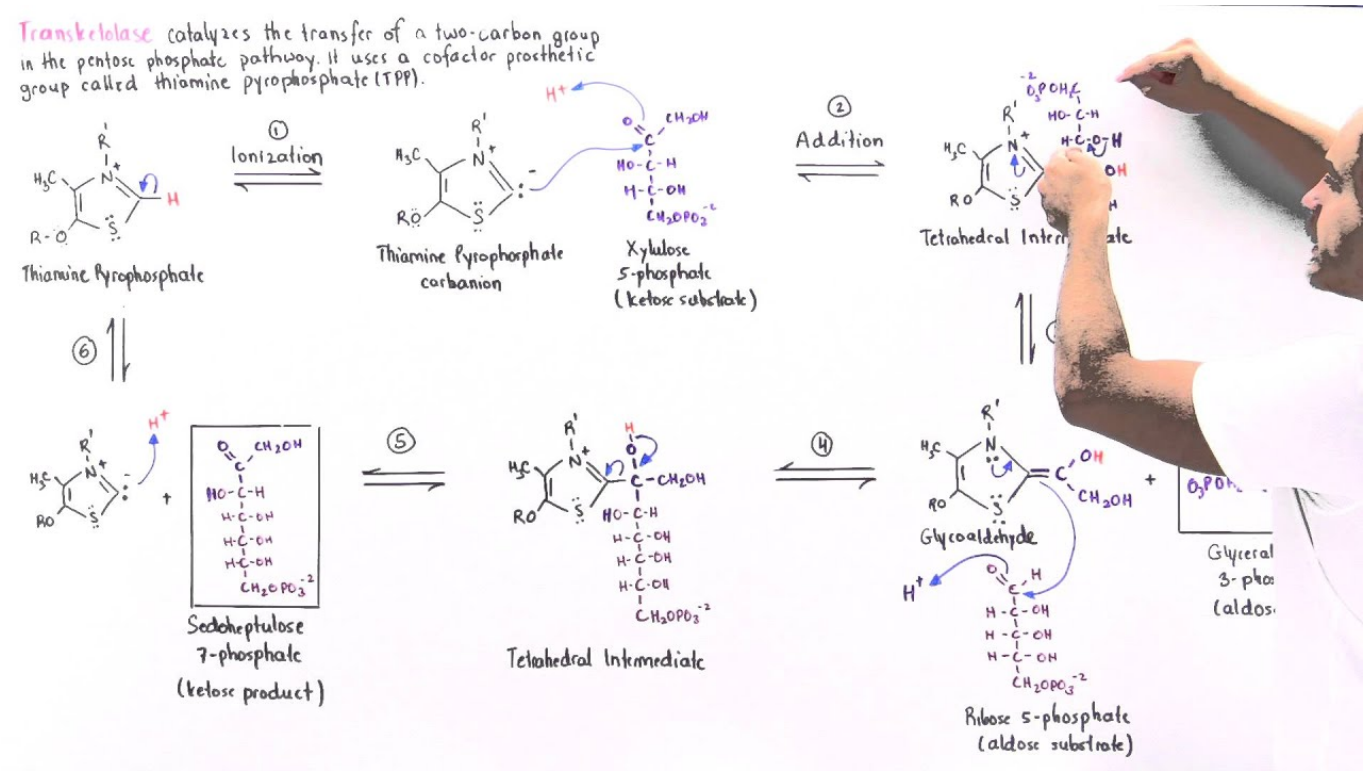

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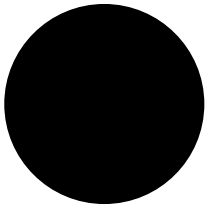

Link

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Thiamine, also known as vitamin B1, is now known to play a fundamental role in energy metabolism. When we are deficient in B1 the mitochondrial matrix suffers from pseudohypoxia. Its discovery followed from the original early research on the 'anti-beriberi factor' found in rice polishings. After its synthesis in 1936, it led to many years of research to find its action in treating beriberi, a lethal scourge known for thousands of years, particularly in cultures dependent on rice as a staple.

Thiamine pyrophosphate (TPP) is the active co-enzyme form of thiamine and it is abundant in human RBC's. For this reason it is a reasonable marker that we can use in mitochondrial matrix failure associated with higher heteroplasmy states. When we see abnormal peripheral smears in patients it signifies that we might want to clinically assess TPP activity and thiamine levels in our patients. Some disease states associated with high mitochondrial density show these clinical features more often than not because certain organs have higher mitochondrial capacity.

When TPP is abnormal so is transketolase in RBC's. Transketolase is an enzyme of both the pentose phosphate pathway in *all organisms* and the Calvin cycle of photosynthesis.

When RBC transketolase is abnormal this is a beacon that RBC might not have a high fidelity signal connecting the sun to our colony of mitochondria. This fosters the development of heteroplasmy in an insiduous way. If one is not looking for it, a relative thiamine deficiency can manifest in many disease or non disease states.

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In humans, transketolase connects the pentose phosphate pathway (EMF 4) to glycolysis, feeding excess sugar phosphates into the main carbohydrate metabolic pathways. Its presence is necessary for the production of NADPH (PPP), especially in tissues actively engaged in biosyntheses, such as fatty acid synthesis by the liver and mammary glands, and for steroid synthesis by the liver and adrenal glands. Thiamine diphosphate is an essential cofactor, along with calcium as a co-factor.

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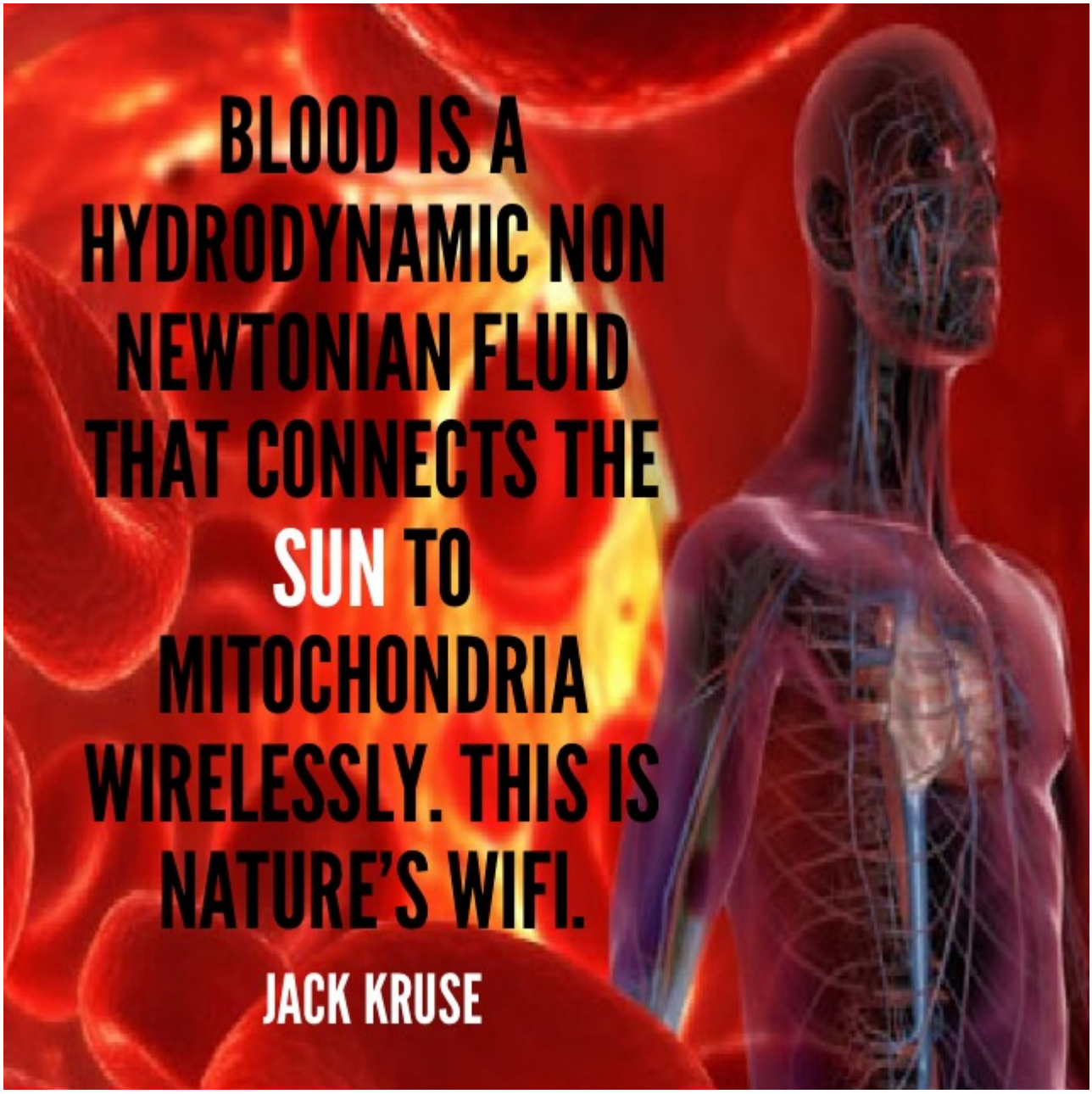

THE KEY LINK:

Thiamine Pyrophosphate (TPP) is the cofactor needed for the following reactions, Thiamine is required for only 4 biochemical reactions in the body 1. Pyruvate dehydrogenase 2. α ketoglutarate dehydrogenase 3. Branched-chain ketoacid dehydrogenase 4. Transketolase

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TPP is involved in energy metabolism. Deficiency of TPP will affect the link reaction and TCA cycle. This leads to reduced ATP production and can alter function of the Pentose phosphate pathways I wrote about in EMF 4 blog post. Red light from the sun can augment this ATP loss from thiamine deficiency.

Transketolase is an enzyme that uses a thiamine pyrophosphate (TPP) as its KEY cofactor to conduct a 2 carbon transfer from ketoses onto aldoses in humans. In the Pentose Phosphate Pathway (EMF 4 blog), it performs both a transfer of carbons from xylulose-5-P onto Ribose-5-P and onto Erythrose-4-P, setting them up for reaction with transaldolase.

Transketolase activity is decreased in deficiency of thiamine and can be used as a marker of heightened heteroplasmy by enlightened physicians.

RBC transketolase activity is reduced in deficiency of vitamin B1, and may be used in the diagnosis of Wernicke's encephalopathy and other B1-deficiency syndromes if the diagnosis is in doubt. Apart from the baseline enzyme activity (which may be normal even in deficiency states), acceleration of enzyme activity after the addition of thiamine pyrophosphate may be diagnostic of relative thiamine deficiency from any causes. This altered activity can be quantified as follows:

a. 0-15% normal

b. 15-25% deficiency

c. >25% severe deficiency

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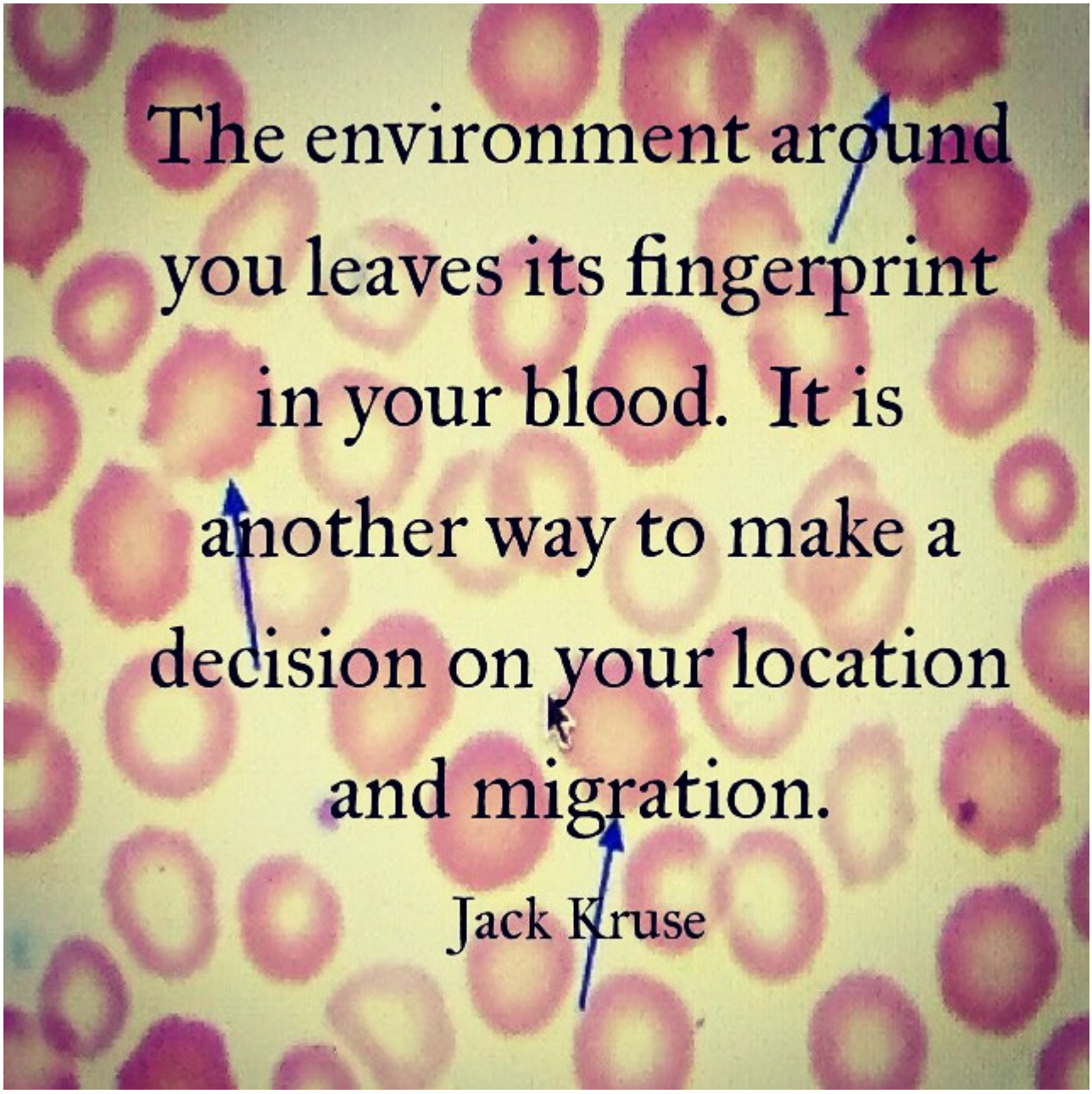

CENTRAL LEPTIN RESISTANCE: LONG LOOP OF BAZAN

Liver disease is one such disease state that causes central leptin resistance (Leptin resistance Part Deux blog). Most cases of liver disease have central leptin resistance associated with them due to damage of the long loop of Bazan (image below). This limits the

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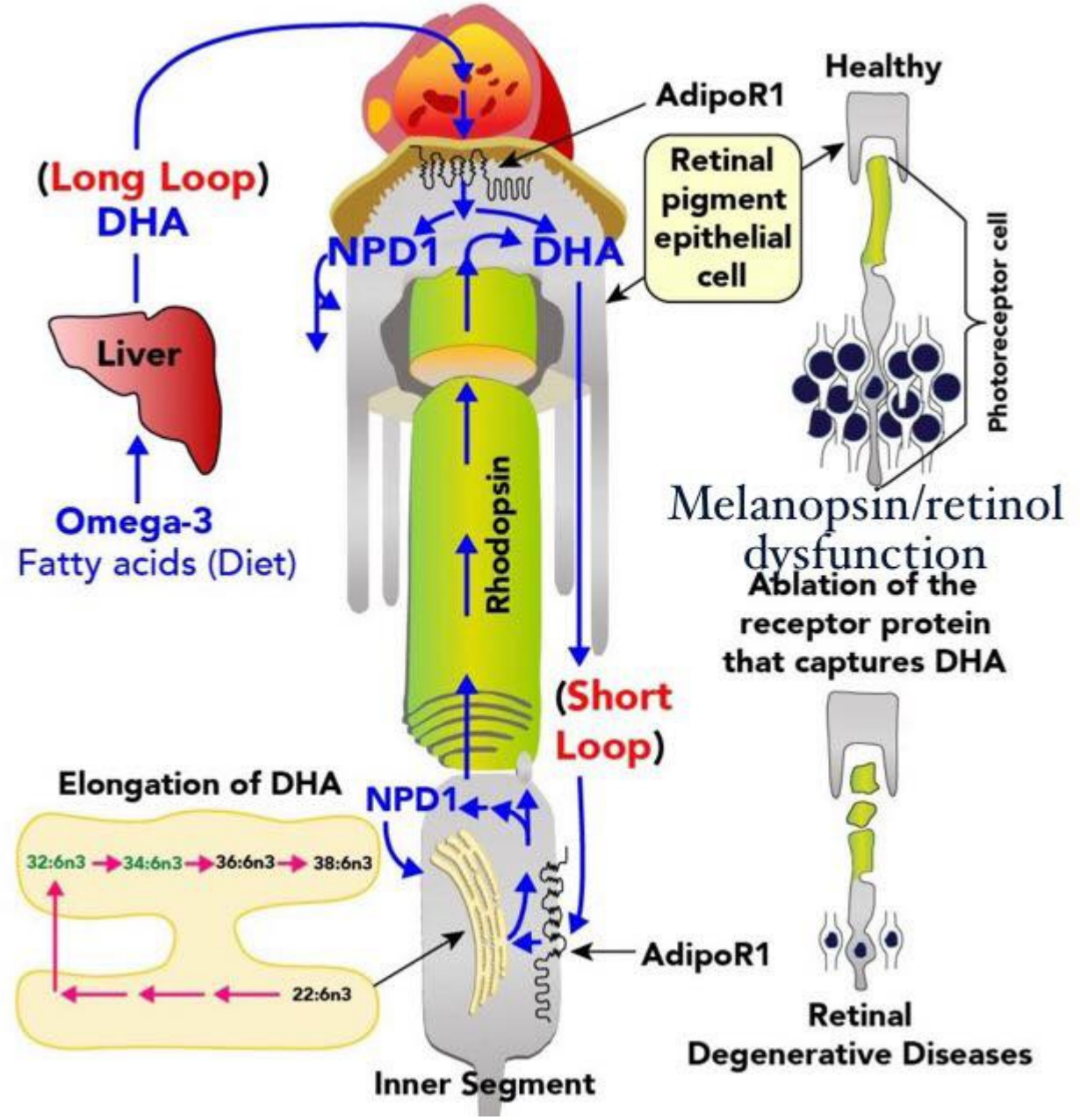
reincorporation of DHA into human cell membranes and fosters inflammation because the elvanoids cannot be made to curtail the inflammatory cascade.



Simultaneously, thiamine can be depleted because of altered matrix functioning. There is

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brisk evidence that thiamine deficiency is found in many liver diseases. The literature reports 58% of patients with chronic liver disease have B1 deficiency, moreover, the incidence is higher in alcoholic than in non-alcoholic hepatic patients. It has also been shown that daily supplementation with high doses of thiamine hydrochloride (200 mg/day) for one week can restores levels of thiamine pyrophosphate (TPP) in most cases. Since TPP is the active co-enzyme form of thiamine, it also stimulates synthesis of the enzyme transketolase. Because of the essential role of TPP as a co-factor in intermediary metabolism of carbohydrates, lipids, and protein it can be a proxy marker for mitochondrial matrix dysfunction. *It maybe a NOVEL new way for us to indirectly measure heteroplasmy levels in humans.*

THE LINK TO TECHNOLOGY:

Because thiamine is a major factor in the metabolism of glucose, it has long been known that ingestion of simple carbohydrates, processed in the body mainly to glucose, automatically increases the need for dietary thiamine. Since Frey and Volkow work, we know exposure to nnEMF also increase AMPK and glucose metabolism it should be clear that technology use and abuse can mimic nutritional problems historically associated with Vitamin B1. Thus, high calorie malnutrition and technology abuse should be commonly associated with a chronic relative thiamine deficiency, irrespective of its fortification in food substances or the diet of any patient. This relative deficit might lead to unusual presentations of disease linked to elevated heteroplasmy in humans.

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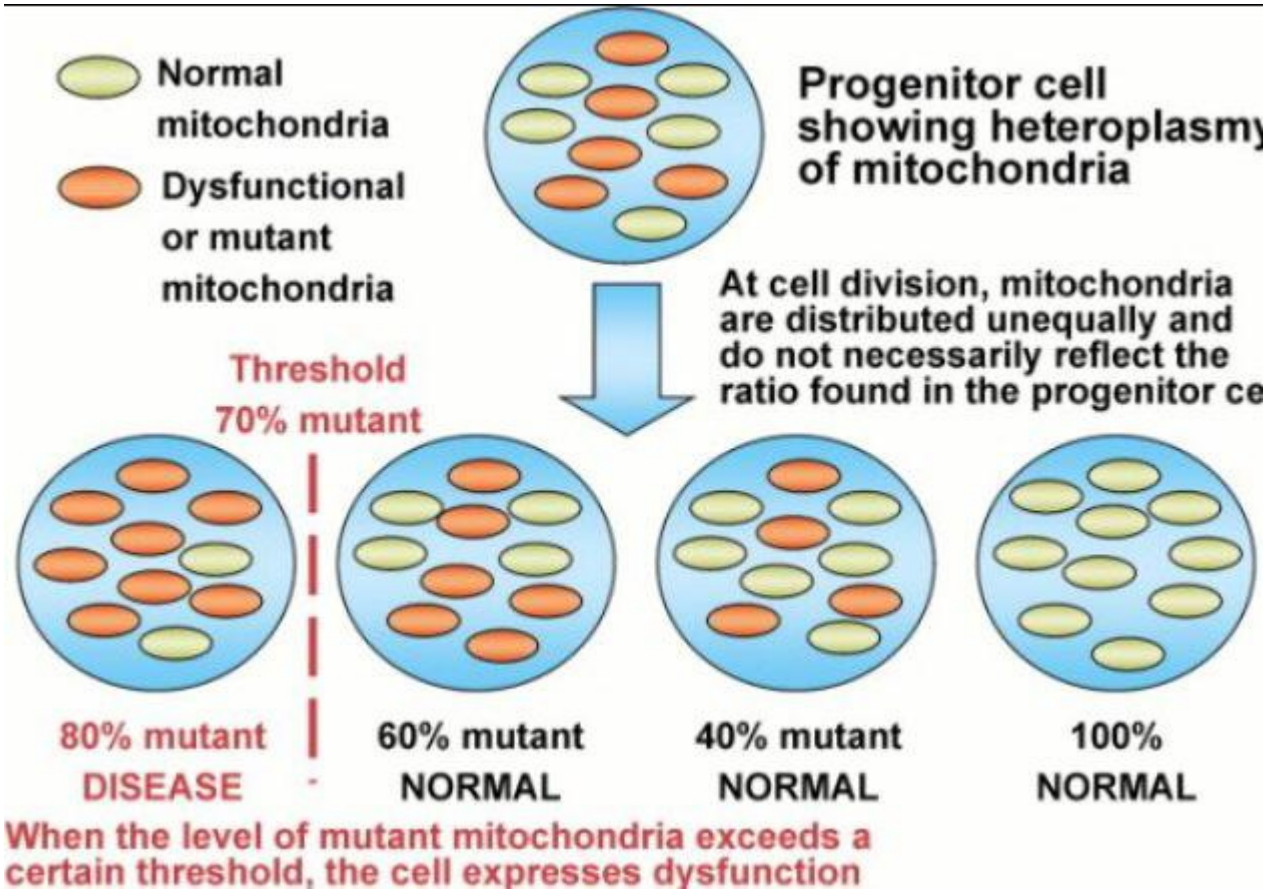

QUICK SUMMARY:

Thiamine is normally present in pastured lean pork and other meats, wheat germ, liver and other organ meats, poultry, eggs, fish, beans and peas, nuts, and whole grains. It is lower in foods like those mentioned above that have been altered by man's input into food webs. Blue light screens and nnEMF field deplete cells of thiamine because of how they affect AMPk pathways and glucose metabolism to mimic high calorie malnutrition. Modern dairy products, fruit and vegetables are not good sources of B1. In fact most of them deplete thiamine stores. Humans only have the ability to store 14-18 days of this essential vitamin. This storage ability is decreased by technology abuse and by vegan/vegetarian diets. The RDA is 0.5 mg per 1000 kcal, adequate for a healthy individual consuming a healthy diet. Considerable losses occur during cooking or other heat-processing of food. Polyphenolic compounds in coffee and tea inactivate thiamine so that heavy use of these beverages could

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compromise thiamine stores in tissues.

CITES:

http://jackkruse.com/emf-4-why-might-you-need-carbs-for-performance/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6435462/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6459027/

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