The Benefits of Carnitine and DHEA for Fat Metabolism

People want energy efficiency in their homes; they need it for their bodies as well. Carnitine is an amino acid that’s critical for converting fatty acids into fuel. Because of this, it has the potential to help people lose weight. Muscles, liver, kidney and heart all require carnitine to convert fat into energy. If carnitine is not available, fatty acids will accumulate in the body. Japanese researchers have demonstrated the concept using high-fat diets. Normally this type of diet elevates cholesterol and triglyceride levels. But adding carnitine reverses the effect. Carnitine works by transporting fatty acids into mitochondria where they are used for energy production. Where fat goes, carnitine must follow.
Fats are one of three sources of energy the body utilizes. Muscles, including the heart, plus the kidney, and the liver depend heavily on fatty acids for fuel. Carnitine functions as a carrier system for fatty acids, shuffling them into mitochondria so they can be oxidized. The "carnitine shuttle" is well-known in biochemistry where it’s recognized as critical for the metabolism of fatty acids. The shuttle relies on several carnitine-related enzymes to work. If any of them are nonfunctional, a build-up of fatty acids in the blood and organs will occur, as the shuttle screeches to a halt. This problem has been documented in people with insulin resistance. Studies show that defective carnitine-related enzymes are involved in this condition. But although dysfunctional enzymes would suggest something genetic, it’s not necessarily the case. Some research suggests that supplemental carnitine may get the enzymes working again.

In addition to insulin resistance, obesity, fatty blood, fatty liver and diabetes also involve abnormal fatty acid utilization. As a consequence, energy production falters, and fatty acids build up in the body. What can be done to get at this problem? There are two supplements and a stack of scientific papers that may provide answers.

**Carnitine lowers lipids**

The effect of carnitine on lipids is dramatically illustrated by experiments on cats. Felines quickly develop a life-threatening condition called “hepatic lipidosis” if they don’t eat. Although it sounds paradoxical, not eating causes fat to build up in the liver. With it, the mitochondria begin to die off, and the energy system collapses. The break-down in metabolism occurs, at least in part, because of insufficient carnitine, which is critical for fatty acid utilization. Without carnitine, which comes from protein, fatty acids cannot be moved into mitochondria where they are used for fuel. Force-feeding is the treatment for this condition. Once protein (a source of carnitine) gets back in the system, lipid levels fall, and the condition reverses itself.

**Weight loss**

Because carnitine moves fatty acids into mitochondria for fuel, it has potential as a weight-loss supplement. Its beneficial effects have been demonstrated in cats where it promotes faster weight loss during dieting. However, a study in humans on an aerobic exercise program showed no results. One of the reasons may be that carnitine’s ability to energize lipids depends on other factors. Hormones are one of those factors.

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Insulin and glucagon are two hormones that affect the metabolism of fatty acids. DHEA (dehydroepiandrosterone) is another. DHEA upregulates carnitine-related enzymes that promote fatty acid utilization. In a study on humans, it was shown that age-related declines in DHEA levels cause carnitine to accumulate and not be utilized. This is similar to the fatty acid problem in cats, where lipids build up in the liver for lack of carnitine to move them. In this case, the lack of DHEA causes enzymes to lie idle, and the carnitine shuttle doesn’t move. When supplemental DHEA is given to the rat equivalent of a post-menopausal woman, carnitine utilization is increased, and so is energy production. Similarly, when DHEA and/or carnitine is added to bone cells that depend on energy to multiply, energy and the production of bone
protein are increased. DHEA clearly promotes fatty acid metabolism.

A lack of DHEA may be one of the primary causes of insulin resistance. This condition interferes with insulin's ability to regulate glucose. Since insulin is one of the hormones that affects fatty acid metabolism, insulin resistance turns up frequently where fatty acid metabolism is abnormal. And, although it is usually associated with obesity, obesity is not always present at the same time as insulin resistance. A study was done on nondiabetic, normal weight men with high blood pressure and insulin resistance. It showed that insulin resistance coincides with insufficient DHEA.

The positive effects of DHEA on insulin resistance have been proven in genetically diabetic mice where it restores insulin sensitivity and reduces the severity of diabetes. In humans, or at least in women, the level of testosterone appears to be critical to whether DHEA will ameliorate insulin resistance and improve fat utilization. This was dramatically illustrated in a study on a woman in Tennessee who had non-insulin dependant diabetes. When she was given the testosterone-suppressing drug, dexamethasone at the same time as supplemental DHEA, there was a 30% increase in insulin binding, and improvement in her diabetic condition. Dexamethasone alone did not have this effect. It was the increase in the ratio of DHEA-to-testosterone that had the beneficial effect on insulin binding.

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Supplemental carnitine and DHEA both increase fat utilization through different, yet complimentary, mechanisms. Unfortunately, no human studies have been published using the two together. Despite this, all signs point to good effects on fat metabolism with this combination.

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References


