

# Carnitine in the crosshairs: A fishy tale?

Lately I've been bombarded with inquiries from anxious patients, listeners and Facebook friends about a highly-publicized story suggesting that carnitine, a popular nutritional supplement, might be harmful.

CBS news trumpeted: "Carnitine Chemical, Not Fat, May Explain Link Between Red Meat and Heart Disease."

Business Insider reports "Researchers Have Found What Makes Steak Unhealthy." And MSN declaims: "Red Meat Is Bad for You, and It's All This Chemical's Fault." And so, in one fell swoop, science and the media have struck a blow against easy targets: greedy, planet-devouring meat-eaters and gullible consumers of nutritional supplements. As stories go, it's a real grabber. I'm all for being vigilant about unanticipated side effects of nutritional supplements we've come to rely on. Occasionally, we need to reign in our enthusiasm if science proves us wrong. But several things about this story strike me as, frankly, kind of fishy. First of all, and what's being missed in all the brouhaha, is the implicit admission that conventional medicine has been wrong all along about dietary saturated fat and cholesterol. Recent studies just don't support the old saw that they're responsible for heart disease. I think that represents significant progress toward understanding the true origins of heart disease. So, this new study represents a feeble attempt to perpetuate the "Meat is Bad for You" hypotheses—but with a new villain. The culprit is said to be TMAO (trimethylamine oxide—not to be confused with LMAO, which some people like to use frequently as an abbreviated comment on a funny text message or Facebook post). TMAO is thought to trigger the activity of macrophages, a type of white blood cell defender that releases harmful free radicals within the arterial wall, contributing to atherosclerotic plaque development. The funny thing about TMAO is that substances other than carnitine are its precursors. Fish is rich in choline, a step away from trimethylamine, which oxidizes to TMAO, giving spoiled fish its fishy odor. Eggs and soy and lecithin also are sources of choline. So therefore, if the new theory holds water, it should be fish, eggs and soy that increase heart disease risk. In fact, they don't. The Cleveland Clinic researchers speculate that carnitine, an ingredient uniquely found in red meat (as in carnivore, chili con carne, etc.), may act as a kind of Miracle-Gro for intestinal bacteria that oxidize trimethylamine into dangerous TMAO. But here's where it gets complicated. Feeding human vegetarians carnitine DOES NOT result in excess TMAO. Feeding carnitine to meat-eaters does. (Head scratch!) The researchers speculate that ongoing meat consumption conditions the intestinal bacteria to make more harmful TMAO. If that's the case, then it's not the meat, or the carnitine, but the bad bacteria that are at fault. Is it possible that plentiful plant food or fiber could suppress these harmful bacteria, even in the presence of dietary carnitine or red meat? Alternatively, could probiotics compete with the TMAO-making bacteria, neutralizing the effects of carnitine or red meat? Instead, the researchers seem to draw the hasty conclusion that we should consider banning carnitine and curtail our consumption of red meat, a conclusion they have gleefully shared in some unduly alarmist print, radio and TV interviews. What happened to proper scientific skepticism in their rush to judgement? Are these normally staid researchers blinded by the light of all the media attention? It's a far leap from the lab bench to real life. NO studies have ever demonstrated a propensity to heart disease in human consumers of L-carnitine—this is pure hypothesis and supposition at this point, based on a hypotheses, admittedly an intriguing one. The experimental animal in question—rats—are neither carnivores nor habitual consumers of carnitine, and their

metabolism is not likely to mirror that of humans. The evidence is circumstantial. Carnitine, in and of itself, is an important amino acid that shuttles fatty acids to the mitochondria (the powerhouse!) of the cell. This enhances our fat-burning metabolism. Anthropological records show that early prehistoric humans ate plentiful red meat, yet heart disease is a modern epidemic. In fact, ancient medical textbooks rarely describe the classic symptoms of heart attacks. In the 1800s, when Americans consumed massive amounts of pasture-raised beef, pork and lamb, and before the introduction of refined grains and massive amounts of sugar in the 20th century, heart disease was an anomaly. People died of tuberculosis, fevers, pneumonia, alcoholism, syphilis and accidents, but there were far fewer cancers and degenerative diseases. Additionally, studies that show a correlation between red meat consumption and heart disease are plagued with methodological problems. Part of the difficulty lies with the fact that many studies of meat eaters include poor-quality processed meats such as hot dogs, luncheon meat and nitrite-laced bacon. Also, many meat eaters in the studies in question ate fewer fruits and vegetables and unrefined grain products than their vegetarian counterparts. No proponent of red meat consumption, least of all me, suggests that you eat it to the exclusion of healthy fruits and vegetables. It's unclear whether a study of people eating organic, pasture-fed meats without preservatives or antibiotic residues along with plentiful fresh polyphenol-rich plant foods would confirm the heart risks seen with less discriminating meat eaters. I offer myself as an experimental subject!

Statistical analysis, by the admission of the Cleveland Clinic researchers, shows that at most, only 11 percent of the variation in heart disease risk could be explained by the carnitine connection. Is this important enough to warrant telling people to stop eating red meat or taking carnitine supplements?

Studies of carnitine metabolism in humans show that TMAO builds up to potentially harmful levels only in consumers of greater than 2 grams per day of carnitine. I ordinarily prescribe 1 or 2 grams per day, which should have negligible effects on heart disease risk, even if the Cleveland Clinic researchers were to be right.

Don't get me wrong: I think that the inclusion of carnitine in energy drinks with sugar, caffeine and all manner of harmful additives is window dressing at best, leading consumers to believe that they're getting some kind of health benefit from what is essentially a sucrose-infused version of old-fashioned caffeine-laced NoDoz pills. If you want a supplement, go buy a good quality supplement and don't hope to improve your health with shots of "Monster" energy beverage.

And if you want a jolt of caffeine, get the documented health benefits of green tea; even coffee is making a comeback with proven protection against diabetes, liver disease and Alzheimer's.

Carnitine is a useful nutrient, with considerable scientific research backing its safety and health properties.

I have found it to be an energizer for patients with chronic fatigue, especially when they're on skimpy vegetarian diets. Studies show it's a great antidote for cancer fatigue, particularly when patients get radiation or chemotherapy.

Teamed with coenzyme Q 10 or Ubiquinol, it can help the pumping action of the heart in patients with congestive heart failure.

Patients with kidney disease seem to "leak" carnitine, and their levels often are very low. They feel considerably energized when supplemented with carnitine.

A form of carnitine called acetyl-L-carnitine seems to have a particular affinity

for the nerves and brain. That's why studies have shown it to alleviate peripheral neuropathy and mild dementia.

Research on carnitine with regard to exercise performance has come up mixed. Some athletes feel it offers them a performance boost, while others consider its effects negligible.

Before we accept the conclusions of this initial study suggesting harm from carnitine, more research needs to be undertaken. I'll keep you posted.

In the meantime, take a look at the latest [here](#).