

Dean Ornish in Defense of the Dietary Fat – Heart Disease Link

Tricia Ward | May 12, 2016

Editor's Note: *At the American College of Cardiology Scientific Sessions, Dean Ornish, MD, clinical professor of medicine at University of California, San Francisco, and founder of the nonprofit Preventive Medicine Research Institute, presented at the session "Lifestyle Medicine: A Little Less Drug, a Little More Sex, and a Lot More Rock and Roll." In the first of two interviews, the best-selling author talks about the diet portion of the Ornish program. The second interview will focus on what he considers the least understood and one of the most important parts of his intensive cardiac rehab program—love and connection.*

theheart.org | Medscape Cardiology: There has been a backlash against the dietary fat and heart disease hypothesis. Are you sticking to your guns on the benefits of a low-fat diet?

Dr Ornish: We still have the only randomized controlled trial^[1] showing that lifestyle changes can actually reverse the progression of coronary heart disease. We showed this using radionuclide ventriculography to measure the ejection fraction response from rest and peak exercise in our study published in *JAMA* in 1983. The Lifestyle Heart trial 1-year data published in the *Lancet*,^[2] and the 5-year data published in *JAMA*,^[3] showed that the percent diameter stenosis improved in the people who went through our program, and it got worse in the control group.

We also found that cardiac PET scans^[4]—and these were all performed and blindly analyzed by independent observers—showed a 300% improvement in blood flow to the heart in the intervention group compared with the randomized control group, and they had 2.5 times fewer cardiac events.^[3] We found a dose-response correlation at both 1 year^[2] and 5 years^[3] between the degree of adherence to both the lifestyle program as a whole and the diet, in terms of cholesterol milligrams and fat grams consumed, and changes in the arteries. These things make a difference. The burden of proof is on others to show that a higher-fat diet can reverse the progression of heart disease.

That said, this is a work in progress. I have been doing work in this field for 39 years, and as new science comes, we modify accordingly. For example, when I did my internship and residency at Harvard Medical School and the Mass General Hospital, the chief of medicine at the time, Dr Alexander Leaf, was my mentor. He had done a lot of the pioneering work with fish oil^[5] and convinced me back then that we should add fish oil to our diet, which we have been doing ever since. Given the preponderance of evidence on the benefits of seeds and nuts, we have added those in small quantities this year.

theheart.org | Medscape Cardiology: What about the benefits of the Mediterranean diet seen in the PREDIMED study?^[6] The intervention diet comprised 9%-10% of calories from saturated fat and 41% from total fat.

Dr Ornish: The headlines in coverage of the PREDIMED study in the *New England Journal of Medicine* a few years ago said that the Mediterranean diet is better than a low-fat diet. It is not correct to say that. I looked at that study very carefully, and it turns out that the control group ate a diet that went from 39% to 37% of calories from fat—hardly any reduction at all—and they tended to replace fat with sugar, which is not a good idea.

When the Mediterranean diet was compared with this "low-fat" control group, there was no significant reduction in cardiovascular events; there was a huge reduction in stroke, and I think it is probably because they were advised to eat three or more servings of fatty fish per week to provide omega-3 fatty acids, which is why we have recommended fish oil, for many years. The omega-3 fatty acids in fish oil tend to inhibit thrombus formation, which is the underlying cause of 90% of strokes.

When they pooled the stroke data with the cardiovascular outcomes data, there was such a reduction of stroke that the net average came down, but if you look at the cardiovascular disease data separately, there was no significant reduction in cardiac events. Our diet is 10%-12% of calories from fat versus going from 39% to 37%. Because there is always a bias in large-scale studies for study participants to tell you what they think you want to hear, I

doubt that they even reduced their dietary fat to 37%. In all of our studies, we found a dose-response correlation with the degree of fat reduction and the degree of changes.

Now, it is entirely possible that perhaps a somewhat higher-fat diet, if it is plant-based fats, might work as well, and those are studies worth doing. If that turns out to be true, I will modify our dietary guidelines accordingly. But fat is not the whole story.

theheart.org | Medscape Cardiology: Do you think there has been too much of a nutrient-based approach to discussing diet?

Dr Ornish: Years ago at the American College of Cardiology meeting, I debated Dr Atkins. He was always the low-carb guy, so I got pegged as the low-fat guy. It has never been just about fat; it is a whole-foods, plant-based diet. It's fruits, vegetables, whole grains, legumes, soy products in their natural, unrefined form, that is low in fat, low in sugar, and low in refined carbs. It is not fat versus carbs.

Many people say, "Americans have been told to eat less fat, yet they are fatter than ever. Low fat is dead." It's true that Americans have been told to eat less fat. We have been told to do lots of things that we are not really doing. I looked at the US Department of Agriculture database,^[7] which has tracked the entire US food supply every decade since 1950. And every decade since 1950, including the current one, we have been eating more fat, more sugar, more meat, and more calories, and we are exercising less. So it's not surprising that we are fatter. It's not because we are eating too little fat but because we are eating too much of everything.

theheart.org | Medscape Cardiology: The NHANES data^[8] suggests a decrease in fat intake among US men and women since the 1970s. Are you saying that people are eating more total calories so the percentage of fat looks lower?

Dr Ornish: In the NHANES study, they are looking at a very selective group. It's about 5000 people as opposed to food consumption in the entire population, which the USDA measures. In NHANES, the decrease in the percentage of calories from fat during 1971-1991 was due to an increase in total calories consumed; absolute fat intake in grams actually increased.

It is true that in the 1980s some people went the low-fat cookie route, where they ate low-fat foods that had tons of sugar. That is not anything we ever recommended. People take this reductionistic idea that it is all fat or it is all sugar. It is not all anything. It is not even all diet. The other three components of our program are stress management, exercise, and the love and social support part. They are all important, and we have found direct, statistically significant correlations between adherence to each aspect of the program and the amount of atherosclerosis in the arteries (or whatever we happen to be measuring).

theheart.org | Medscape Cardiology: What about studies challenging the role of saturated fat, specifically, in coronary artery disease?

Dr Ornish: The *British Medical Journal* published an article^[9] saying that saturated fat is not linked with heart disease but trans fats are. I looked at that article and they present data in two ways: One is the raw data, which is the most accurate because it is not subject to bias, and the other is what they call the adjusted data. The adjusted data didn't show a significant difference, because when you adjust for cholesterol you are invariably adjusting for saturated fat; they both come from animal products.

If you actually look at the raw data in the report (they didn't even put this in the abstract) there was a highly significant correlation between the intake of saturated fat and total mortality, cardiovascular mortality, diabetes, etc.

There is a study by M.E. Levine in *Cell Metabolism*^[10] that found that animal protein is harmful, independent of the whole fat-vs-carbs issue. This may be mediated in part through changes in trimethylamine-N-oxide (TMAO) levels, which Stanley Hazen^[11] at the Cleveland Clinic is working on, and other mechanisms that we don't fully understand. People who eat a lot of animal protein have a 75% increase in premature death from all causes, a fivefold increased risk for premature death from diabetes, and a 400% increase in death from the major forms of cancer (prostate, breast, and colon cancer).^[10]

Walter Willett and his group at the Harvard School of Public Health looked at red meat consumption in an article for the *Archives of Internal Medicine*.^[12] I was invited to write the editorial; the title was "Holy Cow! What's Good for You Is Good for the Planet."^[13] Basically, they looked at more than 100,000 people from both the Physicians' Health Study and the Nurses' Health Study and found that red meat consumption was strongly and significantly correlated with increased risk for premature death from prostate, breast, colon cancer, heart disease, and diabetes.

theheart.org | Medscape Cardiology: It has been suggested that the quality of the meat is a factor.^[14]

Dr Ornish: People say, "Oh, grass-fed beef is okay." There is not a single study showing that grass-fed beef is healthier. The only study done^[15] that has even come close to that was to show that the omega-3 fatty acids were a little bit higher in grass-fed beef, but that is not the best way to get omega-3 fatty acids because you get all of this other stuff that is harmful.

theheart.org | Medscape Cardiology: In terms of the Ornish program, is the approach different if you are trying to reverse angiographically confirmed disease vs trying to lower your risk factors?

Dr Ornish: You don't have to be so intensive to prevent disease. It is the ounce of prevention or the pound of cure, and so it depends where they are. I wrote a book a few years ago called *The Spectrum*, which was based on the finding in all of our studies that the more you change, the more you improve. If you are trying to reverse a life-threatening condition, it requires bigger lifestyle changes than prevention. Part of what I have learned is that as soon as I tell somebody what to do, they want to do the opposite. When I lecture, I joke that this goes back to the first dietary intervention, when God said, "Don't eat the apple." Instead, let's ask them, "How much are you willing to change?"

If someone came to me with bad heart disease, I would say, "You could have a stent, coronary bypass, go on statins, or change your lifestyle much more than you would otherwise." If they said, "I am not interested in changing my lifestyle to that degree," I would respond, "It's your life. I am here to support whatever you choose and to make sure you are making an informed choice," prescribe statins, and consider revascularization. I have no agenda with patients. As soon as I have an agenda, first of all, they are going to push back; second, they are not going to be honest with me. The reason we find such strong correlations between adherence and outcome is because people generally tell us the truth; they know that there is no shame or guilt. We point out that if you tell us you are following the program and you are not, it is going to look like the program is not working.

Now, if you don't have a life-threatening illness, you have a spectrum of choices; it's not all or nothing. Shame, guilt, anger, and humiliation are really toxic. Calling foods "good" or "bad"—the whole language of behavioral change has this moralistic, judgmental quality. In my book, foods are just foods, but some are healthier than others. I categorize them from the most healthy (what I call group 1) to the least healthful (group 5), and what matters most is your overall way of eating and living. If you indulge yourself one day, that is okay. It doesn't mean that you cheated or that you are bad; just eat healthier the next day. Whatever you do, there is a corresponding benefit.

Let's say you want to lose 10 lb or lower your LDL-C by 50 points—instead of saying, "Here is your diet," I ask, "How much are you willing to change?" The response may be, "I don't know. I will eat less of the unhealthy group 4 and 5 foods and a little more of the healthier groups." Then ask, "How much exercise are you doing? How much are you willing to do?" You may hear, "Oh, no one has ever asked me that." We support the degree of change that they are willing to do. We will track it and check back in a few weeks. If that degree of change was enough to accomplish their goals, that's all they may need to do.

If they wanted to lose 10 lb and lost 5 lb, I say, "Look at what great progress you are making. Now just go a little farther and you will get the rest of the way." It is a much more compassionate approach and a much more sustainable one.

All of which is a longwinded answer to a very simple question.

theheart.org | Medscape Cardiology: In terms of the dose-response correlation, do you see all components of the Ornish program as important?

Dr Ornish: They are all important. We have found direct statistically significant correlations between adherence to each aspect of the program and the amount of atherosclerosis in the arteries, or whatever we happen to be measuring. We found the same dose-response correlation between adherence and the degree of change not only in heart diseases but also in prostate cancer,^[16] in gene expression,^[17] and in telomere length^[18] (the ends of our chromosomes that control aging).

It was the same lifestyle intervention, and the more people changed, the better they got at any age. We found an average reduction in LDL-C of 40% in the Lifestyle Heart Trial without drugs. In fact, none of these patients was taking cholesterol-lowering drugs during the 5 years of the intervention in the experimental group.

We know that the program works.

theheart.org | Medscape Cardiology: A criticism of the Lifestyle Heart Trial is that it was very small.

Dr Ornish: The critics say, "You only had 48 patients." What they don't understand is that in science, the whole point is the likelihood of this being a real finding versus a chance finding. Through convention, if there is less than a 5% likelihood that it is due to chance, then it is probably a real finding (a *P* value of $\leq .05$). If you have only 48 patients, then that means you have to have a really powerful intervention and you have to have very accurate and reproducible measures in order for it to be significant; otherwise, you get so much noise that you can't tell much.

Attilio Maseri,^[19] who was one of the pioneering cardiologists who discovered coronary artery spasm, would say that if you need 1000 people to show a significant difference, then that means that the intervention must not be very strong. If you were looking at the effects of penicillin on pneumococcal pneumonia in the 1950s, you don't need 1000 people because the intervention is so strong. You give it to one group and not the other—48 people would be plenty. These people all got better and these people didn't.

We found that the control group got worse after 1 year and their arteries were even more clogged after 5 years, whereas the experimental group showed some reversal after 1 year and even more reversal after 5 years as measured by quantitative coronary arteriography. For the blood-flow analysis measured by cardiac PET scans, because perfusion is a fourth-power function of the diameter, it is an exponential improvement in blood flow. Small changes in blockages can cause big improvements in blood flow. As I mentioned, over 99% of the patients stopped or reversed their heart disease as measured by PET scans. That is pretty good. Only 5% of the control group got better—not subtle.

We published data from almost 3000 patients who went through our program in 24 sites and found significant improvements in all metrics after 12 weeks and after 1 year. Adherence to our program has been more than 85% after 1 year in all sites we've trained.^[20]

theheart.org | Medscape Cardiology: Do you think diets could be personalized on the basis of an individual's makeup or family history?

Dr Ornish: I was on Craig Venter's board of his nonprofit for many years, so I am pretty up to speed on a lot of the genomics work, and it is not really there yet. It may be one day. If you have pancreatic cancer and you are trying to personalize an immunotherapy for a particular cell type, then that is a very different thing. That is brilliant work. In terms of diet and lifestyle, as I mentioned, it wasn't like there was one set of lifestyle recommendations for reversing heart disease and a different one for diabetes or prostate cancer, your genes, or your telomeres; it was the same for all of them.

Our genes are a predisposition, but our genes are not always our fate. We found that comprehensive lifestyle changes caused changes in gene expression in over 500 genes in only 3 months.

I visited one of our sites at Beacon Hospital. I met a guy who was on the heart transplant list due to an ischemic cardiomyopathy. After 9 weeks on our program, his ejection fraction improved so much that he doesn't need a heart transplant. We have had several people like that.

It is almost as if when you give your body the right raw ingredients, it can extract or personalize what it needs in

order to be able to heal in direct proportion to the degree of change that people make.

theheart.org | Medscape Cardiology: In 2014, you were a signatory to a commentary in the *American Journal of Medicine* criticizing the deficiency of nutrition education in medical training,^[21] but even physicians who want to learn about nutrition get frustrated with the seemingly flip-flop headlines: Something is good for you one day and bad for you the next.

Dr Ornish: I think that is because the media like controversy. That is why I spent so much of the past 39 years doing science—because the whole point of science is to figure out what is true and what isn't, what works and what doesn't, and for whom and under what circumstances. There are certain standards and there are certain statistical norms.

The Mark Hyman and Gary Taubes should know better. We have published in the *Lancet*, the *New England Journal of Medicine*, *Circulation*, *Lancet Oncology*, *Proceedings of the National Academy of Sciences*, and *JAMA*, and we presented at meetings like the American College of Cardiology and the American Heart Association numbers of times. They have not.

For example, in his new book, Mark Hyman quoted Kevin Hall's NIH metabolic ward study,^[22] basically parroting Gary Taubes and saying that all calories are not alike, that somehow carbohydrate calories are making you fatter than fat calories. The metabolic ward study found that calorie for calorie, they are not all alike, but in the opposite way: They found that people lost 67% more weight by restricting fat than by restricting carbs. Yet Mark misquotes this study to support his belief that, calorie for calorie, restricting carbs causes more weight loss than restricting fat, yet the conclusion of the study was just the opposite. In any event, both are important, which is why I have always recommended reducing the intake of fat and refined carbohydrates.

In the metabolic ward study they were controlling every calorie that the participants ate. Part of the problem in a lot of these studies, like the A to Z study,^[23] is that there is 25%-30% adherence to the diets. You can't really say much when people aren't following the diet.

Physicians should ask, "What are the facts? What are the data?" No one has ever done a randomized trial showing that a high-fat, low-carb diet can reverse heart disease. It is not enough to say that people lose more weight on a low-carb versus a low-fat diet. You can lose weight in lots of ways that aren't very good for you; smoking cigarettes is a really good way to lose weight, and chemotherapy is a good way to lose weight, but I don't recommend it. The question is, what is happening in the arteries? In the *New England Journal*,^[24] Steven Smith showed that on a whole-foods, low-fat, plant-based diet that is also low in carbs, the arteries are essentially clean vs with your typical American diet or an Atkins-type, Paleo-type, Mark Hyman-, Nina Teicholz, Gary Taubes-type diet where the arteries are severely clogged. While this is in mice,^[25] it's likely true in humans in well. For example, one study found higher flow-mediated vasodilation in those following our dietary recommendations compared with those on an Atkins diet.^[26]

Gary Foster^[27] and others have done studies where they just look at risk factors. They will look at weight or blood pressure and say, "Well, the LDL-C goes down lower in the Ornish diet, but the HDL-C goes up more on an Atkins diet. Tell your patients to eat what they like."

First of all, the fact that HDL-C goes up on an Atkins diet is not necessarily a good thing. We used to have this very simplistic view that HDL-C is good and LDL-C is bad, but several studies have shown that raising HDL-C actually increased cardiac events and increased premature mortality.^[28] Not everything that raises your HDL-C is good and not everything that lowers it is bad. HDL is part of the reverse cholesterol transport mechanism, and it is like garbage men: If you are eating a lot of fat and cholesterol—"garbage"—(as most Americans are), your body has to make more garbage men to get rid of it. But when you reduce the amount of dietary fat and cholesterol by going on a really healthy diet, it is almost as if your body says, "Hey, not as much garbage, don't need to make as many garbage men." It has a very different prognostic significance. We know that because in our studies, the HDL-C did go down a little bit, but the LDL-C went down way more and these patients reversed the progression of their coronary heart disease.

But HDL and LDL are not diseases. The disease is what is going on in your arteries and your blood flow, and that is what we actually measured. What they found [in the Foo study] is that it wasn't always mediated through traditional risk factors like LDL, weight, or blood pressure. There are nontraditional risk factors like endothelial-derived growth factors, nonesterified fatty acids, and things that most people have never heard of that play an important role, like TMAO [trimethylamine N-oxide].

Show me some randomized trial data that the Atkins, Paleo, or whatever kind of diet can reverse heart disease—not just risk factors, but the actual measures of the underlying disease process—and my colleagues and I will modify our recommendations.

theheart.org | Medscape Cardiology: How do you address physician inertia against lifestyle changes when it's easier to prescribe a drug or a procedure?

Dr Ornish: We were trained to use drugs and surgery. We are reimbursed to use drugs and surgery, and so we use drugs and surgery. Through my nonprofit 501(c)(3), beginning in 1993, we trained 53 hospitals and clinics around the country to show that it was scalable. We trained sites around the country, achieved bigger changes in lifestyle and better clinical outcomes,^[20] but some of the sites closed because we didn't have reimbursement. That set me off on a 16-year journey to ask Medicare to pay for this program, which they are now doing in a new benefit category: intensive cardiac rehabilitation.

We train a team of six people: a doctor, a nurse, a stress-management specialist, an exercise physiologist, a dietician, and a psychologist. Medicare will pay for 72 hours of training, which we divide into 18 four-hour sessions. Patients get an hour of supervised exercise, an hour of stress management, an hour of group support, and an hour-long lecture followed by a group meal. CMS is reimbursing this at a high enough level (\$107 an hour) that it becomes financially sustainable.

I learned that no matter how good the science is, if it is not reimbursable, it is not sustainable. If people are interested in learning more about this, they should go to our website, which is ornish.com. Everything on there is free, and there is information on how they can contact us. We are ramping up to train as many people as we can.

Disclosures: Dean Ornish, MD, has disclosed the following relevant financial relationships:

Receives consultancy fees/honoraria from: Healthways, is the author of general-interest books on health, and is paid to lecture on health.

Editor's Note: This article has been updated with corrections and clarifications from Dr Ornish.

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References

1. Ornish D, Scherwitz LW, Doody RS, et al. Effects of stress management training and dietary changes in treating ischemic heart disease. *JAMA*. 1983;249:54-59. [Abstract](#)
2. Ornish D, Brown SE, Scherwitz LW, et al. Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. *Lancet*. 1990;336:129-133. [Abstract](#)
3. Ornish D, Scherwitz LW, Billings JH, et al. Intensive lifestyle changes for reversal of coronary heart disease. *JAMA*. 1998;280:2001-2007. [Abstract](#)
4. Gould KL, Ornish D, Scherwitz L, et al. Changes in myocardial perfusion abnormalities by positron emission tomography after long-term, intense risk factor modification. *JAMA*. 1995;274:894-901. [Abstract](#)
5. Leaf A. Cardiovascular effects of fish oils. Beyond the platelet. *Circulation*. 1990;82:624-628. [Abstract](#)

6. Estruch R, Martinez-Gonzalez MA, Corella D, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. *Ann Intern Med.* 2006;145:1-11. [Abstract](#)
7. United States Department of Agriculture Office of Communications Agriculture Fact Book 2001-2002.
8. CDC. Trends in intake of energy and macronutrients--United States, 1971-2000. *MMWR Morb Mortal Wkly Rep.* 2004;53:80-82. [Abstract](#)
9. de Souza RJ, Mente A, Maroleanu A, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ.* 2015;351:h3978.
10. Levine ME, Suarez JA, Brandhorst S, et al. Low protein intake is associated with a major reduction in IGF-1, cancer, and overall mortality in the 65 and younger but not older population. *Cell Metab.* 2014;19:407-417. [Abstract](#)
11. Tang WHW, Wang Z, Levison BS, et al. Intestinal microbial metabolism of phosphatidylcholine and cardiovascular risk. *N Engl J Med.* 2013;368:1575-1584. [Abstract](#)
12. Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and mortality: results from 2 prospective cohort studies. *Arch Intern Med.* 2012;172:555-563. [Abstract](#)
13. Ornish D. Holy Cow! What's good for you is good for our planet: comment on "Red Meat Consumption and Mortality." *Arch Intern Med.* 2012;172:563-564.
14. Kaluza J, Akesson A, Wolk A. Processed and unprocessed red meat consumption and risk of heart failure: prospective study of men. *Circ Heart Fail.* 2014;7:552-557. [Abstract](#)
15. McAfee AJ, McSorley EM, Cuskelly GJ, et al. Red meat from animals offered a grass diet increases plasma and platelet n-3 PUFA in healthy consumers. *Br J Nutr.* 2011;105:80-89. [Abstract](#)
16. Ornish D, Weidner G, Fair WR, et al. Intensive lifestyle changes may affect the progression of prostate cancer. *J Urol.* 2005;174:1065-1069; discussion 1069-1070.
17. Ornish D, Magbanua MJ, Weidner G, et al. Changes in prostate gene expression in men undergoing an intensive nutrition and lifestyle intervention. *Proc Natl Acad Sci U S A.* 2008;105:8369-8374. [Abstract](#)
18. Ornish D, Lin J, Chan JM, et al. Effect of comprehensive lifestyle changes on telomerase activity and telomere length in men with biopsy-proven low-risk prostate cancer: 5-year follow-up of a descriptive pilot study. *Lancet Oncol.* 2013;14:1112-1120. [Abstract](#)
19. Maseri A, Cianflone D, Paceri V, Crea F. The risk and cost-effective individual patient management: the challenge of a new generation of clinical trials. *Cardiovasc Drugs Ther.* 1997;10:751-758. [Abstract](#)
20. Silberman A, Banthia R, Estay IS, et al. The effectiveness and efficacy of an intensive cardiac rehabilitation program in 24 sites. *Am J Health Promot.* 2010;24:260-266. [Abstract](#)
21. Devries S, Dalen JE, Eisenberg DM, et al. A deficiency of nutrition education in medical training. *Am J Med.* 2014;127:804-806. [Abstract](#)
22. Hall KD, Bemis T, Brychta R, et al. Calorie for calorie, dietary fat restriction results in more body fat loss than carbohydrate restriction in people with obesity. *Cell Metab.* 2015;22:427-436. [Abstract](#)
23. Gardner CD, Kiazand A, Alhassan S, et al. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial. *JAMA.* 2007;297:969-977. [Abstract](#)

24. Smith SR. A look at the low-carbohydrate diet. *N Engl J Med*. 2009;361:2286-2288. [Abstract](#)
25. Foo SY, Heller ER, Wykrzykowska J, et al. Vascular effects of a low-carbohydrate high-protein diet. *Proceedings of the National Academy of Sciences*. 2009;106:15418-15423.
26. Miller M, Beach V, Sorkin JD, et al. Comparative effects of three popular diets on lipids, endothelial function, and C-reactive protein during weight maintenance. *J Am Diet Assoc*. 2009;109:713-717. [Abstract](#)
27. Foster GD, Wyatt HR, Hill JO, et al. Weight and metabolic outcomes after 2 years on a low-carbohydrate versus low-fat diet: a randomized trial. *Ann Intern Med*. 2010;153:147-157. [Abstract](#)
28. Barter PJ, Caulfield M, Eriksson M, et al. Effects of torcetrapib in patients at high risk for coronary events. *N Engl J Med*. 2007;357:2109-2122. [Abstract](#)

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