to younger people (Carlè 2007). Attention to symptoms is therefore essential for detecting persons suffering from hypothyroidism underestimated by standard laboratory testing.

Synthetic T4 (e.g., Synthroid®) is not sufficient to resolve symptoms of hypothyroidism in a substantial proportion of persons with hypothyroidism, since not everyone converts T4 to active T3 with equal efficiency. Indeed, low-T3 has been documented to be a predictor of death in patients with congestive heart failure (Iervasi 2003). T3 supplementation has positive effects on psychological symptoms of hypothyroidism with several studies showing that well-being, mood, and cognitive functions are improved; weight loss may also be greater with added T3 (Saravanan 2002; Bunevicius 1999; Appelhof 2005). T3 supplementation tailored to symptom relief can also address the issue of reverse T3 by providing an external source of effective T3 (Gaby 2004).

Claims that synthetic T4 drugs are superior to natural thyroid drugs in consistency, potency, and stability are false, largely driven by drug industry marketing efforts. The hormones found in porcine (pig) thyroid are chemically identical to that found in humans. Worries over consumption of the animal proteins in desiccated pig thyroid are also unfounded, as they are regularly consumed whenever eating ham, pork chops, or bacon. While this may pose a problem on religious grounds for some people, it offers no impediments on medical grounds. Claims of impurity are also misrepresentation propagated by pharmaceutical marketing. In fact, both Synthroid® (synthetic T4) and Armour® Thyroid (porcine thyroid) have both had similar struggles with potency and stability in past. Unfortunately, many doctors and patients have only heard about problems with Armour® Thyroid. The Department of Health and Human Services has twice issued cease and desist orders to the manufacturer of Synthroid® for misleading advertising. Prior to the development of synthetic T4 in the 1950’s, natural thyroid hormone products were the only treatment for hypothyroidism since their introduction in the late 1800’s. In that time, they have developed an extensive track record for safety and efficacy, and products such as Armour® Thyroid are now produced under the same USP standards as synthetic T4.

In the Track Your Plaque experience, we favor symptom management over TSH, assessment of T3 as well as T4, and the use of natural thyroid preparations like Armour® Thyroid over levothyroxine or Synthroid®. For anyone who wishes to add T3 but wants to avoid the animal-sourced product, a synthetic T3, liothyronine or Cytomel®, is also available (by prescription).

Should reverse T3 (rT3) be present in high quantities (i.e., in the high "normal" or above "normal" range), then consideration is given to increasing the dose of T3 replacement until symptom relief is obtained. In other words, if fatigue and being cold remain prominent symptoms and rT3 is high, then T3 dose can be increased gradually until symptoms improve.
Mild hypothyroidism and the *Track Your Plaque* program: Summary and practical tips

- All levels of hypothyroidism, regardless of how mild, have the potential to increase heart disease risk. Increased risk for cardiovascular events may extend to TSH levels as low as 1.4 mIU/L.
- Synthetic T4 (i.e., levothyroxine, Synthroid®) *only* to treat hypothyroidism does not represent optimal treatment in everyone. Natural hormone therapy (e.g., Armour® Thyroid) that provides T3 replacement or adding T3 (liothyronine) may be more efficacious for many, perhaps most, people with hypothyroidism. Lower levels of T3 and/or less than optimal symptom relief on T4 alone may suggest potential for benefit by adding T3 (liothyronine) or converting to a preparation like Armour® Thyroid.
- Correction of mild hypothyroidism can reduce LDL, oxidized LDL, triglycerides, Lp(a), and homocysteine, and helps normalize endothelial function. Our experience suggests that it also facilitates atherosclerotic plaque regression.
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Chapter 12

Omega-3 fatty acids

Omega-3 fatty acids from fish oil provide a foundation for your plaque-control program through effects on reducing triglycerides, accelerating clearance of postprandial, or after-eating, byproducts, and anti-inflammatory and plaque-stabilizing effects.

Here’s how to gain full advantage from omega-3 fatty acids.

What genuine breakthrough discoveries or revelations have there been over the past 50 years that we can honestly say have altered the course of cardiovascular illness? Not just refinements or minor improvements, but big, ground-shaking discoveries that result in substantially reduced death, heart attack, and stroke?

There are heart procedures like bypass surgery, valve surgery, angioplasty and stents, of course. But what treatments outside of major procedures have resulted in dramatic and life-saving treatments? The prescription statin drugs, whether we like it or not, have become a prominent fixture of the landscape and have, indeed, cut the incidence of heart attack and stroke (though at a price). Aspirin, niacin (vitamin B3) and anti-hypertensive drugs have also had substantial impacts. Omega-3 fatty acids figure prominently on this list.

The case for omega-3s has become inarguably powerful. Safe, effective, and inexpensive, omega-3 fatty acids exert mortality-reducing and health-promoting effects that are, in many cases, superior to prescription agents. Yet they remain woefully underutilized. Ironically, the recent appearance of a prescription form of omega-3 fatty acids (Lovaza®) has “legitimized” the use of
omega-3 fatty acids among physicians. But you can obtain all the benefits of these healthy fats without a prescription.

Don’t underestimate the power of fish oil: A case study

Stacy, a 40-year old physical therapist, was terrified when she saw her most recent cholesterol panel: Total cholesterol 594 mg/dl and triglycerides 2893 mg/dl. Because these values were so high, the LDL and HDL cholesterol values were unobtainable.

I met Stacy in a panic. In tears, she declared “I don’t understand it. I take good care of myself. I don’t eat fatty foods, I exercise, I don’t do anything wrong!”

She was right: Her frightening distortions were not from anything “bad” she’d done. It was a combination of genetics and modest dietary excesses. But these levels of triglycerides and cholesterol pose risk for liver disease and pancreatic damage (pancreatitis), as well as heightened long-term risk for heart disease and stroke. Stacy also showed some features of insulin resistance or metabolic syndrome: blood sugar elevated at 114 mg/dl; blood pressure 140/88; and excess abdominal fat, weighing 160 lbs at 5’5.”

I advised Stacy to take 3000 mg of omega-3 fatty acids, EPA and DHA, from fish oil every day. She chose a low-cost, low-potency fish oil that required 9 capsules per day. She accomplished this by taking three capsules, three times per day with meals. (Stacy could have chosen a more-concentrated fish oil, allowing her to take fewer capsules to achieve the same dose of omega-3s.)

I also counseled Stacy on reducing her intake of processed carbohydrates like crackers, pretzels, breakfast cereals, and other wheat-containing products; avoiding the food additive high-fructose corn syrup, since it causes triglycerides to skyrocket; and to reduce weight by at least 20 lbs. Stacy accomplished all this. Her most recent panel showed a total cholesterol of 165 mg/dl, triglycerides of 144 mg/dl, HDL 70 mg/dl, and LDL 66 mg/dl.

Though an extreme case, Stacy’s experience showcases just what a powerful tool omega-3 fatty acids can be.

The cardiovascular power of omega-3 fatty acids

Omega-3 fatty acids from fish oil have the advantage of a substantial scientific basis. Thousands of studies have now documented the broad range of beneficial effects provided by EPA, eicosapentaenoic acid, and DHA, docosahexaenoic acid.

Among the benefits that omega-3s provide:

Omega-3s stabilize heart rhythms

At first, the results were puzzling. Clinical trials confirmed that eating fish reduced likelihood of heart attack, but reduced sudden cardiac death even more.
Thus it was suspected that omega-3 fatty acids of coldwater fish stabilize cell membranes of heart muscle cells and turn-off abnormal heart rhythm activity.

The Diet and Reinfarction Trial (DART) was the pioneering treatment trial revealing this paradox. 2033 men with prior heart attack either ate fish twice per week or did not (fish consumption vs. “placebo”), resulting in a 29% drop in cardiac death among those advised to eat fish. There was no decrease in the number of heart attacks, but only death from heart attack. The investigators reasoned that fish oil suppressed abnormal rhythms generated by damaged heart muscle (Burr 1989). This was further supported by the observational Physicians’ Health Study showing that participants dying from sudden cardiac death had lower blood levels of omega-3 fatty acids than those who did not. Protective levels were obtained by eating two or more servings of fish per week, yielding a 52% reduction in the risk of sudden cardiac death (Albert 1995).

In 1999, the 11,000-participant GISSI-Prevenzione trial brought remaining naysayers to their knees (GISSI-Prevenzione 1999). Participants taking 1000 mg of the omega-fatty acids, EPA and DHA, experienced 30% reduction in cardiovascular death and an astounding 45% reduction in sudden death compared to placebo; protective benefits began as early as three months after initiation of omega-3 supplementation.

The rhythm-suppressing properties of fish oil are so effective that some cardiologists now recommend that patients with implanted defibrillators (for life-threatening heart rhythms) take omega-3s to reduce rhythm instability and cut back on defibrillator firings (which are painful and frightening) (Leaf 2005; Christensen 2005).

People with the common though troublesome rhythm, atrial fibrillation, have lower levels of omega-3s in their blood. Fish oil has impressively suppressed atrial fibrillation in experimental non-human preparations, as well as in human patients, with one study demonstrating a 54% reduction in atrial fibrillation that is common after bypass surgery (Calo 2005).

**Omega-3s shut down inflammation**

Hidden, imperceptible inflammation is a fundamental process that triggers the chain of events leading to heart attack, stroke, cancer and diabetes.

Omega-3 fatty acids suppress multiple steps in inflammatory pathways, including inflammatory cytokines IL-1, IL-2, tumor necrosis factor, COX-2 and others. Inflammatory joint diseases like rheumatoid arthritis serve as a therapeutic model of omega-3 anti-inflammatory benefits. Omega-3 fatty acids reduce blood markers of inflammation and ease the severity of arthritis (Watkins 2001; Ciubotaru 2003; Adam 2003). In contrast, the non-steroidal anti-inflammatory drugs (NSAIDs) and COX-2 inhibitors (e.g., Celebrex®), commonly prescribed for arthritis, increase production of inflammatory cytokines and
increase risk of cardiovascular events (Solomon 2005; Hippisley-Cox 2005). Omega-3 fatty acids, unlike arthritis drugs, do not cause ulcers, nor do they impair kidney function. Interestingly, long-term supplementation of omega-3 fatty acids may allow up to 50% reduction in NSAID usage in chronic arthritis sufferers (Cleland 2006).

The misguided advice of the 1980s and ‘90s that polyunsaturated oils were good for us has resulted in an American diet appallingly overloaded with omega-6 fatty acids. Omega-6 fatty acids increase inflammation by activating production of arachadonic acid and related inflammatory prostaglandins. Omega-3 fatty acids compete with omega-6s, thereby slashing production of inflammatory prostaglandins and increasing anti-inflammatory prostanoids and leukotrienes (Leaf 2002).

Omega-3 fatty acids may provide special benefits to people with the metabolic syndrome, the collection of low HDL cholesterol, increased triglycerides, high blood pressure, resistance to insulin, and high C-reactive protein (CRP), that afflicts 47 million U.S. adults. Excess weight is an important trigger for hidden inflammation. Fat cells in the body produce a signaling molecule called interleukin-6, which provokes CRP release from the liver. CRP is a popular method for measuring hidden inflammation, with levels of 3.0 mg/l or greater yielding a tripling of heart attack risk. Overweight and obese people have higher levels of CRP and suffer far diabetes, cancer, and heart disease driven by inflammation. Omega-3 supplementation provides outsized anti-inflammatory and cardiovascular benefits in people with this condition (Bassuk 2004; Menuet 2005).

**Omega-3s prevent blood clots**
Most heart attacks and many strokes result from the sudden appearance of blood clots that form on the surface of atherosclerotic plaque. That’s why treatments like aspirin that inhibit blood clotting reduce the likelihood of these events. It’s also behind the push for new blood-thinning agents that are pouring into the marketplace, like Plavix® and others. Omega-3s have a similar effect.

Omega-3s reduce blood clotting proteins, fibrinogen and factor V, and inhibit platelet aggregation, all of which reduce the likelihood of clot formation on active, ruptured coronary plaque that would otherwise result in heart attack (Vanschoonbeek 2004). In all practicality, the blood thinning effect is modest and almost never sufficient to result in excess bleeding or bruising.

**Omega-3s correct triglycerides and lipoprotein disorders**
While the world obsesses over cholesterol, an important cause of atherosclerosis is neglected: triglycerides. Triglycerides are a potent driving factor behind heart disease and stroke.

Few treatments provide the dramatic triglyceride-reducing power of omega-3 fatty acids, yet they are woefully underutilized by physicians. Omega-3s
block triglyceride production that, in turn, reduces formation of abnormal lipoproteins created from triglycerides, especially “small LDL” (Balk 2006). Increased triglycerides and small LDL have ballooned in importance as a cause for heart disease as more and more Americans develop the metabolic syndrome, or pre-diabetes. Omega-3s provide substantial correction of the lipoprotein abnormalities triggered by the metabolic syndrome (Menuet 2005).

An exciting area of emerging research is that of “postprandial hyperlipidemias,” the after-eating flood of lipoproteins in the blood that follows every meal. In several studies, this has proven to be a potent cause of atherosclerosis. Omega-3 fatty acids accelerate clearance of postprandial particles like chylomicrons, chylomicron remnants, and VLDL that persist in the blood after eating, reducing blood levels by 50% (Karpe 1994; Westphal 2000).

**Omega-3s stabilize atherosclerotic plaque**

Not only do omega-3s reduce the factors that cause atherosclerotic plaque, but they also directly modify plaque structure and activity.

Omega-3s slow atherosclerotic plaque growth by blocking cellular growth factors and adhesion molecules, inhibition of smooth muscle cell growth and migration of inflammatory monocytes (Connor 1997; Abeywardena 2001). All of this contributes to a slowdown in plaque growth and suppressing abnormal activity that triggers stroke and heart attack.

A cleverly designed British study led to the fascinating observation that omega-3s transform atherosclerotic plaque composition. 150 people with severe carotid plaque scheduled for carotid endarterectomy (surgical removal) were given either fish oil or sunflower oil while waiting for their procedure. (Delays in the British health system permitted this study design.) Plaque was surgically removed several months later and examined. Participants taking fish oil had reduced plaque inflammation and thicker tissue covering the fatty core, markers for stable plaque. Those taking sunflower oil had unstable plaques with greater inflammation and thinner, more fragile overlying tissue. This suggests that omega-3s from fish oil taken for just a few months stabilize carotid plaque, making it less prone to rupture and fragment (Thies 2003).
Omega-3 Index: The higher, the better?

So you take a few fish oil capsules every day and eat fish once or twice a week. What are the blood and tissue levels of omega-3 fatty acids generated by your habits?

A number of variables enter into the equation. For instance, if you take fish oil capsules, what is the concentration of omega-3 fatty acids? How well are the contents absorbed? After absorption, how effectively are omega-3 fatty acids incorporated into cell membranes?

Even if you take fish oil supplements, it is hard to know just how much you’ve increased blood levels. However, it is now possible to measure the amount of omega-3 fatty acids in your bloodstream, a value called the "omega-3 index." Too little and you might still be at risk for cardiovascular events.

Two large studies have demonstrated that higher omega-3 blood (the level in red blood cells, or RBCs) levels were associated with reduced likelihood of sudden cardiac death. The risk for sudden cardiac death was 10-fold higher for the lowest omega-3 RBC levels compared to the highest (Harris 2008; Siscovick 1995; Albert 2002).

Most Americans have omega-3 RBC levels in the 2.5-4.0% range, associated with greatest risk for sudden cardiac death. People with heart disease can have levels as low as 1%.

The omega-3 index has greater power to discriminate who will have a heart attack or die from sudden cardiac death better than many other common laboratory measures of coronary risk, including LDL cholesterol, HDL cholesterol, triglycerides, total cholesterol to HDL ratio, homocysteine, and c-reactive protein.

Just as hemoglobin A1c offers a 3-month look into blood glucose levels, the omega-3 index reflects your long-term omega-3 intake and absorption. The quantity of RBC omega-3s also closely parallels the quantity of omega-3s in heart tissues.

What is an ideal omega-3 index?

Studies relating RBC omega-3 levels and sudden cardiac death suggest that a level of 6.3-7.3% is associated with fewer fatal events—but events are not eliminated at this level. Is there even greater benefit with levels higher than 6.3-7.3%? A recent analysis of females from the Harvard School of Public Health suggests that RBC omega-3 levels as high as 8.99% are still associated with non-fatal heart attack (myocardial infarction), compared to 9.36% in those without heart attacks. This suggests that even higher levels may be necessary to prevent non-fatal events.

Should we target 10%? 12%? Maybe higher? Any higher and we are toeing the level achieved by the Inuits, the “Eskimos” of Greenland, northern Canada and Alaska who have been observed to have low rates of heart disease (before the introduction of Western foods).

Most recently, another study comparing 50 people after heart attack with 50 controls showed that people with heart attack had omega-3 indexes of 9.57% versus 11.81% in controls—even higher. (This study was in a Korean population with higher fish consumption. There was also a powerful contribution to risk from “trans” fat RBC levels.) The investigators concluded: "The area under the receiver operating characteristic curve of fatty acid profiles was larger than that for traditional risk factors, suggesting that fatty acid profiles make a higher contribution to the discrimination of MI cases from controls compared with modified Framingham risk factors."
Omega-3s: How to get what you need for vascular health

Fish oil is the most concentrated source of omega-3 fatty acids, EPA and DHA, obtained from coldwater fish and phytoplankton.

A secondary, much less concentrated, source is alpha-linolenic acid (ALA) from flaxseed and flaxseed oil, walnuts, and canola oil. Only 10% of ingested linolenic acid, however, is converted into active EPA or DHA. Although linolenic acid may provide unique health benefits of its own, fish oil remains by far the most confident source of effective omega-3s. Linolenic acid sources cannot be used in place of fish oil.

Dosing for fish oil is a perennial point of confusion. However, it's quite simple.

The outsized heart benefits of omega-3s start at 1200 mg omega-3 per day, usually provided by taking 4000 mg of fish oil per day (300 mg EPA+DHA per capsule) and go up to 6000 mg of omega-3. Consider a concentrated fish oil preparation with greater content of EPA + DHA per capsule if you and your doctor decide that a high dose is necessary, such as for reduction of lipoprotein(a). If you suffer fishy belching with your fish oil capsules, try refrigerating the capsules. This usually minimizes the effect. Also, consider a different preparation; sometimes, overly-oxidized (rancid) fish oil can cause belching. Lastly, people who are wheat-free tend to tolerate fish oil far more easily than wheat-consuming people, likely because wheat is destructive to the gastrointestinal tract. Fish oil is also best taken with meals. Doses are also best divided into two (e.g., three capsules twice a day).

Since the active ingredients in fish oil are DHA and EPA, anything else in your capsules, such as omega-6, omega-9, or linolenic acid, should not count towards the sum of EPA + DHA, since they do not exert the same benefits as the omega-3s.
The basic suggested starting dose for the Track Your Plaque program is 1800 mg of EPA+DHA per day. This is usually provided by taking 6 x 1000 mg capsules of fish oil, providing 180 mg EPA, 120 mg DHA per capsule (300 mg per capsule), for a total of 1800 mg EPA+DHA. Fewer capsules are required if more concentrated fish oil capsules or liquids are used.

The ideal daily intake of EPA + DHA for our Track Your Plaque purposes, however, is 3000 mg per day. This dose is best managed by using more concentrated fish oil preparations, e.g., 360 mg EPA, 240 mg DHA per capsule, for a total of 600 mg EPA + DHA per capsule. This preparation will allow achieving our ideal omega-3 intake by taking five capsules per day.

If you ever decide to change your fish oil preparation, or if you switch to a more concentrated form or another form such as liquid fish oil (e.g., Carlson’s, Barleans, Pharmax, Nordic Naturals) or fruit-flavored emulsions (e.g., Coromega®, Pharmax), then you will need to examine the label to determine the content of EPA + DHA. If, for instance, a teaspoon of liquid fish oil provides 1050 mg EPA and 750 mg DHA, that’s a total of 1800 mg omega-3s per teaspoon. If your desired EPA + DHA dose is 3000 mg per day, then approximately 1½ teaspoons per day should provide it. Adding up the EPA+DHA content of whatever preparation you choose will therefore allow you to mix, match, or change your dose whenever you like.

How much is enough?

1800 mg EPA+DHA is our basic Track Your Plaque starting dose, generally obtainable by taking 6 capsules of 1000 mg of fish oil, since the majority of preparations contain 180 mg EPA and 120 mg DHA per capsule. Anyone with a positive heart scan score (above zero), established coronary disease (e.g., stents, angioplasty, heart attack, or bypass surgery), or starting levels of triglycerides >300 mg/dl should consider the Track Your Plaque “ideal” dose of 3000 mg EPA + DHA per day, the dose more likely to generate the most protective blood (RBC) levels of omega-3 fatty acids. More concentrated fish oil capsules or liquids are best used to obtain this higher dose.

How will you know if even higher doses wouldn't be even better? The principal measure to look at is triglycerides. If triglycerides remain above 60 mg/dl, then an increase in omega-3 intake from fish can be considered.

If starting triglycerides are, for instance, 500 mg/dl, then even the 3000 mg EPA + DHA dose may be insufficient. Recall that we aim to reduce triglycerides to 60 mg/dl or less. This is important to suppress the formation of abnormal triglyceride-containing lipoprotein particles, especially small LDL and VLDL. For reduction of high triglycerides, EPA + DHA daily intakes of 3600-6000 mg per day may be necessary. However, this is best undertaken under supervision of
your healthcare provider. Obviously, more highly concentrated preparations of fish oil will be necessary.

Incidentally, we never use the prescription form of fish oil, Lovaza®. In my view, there is only one reason to take the prescription form of fish oil: to make the drug manufacturer rich. You can match or exceed the effects of prescription fish oil at far lower cost by taking one of the many excellent preparations of fish oil available over-the-counter. Even in patients with very high triglycerides of >1000 mg/dl, I never use prescription fish oil, yet obtain excellent results.

**Fish oil, mercury and pesticides**

Manufacturers of fish oil make claims that this product or that ("super-concentrated," "pharmaceutical grade," "ultra-purified," etc.) is purer or less contaminated than competitors’ products. The manufacturers of prescription fish oil, Lovaza®, have added to the confusion by suggesting that their product is the purest of all, since it is more concentrated than some of the lower concentration fish oils available over-the-counter (842 mg EPA+DHA per Lovaza® capsule). They claim that "LOVAZA® is naturally derived through a unique, patented process that creates a highly concentrated, highly purified prescription medicine. By prescribing LOVAZA® (omega-3-acid ethyl esters), a prescription omega-3, your doctor is giving you a concentrated and reliable omega-3. Each LOVAZA® capsule contains 90% omega-3 acids (84% EPA/DHA). Nonprescription omega-3 dietary supplements typically contain only 13%-63% EPA/DHA."

How much truth is there in these concerns?

Let's go to the data published by the USDA, FDA, and several independent studies. Let's add the independent analyses provided by Consumer Reports (2003) and Consumer Labs (continually updated; www.consumerlab.com). How much mercury has been found in fish oil supplements?

Virtually none.

Consumer Labs, for instance, has tested dozens of fish oil preparations for mercury content and found none to contain unsafe levels above 10 parts per billion; most contain near zero. This is different from the mercury content of whole fish that you eat that typically contain many times this amount. Predatory fish that are at the top of the food chain, consume other fish and thereby concentrate organic methyl mercury, the toxic form of mercury, do indeed have higher levels of mercury in their fatty tissues. Thus, predatory shark, swordfish, and King mackerel are higher in mercury than sardines, herring, and salmon. Farm-raised fish also contain higher levels of mercury.

The mercury content of fish oil capsules have less to do with the method of processing and more with the animal source of oil. Fish oil is generally
obtained from sardines, salmon, and cod. Fish oil capsules are not prepared from swordfish or shark.

Thus, concerns about mercury from fish oil—regardless of brand—are generally unfounded. Eating whole fish—now that's another story for another time. But we can take fish oil to reduce triglycerides, VLDL, IDL, small LDL, and heart attack risk without worrying about mercury.

Likewise, other contaminants like polychlorinated biphenyls (PCBs), dioxin, and furans have not been found to be present to any substantial level in fish oil. They are certainly found at lower concentrations than that found in eating the fish itself (Melanson 2005). (Cod liver oil, however, is different; some preparations have been found to contain small quantities of PCBs (Storelli 2004). For this and other reasons, including vitamin A content, I do not recommend cod liver oil in the Track Your Plaque program.)

The one issue that has, however, proven to be a tripping point for some brands of over-the-counter fish oil has been oxidative breakdown products, i.e., rancidity. Consumer Labs, for instance, has found excessive quantities of oxidative breakdown products in several products. (An annual subscription to their service is low-cost and well worth the few dollars per year.)

Another easy way to test fish oil for oxidative breakdown products: Make sure your fish oil passes the “sniff test.” Most fish oil will smell slightly fishy. The most purified fish oils, such as the Pharmax preparation of liquid fish oil, has no fish smell when first opened. If a fish oil preparation you purchase smells really fishy, then that batch may be oxidized or rancid, an occasional occurrence when there was lax preparation or if the bottle sat on the shelf too long. Discard or return any preparation with too fishy an odor. Also, all liquid forms of fish oil, because they lack a protective coating, should be refrigerated after opening.

While various manufacturers continue to make extravagant claims of purity, no unbiased testing has yet proven that most of these claims hold any truth. Having gone through the FDA approval process also does not necessarily mean that Lovaza® provides any advantage.

**Fish oil: The choices**

Here’s a list of fish oil products that we have used successfully that have consistently provided high quality at reasonable cost and pass the “sniff test.” They have also not been identified as having any problems by any independent analysis.

Barleans
Carlsons
Coromega
Costco
GNC
Life Extension
Members’ Mark (Sam’s Club)
Nordic Naturals
Nuvite
PharmaNutrients
Pharmax
Pure Encapsulations
Swanson
Trader Joe’s

This list is not meant to be exclusive. There are likely many other high-quality brands available.

**Alternatives to fish oil capsules**

Occasionally, someone will be unable to take fish oil due to the large capsule size, fishy belching, or stomach upset.

Here’s a list of products we’ve used with success. Some cost more than the most common low-potency fish oil capsules, but omega-3 fatty acids from fish oil are so crucial to your plaque control efforts that it really pays to search out alternatives.

**Liquid fish oil**—Nordic Naturals, Barleans, and Pharmax produce highly concentrated fish oil liquids with 1800-2100 mg EPA + DHA per teaspoon. Carlson’s liquid fish oil is widely available but is less concentrated (1300 mg EPA + DHA per teaspoon). Most liquid fish oil comes flavored either lemon or orange. Note that liquid fish oil must be refrigerated after opening.

**Coromega®**—a fruit-flavored emulsion available in some health food stores. Coromega® comes in single-serving foil dispensers.

**Frutol®**—A re-formulated emulsion of fish oil that makes it water-soluble and non-oily. The Pharmax company has put their fish oil into a fruit flavored base that has a pleasant taste and is not too expensive. See the Track Your Plaque Marketplace for more information.

**Krill oil**—Krill oil is extracted from Antarctic krill, a type of plankton rich in omega-3s. Krill oil is not a replacement for fish oil, despite extravagant claims of superiority to conventional fish-sourced fish oil by the manufacturer. Krill oil may have unique properties of its own, e.g., astaxanthin content, a carotenoid unique to shellfish and other sea animals, but cannot be used to replace fish oil.
Regardless of what preparation you choose, you can determine the dose needed by adding up the EPA+DHA content.

**Beware: Fish oil and marketing**

Craig was absolutely convinced that his fish oil was the best available in the world: purer, uncontaminated by mercury or pesticides—"not like that other crap on the shelves!" When asked how he knew this, he proclaimed, "They said so."

Craig fell for the marketing. While there may be some truth in the manufacturer's claims, you can't believe it from the mouth of the manufacturer. True judgments about quality and purity have to come from independent sources like Consumer Reports or Consumer Lab.

The FDA doesn't regulate the quality and purity of nutritional supplements. On the positive side, this has allowed supplement manufacturers to keep costs down, not having to navigate arcane and complex regulatory restrictions.

On the negative side, some supplement manufacturers get away with 1) producing supplements that fail to contain the stated amounts of ingredients, 2) contain contaminants like lead, and 3) make extravagant and often unfounded claims like "superior," "pharmaceutical grade," and "purer."
Thankfully, analyses like those conducted by Consumer Reports and Consumer Lab are reassuring. The great majority of fish oil preparations on the market are high-quality, free of contamination, and provide the benefits you desire. Just be sure that you purchase your product from a supplier that turns over its inventory with some frequency. A dust-covered bottle from the back of
the shelf is probably not a good idea, nor is a bottle that, when opened, emits a powerful odor of rotten fish. Both point towards rancidity.

Conclusion

If omega-3 fatty acids are not a part of your heart disease and stroke prevention program, then you're missing a critical ingredient for health. In fact, we can include omega-3 fatty acids among the most significant health discoveries of the 20th century.

Omega-3 fatty acids from fish oil reduce triglycerides more than any prescription agent. New research is showing that omega-3s provide the unique benefit of reducing the after-meal, or postprandial, flood of lipoproteins that are potent triggers for cardiovascular disease. Omega-3 fatty acids provide direct atherosclerotic plaque-stabilizing benefits throughout all the arteries of the body and yield reductions in death from cardiovascular disease unmatched by any other treatment, prescription or otherwise. The anti-inflammatory effects of omega-3s complement and, in many regards, surpass that of prescription anti-inflammatory drugs, without the threat of adverse cardiovascular effects.
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Chapter 13

Control Plaque with Exercise

Exercise and physical activity facilitate success in your Track Your Plaque program. While not sufficient as a standalone strategy, exercise improves insulin response, reduces blood sugar, partially corrects lipoprotein abnormalities, reduces blood pressure, and helps control weight.

Step 3 of Track Your Plaque is to correct the lipid/lipoprotein and other abnormalities causing coronary plaque. If you’re eager to begin, exercise is a tool you can start almost immediately.*

I can almost always tell who does and who doesn’t exercise when they walk into the office. The step of the exerciser is brisk and confident; they sit erect while we talk. Non-exercisers, in contrast, often drag their feet as they walk in and slump while they sit. The difference goes deeper than physical appearances. Exercisers are more upbeat and positive, less apt to stumble over the hurdles encountered in day-to-day life. They are more likely to deal effectively with change, both in their program as well as life in general. To non-exercisers,

* Before starting an exercise program, you should discuss the specifics of your program with your doctor first. Your doctor will be able to assess whether various exercises are safe or not, given your particular starting condition. This is especially true if you have a positive heart scan score or a prior history of heart attack, angioplasty/stent, or bypass surgery, even if you are without symptoms. The actual risk of heart attack on initiating exercise when you start from a sedentary lifestyle is small, but it does happen. If your calcium score is >100, then a stress test prior to exercise is mandatory—not to detect coronary disease, but to assess the safety of your response to exercise. This is true even if you don’t have symptoms of chest pain, abnormal breathlessness, or neck or arm pain. If you do have any of these symptoms, a discussion with your doctor before undertaking exercise is an absolute necessity.
hurdles in life often seem overwhelming, put there as obstacles to defeat them. When presented with change, they often resist and try to find an escape or excuse.

I’ve witnessed the transformation from a non-exerciser to an exerciser hundreds of times, but I am still amazed at the power in this process. Mike L. is a good example.

I met Mike at age 57. Two years earlier, Mike had a heart attack that converted the bottom third of his heart muscle to scar. His cardiologist at the time told him that the damage was done and the closed artery causing the heart attack could not be opened. They would watch him for signs of an impending second heart attack. Mike accepted this answer but was dissatisfied with the lack of direction he was given in dealing with the causes of his heart disease.

When he came to me for a second opinion, Mike didn’t walk, but lumbered slowly into the office. He plopped his 303 lbs. into a chair and slumped against the wall, huffing from the modest effort of walking 25 feet down the hallway—clearly not a physically active guy. Mike and I talked at length about how he needed to change his lifestyle, eating habits, correct his lipoprotein patterns (of which he had many), etc. I wasn’t initially very hopeful that Mike could succeed in sacrificing his sedentary lifestyle dominated by snack-filled TV watching.

To my surprise, Mike seized the exercise advice and launched a vigorous program of treadmill walking, the elliptical machine (a cross between a stair-stepper and stationary bicycle), and weight-training with light weights and high repetitions. He varied his routine but managed 45-60 minutes, five days a week. Over a period of months, I saw Mike three more times. The transformation on each visit was impressive. By the end of six months, Mike had lost 60 lbs. while visibly gaining muscle. He strode confidently into the office. He’d sit upright, eager to talk about his health. He learned to love exercise, as he experienced the power it had to change his health, mood, and appearance. For Mike, exercise provided a powerful tool for this tired, lethargic, and overweight man to regain control of his life.

For Mike, exercise provided life-changing power. The combination of exercise and weight loss also yielded substantial improvement in his lipoprotein abnormalities. Of course, Mike still required other efforts to correct his lipid/lipoprotein patterns—he did suffer a heart attack, after all—but he viewed this as small price to pay for continued vigorous health.

Exercise can be a great self-empowering strategy for your program, something you can control. You can do a little or you can do a lot: it’s entirely up to you. It’s also a natural method that costs little and can be fun. Weight loss can likewise achieve many of the same benefits. Combine the two, and you have a synergistic combination that yields substantial control over your heart disease.
future. Participants in the *Track Your Plaque* program who exercise and maintain near-ideal weight do enjoy greater success in controlling their heart scan score.

The discussion of how to exercise could fill an entire book by itself. There are many wonderful books, videos, and exercise programs that much more completely discuss the nuts and bolts of how to exercise. We will not cover these issues here. Our discussion will center around how exercise fits into the *Track Your Plaque* program to achieve improvements in lipid/lipoprotein patterns.

First, let’s dispel some common myths about exercise.

**Exercise myth busters**

*“If I exercise hard enough, I’ll never have a heart attack!”*

Don’t get the wrong idea: Exercise is a means of minimizing, *not eliminating*, your need for other strategies.

Men in particular are frequently guilty of this misguided attitude. They convince themselves that heart disease risk can be completely overpowered by vigorous exercise. It explains why we hear about exceptionally fit men, such as marathon runners, who unexpectedly suffer heart attacks or sudden cardiac death.

Unfortunately, exercise alone will *not* reduce your heart scan score, nor will it completely turn-off your heart attack risk. Exercise is important, but is insufficient by itself, no matter how vigorous or long.

*“If I eat right and do everything else, I won’t have to exercise.”*

Conversely, can you succeed in shrinking your plaque yet be a complete “couch potato”?

Perhaps. But the price you’ll pay will be more medication, a stricter diet, and an all-around greater effort to achieve the same results.

Inactive people struggle to control their health and are less likely to “turn-off” an increasing heart scan score. When I say “active,” I don’t mean running marathons or performing at athlete levels. The level of physical activity required to succeed is modest, a level that just about anybody can reach.

Luck eventually runs out for the “couch potato” if sedentary habits lead to substantial weight gain, as it nearly always does. Once inactivity leads to obesity, it frequently leads to pre-diabetes and diabetes, and control of your score becomes much more difficult. Even if obesity isn’t yet present, inactivity all by itself can still result in insulin resistance (a prerequisite for developing diabetes) and all its associated lipoprotein disturbances.
Committing yourself to a program of exercise or physical activity truly powers your overall effort, making multiple facets of your program more readily achievable.

“I don’t want to be an athlete!”
Let’s distinguish two different goals in an exercise program.

The goal for Track Your Plaque purposes is to use exercise to help reduce risk of heart attack and shrink coronary plaque. This requires a level of exercise that just about anyone can achieve with modest commitment of time and effort. A very different goal that does not concern us is to reach a high level of cardiovascular fitness, like that achieved by competitive athletes. While being exceptionally fit and able to run 26.2 miles in 4 hours is admirable, this sort of performance is entirely unnecessary for our plaque-control purposes.

This distinction may seem obvious. But many people, when a modest exercise program is suggested to reduce cardiovascular risk, instead hear “I want you to achieve extreme levels of cardiovascular fitness.” They are intimidated, fearing hours upon hours of exercising to exhaustion, and give up before they’ve even started. Extreme fitness is simply not necessary to obtain significant cardiovascular benefits. The truth is that almost anybody can reach the levels of physical effort required to achieve your goals in Track Your Plaque.

The bulk of protective benefits of exercise come from walking, light yard work, and other similar but modest levels of everyday activities. The ability to walk at a moderate pace and incline on a treadmill for several minutes is a level that nearly maximizes the cardiovascular benefits of exercise (Lee 2010). Surprisingly, higher levels of fitness, such as long-distance jogging, long-distance biking, etc. provide only a modest additional decrease in heart attack risk.

The broad health-enhancing benefits of exercise are undisputed. But let’s now talk more specifically about how exercise and weight control can be used as tools to keep your heart scan score from increasing. We achieve this, once again, by using exercise to help correct your lipid and lipoprotein patterns.

Exercise: A treatment for lipids/lipoproteins
Exercise exerts a broad range of beneficial lipid and lipoprotein effects:

- Exercise increases HDL cholesterol—When going from a sedentary “couch potato” to moderate activity most people increase HDL by around 5 mg/dl. This effect lasts about two weeks and therefore requires continuing effort. The majority of people can achieve this level of benefit without expending extraordinary effort. Extreme levels of exercise can raise HDL 10 mg/dl if sustained. Exercise increases the most beneficial
HDL2 fraction or “large” HDL more than the less beneficial HDL3 or “small” HDL.

- Exercise decreases triglycerides—This effect can be substantial, particularly if exercise leads to weight loss. Drops of 100 mg/dl are not uncommon if exercise is sustained and consistent.
- Exercise decreases LDL cholesterol and LDL particle number/apoprotein B—A modest effect. Reductions in LDL of 10% are common, enough to help when part of a more comprehensive effort, though usually not enough to stand alone.
- Exercise increases LDL particle size—This effect goes hand-in-hand with the rise in HDL cholesterol. Exercise by itself will rarely correct this pattern fully since diet is the dominant influence, but exercise can be a useful adjunct.
- Exercise reduces blood pressure—Exercise generally yields a drop of 2 to 4 mm when sustained and consistent.
- Exercise reduces blood sugar—Many people with borderline diabetic tendencies can improve sensitivity to their body’s insulin with consistent physical activity. Even diabetics will experience reductions in blood sugar and/or reduced need for medication to control blood sugar.
- Exercise helps control weight—Weight loss magnifies all of the above listed lipoprotein benefits and reduces blood sugar, as well. Exercise accelerates weight loss by contributing to the 3500 calories required to lose a pound of weight. Even if you’re already at your desired weight, exercise makes maintaining weight much easier.

(Kokkinos 2010; Bassuk 2010; Whyte 2010)

Exercise also makes you feel better. Once you make exercise and/or physical activity a consistent activity several times a week, you will likely experience improved mood, deeper sleep, greater alertness, clearer and more optimistic thinking. People who are brighter in their overall outlook tend to be more successful in other spheres of life. They are also less likely to indulge in mood-elevating but health-defeating habits like smoking, excessive drinking, and too much caffeine. All this can translate into greater success in correcting lipoprotein abnormalities.

There’s a downside: Benefits of exercise are not permanent. Even if you were an Olympic-level athlete in high school or college, the benefits of training dissipate over a period of months after you stop.

It’s not so much what you do, as how long and consistently you do it. Modest efforts carried out over a period of 10 years yield greater benefits than intensive exercise for six months followed by sporadic efforts. Some of the most successful participants in Track Your Plaque are people who’ve committed themselves to modest—yet realistic—exercise and/or physical activity routines, such as walking with a spouse for 30 minutes four times a week. This is not
physically challenging for most people, but does require a commitment to follow-through with consistent effort week after week, month after month, year after year.

**The “secret” to a successful exercise program**

Take a practical, long-term perspective and answer the following question: “What sorts of physical activities can I perform and stick to for the next 30 years—and enjoy doing it?” The key here is not the form or intensity of effort. **The essential ingredient is that you derive some form of enjoyment from the activity.** If you force yourself to pedal five miles a day on your stationary bicycle in your basement but despise every moment of it, you’re unlikely to succeed over the long haul.

People who discover enjoyment and satisfaction in their exercise will stick to their program even when stressful distractions crop up in other parts of their lives. It’s the difference between jumping out of bed in the morning and looking forward to learning to salsa in your dance class versus dreading the 30 minutes you force yourself to endure on the treadmill.

Some people prefer solitary activities that provide quiet moments for contemplation. You might therefore enjoy riding a stationary bicycle, walking a treadmill or using an elliptical machine. Others prefer the camaraderie and sense of shared experience that is only possible in groups. For these people, aerobics class, Zumba® class, group yoga instruction, “spinning” classes and other organized group activities might be best. Still others might most enjoy the close company of a partner on walks, tennis, golf, etc.

If you prefer visual stimulation, you might do better by watching the news on TV while on your stationary bike. If you prefer auditory stimulation, listen to some of your favorite CD’s, MP3, or the radio while you walk a treadmill. You might vary your choice of exercise over the course of a week: Bike once a week, walk twice a week, swim once a week, play tennis once a week.

Maybe you need to be mentally stimulated. You’re bored easily and struggle to exercise more than a few minutes. Or, perhaps you are “goal oriented” and need to accomplish something concrete with your physical activity. Then consider physical activities in which you fix, clean, learn, or create something. Rake leaves, aerate your lawn, enroll in dance classes, ride your bicycle to work, run errands by foot, etc. Turn the clock back 100 years and give up taking the car to the grocery store, replace your power lawnmower with an old-fashioned rotary-blade mower, walk the kids to school instead of driving them.

The key is to discover your own path and not just resign yourself to a routine you dread, finding every excuse to put it off until “later.”
What happened to “No pain, no gain?”

What constitutes a sufficient level of physical activity? For instance, is light to moderate gardening good enough? How about cutting grass? Do you have to exercise to exhaustion or is just breaking a light sweat enough?

You can decide for yourself whether an activity is sufficiently stimulating to the cardiovascular system if you raise and sustain your heart rate to 70% of your age-predicted maximum heart rate or greater.

This seems like an awfully complex rule, but it’s really simple to calculate. To get your 70% target—the minimum heart rate you would like to achieve and sustain—start with the number 220 and subtract your age; multiply the result by 0.7 (70%). This will give you the approximate minimum heart rate required to yield the sorts of benefits listed earlier. (This applies to both men and women.) For example, if you’re 50 years old:

\[
220 - 50 = 170 \\
170 \times 0.7 = 119
\]

You therefore need to maintain your heart rate at 119 beats per minute or greater. A heart rate of 119 is easy to attain by a brisk walk, light- to moderate-effort biking, walking on your treadmill with a modest incline and moderate pace (say, 3% grade at 3.4 miles per hour), raking leaves, singles tennis, ballroom dancing, etc. Measure your heart rate by feeling the pulse in your wrist (just below the base of your thumb): Count the number of heart beats in 15 seconds and multiply times 4, and this yields heart beats per minute. You can also measure your heart rate by using one of the many heart rate monitors available today or that come built-in with exercise machines. These devices are reasonably accurate (especially if there is moisture, i.e., sweat, between your skin and the metal sensor). If you have cause to doubt a device’s accuracy, just compare the heart rate readout to the value you obtain by taking your pulse. You can, of course, exceed this 70% level (provided your doctor agrees). You will burn more calories and lose more weight, but the additional improvement in your lipids and lipoproteins will be relatively small.

The “no pain, no gain” mantra does not apply to you. This is meant for people whose aim is to achieve higher levels of fitness for competition, or other such purposes that have little to do with our goals. Pain is not required to achieve success in correcting lipids/lipoproteins or losing weight. Much of the benefit from exercise comes by devoting sufficient time to your efforts. Two hours a week of a 70% effort is the minimum, preferably divided up into three or four sessions. This amount provides 90% of the lipid/lipoprotein benefits achievable with exercise. Less than this and you might not obtain the improvements in lipoprotein patterns you desire. More than this, and there are modest additional increments of up to
10% in your lipoproteins. (This does not mean that lipoprotein abnormalities are 100% correctable with exercise. It simply means that vigorous exercise can improve lipids/lipoproteins, and exercising at the level described above will achieve 90% of this amount.)

The message is that intense exercise is not necessary for you to feel better, lose weight, or correct your lipids and lipoproteins. Don’t obsess about heart rate targets. Just be aware that achieving a heart rate of around 70% of your maximum for age should be part of the overall equation for cardiovascular benefit.

**Lose weight faster with exercise**

The closer you are to your ideal weight, the more likely you’ll succeed in controlling the growth of plaque in your coronary arteries. Conversely, if you are overweight, success is still possible but other efforts (exercise, diet, medical treatments, etc.) will need to be correspondingly greater.

Excess fat (particularly when concentrated in the abdomen, or “visceral,” fat) produce several evil substances known as “adipocytokines.” These molecules are responsible for many of the negative effects of excess weight, particularly high blood levels of fatty acids and insulin resistance. Research in this same area yielded the recent discovery of leptin, a hormone that may be responsible for appetite control. This will be an exciting area of investigation in the coming years.

Some lipoprotein patterns are very sensitive to body weight. If you begin with a low HDL, high triglycerides and small LDL particles, all these abnormalities improve with weight loss. (If you have these abnormalities but are not overweight, then no further benefit will be obtained by losing weight.) The number of pounds required to correct each pattern varies widely from person to person. In general, however, the effect can be substantial. Increases in HDL of 5-10 mg/dl and triglyceride decreases of >50 mg/dl are common when losing 10-20 pounds, for instance.
Losing weight is almost always best accomplished with a combination of diet and exercise. It is very difficult to lose more than a few pounds relying exclusively on diet only while neglecting exercise, or vice versa. Remember that losing one pound means you need to burn 3500 calories more than you consume in food. Achieving this with calorie restriction alone is tough. Few people successfully lose weight and keep it off using diet alone. This principle was demonstrated very clearly by Mike and Darlene:

Remember Mike’s transformation from an overweight, lethargic, survivor of a heart attack to the vigorous, energetic exercise enthusiast?

Mike, 57, and his wife, Darlene, 58, were both substantially overweight when I first met them. At the start, Mike, at 6 feet 2 inches, was 303 lbs. Darlene, at 5 feet 1 inch, was 203. Both had multiple lipoprotein abnormalities that could improve markedly with weight loss and exercise, including low HDL, small LDL, excess VLDL and triglycerides. In addition, Mike was borderline diabetic and had already been started on a diabetic medication by his family doctor.

This close couple, married over 20 years, had committed to better health together and wished to proceed through the program as partners. The couple was interested in minimizing the use of medication to correct their lipoprotein disorders and so jumped wholeheartedly into a nutrition program (like the one

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Am you overweight?

Let’s face it: Most of us know when we are overweight. But if you need a measure to establish a baseline and track to assess your results, one helpful number is the Body Mass Index, or BMI. Many studies examining the health risks of excess weight use the BMI.

This measure is calculated depending on your height and weight. What it does not take into account is your relative bone structure (“big-boned”) or the relative proportion of muscle mass compared to fat that you have. Different races can also have moderate distorting effects on the BMI. In other words, the BMI is a rough index that tends to lump many different body types together based purely on size. If we were to rely on BMI, a value of >27 is regarded as overweight, >30 is defined as “obese”. To determine your BMI:

1) Weight in kilograms (kg) = weight in pounds/2.2
2) Height in meters (m) = height in inches x 0.025
3) BMI = weight (kg) / height (m)^2

A BMI of 20-25 is ideal, 25-30 is overweight, and >30 is obese.

Another helpful measure that eliminates the differences in body types is body fat percent, most readily obtained with bioimpedance devices (“body fat analyzers”), now inexpensive and widely available in health clubs and stores. If your percent body exceeds 19% for males or 21% for females, then you are overweight, and this likely contributes to your lipoprotein abnormalities (Pajunen 2010; Marques-Vidal 2009). Percent body fat can provide a great measure to track your results as you proceed through your exercise and weight loss efforts.
described in chapter 9) and exercise. The couple exercised for 45-60 minutes (more than the recommended time) five days a week: Darlene by walking, biking, and aerobics; Mike by a combination of walking a treadmill, elliptical trainer, and weight-training. After 6 months, Mike had lost 60 lbs; Darlene lost 50 lbs—phenomenal results. The couple was ecstatic and would walk into my office beaming with pride over their success.

Unfortunately, family stresses entered their lives that took the greatest toll on Darlene. Their 29-year old daughter with three young children was going through a messy divorce. Darlene had no choice but to devote much of her time and emotional energy to helping her daughter deal with this life-shattering change. Darlene sacrificed her exercise efforts in the process. At the same time, Mike tried to do his part but still managed to maintain his exercise routine. Another six months later, Mike was looking great, having maintained his ideal weight and avoided regaining the 60 lbs. he’d lost. Darlene, on the other hand, because of the unselfish devotion to her daughter’s children, regained the entire 50 lbs over the next year, even though she’d adhered to the diet. She felt overwhelmed and defeated.

The lesson: Despite good nutritional habits followed by both, it was the exercise that allowed Mike to maintain his weight, while Darlene’s (understandable) lapse in exercise led her right back to her original overweight condition. Darlene’s experience is somewhat extreme but nonetheless an example of how the combined effects of exercise and diet yield the greatest results.

The key in using exercise to burn off calories and lose weight is more the duration of exercise, and not the intensity. Losing weight will require you to exercise longer than if you were just trying to maintain weight. Exercising 30 minutes per day is best for maintaining weight; longer periods are required to burn the additional calories to achieve weight loss. However, intermittent bursts of more intense activity, e.g., walking briskly up a hill during a walk, increasing the incline on a treadmill for a minute or two, increasing speed and resistance on a stationary bicycle or elliptical device, will further accelerate weight loss and increase fitness.

Muscle burns calories

If you’re trying to lose weight, or if you’d like to further enhance overall health, consider weight or strength training.

As we age, we all lose muscle mass and gain fat. At age 65, the average man has lost 30% or more of his muscle mass, a woman at least 20%. Even if you weigh the same as you did at age 20, you have considerably less muscle and more fat.
Muscles are calorie furnaces and a youthful quantity of muscle can help lose fat and maintain weight. Greater muscle mass increases your body’s basal metabolic consumption of calories. In other words, muscle consumes calories just to meet its energy requirements. This means you’re burning up calories without trying, just from the heightened metabolic requirements of muscle. You don’t have to be a bodybuilder to achieve these modest benefits, and neither will you bulge with muscles. Increased muscle mass and strength also correlates with better bone health, less osteoporosis (Marin 2010).

The “Slow Burn” method: Greater muscle, greater metabolic effects, less injury

Personal trainer, Fredrick Hahn, and coauthor of the book, The Slow Burn Fitness Revolution, along with Dr. Michael Eades (of Protein Power fame), have helped popularize the practice of slow-motion strength training, what they call “slow burn.”

Unlike the relatively brisk motions of conventional strength training, the slow burn movements are conducted in slow motion, requiring approximately five seconds for each “positive” and “negative” movement, e.g., five seconds to pull down the latissimus machine, then five seconds to return to neutral position. A helpful animation that demonstrates the pace and motion of the “Slow Burn” method can be viewed here: http://www.seriousstrength.com/home/about.php. Interestingly, Hahn advocates only one set per muscle group using a weight sufficient to perform 5-6 repetitions. The unusually slow motion with no jerking at the positive-negative transition means virtually no risk for injury, unlike more forceful conventional strength movements.

The motion requires some getting used to and will leave you unusually sore the first few times you try it, even if strength training is already part of your exercise habits. The muscle stimulating effects of the slow burn technique are vigorous, yielding increases in muscle growth and strength in a short amount of time.

More information is available at Fred Hahn’s website and blog, found at www.seriousstrength.com. The 3-DVD video produced by Hahn is especially helpful (http://www.seriousstrength.com/home/shop-Videos.php).

You can obtain the calorie-burning and health-enhancing effects of muscle with a modest time investment by weight training once or twice a week for 20 minutes per session. If you’re unfamiliar with the machines or free weight movements, most health clubs can provide instruction to get you started. Once you’re shown the basic movements, the rest is easy. Follow a “circuit-training” approach using higher repetitions (10-20) at each station with little or no rest in between each set and the 20 minutes you devote to weight training will also suffice as aerobic exercise. (Circuit-training refers to following a sequence of strength exercises in a cycle that you can repeat several times; ask your trainer specifically about this technique.)
If you incorporate strength training, you will add muscle weight—often several pounds—and your net weight loss from fat may not seem as dramatic. Calculating BMI won’t be helpful, since it only incorporates weight and height, regardless of the percentage fat vs. muscle. If you choose to add weight or strength training to your program, assessing just how much fat-weight reduction you’ve achieved is easy. There are several ways to do this:

1. **Measure body fat percent**—Convenient devices to measure body fat are widely available nowadays. Reliable units cost as little as $50. (Tanita manufactures several great products.) Many health spas and clubs now have them, as well. Body fat is measured using “bio-impedance,” which makes use of the differing electrical conductivity of fat and muscle. (Muscle is a conductor of electricity; fat is an insulator.) A very small quantity of electrical current (which you cannot feel) is passed through the body and the device calculates how much fat and muscle is encountered in the path of the current. Devices that pass the current through the feet (“foot-to-foot”), as opposed to the hands, are more reliable. You stand on these devices much like a conventional scale. Current passed from one hand to the other is a better measure of fat and muscle in the thorax, but neglects much of the abdomen. Foot-to-foot bio-impedance includes the abdomen and tends to be more accurate, particularly if you have excess abdominal fat characteristic of the “metabolic syndrome”. Body fat can also be approximated by using calipers and measuring the thickness of fat in several skin-folds around the body. This is best performed by someone who uses this technique frequently, such as a personal trainer. If you do it yourself, there is just too much room for “fudging” results.

2. **Look in the mirror**—This may sound obvious but it’s a method you can use anytime. If you gain muscle and lose fat, you’ll notice a more erect posture; broader shoulders; a flatter, more muscular abdomen, less “love handle,” firmer thighs. After all, most people are very interested in looking good and weight training can yield great improvement in appearance.

**Caloric expenditure—Keeping you honest**

Time after time, people say, “I don’t understand it. I’m eating healthy foods and exercising, but I’m just not losing any weight!”

Measure the number of calories these people burn in physical activity during the course of a day, and they commonly fall far short of the average person’s daily energy usage. In other words, many people have gotten overweight because they’ve become very economical in their physical activity.

The next time you’re at work or in public, observe how overweight people move. They will often be very spare in their motions: They’ll be seated at a desk and scoot about in their wheeled desk chair, rather than get up. They will reach
for things, rather than get up and walk to get it. They'll use more hand motion rather than their body and legs. Each one of these economies of movement may conserve only a fraction of a calorie, but compounded hour after hour, day after day, and it all adds up to a progressive gain in weight. These people are very efficient in their calorie burning and the unburned energy is saved as fat. When you measure the amount of energy people burn, overweight people burn far less calories, often half or less the calorie expenditure compared to a non-overweight person (Ewbank 1995; Slentz 2004).

You can measure your calorie expenditure using a pedometer. Pedometers have come a long way since the crude mechanical devices available when we were kids. I’ve used several excellent models that cost between $10-$20. You can find them in sporting goods stores, health clubs, and in some department stores. Present day pedometers can measure horizontal motion fairly accurately and convert this to number of calories burned. You can therefore use a pedometer to measure how active you are and how many calories you’re actually expending. Sedentary, overweight people who wear a pedometer commonly burn only 100-150 calories in a typical 12-hour day (over and above the basal metabolic rate requirement). Active people, on the other hand, commonly burn 350-500 or more calories. Very active people burn 500 or more calories per day. The daily difference is not great, perhaps 200 calories. But the 200 cal/day difference leads to a 1 lb. weight gain over 17.5 days (3500/200 = 17.5), Over a year: 21 lbs.! That’s 21 lbs. just from sub-consciously limiting movement through an ordinary day.

Another common difficulty is that, when you cut your calorie intake, your body’s survival instincts kick in and you unconsciously cut back on physical activity. Some call this re-setting your “set-point.” This instinctive behavior served humans well when we had to forage for food during times of starvation. A reduction in calorie intake triggers our bodies to conserve energy. We reduce energy expenditure in almost imperceptible ways. Like the people who gain weight because of more economical movements, you will also limit your energy expenditure by cutting corners in your daily physical activities (Farias 2010). However, if you are aware of this sub-conscious phenomenon, you can use it to your advantage by consciously resisting the inertia to physical activity: get up and get going!

Before starting your weight loss program, wear the pedometer and record the number of calories you burn each day over a specific period. For example, reset your pedometer starting at 8 a.m. then go through your day. At 8 pm, write down the total calories burned. This provides a good idea of how many calories you’ve expended through the most active part of your day. If you’ve used only 175 calories, you know that you’ve had a relatively inactive day. If you’ve used 350 calories, this has been a moderately active day that will help control weight. Repeat this process for three successive days and average the daily value.
Once you’ve determined the average number of calories you burn each day, you can now set your goals higher. A goal of 350 calories in a 12-hour period is realistic. If you fall short, you can review your day and see how you might do better the next day: avoid shortcuts when you’re shopping, parking farther away at the office, taking the stairs, etc. Increasing your energy expenditure by only 100 calories a day will result in a loss of one pound a month or 12 pounds a year, significant over a long period. (Unfortunately, some activities, like swimming or biking, cannot be quantified by most pedometers.) If you are in a hurry to lose weight, you can increase your daily expenditure accordingly. For example, adding another 230 calories of calorie expenditure per day (it’s really not that tough) will result in a weight loss of two pounds per month over and above what you achieve by other efforts.

Summary

• They key to exercise is to choose activities that you enjoy, or to find some means to make it enjoyable. Otherwise, you will simply find every excuse not to exercise.
• Exercise helps correct multiple lipid and lipoprotein patterns that cause coronary plaque to grow. It magnifies the benefits you obtain through diet.
• Losing weight is most effectively accomplished through a combination of diet and exercise. Losing weight as opposed to maintaining weight requires a longer duration of exercise.
• Keeping count of calories you expend using an inexpensive pedometer can be a simple though powerful method of ensuring that even ordinary activities add up to a quantity of physical effort that provides weight control benefits.
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Sometimes the best way to convey a message is to tell a story.

Too often, people shy away from beginning a program like this because they're afraid of what they might find, or that it might lead to frightening, risky treatments. So here are several real life profiles of several participants in *Track Your Plaque* who navigated through the program successfully. The people and stories are real but names changed to maintain privacy.

Of course, not everybody who follows the program will achieve the successes detailed here. I tell these stories to show that it *can* be done following the *Track Your Plaque* strategies, often requiring no extraordinary commitment, using tools that you already have available. Some of these people also had to overcome hurdles, barriers put in their way, to succeed. Sadly, most of these barriers to success were put there by their doctors.

Nonetheless, these stories are meant to instruct and inspire. Perhaps we can add *your* story.

**LindyBill: 32% reduction**

The irrepressible *Track Your Plaque* online Member, LindyBill (his online nickname), posts this tale of the ups and downs of achieving coronary plaque reversal in the modern age—in an HMO, no less!
LindyBill’s initial struggles and eventual success highlight how to get the job done, even when the doctors around you do their best to booby-trap your best efforts. LindyBill handled it all with grace, despite the fact that he had acquired deeper appreciation for the finer points of coronary plaque reversal than his doctors. Here is a true life testimonial in his own words.

I was born in Saginaw Michigan in 1934. My Father built boilers for electrical power companies all over the Midwest and so I spent my childhood on the road. After a couple of years in the Army in the ‘50’s, I graduated from San Diego State in 1956 and spent my productive years as a salesman and small businessman in California. I raised a family of three kids and retired to Waikiki in 2001.

I was skinny as a rail and never worried about my health. I smoked for 20 years and finally quit using “Shick Shadel” in 1974. I drank heavily for 40 years and quit, again using “Shick Shadel,” in 1995. (Schick Shadel is a hospital in Seattle that uses aversion therapy to get people off booze and cigarettes. They also used it for drugs. It got quite big in the ’70s and they had hospitals all over the country. Mainstream medicine was against them and lawyers had a field day suing them. They have one facility left. Worked great for me.)

By 2007, I was 72, obese (32 BMI), hypertensive (on BP meds) and concerned about my health. I was asymptomatic for any disease, but researching the Internet for medical info. The “Futurepundit” blog had an article on Vitamin D that was interesting and in following it I discovered Dr Davis’ blog. His explanation of heart disease fascinated me and I decided to get a scan. The only EBT machine around was located at Holistica in Waikiki, where I lived, and I scheduled a heart and body scan there in October of 2007.

The scan revealed that I had a 322 calcium score. Plus, I had a bad looking cyst on my kidney. My primary care Doctor at Kaiser Hawaii had me do a stress test which came back negative, and put me on a statin. The kidney cyst was malignant and was removed.

Upon finding the heart disease, I immediately started the full range of Track Your Plaque supplements, including an 8000 IU dosage of vitamin D. My doctor had no interest in my supplements. My lipids at that time were HDL 34, triglycerides 98, and LDL 119. Kaiser had a contract with VAP but had never ordered a test from them. The attitude of the cardiology dept was that the lipoprotein analysis was “an experimental test which had no clinical use.”

In the summer of 2007, I joined the TYP [Track Your Plaque] site and finally faced up to my obesity. I was very put off by the concept of not eating bread and doubted my ability to do this. But when I stopped wheat, corn, sugar, rice and potatoes, I found it very easy to stay off. To my surprise, I found myself
losing a pound a week. I was 252 lbs. when I started and by January of ’09 I was down to 222, at 28 BMI.

I participated very heavily in posting on the TYP website and my learning curve was accelerated by the input from Dr. Davis and others there. The more I learned, the more I realized how behind the times mainstream medicine was on preventive treatment of heart disease. They were doing a great job when you had angina or a heart attack, but preventing those events from happening were not a point of major interest for most doctors. It seemed to be a “lose weight, take a statin and call me if you have chest pain” attitude. Details of diet and use of supplements were never mentioned.

These last two years of concentrating on improving my health, posting on TYP, and researching has made me realize that the only person who is interested in improving my health is me. I quit worrying about finding the "right" doctor and now spend my time figuring out what I need to do next, doing it, and getting the doctor to help if it involves a test or script. I count on the doctor to find things I am not aware of, but sense that what I am aware of is much better taken care of by me. A doctor spends 20 minutes a few times a year going over my health. I spend 24/7/365 on it.

In October of 2008, I finally convinced my Kaiser doctor to order a heart scan and was astounded when it came back with a score of 219, a 32% reduction from my first score. My doctor there and the Kaiser cardiologist I talked to were not interested. They don’t “believe” in heart scans.

In March, I changed my primary care doctor and the new one had no interest in heart scans or advanced lipid panels. But he at least was willing to accommodate me. I negotiated a VAP [Vertical Auto Profile] lipoprotein test by agreeing to pay the $40 additional cost. When this came back I was again astounded by the major difference in my lipids. My 34 HDL was up to 46. My 98 triglycerides were down to 48. My LDL has dropped from 119 to 52. In addition, I found my small dense LDL, which I believe used to be high, is in the A/B 50% position. My Lp(a) is 9. My apoB/apoA ratio is 0.4.

I am continuing my efforts to reduce my weight, my small LDL ratio, and my calcium score by staying on the TYP diet and supplements. Without my reading of Dr. Davis’ blog and joining his site I would be sitting here with undiagnosed heart disease, waiting for angina or a heart attack to happen.

The active resistance by doctors to practicing "best medicine" on heart patients is discouraging, to say the least. It makes you wonder in what other areas of medicine their practice is also behind the times. They want to use new diagnostic tools when they are "sexy," such as procedures. And they want to do what the other doctors they hang out with do. Other than that, if it wasn't taught to them in medical school, they are not interested. Most have little or no interest in
non-prescription drugs. Their prejudice against supplements is just as bad as the "back to nature" type's prejudice against scripts.

There's more . . .

My natural reaction, and that of most of us here, is to preach to the world. I call it UBF: the "Universal Blab Force" we are all afflicted with. Counterproductive and a waste of time. If your doctor wants your opinion, he will beat it out of you.

I realized even then that what I needed to do was not to argue with them but to figure out the treatment I wanted and convince the doctors to give me the tests and scripts I needed.

When I came back to my primary care doctor in '07 with my kidney and heart scans, I never said a word to him about the fact that he should have found the problems. He wanted me to take a statin and get a stress test? Fine. Gave me a one-page handout that told me to lose weight? Fine. I knew the stress test was just defensive on his part, but it cost me nothing but my time and I was interested in learning about the procedure. I knew then that he was a "weight and statin" doctor, with no other preventative measures in his arsenal. So what? All the others there were probably the same. I did force him into ordering a heart scan when Kaiser got a 64-slice machine in stock.

When I got Afib [atrial fibrillation], he was content to leave me on warfarin for life. Told me my "compliance" made me a good candidate for it. That was the end of him for me. I found out who the electrophysiology cardio at the hospital was and set up an appointment with him. He did an excellent job of cardioversion for me. He was a Boston doctor who had taught at Harvard and thought he passed "Nun's farts." Tried to hide his arrogance with the patients but was not very good at it. He was brought in to run the whole electrical end of the cardiology dept for Kaiser Hawaii. I had two fun exchanges with him. First time I saw him, my calcium score had just been posted and I asked him to look it up. When he told me it was 219, a 32% regression in a year, I couldn't hold in my excitement. It was obvious this was the first Agatston score he had ever looked at. After he looked down his nose at me and told me he had no use for calcium scores, his response to my "why?" was "they use too much radiation." I responded, "they only use about eight X-rays" and he backed right off. His lack of knowledge and interest was obvious.

Later in our interview I told him the main reason I was so interested in getting off warfarin was that it caused an increase in plaque. He made no response to this statement but four weeks later when he did the cardioversion and he visited me during the recovery period it was obvious that this claim of mine had stuck in his mind and he had researched it. He brought the subject up and said, "Do you want a stroke or a heart attack? A stroke is much worse." I told him, "That's false alternatives. I don't want either." I never told him that I had balanced my warfarin with 90 mcg of K2 every day. I knew this would make him
have a cow. But I found it made it even easier to maintain a 2.4 INR level. And I hoped it held down plaque increase.

I became friendly with the urologist who removed my kidney cyst and who had discussed dieting with me before and after I had lost 30 pounds. This led to letting loose to him with my UBF about cardiologists. Which resulted in the change in my primary care doctor to the one I have now. It was this late in the game that I finally realized that seeing a cardiologist was a waste of time unless I needed a procedure. They are all "cath lab" oriented.

So now I negotiate tests and scripts with my new primary care. I know he is a "weight and statin, call me if you have chest pain," doctor who practices 1985 cardiology otherwise. That's fine because he is friendly to my outlook and is willing to order most of the tests and scripts I need. I just got the VAP. I will get another heart scan this October and another VAP next year. In the meantime I will take my supplements, dance, ocean walk daily and generally enjoy life. I tell you, the girls on the beach at Waikiki get better looking every year!

Dr. Davis: Coronary plaque reversal couldn’t have happened to a nicer guy.

I can’t describe the tremendous satisfaction I get when someone like LindyBill, encountering nothing but indifference towards this life-changing (sometimes, life-ending) question, discovers the solution and vastly improves on what any of the doctors around him could have achieved.

Had LindyBill started out by asking his doctors if coronary plaque could be reversed, he likely would have received the usual snide comments, scoffs, or dismissals. Instead, LindyBill sought his own answers—and found them.

Amy A: 63% reduction
At age 52, Amy was at the height of her career.

Amy had started a small business with her husband seven years earlier, and they were now enjoying enormous success. She and her husband, Tim, decided it was time to devote less time to their flourishing business and more time to travel, health, and just sitting back and enjoying life.

Both got heart scans. While Tim’s heart scan score was a modest 21, Amy’s was higher at 117. For a woman, this put Amy in the 90th percentile (worst 10% for her age group). The score came as a surprise to her, despite the fact that her brother had his first heart attack at age 48 and her mother had a heart attack at age 50. As an entrepreneur with a big success under her belt, perhaps she felt invincible.

Lipoprotein (a), Lp(a), proved to be the crucial cause behind Amy’s
coronary plaque, along with a marked excess of small LDL. (Seasoned Track Your Plaque followers will tell you that this combination is an especially potent trigger for coronary plaque.) We advised Amy to embark on a program of fish oil, vitamin D, and niacin for Lp(a). We advised Amy on how to use diet to correct the small LDL pattern.

Amy promptly lost 10 lbs with these changes and was well on her way. However, Amy and Tim’s daughter unexpectedly suffered a fatal injury in a work-related accident.

The parents were devastated. Amy returned to us after a few months, shaken but bolstered by the support of her husband, her family that came back together with the tragedy, and friends. We picked up where she left off.

A second heart scan 15 months after her first revealed a score of 43—a 63% drop.

**Dr. Davis:** In the Track Your Plaque program, we’ve come to respect the profound effects of stressful events, particularly one as exceptionally difficult as the loss of a loved one.

It is powerful testimony to this woman’s strength that she maintained her health program after picking up the pieces of her life. Thankfully, she had the support of a loving husband and family.

I believe that the lesson we need to learn from Amy’s important story is that, while great stress like the death of someone close to you, divorce, financial ruin, etc., can have enormous effects on health—drop in HDL, increased inflammatory measures, increased cancer risk, loss of control over heart disease, even profound reductions in heart muscle strength (a phenomenon that was validated by studies performed in New York City immediately post September 11)—it does not necessarily have to overwhelm you and undo all the good still achievable. In fact, despite the odds, Amy achieved among the largest percentage decreases in score of anyone ever participating in the program.

Perhaps other people experiencing the same severity of stress that Amy experienced would not have had the same level of success that she did. But I do believe that this is yet another example of what is possible in the big picture of heart disease reversal.

Nothing can replace the loss of a loved person in our lives. But the Track Your Plaque approach allowed Amy to continue her life uninterrupted by the devastation of heart disease.
Freddie: Record-breaking 75% reduction

At the start, Freddie had disastrous lipid values:

- LDL cholesterol 263 mg/dl
- HDL 26 mg/dl
- Triglycerides 323 mg/dl
- Total cholesterol 354 mg/dl

Lipoproteins (NMR) were worse:

- LDL particle number 3360 nmol/L
- Small LDL 2677 nmol/L

Heart scan score: 732

Interestingly, Freddie had virtually no vitamin D in his body, with a 25-hydroxy vitamin D level that was unmeasurable.

Freddie was miserably intolerant to statin drugs, with even the smallest dose resulting in intolerable muscle aches. That's when his doctor sent him to me.

Because I felt that the dominant abnormality in Freddie's lipids and lipoproteins was small LDL particles, representing 80% of total LDL particle number, we focused his program on correcting this measure. Freddie's program was therefore focused on elimination of wheat, cornstarch, oats, and sugars, along with an eventual vitamin D dose of 20,000 units to finally achieve a 25-hydroxy vitamin D level of 66 ng/ml. No statin drug in sight.

Freddie was not the most motivated person. He did make big changes in diet, but did not go the "full mile," choosing to indulge several times per week in our "do not eat" foods, especially convenience foods.

A repeat lipoprotein panel showed a marked reduction in small LDL:

- LDL particle number 2988 nmol/L
- Small LDL 688 nmol/L
- LDL (calculated) 188 mg/dl
- HDL 48 mg/dl
- Triglycerides 73 mg/dl
- Glucose 102 mg/dl
- 25-hydroxy vitamin D 72 ng/ml
- Free T3 2.5 pg/ml
- Free T4 1.18 ng/dl
- TSH 1.29
43 lbs of weight loss and 18 months later after the start, a second heart scan score: 183—75% reduction.

You can see that what makes Freddie's experience incredible, apart from the dramatic reduction in score, was that he did it without achieving "perfect" lipids and lipoproteins. Does this mean that it's not the absolute values achieved, but the magnitude of relative change? In other words, Freddie's starting values were so awful that just partial correction might have been sufficient to succeed. I don't think that we should try to squeeze too much out of the experience of one person, but it makes for an interesting possibility.

The zero vitamin D at the start also makes you wonder if much of his success was simply from correction of his profound deficiency. Another twist: Freddie is African American. I don't know what difference this introduced, but it may mean somewhat different response than non-African Americans in certain parameters.

While the rest of the world continues to insist that coronary calcium (heart scan) scores cannot be reduced, I am seeing records being broken. I add Freddie's experience to the growing list of people who have not just stopped coronary plaque from growing, but are seizing control and reducing it, sometimes to dramatic degrees.

Another tantalizing possibility: Would "correction" of Freddie's low T3 value have yielded an even larger reduction in score? We have to be careful here, since recent weight loss will reduce free T3. But, in future, it may be something to consider for Freddie's program to further reduce LDL and, perhaps, heart scan score.

**Neal T: 51% Reduction**

Neal, a 40-year old school principal, went to his doctor because of chest pain.

Slender and physically active, at first he refused to believe that it could have represented heart disease. He started to really worry when he had to stop mid-step while coaching basketball. The pain passed within 30 seconds, but Neal mentioned it to his wife, who promptly insisted that he discuss it with his physician.

His primary care physician, skeptical of heart disease, had Neal undergo a simple stress EKG, i.e., a stress test without nuclear or ultrasound imaging. While it was normal, Neal did experience some of his chest discomfort. To help clarify the issue, Neal's primary care physician asked him to undergo a CT heart scan. His score: 339, in the 99th percentile for men in his age group. Even worse, 200 of the 339 points of plaque scoring were in the left main stem artery, the shared trunk of the left anterior descending and circumflex coronary arteries.
Heart attack here is fatal immediately.

Neal ended up with a heart catheterization because of the crucial location of his plaque, as well as the equivocal symptoms and stress results. Thankfully, only mild plaque of no more than 30% severity in the left main stem artery was identified. Thus, it was unlikely to account for Neal’s symptoms and there would be no benefit from a procedure like bypass surgery. So we were free to pursue his program of prevention.

Through lipoprotein testing, Neal proved to have high LDL cholesterol comprised almost entirely (>90%) of small LDL particles, along with a moderate to severe deficiency of vitamin D.

One year of effort to correct his patterns included fish oil, niacin for small LDL, and changes in food choices. A repeat heart scan 15 months later showed a score of 161—a 51% reduction.

**Dr. Davis:** After the initial gut-wrenching scare to Neal and his family on first learning of his high heart scan score at age 40, the enormous drop in his score brought a big sigh of relief.

I tell critics that, not only is reversal possible, but incredible amounts of reversal can be achieved in many people.

Just a few short years ago, even I didn’t believe this much reversal was possible. The proof is in the pudding.

**What does a dropping heart scan score look like?**

Most of the time, when someone drops their CT heart scan score, it’s tough to tell the difference with the naked eye just by looking at the scan images. You can hold up the “before” and “after” images of plaque side by side, yet often not be able to tell. The difference can be subtle and tough to distinguish just by looking.

While a computer has no difficulty in distinguishing a reduction in score, differences of 5, 10, 15% are difficult, perhaps impossible, to gauge with the naked eye.

Here are sample images from Neal’s heart scan that show the visible regression of plaque:
You’ll notice that white plaque (centered on each image) has shrunk visibly in length, with the current length roughly half that of the original on the first scan. (Several additional cross-sectional “slices” that are not shown displayed a similar phenomenon. The slightly wider appearance of the plaque on the “after” image is a result of different image position.)

The magnitude of plaque reversal was so significant that it is immediately obvious even to the naked eye.

**Ron P: 28% reduction**

When Ron first received the result of his heart scan, he started having pain in his chest. He became aware of pounding when he sat still. In fact, he became acutely aware of every little blip or flip of his heart. In short, it kind of made him nuts.

After all, Ron knew for years that he had several substantial abnormalities in his cholesterol panel. Ron’s triglycerides ran as high as 700 mg/dl, HDL as low as 26 mg/dl. So when he had his first heart scan at age 53 and it showed a score of 135, neither Ron nor his doctor was surprised.

Nonetheless, such concrete evidence of potential for heart attack really shook him up. Ron’s dad had experienced his first heart attack at age 52; his second proved fatal.

I met Ron because of the chest pains. Although the statistical likelihood that a heart scan score of 135 was the culprit for his chest pain was rather small (<5%), I had Ron undergo a stress test. It proved normal with no evidence of
poor blood flow in any area of the heart. Curiously, just knowing this provided Ron with relief from his symptoms.

We proceeded to study Ron’s lipoprotein patterns more deeply. Not surprisingly, a number of important hidden factors were identified beyond the high triglycerides and low HDL, including a flagrant excess small LDL particles, zero large healthy HDL, and lipoprotein(a).

Ron initially took my advice and added high dose fish oil, sharply reduced processed carbohydrates, lost a few pounds, added niacin. But he progressively began to doubt that he would ever change the course of his future and became obsessed with the notion that he was destined to follow the footsteps of his father. He ended up stopping many of the strategies we started, regained the lost weight and more. I urged Ron to get another heart scan. Score: 350, representing 159% increase over two years, a much greater rate of plaque growth than average.

But then something happened. I’m not sure what internal dialogue Ron had, but he really started to apply himself. He resumed all the changes in lifestyle we discussed, exercised, added vitamin D, took his fish oil, reduced his weight 15 of the 25 lbs I advised, etc. One year later, yet another heart scan. This time, the score: 253, a 28% decrease.

Dr. Davis: There may be a number of important lessons Ron’s experience might teach us.

One, attitude is everything. Being optimistic can spell the difference between dropping your score hugely and watching it rise while you watch helplessly. Ron’s change in attitude was undoubtedly a crucial factor.

Two, coronary disease is, for many or perhaps most of us, a very controllable process. Once you have the right tools combined with the proper attitude, dramatic results are possible. For Ron, it was the difference between an extraordinary rate of plaque growth vs. a precipitous amount of reversal.

You can’t argue with success.

Tim T: 23% reduction
Tim’s diabetes hit him like a truck.

At age 62, Tim’s weight dropped unintentionally 25 lbs. over two months. Although he liked the weight loss, he felt awful: exhausted, even when he first got out of bed; lightheaded; and urinating large volumes. He was barely able to function in his job as CEO of a small manufacturing company.
Tim’s primary physician diagnosed diabetes and put him on two medications, one of which was Actos® (pioglitazone). Within days, Tim felt normal again, though a good deal thinner. But his doctor warned him that the episode he suffered could have become very dangerous, sometimes even resulting in a condition called “hyperosmolar coma,” a state of profound dehydration so severe it can be fatal.

Tim took the advice to heart and started taking his health more seriously. He got himself a heart scan to see if hidden heart disease was also lurking. His score: 318, in the 80th percentile compared to other men in his age group.

Tim’s primary physician put him on a statin cholesterol drug (Vytorin®). LDL cholesterol was reduced by the drug to a favorable level of 66 mg/dl, verified by a LDL particle number of 813 nmol/l (Track Your Plaque target <700 nmol/l). His lipoprotein analysis (NMR, Liposcience) on both Vytorin® and Actos® (an unusual diabetes agent in that it partially corrects lipoprotein patterns) also revealed:

• HDL 43 mg/dl, with only 3 mg/dl of effective large HDL
• Small LDL representing 80% of all LDL particles

Vitamin D supplementation increased 25-OH vitamin D3 from 22 ng/ml to 54 ng/ml.

We also counseled Tim on reducing processed carbohydrates, wheat products, and increasing reliance on proteins and fats reduce small LDL. Over time, this also resulted in reduced need for diabetes medication, since reduced carbohydrates means less need to control high blood sugars.

One year later, Tim’s heart scan was repeated. His score: 244, a 23% reduction.

**Dr. Davis:** Tim is a diabetic who beat the odds.

Conventional medical wisdom is that diabetes represents a profound risk for heart disease. Rates of plaque growth of 50% per year are not uncommon when causes remain uncorrected. Tim is an example of how this is not necessarily true if the proper steps are taken. Not only did he diminish risk, he reversed coronary atherosclerotic plaque—dramatically.

Tim’s case is somewhat more complicated in that he was taking a diabetes medication that may have influenced lipoprotein patterns. This is, of course, not a strategy that should be used by non-diabetics.

But the lesson learned from Tim’s case is that diabetes is not necessarily a death sentence from heart disease, as conventional medicine dictates. Tim’s
substantial reduction in heart scan score will translate into a profound reduction in risk for heart attack, as well as dramatically slashing the likelihood of ever needing a heart procedure.

**Dr. Sam L: 18% reduction**

Dr. Sam L. is a 61-year old physician. Sam’s father had a heart attack at age 55, so Sam had tracked his own cholesterol panel religiously, up to several times a year. His LDL ranged between 130–170 mg/dl, HDL 41–48 mg/dl. Sam prided himself on remaining slender—certainly more slender than many of his colleagues—and fit. He felt wonderful during his three time-per-week exercise efforts. He’d also asked a cardiology colleague to perform a nuclear stress test every year, and he’d consistently passed all of them.

Sam was therefore pretty shaken, even shocked, to learn of his CT heart scan score of 617. He immediately put himself on Lipitor® to reduce LDL cholesterol. On the drug, his LDL was reduced into the 85–100 mg/dl range.

After a year and a half, Sam got himself another heart scan score: 744, a 20% increase. His cardiologist colleague dismissed the result. “What difference does it make? Your stress test is still normal. I don’t think that you don’t need a cath yet [heart catheterization]. We could do one if it would help settle your mind.”

Sam wasn’t sure what to make of all this. So he took the easy way out: he pushed it out of his mind and went about his busy schedule. One year later and another heart scan: score 880, another 18% increase.

Sam sought an opinion from another cardiologist. This cardiologist reassured him and suggested another nuclear stress test. “We want to catch it in time!” he declared. That’s when Sam realized that his cardiologist friends had no insight whatsoever into how to manage his escalating score and heart attack risk. They only knew how to watch and wait for the time when procedures were justified. So Sam enrolled in the *Track Your Plaque* program.

Among the patterns Sam showed on his enrollment lipid/lipoprotein analysis (partially corrected on Lipitor®):

- HDL cholesterol 47 mg/dl (TYP target ≥60 mg/dl)
- Small LDL particles 65% of total LDL (TYP target <10% of total) LDL
- Triglycerides 96 mg/dl (TYP target ≤60 mg/dl)
- Vitamin D 30 ng/ml (TYP target 60-70 ng/ml)

We showed Sam how to raise HDL and reduce small LDL particles using diet and niacin, drop triglycerides using omega-3 fatty acids, and raise vitamin D with vitamin D supplements. He also brought his blood pressure into normal
range using a combination of nutrition, modest weight loss of 7 lbs, and one medication.

Another year later, Sam’s heart scan score: 719.9, an 18% decrease.

**Dr. Davis:** Since then, Dr. L. has become a vocal advocate for the *Track Your Plaque* approach. I suspect that he came within months of a heart attack or truly requiring a heart catheterization (that is, a procedure necessary for real reasons, not for the financial enrichment of his cardiologist friend, nor just for peace of mind).

Dr. L. is living proof of the limited effectiveness of conventional answers to heart disease prevention. Taking Lipitor® and following a “heart healthy diet” yielded 18–20% yearly increases in his score. If that were your bank account or mutual fund, you’d be hobnobbing with Bill Gates within a few years. But it’s coronary plaque. That rate of growth is simply not sustainable for any length of time without catastrophe.

The majority of physicians regard prevention of heart disease as “fluff” and not worth their effort. Procedures yield quick answers and lots of financial incentive. But that approach makes no sense, particularly when heart disease prevention can truly be powerful.

Only when he was introduced to the *Track Your Plaque* concepts did Sam manage to put a halt to his rapidly growing plaque and dramatically turn it around.

**What does this mean?**

Step back for a moment. What does reversal to the degrees enjoyed by the people in the above stories really mean? It may mean, to the properly informed, an end to heart disease.

You and I will encounter tremendous resistance to this notion. After all, testing and procedures for coronary heart disease are the number one source of revenues for hospitals in America. We are now proposing to have a solution that shuts off this considerable flow of revenue, totaling billions of dollars.

That’s how revolutions are started: An inkling that things could be different, that there may be a better answer than the one offered every day in countless offices, hospital rooms, and catheterization labs.

Seeing is believing. Let’s start a revolution.
Now that it is possible to—readily, easily, inexpensively—measure silent coronary plaque, there is no reason to wait for coronary disease to reach life-threatening proportions. No need for angioplasty. No need for stents. No need for bypass surgery or the entire array of procedures and devices that have cropped up due to the misguided advice of the last 40 years.

This is the earth-shattering revelation of the 21st century in heart health: No longer do we need to submit to unnerving, unexpected, and life-changing—life-ending?—heart attack, angioplasty, stents, bypass surgery, defibrillators. No more $100 million hospital additions, no more worrying that you Dad had a heart attack at age 51 . . . no more.

At this point I hope that you're eager to get underway and begin tracking your plaque.

But where to start? How do you assemble all the pieces of the Track Your Plaque program for maximum reduction—or elimination—of heart disease risk in your city or neighborhood?

In this brief chapter, we discuss the practical steps to create your own Track Your Plaque program.

Putting together your own program is not tough. Taken step by step, you will likely find that one step logically leads to the next. Start with a heart scan and you will begin to understand how knowing your score crystallizes awareness of
heart disease. You’ll also find that just detecting plaque is not enough. It must be precisely quantified. A heart scan score provides an understandable, measurable, “real” feel for heart disease and the potential dangers ahead, far better than any other measure like cholesterol.

Once you know your score, you will want to know why you have plaque in the first place. That’s where lipids, lipoproteins, and other measures come in. Next you will want to know what to do about your plaque to keep it from growing.

That is precisely how the logical sequence of Track Your Plaque proceeds, as well.

Can you construct a program where you live, even if you have to travel a distance to obtain the pieces? You absolutely can. The number of scanners is booming nationwide and a scan center is bound to be within driving distance of your home. The need for a heart scan is occasional and so a scanner does not have to be in your own backyard to play a role in your program.

Lipoprotein testing may also require travel to consult with a physician who can interpret results and advise you on potential treatments. The number of physicians well versed in this technology is growing. What if you are unable to find a healthcare provider able or willing to help you? Interestingly, we’ve also observed a growing number of people who have engaged in the program online, obtained lipoprotein and other testing on their own—without their healthcare provider—just using the resources like this and on the Track Your Plaque website. (That may, in fact, be the emerging paradigm for future healthcare: self-management of health, with assistance from unbiased online tools to help you navigate all the intricacies.)

Let’s get started on creating your own personal Track Your Plaque program. Just follow the 3-steps:

Step 1: Get a coronary calcium score with a heart scan
It makes no sense to pursue a program of heart disease prevention, particularly one as powerful as Track Your Plaque, unless you are truly at risk for heart disease in your future. You therefore need to start out by obtaining a heart scan score.

You can refer to Scan Directory (http://www.scandirectory.com) to locate a scanner in your area. Alternatively, unless a local hospital or clinic makes it easy by advertising their scanning services, you may have to call major hospitals or clinic in your area to ask whether they offer heart scan services. (Of course, you want a CT heart scan to obtain a coronary calcium score, not a CT coronary angiogram. Don’t be surprised if the hospital or clinic you contact tries to “upsell” you to a higher-paying CT coronary angiogram. All you need for your Track Your
Plaque program is the simple heart scan coronary calcium score: low-cost, low-radiation, unlike CT coronary angiograms. Stand firm.)

(In the previous edition of Track Your Plaque, we provided a directory of scan center locations. However, it became clear quite quickly that this was an impractical service, since scan centers change rapidly, opening and closing faster than we could keep up. Perhaps as the "market" stabilizes in future, we will be again able to provide a starting place. Until then, a little bit of work will be required to find your own scan center.)

If your score is >0, meaning coronary plaque is present, then proceed to Step 2, lipid or lipoprotein testing. If your score is 0, meaning no detectable coronary plaque is present, no immediate action is required. You can still choose to proceed onward, but the intensity of your program will not be as great, since your genetics and lifestyle have not yet succeeded in creating any measurable plaque.

Step 2: Get your lipids/lipoproteins and other measures tested

Lipoprotein testing reveals hidden causes of heart disease in 98% of people with positive heart scan scores. Unfortunately, lipoprotein testing is still relatively new and has not yet been widely adopted nor understood by most practicing physicians.

If you are interested in obtaining lipoprotein testing, start with your current doctor and ask whether he/she can provide blood drawing and interpretation services for lipoprotein analysis. Don’t be disappointed if he/she cannot, since this is the rule. If not, refer to the customer service phone numbers for the lipoprotein testing laboratories listed in Appendix A. The customer service representative for each lipoprotein testing service will be able to refer you to healthcare providers in your area who can provide both blood drawing and interpretation.

If your doctor agrees to order the blood draw but cannot provide the service him/herself, the customer service representative for the various lipoprotein testing laboratories can direct you to designated “draw sites,” sites located nationwide that will be able to draw your blood samples and prepare them properly.

The customer service numbers listed in Appendix A can help you locate a draw site. Should you choose this route, you will still need interpretation and advice for treatment based on lipoprotein testing results. The customer service representative can help you identify a physician in your area who is familiar with the technology and may be seen in consultation for advice. If you choose to use the services of a physician who is new to you, remember to first check with your insurance company to ensure that you are covered for these services (“consultation for hyperlipidemia”).
The direct-to-consumer services that can help you obtain lab tests without your healthcare provider are also listed in Appendix A.

**Step 3: Correct your lipid/lipoprotein abnormalities, add the other components**

Interpretation of your lipoprotein analysis should come with advice for treatment. Chapter 8 of *Track Your Plaque* details treatments for each specific abnormality that you and your healthcare provider can use as a reference. Refer to chapter 9 for nutritional approaches to treat lipids and lipoproteins.

Vitamin D and omega-3 fatty acids from fish oil need to be addressed. Ideally, the dose of vitamin D supplementation should be based on your initial 25-hydroxy vitamin D level. The dose of omega-3s depends on your goals, but the ideal dose of EPA + DHA from fish oil is 3000 mg per day. Don’t forget to address thyroid issues.

The entire starting laboratory panel for the *Track Your Plaque* program therefore includes:

- Advanced lipoprotein testing
- Lipoprotein(a)
- 25-hydroxy vitamin D
- TSH, free T3, free T4
- Fasting glucose, hemoglobin A1c (HbA1c)
- C-reactive protein

Those are the 3 steps of the *Track Your Plaque* program. Once you’ve successfully assembled these core pieces, you can move on to the next phase that will carry you into the future months and years.

**Monitor your progress**

Your healthcare provider can help decide whether your program is achieving the improvements in lipids or lipoproteins you desire. Conventional lipid analysis (total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides) can be used to assess progress.

When you and your healthcare provider feel that lipids have been corrected to goal, a lipoprotein analysis can be repeated to assess whether the full extent of abnormalities has been corrected. Your primary physician, the consulting physician you’ve chosen, or *Track Your Plaque* online resources can help decide whether desired endpoints have been achieved.
Repeat your heart scan score
Consider a repeat heart scan no earlier than one year after your lipids or lipoproteins have been corrected to goal.

Your second heart scan will usually be more than one year after your first heart scan, as lipids and lipoproteins don't get corrected immediately. It is not unusual to require several months, even one to two years, to fully correct lipoprotein abnormalities. For instance, if you are 80 lbs overweight at the start of your program, weight loss won't happen within a week. On the Track Your Plaque program, it develops faster than under other circumstances . . . but it still requires time.

If your heart scan score has not increased, congratulations! You simply need to continue your program for the next 50 years. (You will still need monitoring of lipids and liver function on occasion along the way.) If your score has decreased, spectacular! You have achieved what the majority of my colleagues have declared impossible: reversed coronary plaque. Just continue in your program—but don't back down. Turn your back and plaque will come back, so just continue on your program.

If there has been a substantial increase in score (>20%), you or your healthcare provider should re-examine your lipid/lipoprotein analysis and other measures to identify deficiencies in your program. Lipoprotein endpoints for treatment may need to be re-adjusted.

If your score increased 10-20%, you may just need to “fine-tune” your lipoprotein treatment modestly (e.g., aim for further reductions in small LDL, achieve higher HDL, or reduce Lp(a) further, etc.) and continue for another year, at which time another heart scan score should be considered. The process is then repeated, depending on the rate of increase of your heart scan score.

That’s the program in a nutshell. For updates, new insights, breaking research information, and practical tips on succeeding in your own personal Track Your Plaque program, please go to our website at www.trackyourplaque.com.

We also want to hear your story; your story adds to the Track Your Plaque experience that is helping to vanquish coronary heart disease from the human experience. The Track Your Plaque Member Forum is a great place to share your story and talk to other people engaged in the program.

Good Luck!
To obtain advanced lipoprotein testing, along with the other starting measures for the *Track Your Plaque* program, the best place to start is with your own family physician, internist, cardiologist, or other healthcare provider. More and more doctors have at least heard about these tests.

If your doctor is unable to provide this service, consider asking to be referred to another physician with expertise in lipoproteins. If your doctor is unaware of an expert in your area, contact the lipoprotein testing laboratory. Customer service representatives for the testing laboratories can identify practitioners in your area who can both draw the blood sample and provide interpretation. Individual practicing physicians are not listed here, but the lipoprotein testing companies below can point you towards practitioners who may be able to assist you. As another option, ask the staff at the heart scan center you use whether lipoprotein testing is offered, also, since some centers have added blood drawing and physician interpretation of lipoproteins to their scanning services.

If your physician is willing to obtain the blood sample for you but is unfamiliar with the procedure, you can refer him/her one of the resources listed below. The staff at the testing laboratories can help your physician and staff prepare the blood sample for testing. (The specimen needs to be prepared in a specific fashion.) Alternatively, you can be directed to a “draw site” in your area for the blood sample to be taken. These are general laboratories with staff who’ve received instruction on the proper procedure to follow, though they don’t actually run the test. After the blood sample is obtained and submitted to the testing laboratory, you will then need to see a healthcare provider for interpretation of these results.
If you choose to see a physician identified by one of these testing labs, you should check with your health insurance carrier first to see whether you can see this practitioner within the restrictions of your health plan. Otherwise, you might be personally liable for part or all of the expenses incurred.

For physicians interested in learning more about lipoprotein analysis and its clinical application, the resources listed below can point you in the right direction. All provide excellent educational materials and help professionals obtain the learning and experience necessary to begin applying these technologies.

**Can’t I just do it myself?**

Yes, indeed. You can now obtain advanced lipoprotein testing on your own without the assistance of your healthcare provider. You can choose to pay yourself (these services are substantially less expensive) or bill your insurance company.

Members of *Track Your Plaque* online program have used the following services with favorable results:

**DirectLabs** ([www.directlabs.com](http://www.directlabs.com))

Both NMR and VAP advanced lipoprotein panels are available. The other components of the *Track Your Plaque* program, such as 25-hydroxy vitamin D, thyroid tests, and HbA1c, are also available.

**Private MD Labs** (PrivateMDLabs.com)

NMR, VAP, and electrophoresis (“lipoprotein phenotyping”) are available, as well as all the other components of the *Track Your Plaque* program.

Listed below is contact information for the various advanced lipoprotein testing laboratories; they do not actually draw the blood sample, but perform the actual test. They can assist you in getting the blood drawn, however, if your healthcare provider is unable or unwilling to do so. Note that, while it varies, these labs also offer additional testing, such as thyroid testing, that may spare you another effort to obtain lab measures outside of lipoproteins.

**NMR Spectroscopy**

The Liposcience website can be found at [www.liposcience.com](http://www.liposcience.com). You can e-mail the company to contact your local representative, who can then steer you towards physicians using the technology in your area. Customer service can be reached via [inquiries@liposcience.com](mailto:inquiries@liposcience.com) or call 877-547-6837.
Electropheresis
The Berkeley HeartLab website can be found at www.bhlinc.com. Information on where to have blood drawn and/or health practitioners in your area who use their technology can be obtained through their customer service representative. Contact them through email at customersupport@bhlinc.com or by phone at 800-432-7889, Option 2.

Note that electrophoresis is also available through Private MD Labs (above) as a direct-to-consumer service.

VAP
Vertical Auto Profile, or VAP, is offered by Atherotech. Their customer service representatives can be reached by email at http://www.atherotech.com/contactus/?page=contactus or by phone at 800-719-9807.
Index

A
Adult Treatment Panel-3, 11, 133
Advanced glycation end-products, 144, 146, 147
Agatston score, 70, 231
Agatston, MD, Arthur, 70
Almonds. See Nuts
American Heart Association diet, 125
Heart-Check Mark program, 126, 154
policy statement on stress testing, 44
Ankle-brachial index, 61
See also LDL particle number
Atherotech, 94, 249
Atorvastatin, 105
Avocado oil
Track Your Plaque diet and, 151

B
Barnes, Dr. Broda, 189
Beans, 134
Berkeley HeartLabs, 95, 107, 119, 120, 249
Blankenhorn, Dr. David, 31
Blood pressure, 5-6, 60, 61, 113, 142, 146, 149
coenzyme Q10 and, 160
exercise effects on, 135, 213, 217
vitamin D and, 163, 167, 172
weight loss and, 135
Body fat, 214
Body mass index (BMI), 221
Boyd, PhD, Douglas, 56-57
Butter, 144, 147
Bypass surgery, 26-28

coronary calcium score and, 73-74

C
C-reactive protein, 115
calcium score and, 116
small LDL and, 116
treatment of, 116
Calcium
and atherosclerotic plaque, 19, 38, 57
coronary, 57
Calcium score. See coronary calcium score
Calorie expenditure, 224-226
Canola oil, 141
Carbohydrate reduction, 104
Carotid
intimal-medial thickness (CIMT) and coronary plaque, 60-61
ultrasound, 59
Catheterization, 45, 47, 53
coronary ultrasound and, 47, 55
Cheese, 127, 133, 138, 147, 155, 162
Cholesterol, 105-106
failure to identify silent heart disease, 51
HDL. See HDL cholesterol
heart attack and, 51-53
lipoproteins and, 94-95
LDL. See LDL cholesterol
Coconut oil, Track Your Plaque diet and, 151
Computed tomography (CT), 18, 19
64-slice, 73
calcium detection, 48
coronary angiography, 77
cost of, 77-78
electron-beam (EBT), 57, 59, 73
history of, 56-59
multidetector (MDCT), 73
plaque detection, 63
radiation exposure from, 76-77
spiral, 73
Cordain PhD, Loren, 127
Cornstarch, 134
Corn syrup, high-fructose, 104, 131, 152, 155
Coronary artery
anatomy, 36
remodeling, 39-40
Coronary bypass surgery, 73-74
Coronary calcium, 38, 56, 93, 119
Coronary calcium score, 18, 20, 48, 57, 58, 243
Agatston, 70
age and, 70
bypass grafts and, 76
CRP and, 116
cost, 77-78
heart attack prediction and, 53, 54, 56, 58, 72, 81
LDL cholesterol and, 90, 101
measure of plaque, 38, 45, 59
osteoporosis and, 160
percentile rank and, 71-72
radiation exposure and, 76-77
rate of increase, 74-75, 81
"red flags," 76
severity of blockage (stenosis severity), 58, 72
stents and, 75
stress testing and, 20, 54, 58, 74
vitamin D and, 168
reduction of, 82
Coronary calcium scoring, 20, 56, 58, 68, 70, 76, 77, 90
Coronary plaque
as coronary risk factor, 17-18, 46, 53-54
carotid intimal-medial thickness (CIMT) and, 60-61
causes of, 89-90, 92-93, 100-122
cholesterol and, 90
composition of, 37
heart attack and, 17-18, 46, 53-54
measurement, 62
MRI measurement of, 62
minor, 40
normal stress test and, 44-45
remodeling, 39
reversal, 82-84
rupture, 40, 53
silent, 45-46
ultrasound of, 47, 55
Coronary artery disease
causes of, 92-94
cost of, 28
hospital revenues and,
plaque and, 90
thyroid and, 97
Coronary Artery Surgery Study, 53
Coronary bypass surgery
coronary calcium score and, 76
cost of, 28
hospitals and, 27-29
hospital revenues and, 28
number performed in U.S., 26
COURAGE Trial, 14
Crestor®, 105, 108

D
Dairy products, 138
DHEA, lipoprotein(a) and, 114-115
Diabetes
diet and, 125, 127, 132, 140, 145
exercise and, 215
grains and, 127
inflammation and, 199-200
nuts and, 149
plaque reversal and, 238-240
processed foods and, 152
vitamin D and, 166
Diet
American Heart Association, 125-126
Atkins, 125, 128
carbohydrate reduction, 104
cholesterol and, 93
cured meats and, 116
fat content of, 129
HDL cholesterol and, 102-103
hydrogenated oils and, 128
LDL cholesterol and, 101-102
LDL particle number and, 108
linolenic acid and, 143
low-carbohydrate, 19,129
low-fat, 32, 83 125, 126, 139
Mediterranean, 125, 141, 142
monounsaturates and, 141
nuts and, 149
omega-3 fatty acids and, 142
paleolithic, 127
plaque reversal and, 82, 83, 86
polyunsaturates and, 128-129
processed foods and, 152
saturated fat and, 129-130, 145-147
small LDL and, 96, 109-110
Track Your Plaque nutrition principles and, 125-155
vegetables and, 147
vegetarian, 126-127
vitamin D and, 162-163
weight loss and, 221
whole grains and, 127, 130
Zone, 125

E
Electro-convulsive therapy, 4
Electron-beam tomography, 57, 59, 73
Electropheresis, 95, 111, 120-121, 249. See also Berkeley HeartLabs
Enduracin® 110. See also niacin.
Esselstyn, Dr. Caldwell, 32
Exercise, 213–226
   blood pressure effects, 217
   blood sugar effects, 217
   body fat measurement, 224
   blood sugar, effects on, 135
   C-reactive protein and, 116
   heart rate and, 219
   HDL, effects on, 216-217
   LDL cholesterol, effects on, 217
   LDL particle number, effects on, 217
   LDL particle size, effects on, 217
   lipid and lipoprotein effects of, 216-217
   pedometer and, 225
   Slow Burn, 225
   triglycerides, effects on, 217
   weight control, 217, 220-222
Exorphins, 136
Ezetimibe, 102, 116

F
Familial hypertriglyceridemia, 104
Fat
   hydrogenated, 101, 104, 108, 128, 139, 140-141, 145, 149, 152
   monounsaturated, 103, 141, 151
   omega-3. See Omega-3 fatty acids.
   polyunsaturated, 128-129, 139, 141
   saturated, 127, 128, 129-130, 139, 144-147
   Track Your Plaque diet and, 139-143
Fiber, 101, 102, 116, 135, 147, 149,
   soluble, 154
   wheat, 154
Fibrinogen, 94, 120, 142, 200
Fenofibrate, 105
Fish oil. See Omega-3 fatty acids.
Flavonoids, 147, 148, 152
Flaxseed, 155, 157, 162
Flaxseed oil, 105, 142, 151, 203
Food
   animal sources, 127
   appetite triggers, 131
   blood sugar and, 134-144
canned, 148, 184
carbohydrate-rich, 140
convenience, 140
“cure” for heart disease, 19
fermented, 127
frozen, 140
gluten-free, 134
habits, 154-155
processed, 104, 141, 148, 152-153
pyramid, USDA, 125-126
raw, 144
snack, 134
unprocessed, 152-153
vegetables, as perfect food, 147
Framingham
risk score, 11-12
trial, 52
Fruit, Track Your Plaque diet and, 148
Fuster, Dr. Valentin, 62

G
Garlic, 147
GISSI-Prevenzione trial, 199
Glagov phenomenon, 39
Gluten-free foods, 134
Glycemic index, 134, 152
Grains, 32, 109-110, 126, 127-128, 130, 132, 135, 140, 145
non-wheat, 138
Track Your Plaque diet and, 130, 132-138
Grimes, Dr. David, 160
Gugulipid, 102

H
Hahn, Fred, 223
HDL cholesterol, 95, 102-103
exercise effects on, 102, 216-217, 220
large, 111-112
lipoproteins and, 95
low-fat diet and, 33, 103, 126
omega-3 fatty acids and, 103
phytonutrients and, 148
saturated fat and, 145
thyroid and, 186
Track Your Plaque “Rule of 60” and, 106
unprocessed foods and, 152
vitamin D and, 102, 164, 170-171
weight loss and, 217
HDL subclasses, 111-112
Healthcare expenditures, 14

Heart attack
apoprotein B and, 107-108
as failure of prevention, 30-31
C-reactive protein and, 115
calcium and, 172
cholesterol and, 50-54
coronary calcium score and, 71-72, 81

crisis management of, 9
flavonoids and, 152
exercise and, 215
Framingham risk score and, 11-12
hypothyroidism and, 97, 190
increasing coronary calcium score and, 74-75, 81, 83-84
LDL particle number and, 107-108
lipoprotein(a) and, 112-114
lipoproteins and, 20
Mediterranean diet and, 141
mild plaque and, 45, 53
normal stress test and, 1, 3, 13, 44
nuts and, 149
omega-3 fatty acids and, 142, 198-199
Ornish program and, 32-33
percentile rank and, 72
plaque and, 1, 17-18, 19, 45-48, 53-54, 58-59, 90
plaque rupture and, 53, 93
prediction of, 11-12
saturated fat and, 129, 145
small LDL and, 108-109
statin drugs and, 108
Track Your Plaque and, 21
vitamin D and, 160, 166

Heart disease. See also heart attack.
reversal, 31-32
silent, 45-46

“Heart healthy” scams, 154
Heart rate, exercise and, 219
Heart scans. See Coronary calcium score
High-fructose corn syrup, 104, 131, 152, 155
Hodis, Dr. Howard, 60
Homocysteine, 202
hypothyroidism and, 186-187, 193, 202
Hydrogenated fat, 101, 104, 108, 128, 139, 140-141, 145, 149, 152
Hypothyroidism, 178-180, 181-193
Armour® thyroid and, 190, 192
basal body temperature and, 188-189
coronary plaque and, 190-191
diagnosis, 182-183
homocysteine and, 185-186
lipids, lipoproteins and, 185-187
lipoprotein(a) and, 187
symptoms of, 182-182
Synthroid® and, 192
Track Your Plaque approach to, 191-192

I
Inflammation. 37, 82, 84, 93, 140, 148. See also c-reactive protein
hydrogenated fats and, 140
omega-3 fatty acids and, 199-200
plaque rupture and 115
thyroid and, 183
vitamin D and, 163, 172
Inositol hexaniacinate, 110
Insulin
  advanced glycation end-products and, 146
dairy products and, 138
diet and, 134-135, 140
exercise and, 213, 217, 220
fats and, 140, 141
metabolic syndrome and, 133
niacin and, 113-114
nuts and, 149
oils and, 151
omega-3 fatty acids and, 200
phytonutrients and, 148
resistance, 113, 135, 215, 220
small LDL and, 133
unprocessed foods and, 152
vitamin D and, 96, 164, 167
weight loss and, 135
Iodine, 184-185
K
K2, vitamin, 127, 138
Koch, Dr. Robert, 4
Kondos, MD, George, 71
Krauss, MD, Ronald, 95
L
L-arginine, 167, 187
LDL cholesterol, 94, 101
  American Heart Association diet and, 125
  average, 51
  C-reactive protein and, 115-116
dietary strategies to reduce, 101-102
  exercise and, 217
  ezetimibe and, 102
  flaxseed and, 101
  Friedewald equation and, 101
gugulipid and, 102
  heart attack and, 51
  hydrogenated fats and, 140
  LDL particle number and, 107
  lipoprotein(a) and, 113
  low-fat diet and, 33
  monounsaturates and, 141
  nuts and, 101, 149
  pantetheine and, 102
  pectin and, 102
  phytonutrients and, 148
  policosanol and, 102
  polyunsaturates and, 129, 141
  red yeast rice and, 102
  saturated fat and, 129-130, 145-146
  statin agents and, 102
  thyroid and, 185-186
  Track Your Plaque diet and, 139
  Track Your Plaque "Rule of 60" and, 106
treatment goals, 101, 117, 121
unprocessed foods and, 152
wheat elimination and, 101, 110
Zetia® and, 102
LDL particle number, 106-108
exercise and, 217
LDL cholesterol and, 107, 109
saturated fat and, 146
suggested lipoprotein panel, as part of, 120
treatment of, 117
treatment goals, 212
LDL, small, 108-111, 133
atherogenicity and, 145
carbohydrates and, 109, 130, 133
genetically determined, 108-109, 110
grains and, 130, 140
niacin and, 110-111
insulin and, 133, 167
omega-3 and, 200-201, 204-205
plaque growth and, 109, 145
saturated fat and, 130
thyroid and, 110-111
triglycerides and, 109
vitamin D and, 111
VLDL and, 112
weight loss and, 109, 191, 220
wheat elimination and, 109-110, 130, 134-135
Lifestyle Heart Trial, 32
Linolenic acid, 138, 142-143, 151, 203
Lipids, 94-96, 116-118. See also LDL cholesterol and Lipoproteins
diet, gauging success, 132
exercise and, 216-217
lipoproteins vs., 94, 117-118
thyroid and, 185-186
Lipitor®, 83, 105, 108
Lipoprotein(a) 112-115
as part of lipoprotein panels, 120
coronary risk factor, 112-113
DHEA and, 114
estrogen and, 114
fish oil and, 113-114, 142, 203
foods and, 115
genetic transmission of, 113
heart attack and, 112-114
hydrogenated oils and, 140
LDL cholesterol and, 113
niacin and, 113-114
omega-3 fatty acids and, 113-114, 142, 203
saturated fat and, 129-130
testosterone and, 114
thyroid and, 114, 178, 187, 191, 193
Lipoproteins, 20, 82, 94-95
electropheresis, 95, 249
exercise effects on, 138, 216-217
fish oil, effects on, 150
hypothyroidism and, 185-187
lipids vs., 94, 116-118
low-fat diet and, 126
measurement, 94-96
nuclear magnetic resonance (NMR) of, 95, 248
omega-3 fatty acids and, 142, 200-201, 210
plaque and, 92-94
testing, how to obtain, 118-119, 247-248
Track Your Plaque suggested starting panel, 120-121, 245
treatment goals, 121-122
Vertical Auto-Profile (VAP), 95, 249
Vitamin D and, 164
Liposcience, 95, 248
Low-fat diet, 32, 83
Ornish, 31-33, 126
Lyon Heart Study, 141

M
Magnetic resonance imaging, (MRI), 62
Metabolic syndrome, 84-85, 103, 133
Adult Treatment Panel-3 definition of, 133
body fat measurement and, 224
carbohydrates and, 134
omega-3 fatty acids and, 200-201
processed foods and, 153
wheat elimination and, 134
Monounsaturated fatty acids, 103, 141, 151
Multi-detector CT (MDCT) scanners, 59
radiation exposure and, 77
Multiple Risk Factor Intervention Trial (MRFIT), 57

N
National Cholesterol Education Program, 17
LDL cholesterol and, 47
Niacin
HDL cholesterol and, 113
“hot flush” and, 110-111
immediate-release and, 110
lipoprotein(a) and, 113-115
“no-flush,” 110-111
slow-release, 110
small LDL and, 110-111
triglycerides and, 105
Nitric oxide, 148, 167, 187
NMR. See Nuclear magnetic resonance
Nuclear magnetic resonance, 95, 248
Nuts
C-reactive protein and, 135
fiber intake and, 135
LDL cholesterol and, 101
LDL particle number and, 108
linolenic acid and, 143, 203
monounsaturated fats and, 104, 141, 149
polyunsaturates and, 149
raw, 149
Track Your Plaque diet and, 148-150
Oat protein B and, 108
celiac disease and, 138
LDL particle number and, 108
small LDL and, 110
Track Your Plaque diet and, 132-133
triglycerides and, 104
Olive oil, 103, 141, 151, 155
Omega-3 fatty acids
  alternative preparations, 207-208
  arrhythmia and, 198-199
  available preparations, 204, 206-208
  blood clotting and, 200
dose, 203-205
  flaxseed oil, 105, 142, 203
  GISSI-Prevenzione trial and, 199
  HDL and, 103
  heart rhythm and, 198-199
  inflammation and, 199-200
  linolenic acid, 138, 142, 143, 151, 203
  lipoprotein(a) and, 113
  lipoproteins and, 200-201
  Lovaza® and, 205-206, 209
  mercury and, 205-206
  omega-3 index, 202-203
  plaque and, 201
  postprandial hyperlipidemias and, 201
  small LDL and, 111
  Track Your Plaque diet and, 142
  triglycerides and, 112, 198, 200-201
  VLDL and 112
Ornish diet, 31-33, 126
Ornish, MD, Dean, 31-33
Otvos, PhD, James, 95

Pantetheine, LDL cholesterol and, 102
Pectin, LDL cholesterol and, 102
Pedometers, 224-226
Percentile rank. See Coronary calcium score
Phytonutrients, 148
Pistachios. See Nuts
Plaque. See Coronary plaque
Plaque rupture. See Coronary plaque rupture
Policosanol, 102
Polyunsaturated fat, 128-129, 139, 141
Potatoes, 134

Radiation exposure, 76-77
Raggi, MD, Paolo, 54, 71
Red yeast rice 102
Reverse cholesterol transport, 93, 102, 111
Rice, 104, 110, 134, 138, 147
Ridker, MD, Paul, 115-116
S
Saturated fat, 127, 128, 129-130, 139, 144-147
Seeds, Track Your Plaque diet and, 150
Simvastatin, 108
Sloniacin®, 110. See also niacin
Slow Burn, 223
Small LDL. See LDL, small.
Smoking, 22
South Beach Diet, 70
Statin agents, 108, 197
  Adult Treatment Panel-3 and, 11-12
  C-reactive protein and, 116, 163
  heart attack and, 108
  LDL cholesterol and, 102
  plaque growth and, 82-83
  red yeast rice and, 102
  triglycerides and, 105
Stents, 6, 12, 14, 18, 27, 53, 75, 76, 197, 204, 242
Strength training, 222-224
Stress tests, 13, 17, 44, 74
  American Heart Association guidelines and, 13
  based on heart scan score, 20
  limitations, 10, 13
  normal, 13, 17, 18, 44-45
  plaque and, 18-19, 54
Superko, MD, Robert, 95
T
Taubes, Gary, 128
Testosterone, lipoprotein(a) and, 114
Thyroid, 178-193. See also hypothyroidism.
  basal body temperature and, 188-189
  coronary plaque and, 97, 190-191
  hypothyroidism, 191-192
  iodine and, 184-185
  plaque and, 97
  reverse T3, 180-181, 192
  T2, T1, 181
  T3 (triiodothyronine), 180
  T4 (thyroxine), 180
  thyroglobulin antibody, 183
  thyroid peroxidase antibody, 183
  Track Your Plaque approach to hypothyroidism, 191-192
Track Your Plaque
  5 Stages of Reversal, 82-84
  Rule of 60, 106
  study, 85
Trans fatty acids, 140, 141
Triglycerides, 103-104
  diet and, 104
  exercise effects on, 217
  high-fructose corn syrup and, 104, 152
  hypothyroidism and, 186, 191, 193
insulin and, 164
metabolic syndrome and, 200
omega-3 fatty acids and, 42, 105, 142, 200-201, 204, 210
weight loss and, 220

Tuberculosis, 4, 6

U
Ultra-fast CT. See electron-beam tomography (EBT)
Ultrasound
carotid, 59-61, 160
coronary, 45, 47, 55
Unprocessed foods, 152-153
USDA food pyramid, 125

V
Vegetables, Track Your Plaque program and, 147-148
Very low-density lipoprotein (VLDL), 112
carbohydrates and, 131
HDL cholesterol and, 102-103
omega-3 fatty acids and, 201-204
processed foods and, 152
triglycerides and, 104
Vitamin B12, 127
Vitamin C, 147, 184
Vitamin D, 2, 96-97, 159-173
anti-inflammatory effects, 163, 164, 167
blood pressure and, 163, 167
calcium and, 172
cancer and, 163
carotid plaque and, 160
cholecalciferol (D3), 165
deficiency, 162, 164-165
diabetes and, 164
dose, 165-166
endothelial response and, 167
ergocalciferol (D2), 165
food and, 169
HDL cholesterol and, 170-171
heart attack and, 160
heart disease and, 166-168
hyperparathyroidism and, 167
kidney disease and, 160-161
multiple sclerosis and, 164
osteomalacia and, 161-162
osteoporosis and, 160, 164
Recommended Daily Allowance (RDA), 162
toxicity and, 171
Track Your Plaque target level, 165
Vitamin K2, 127, 138

W
Walnuts. See nuts
Weight loss, 130-131
C-reactive protein and, 116
exercise and, 217
HDL cholesterol and, 103
inflammation and, 163-164
lipoprotein effects and, 220
oils and, 141-142, 151
pedometers and, 225
small LDL and, 109-111
strength training and, 224
thyroid and, 97, 191, 192
Track Your Plaque and, 20, 113, 130-131
triglycerides and, 217, 220
wheat elimination and, 135-136

Wheat, 130, 133-134
addiction to, 135-136
exorphins and, 136
Track Your Plaque diet and, 130, 132-138
withdrawal from, 135-136

Z
Zetia®, 102, 116
Zocor®, 108
Zone diet, 125