



# communicating Food for Health

## For Heart Attack Prevention, is Diet More Important than Statins?

If elevated low density lipoprotein cholesterol (LDL-C) levels were the only source of cholesterol deposited in the artery wall, then high doses of potent statins should be reversing (rather than reducing) the build-up of atherosclerotic plaques, largely eliminating deaths from coronary heart disease (CAD). Sadly the number one cause of death in Americans taking statins to lower their elevated LDL-C to prevent heart attacks is still heart attacks!

Yes, statin drugs are very effective for reducing high LDL-C levels, and they do slow the progression of cholesterol-filled plaques. However, they rarely reverse the build-up of cholesterol in the artery wall. More importantly, statin drugs alone do not come close to eliminating the risk of heart attacks and most strokes despite impressive reductions in LDL-C levels. Research now shows that other lipoproteins besides LDL particles can and do carry

cholesterol from the blood into the artery wall, promoting the growth of cholesterol-filled plaques and CAD. These lipoproteins are neither LDL-C or high density lipoprotein cholesterol (HDL-C), but rather consist of the cholesterol-rich remnants of triglyceride-rich lipoproteins produced by the liver (VLDL) and the small intestine (chylomicrons)(1). Both genetic factors and dietary factors influence the amount of these triglyceride-rich lipoproteins produced and also the amount of cholesterol-rich remnant particles derived from each of them in the blood. Fat and cholesterol-rich meals can dramatically increase the production of chylomicrons and lead to greater amounts of cholesterol-rich chylomicron remnants in the blood for several hours after each fat-rich meal (2).

Dr. Borge Nordestgaard's recent study followed nearly 12,000 people with established CAD in Denmark and found

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November '13

*Professional Member Edition*

### Research

Dr. James J. Kenney explores heart attack and statin research.

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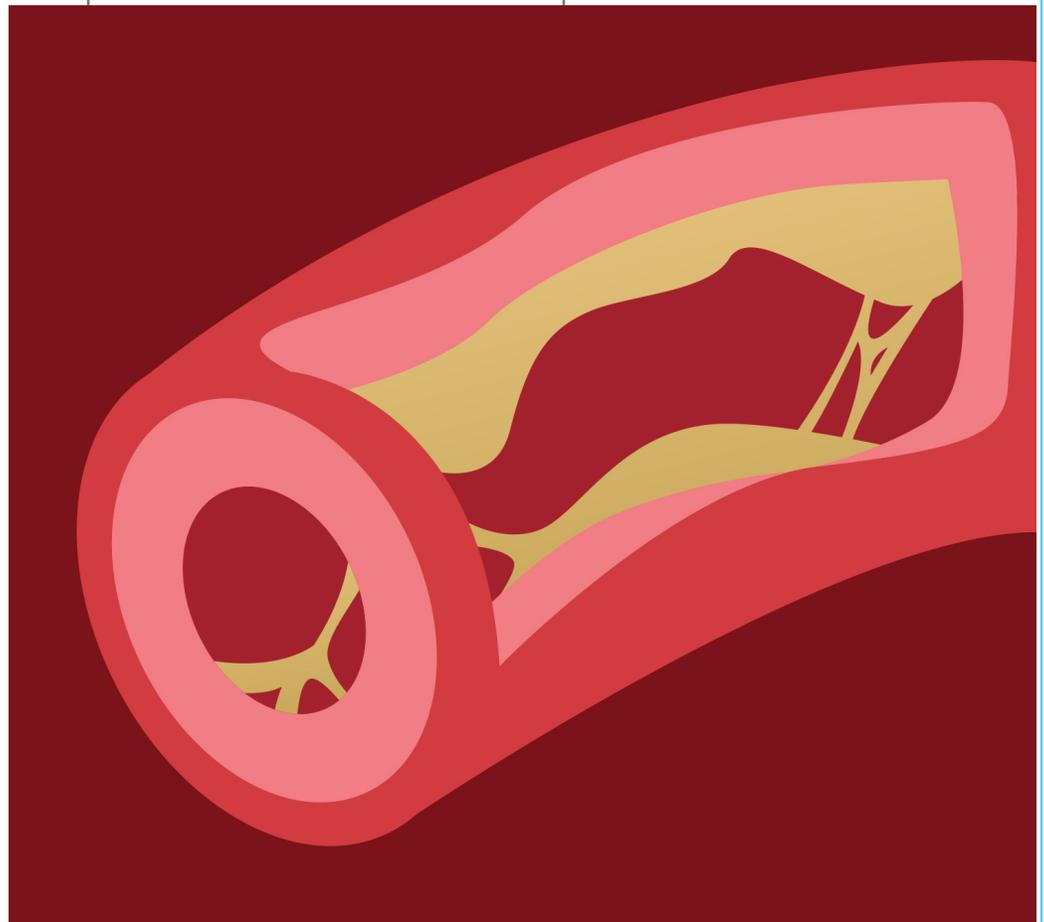
*Of course, we now know HDL-C particles can actually become proinflammatory and proatherogenic "bad" HDL particles.*

that each 1 mmol (38.7 mg/dl) increase in non-fasting remnant cholesterol caused 2.8 times greater risk of a CAD event that was independent of HDL-C levels. The increased causal risk of CAD from elevated cholesterol remnant particles appeared much stronger than for changes in either LDL-C or HDL-C levels (3). Most doctors (MDs) now check only fasting blood lipids and focus largely on LDL-C and HDL-C to assess their patient's future CAD risk. This was based on the simplistic notion that it was only the LDL-C particles delivering cholesterol to the artery wall, making it the "bad" cholesterol, while the HDL-C particles were removing the cholesterol from the artery wall and bringing it back to the liver, making their cholesterol content "good". Of course, we now know HDL-C particles can actually become proinflammatory and proatherogenic "bad" HDL particles, perhaps partly in response to biochemical changes in the HDL particles

triggered in part by chylomicrons and other remnant cholesterol particles in the blood.

Chylomicrons and their cholesterol-rich remnants remain in the blood for several hours after each fat-rich meal and likely play a major role in promoting inflammation (by increasing IL-6 & CRP), thrombosis (by activating clotting factor VII), and atherosclerosis (by delivering more cholesterol-rich remnant particles to the ar-

tery wall). The fact that damage to the endothelium (inside "skin" of the artery wall) as evidenced by reduced flow mediated dilation (FMD) occurs to a much greater extent after a single fat-rich meal than after a meal high in carbohydrate points to the fact that pathological changes must be occurring in the artery wall in response to fat and cholesterol-rich particles coming from the intestines (4). Indeed, this reduced FMD is likely the



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*Reducing LDL-C levels with statin drugs alone is insufficient for stopping and reversing CAD and preventing most heart attacks and strokes.*

main reason why many people with angina tend to experience far more chest pain after a large, fat-rich meal than they do after a meal high in carbohydrate-rich plant foods. The only legitimate debate is not whether LDL-C or other cholesterol-rich remnant particles promote atherosclerosis and increase the risk of CAD, but rather which is more atherogenic. Clearly both LDL-C and other remnant lipoprotein particles deliver cholesterol to the artery wall and promote foam cell formation and atherosclerosis. Unlike LDL-C particles (which must first be oxidized), remnant cholesterol particles are readily taken up by scavenger receptors of macrophages in the cell wall to form foam cells (5,6). Increasing evidence suggests that damage to the artery wall from cholesterol-rich remnant particles appears to be at least as important as either fasting LDL-C or HDL-C levels for predicting future CAD events.

It should be noted that diets high in refined carbohydrates (particularly large amounts of refined sugars) combined with inactivity can contribute to a marked increase in the liver's production of VLDL particles because the liver converts some of the excess carbohy-

drate (especially fructose) into triglyceride. This leads to more triglyceride-rich VLDL particles being released into the blood, which then degrade into cholesterol-rich remnant particles and eventually also LDL particles. This is particularly true in people who are genetically prone to develop insulin resistance and type 2 diabetes and who experience significant increases in fasting triglyceride levels as visceral fat stores accumulate.

**Bottom Line:** Reducing LDL-C levels with statin drugs alone is insufficient for stopping and reversing CAD and preventing most heart attacks and strokes. A diet low in fat, salt, cholesterol, and refined carbohydrates coupled with increased activity and loss of excess weight may also be necessary to stop and reverse CAD in part by reducing remnant cholesterol levels in the blood.

*By James J. Kenney, PhD, FACN*

**Sources:**

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## Insulin Resistance Dodgeball

For this activity, you will need a basket or tub, foam balls or bean bags in two distinct color sets, a blindfold for each participant, and a space that is safe to use for flying objects (i.e. without delicate, easily breakable items).

Divide participants into two even teams. Give each team one set of the balls/bean bags. Each team should have a different color set. Explain that this activity involves them throwing their balls/bean bags into a central basket. The projectiles represent glucose, one of the body's main energy sources, the basket represents the body, and the players represent insulin, which transports glucose into the body's cells. (Whether or not you explain the symbolism at the beginning is up to you -- you can lay it all out right away or use it as a

great "reveal" during your subsequent discussions).

Teams will have one minute to throw as many of their balls into the basket as they can. Once the time is up, the round will end. The team with the most balls in the basket wins. Pretty straightforward, right? What's happening here is a representation of typical body function. Participants are acting out the regular role of insulin – transporting glucose into the body's cells.

Have teams return to their starting spots, and pass out the blindfolds. Explain that this time they are going to do the same thing, only blindfolded. Have everyone assist each other in putting on blindfolds, checking to make sure that it isn't easy to peek. Repeat the activity for one minute, then tally the number of balls in the basket. Explain that the number of balls in the basket is

much lower this time, with many more balls lying discarded around the room. Tally points, announce a winner, and have participants return to their seats.

Once everyone is sitting down, discuss the concept of insulin resistance and its relationship to diabetes. Have participants volunteer their guesses about the game they just played and its connection to insulin resistance. Explain the connection to insulin resistance and answer any questions that may arise.

*This presentation idea is an excerpt from our brand-new educational program, [The 12 Lessons of Diabetes](#). This program follows the same format as the other fantastic [12 Lessons packages](#) and is chock-full of insight from a wide variety of health and nutrition professionals.*

### Communicating Food for Health

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