Aging's Cellular Energy Crisis

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There is growing evidence that one of the most common effects of aging is a progressive loss of mitochondrial function — that is, our cells have more and more difficulty making energy efficiently. When this occurs, the mitochondria begin to produce enormous numbers of free radicals.

A breakdown in mitochondrial function is strongly linked to free-radical generation, which damages cells. Mitochondria are the cell's energy powerhouses and produce about 90 percent of all energy. They are also the source of most free radicals, which is the cost of energy production.

Ironically, chronic inflammation has been shown to be a major cause for mitochondrial failure. In addition, we are all exposed to a great number of mitochondrial poisons:

• Pesticides
• Herbicides
• Fungicides
• Industrial chemicals
• Vaccinations
• MSG (and other forms of glutamate food additives)
• Mercury
• Lead
• Cadmium
• Aluminum
• Fluoride

Most of us are exposed to all of these, and many of these poisons have synergistic toxicity. Many of these produce mitochondrial damage by triggering inflammation. (For a detailed discussion on inflammation and its role in many diseases, see my newsletter "Inflammation: The Real Cause of Most Diseases.")

When our cells begin to have difficulty making energy, we become weak and listless — we sleep a lot during the day and tire easily. Also, our organs begin to fail and are increasingly subject to disease and toxins from the environment, as well as toxic foods.

Mitochondrial failure in our brain is even more ominous. A number of studies have shown that when the mitochondrial energy production falls, even modestly, the brain cells become hypersensitive to the toxic effect of glutamate (excitotoxicity).

Even normal levels of brain glutamate can cause destruction of brain connections and actual brain cells. We see elevated brain glutamate with Alzheimer's disease, Parkinson's disease, ALS, migraine headaches, multiple sclerosis, strokes, and head injuries.

In all of these conditions, brain energy levels are low because of mitochondrial malfunction, and they malfunction long before symptoms develop. Our mitochondria also contain DNA, and this DNA is more sensitive to damage by free radicals and lipid peroxidation products because mitochondrial DNA has very few repair enzymes.

It has been shown that at age 70 our nuclear DNA oxidizes 10 times faster and mitochondrial DNA oxidizes 15 times faster than when we were young. So, not only do we produce more free radicals as we age, we are more vulnerable to damage by these radicals.

This is why we should increase the strength of our antioxidant network as we age. The antioxidant network is a collection of chemicals and enzymes found in every cell in the body. The network neutralizes free radicals and things that can become free radicals, such as hydrogen peroxide.

Fortunately, there are a number of ways to boost mitochondrial function, which has been shown to protect the brain and body from free radical damage. These supplements do just that:

- Vitamins B1, B6, B12
- Niacinamide
- Riboflavin
- Folate
- Vitamin K
- R-lipoic acid
- Acetyl-L-carnitine
- CoQ10
- Magnesium
- Zinc
- Pyruvate

Antioxidants also protect the mitochondria from damage by the free radicals it produces. Of particular concern are the lipid peroxidation products, such as acrolein and 4-hydroxynonenal (4-HNE), which are very destructive. We see high levels of these lipid peroxidation products in Alzheimer's disease, Parkinson's disease, ALS, heart failure, head injuries, and strokes.

Learn even more about the benefits of supplements by reading my special report "Key Vitamins That Save Your Heart, Prevent Cancer and Keep You Living Long."

These caustic oxidized fats are resistant to most antioxidant vitamins, but are neutralized by flavonoids such as curcumin, quercetin, ellagic acid, luteolin, and proanthocyanidins (from grape seed extract). Most of these are available as supplements, and the others can be taken as vegetable blends.

For more of Dr. Blaylock's weekly tips, go here to view the archive.

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