New Research Information on Carnitine

There is a lot of research information available now on carnitine, that wasn't around when we first started looking at carnitine supplementation for polios in 1994 and recording carnitine blood levels via PMH. Answers in smaller print to these questions are straight from the literature. These studies have been done on the normal population not on polio survivors. Carnitine is even more relevant in neuromuscular diseases, including post-polio. **Tessa Jupp RN**

Q. 1. What is carnitine?

A. Carnitine is an amino acid found in the diet primarily in red meat that transports fatty acids through the cell membrane into the mitochondria (energy producing part) of cells. Fatty acids from protein (animal) foods provide longer energy than carbohydrates (plant). It also transports the waste products of energy production back out of the cell to be excreted. **Red meat** is the best source of carnitine in the diet. Chicken and fish contain little carnitine. **Avocado** is the only reasonable plant source.

So carnitine enables **energy to be produced** to make muscles work and also **gets rid of waste** that can cause muscle pain.

New research is now showing that carnitine is also needed for **nerve function** and **signalling to muscles,** other enzyme signals and transports including **protecting the brain** and **lens of the eye**.

"Muscle growth and differentiation, atrophy and regeneration capacity, aging, disease and injury and responses to hormones and nutrients all involve control and regulatory steps at the carnitine system level. 1
Eight different carnitine acyl-transferases have been characterised which are located subcellularly other than in the mitochondrial energy pathways. 1
Carnitine has been shown to protect the lens from ocular oxidative stress from glucose and protein in ocular fluids. Taking carnitine counteracts the protein destabilising effects of ammonia thus preventing encephalopathy. 1

Q. 2. What are the symptoms of carnitine deficiency?

Fatigue	Lack of endurance
Run out of energy leg/arms	Trouble maintaining posture
Muscle weakness	Irregular heart beat/murmur
Muscle pain	Shortness of breath
Poor muscle tone	Brain fog
Rounded shoulders	Extreme exhaustion

"Many of the symptoms occurring in congenital or experimentally induced L-carnitine deficiency can be attributed to a breakdown of the L-carnitine shuttle. These symptoms include the accumulation of fat in muscle in the form of lipid drops (myolipidosis), pronounced muscle weakness, rapid fatigue including the cardiac muscle and muscular pain (myalgia). Progressive cardiomyopathy (weakness of the cardiac muscle) soon develops. ANGELINI and VERGANI, 1996; FRITZ and ARRIGONI-MARTELLI, 1993). 3

Q. 3. Are carnitine levels affected when you first get sick with polio?

A. It is now well known that any illness will deplete carnitine levels so it stands to reason that everyone would have had lower levels of carnitine at the time of acute polio and this could have affected recovery.

"The metabolic process in trauma and illness includes greatly accelerated protein loss in skeletal muscle. It is known that sepsis patients have depleted carnitine stores at the cellular level. The pathophysiology of mediated tissue damage may involve the interplay of reduced carnitine levels and pathogenic requirement of carnitine for growth and survival. The internal carnitine pool could be a major determinant of mounting an effective immune and inflammatory response towards invading pathogens. There has even been suggestion that maintenance of normal carnitine levels might inhibit muscle wasting. 4

Q. 4. Does carnitine help with muscle weakness, pain and increasing muscle loss? What about fatigue?

A. Ongoing trials using carnitine supplementation for post-polio since 1994 in Switzerland (Dr T Lehmann) and Western Australia (T Jupp RN, Dr J Niblett) have shown improvement in endurance, fatigue, muscle strength, weakness, walking gait and slowing muscle wastage. Serum carnitine confirms low levels.

"With lipid (fat) accumulation in type 1 muscle fibres, reduced contractile efficiency, weakness and fatigue are underlying features. 1

Endurance type 1 "muscle damage" is characterized by recurrent episodes of muscle pain and rhabdomyolysis (breakdown and destruction of skeletal muscle). This deficiency is often triggered by heavy exercise, and can also manifest as a result of exposure to cold, infection, emotional distress, and/or fasting. In a clinical trial of carnitine-treated elderly patients there was significant improvement in total fat mass, total muscle mass, total cholesterol and triglycerides, with concomitant decreases in physical and mental fatigue.

Muscle fatigue is measured as a decrease of force development which follows repeated contractions. Muscle phenotype related to carnitine ie type 1 fibre, deals with resistance to fatigue. Fatigue results from abnormalities in carnitine metabolism."

Q. 5. Does loss of nerves and muscle, as in original polio and new muscle atrophy, affect carnitine levels?

Loss of nerve activation lowers carnitine levels in muscle. 85% of carnitine in the body is stored in muscle. If there is less muscle due to polio then carnitine storage space is also lost. Intense exercise quickly uses these stores and they fall to 20%.

Without higher blood levels from daily carnitine supplementation, these fewer muscles from polio damage that are working at a faster rate to accomplish normal activities, are no longer able to keep working at that rate and polios "hit the wall", having to rest until carnitine levels are restored by transfer of carnitine from other parts of the body.

"Following a few minutes of high intensity exercise, skeletal muscle free carnitine content is reduced from approximately 75% of the total muscle carnitine pool at rest to around 20%, with almost all of the reduction being attributed to formation of acetyl-carnitine (Foster & Harris, 1987; Sahlin, 1990).

Normal innervation (nerves in place) is shown to influence carnitine muscle concentration as evidenced by the early marked decrease in total carnitine and acyl-carnitine in rat denervation (loss of nerves) after sciatic nerve lesion. Removal of carnitine induced growth inhibition of muscle cells suggesting this (maintaining muscle cell bulk) is dependent upon the presence of carnitine. 1

Q. 6. Does taking extra carnitine help polio survivors?

A. Taking good quality carnitine as a loading dose every morning helps to increase the carnitine pool in the body that has been lowered by less muscle storage capacity. Some people may need to top up with a bit more carnitine in the afternoon or by eating dietary sources for lunch. New research is suggesting taking fish oil with carnitine may increase cell storage. Eating foods (carbs) that increase insulin release eg vegetables with red meat, will help retention of carnitine within the cells. So eating healthy meals is so important for energy production.

"In order for your body to actually "load" carnitine that you take in supplement form into the muscles where you want it, it has to be accompanied by insulin.

Omega-3 fatty acids (fish oils) come into the equation because they will improve the health of every cell in your body if you get enough of them. The cells are made up of two layers of lipids or fats, which will be composed of good fats or bad fats depending on the type you eat. If the cell lipid layers are made up of omega-3s, the cell will be generally healthier, and it will also make them more sensitive to insulin. Having your cells be more sensitive to insulin allows your body to get the most energy production out of supplemental carnitine.

Carnitine doesn't accumulate or "load" in muscle unless accompanied by high insulin concentrations. This explains why many previous studies have NOT shown increases in muscle carnitine after taking it in multi-gram doses. In this study the carnitine was taken with carbs because the carbs triggered insulin release, which allowed the carnitine to load into the muscle. Take note that there is a big focus in the health world on minimizing insulin secretion from carbs for optimal body composition. This is critical, but be aware that insulin is actually a potent anabolic (building) hormone if the cells are sensitive to it and if appropriate levels are present.

Assuming that the greater whole-body retention of L-carnitine following daily L-carnitine and carbohydrate feeding resided within skeletal muscle (the major carnitine store within the body), this would suggest that an insulin mediated elevation of muscle total carnitine can be sustained in the long term. These findings are important, as they demonstrate that effective whole-body retention of carnitine can be achieved by L-carnitine feeding in a day-to-day setting. 2

From the research described above, it is apparent that carnitine plays a central role in both fat and carbohydrate metabolism.

Q. 7. Is it safe to take carnitine with other diseases?

A. Carnitine is very safe. It is made by the body in the liver. 75% of our needs come from our diet. It is added to baby formulas to equal that in breast milk.

"In studies patients with diabetes and high blood pressure were given daily carnitine. After 45 weeks, irregular heartbeat and abnormal heart functioning decreased significantly compared with non-supplemented patients. A number of studies have shown marked improvement in congestive heart failure. For instance, a 26% increase in exercise capacity after six months. Carnitine taken twice per day for two to four weeks led to positive changes in lung function **(shortness of breath)** during exercise. 4

L-carnitine has been shown to have favourable effects in patients with severe **cardiovascular disorders**, such as coronary heart disease, chronic heart failure and peripheral vascular disease. In patients with chronic heart disease, administration of L-carnitine over 12 months reduced incidence of chronic heart failure and death. 4

In **dry eyes,** damage incurred on the ocular surface of dry eye patients may be due to a lack of carnitine in the tears of these patients, particularly with **diabetic damage** to the **eye lens** and **loss of carnitine in tears** is thought to lead to **triggering of cataracts**. 4

With **osteoporosis,** it has been shown that bone building cells generate 40-80% of their energy demands through fatty acid oxidation and that supplementation of carnitine can influence bone density and slow the rate of bone turnover by slowing bone loss. The study reported that benefits of carnitine are comparable with other drugs of choice in terms of effectiveness in preventing bone mineral loss due to aging. 4

References:

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