Raymond Peat, Ph.D.

Cholesterol and saturated fats

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(Transcribed by Amazionac, verified by Burtlancast)

JR: *Today's show, Cholesterol and Fats. Huge topic. Of course we're going to get Ray Peat's philosophy on this, we'll cover a lot of things from why cholesterol is so important, why doctors try to lower it, why our body needs it, our physiology needs it, and what can happen, what it means when it's high or low, and what we can do to actually lower it. As well as, why saturated fats - yes, the right types of saturated fats - are so important in our diets.*
So, enough of me, I want to get Dr. Ray Peat on the show, maybe let him do an intro of himself this time because I feel like I never do enough honor.
Just one more key piece before we hook him in, I always say this: we do the show, he volunteers his time, and I know a lot of people are emailing him left and right. So, please, please, please; I don't care if you send him a check for a dollar, 10 dollars, 5 dollars, 20 dollars, 100 dollars; whatever you want to send him to say thanks. If you are emailing him, send him a check. I know people that send him checks every month, we send him checks all the time. I just think it's important because he is donating 1-2 hours of his time, plus emails every month. So, don't want to say anymore of that but hopefully you can take that and say thank you to Dr Peat. Ray, are you there?

RP: Yes, hi.

JR: *Introduce yourself with a brief background.*

RP: Before I studied biology I was mostly studying language, literature and painting. I've been interested in biology for a very long time, but I had a bad impression of academic biology from my high-school experience (mostly), but then seeing that in the 1940s there was a very big change in the nature of American biology…and that was powered by industry and government promotion of the gene theory, as opposed to the field theory and development, and health, and so on. So, I waited a long time until 1968 to actually enter a graduate program in biology and I had learned enough about universities and biology to manage to get through the program without having too many conflicts with the professors. Most of the professors were very committed to the genetic determinist idea of what an organism is. And I was interested in the field concept, the environmental influences on the organism. Nutrition is one of the most important environmental influences on the nature of the organism. So I see our nature as being a matter of potential being realized, according to the conditions of the environment, rather than the expression of a pre-determined genetic program.

JR: *In general, why do you think Americans are so afraid of cholesterol and saturated fats?*

RP: I think it's driven primarily by the food industry wanting to sell their products. The seed oils, they were already seen to be toxic, causing sterility, degeneration of the brain, and the gonads and so on. Probably from 1925, but especially in the 1930s and 40s, the very oxidizable seed oils were know to be toxic. And that was one of the factors in researching Vitamin E as something to counter-act the toxicity of the seed oils' unsaturated fats as well as the toxicity of estrogen, which happens to interact, synergize with unsaturated fats. The pig industry had been looking for ways

to make their animals gain weight and eat less food. George Burr, who's identified as the inventor of the essential fatty acids concept himself, discovered that the essential fatty acids slow the metabolic rate tremendously. “Deficient” animals eating sugar instead of unsaturated fats had about a 50% higher metabolic rate; various people noticed that effect, and they found that feeding soy and corn to the pigs, because of the unsaturated fats suppressing their metabolic rate, increased the profit: less food in, more pig out. At the same time, they noticed that it was lowering cholesterol, just as one of its biological effects (a toxic effect, basically). The paint chemists, the oil chemists discovered that instead of using the expensive seed oils that were now being sold largely as pig food….the chemists discovered how to make paints and plastics from petroleum, around 1950. That decreased one of the big markets for soy beans, in particular. Henry Ford had imported soy beans to use in the car industry; he made a car body entirely out of soy bean plastic, and hoped to replace iron with that. There's a picture of him using a sledge hammer to pound on one of his cars showing that they were very strong. But by the late 1930s, only door knobs and steering wheels where still made out of the soy bean plastic. But when the petroleum chemists learned how to use petroleum, that freed up the soy bean market for pigs. And their production was great enough they wanted to find new markets to replace the paint and plastics market. So they started promoting the health idea of their soy oil, cottonseed oil, safflower seed oil, and so on. The only biological effect other than sterility and dementia that were established for the seed oils was that they lowered cholesterol. For several decades people had been noticing that cholesterol accumulated in walls of blood vessels (atherosclerosis). Although the first person who discovered that in rabbits had dissolved the cholesterol in unsaturated vegetable oil, he realized that cholesterol by itself wasn't causing the disease. But there had been these experiments that gave a basis for the seed oil people to say cholesterol is found in the hardened arteries and in the blood, and their oil lowers the level in the blood. So they argued guilt by association that if it's in the blood vessel and the blood, then you can lower it in the blood and prevent the disease. But the very people who had made those discoveries had realized that it wasn't so. In fact, in the 1930s people had found that thyroid was enough to prevent the atherosclerosis and at the same time had lowered cholesterol. Over the last 10 or 15 years, a Danish doctor named Uffe Ravnskov has gone through the literature and shown beautifully that there's no connection between atherosclerosis, heart disease, and the triglycerides and cholesterol in the blood; they were associated, but it isn't causal. The experiments that had been done in the 30s showed that hypothyroidism and the associated changes caused by the lack of thyroid and metabolism were what caused the atherosclerosis. The cholesterol seems to be there as a defensive reaction to protect the injured blood vessels.

JR: *Elaborate on the difference between unsaturated and saturated fats.*

RP: It refers to the chain of carbon atoms in the fatty acid. In one case they have all of the hydrogen atoms attached to them that’s possible, so they are saturated with hydrogen. And if some of the carbons atoms lack the hydrogen, they just have their electron bonds more exposed to the environment. The absence of the hydrogen makes them more flexible instead of being sort of like a bottle brush bristling with the hydrogen surrounding the chain of carbons. It's more like a necklace of beads, flexible, where the hydrogens are missing; the bonds can rotate more freely. You get 2 or 3 or 4 or 5 of those double bonds, and the chain is bent at each place there's a double bond. And at the single bonds, the molecule rotates freely so you can get very flexible shapes when you have the highly unsaturated molecules. That flexibility you can see when you put a bottle of oil in the refrigerator: coconut oil is hard when it is just a little cool, just below room temperature or even at room

temperature. But olive oil has to be much cooler before it starts to solidify; that's because it's relatively saturated. Canola and corn oil have to be extremely cold before they all solidify. That's biologically important; one of its meanings is that if the organism lives at 40ºF for example and it contained butter fat or olive oil, it's fats would be solid and wouldn't be manageable. For example, if an animal contained 30% fat and it all hardened, it's subcutaneous fat would become stiff, just the way a steak, when it's in the refrigerator, the fat is stiff; when it's in very warm conditions it becomes soft and flexible. So, in the tropics, even fish in the Amazon river for example, have fat as saturated as butter fat. If you grow soy beans or corn in a very warm climate, their fat is saturated according to the temperature. Chocolate and coconuts grow in a place where the temperatures are probably averaging close to 90ºF, and so they need to have very saturated fats just for the biochemical manipulation (the molecules to happen). And if you had unsaturated fats, like is necessary for the cold water polar fish, for them to be flexible, they have to have triply or quadruply unsaturated fats. But if you had those in a fish in the Amazon river, the fish would oxidize its fats in just a couple of days. Even with a median unsaturated oil, such as soy oil or corn oil, if you put a rubber hose into a cork at the top of the bottle and put the other end of the hose in a cup of water, and leave the bottle at room temperature; the water will rise up the tube as the oil is consuming oxygen; it's just a constant process even at room temperature. And if you raise it to body temperature 95-100ºF then the process is much faster and you'll have sticky, rancid oil very quickly. So it's a biological adaptation. You can make a pig have subcutaneous unsaturated fat depending on the temperature of the weather that it's exposed to. If you put a sweater on the pig, it'll get more saturated.

JR: *Thoughts on atherosclerosis, cholesterol and heart disease ?*

RP: Polyunsaturated fats, that we unavoidably get in our diet, they are constantly breaking down, producing free radicals, tending to cause inflammation and tissue injury. And if anything's disturbing the metabolism of a cell, that's more likely to happen. For example, the normal healthy oxidation through the mitochondrion keeps the electrons tightly controlled while they are being transferred from, let's say, a glucose molecule to an oxygen molecule. But if you take away some of the oxygen, the electrons in the mitochondrion will have no proper place to go, and they will escape as free radical potentiators. And so the cell becomes more oxidized in the absence of sufficient oxygen. So, any stress of a cell puts out these electrons that can attack the exposed electron bonds in unsaturated fats. But the saturated fats aren't open for stray electrons. So, every time you are limited in your delivery of oxygen to a cell, you're putting the unsaturated fats in the way of these stray electrons. Those will then start chain reactions, that at the extreme, they form lumps of material called age pigment, or lipofuscin. These free-radical oxidized fat fragments attach to proteins and they collect bits of iron and other breakdown products from the free radical attacks on whatever errs around in the cell. These lumps of age pigment, because of their iron and fat fragments, are able to catalyze the consumption of energy, and deliver energy from the enzymes to oxygen, so they can create a false mitochondrial effect consuming energy and wasting oxygen. And then that accelerates the process because the real mitochondrion is furthered deprived of oxygen by these age pigment lumps. But before that happens, (that tends to be at the end of the life) you get degrees of that happening, in which oxygen deprivation and stress cause lower degrees of damage and inefficiency. Signals go out, changing the conditions of the organism, for example sending signals that more energy is needed, activating hormones such as cortisol, estrogen, and growth hormone. These three hormones are very closely connected to the rise in triglycerides and the damage to the blood vessel wall. And they tend to interact, cortisol increases estrogen, estrogen

increases cortisol and they both increase growth hormone. All of these tend to breakdown triglycerides in the free fatty acids which are more reactive, tend to become oxidized more quickly, cause more cellular injury. So the circulating fats in the bloodstream are in first place activated by stress and unsaturated fats, and then these three main hormones increase the damage they can do, even though they normally would be part of a defensive repair process, in the presence of the unsaturated fats, they magnify the damage.

JR: *Why is cholesterol so important for our physiology, and why does it go up when it's stressed ?*

RP: The whole stress hormone system, the things that Hans Selye popularized and studied for many years, he was concentrating on the steroid system, that's the most powerful stress-induced system. The pituitary senses something wrong in the organism, increases it's ACTH, which drives the adrenal glands in particular to take up cholesterol and to synthesize cholesterol from raw materials, and to direct the cholesterol into the mitochondrion to turn it into pregnenolone, progesterone, DHEA and finally cortisol. And cortisol is the thing which has a short term defensive reaction against stress by… One of its main effects is to turn protein into sugar, so that the sugar can be used for energy to increase adaptive ability to handle the stress. But if you keep your cortisol up too long, you'll destroy too much of your protein tissues. So, before the cortisol stage of stress, the continuously activated adrenal tissue will be first producing pregnenolone, progesterone and DHEA. It's only when those aren't enough to handle the stress that the progesterone will be further converted into the cortisol. So the production of the cholesterol is the first stage of handling stress reaction, and then the cholesterol has to be converted into pregnenolone to further the defensive reactions. But the increased production of cholesterol itself is a primitive defensive anti-stress system, and the later stages (pregnenolone, progesterone and DHEA) backup that basic function of cholesterol, giving it more dimensions of protection. Starting probably around 1920, people were trying to purify cholesterol and see what its biological effects would be. They found that, I think they used about 10 different toxins, everything from pus from an infection, to snake venom, heavy metals, chlorinated solvents, everything that occurred to them they would give a lethal dose of the poison to an animal, and if they give it a good dose of cholesterol, it would survive an otherwise lethal dose. That has been done repeatedly. There'd been some more specific things, for example the testes are stimulated to more efficiently produce sperms in the presence of cholesterol, and the cholesterol has a direct activating effect on producing more of the defensive steroids and helping the cell do what it should do, as well as interfering with the toxic effects of chemical stresses and physical stresses. For example, hyperthermia that will cause your red cell to break down, if you add enough cholesterol the cells won't be hurt by an otherwise deadly temperature. So it's just an incredibly broad spectrum protective substance.

JR: *So, cholesterol naturally rising during stress is the body's natural response in fighting this stress. In some people, it keeps rising without going down; what do you need to make the conversion from cholesterol to pregnenolone ?*

RP: I guess it was about 40 years ago that someone was looking at the effects of varying the amount of cholesterol going into either in an ovary or an adrenal gland; and they found that if the thyroid hormone and the Vitamin A nutrition were adequate, the amount of progesterone coming out exactly corresponded to the amount of cholesterol going in. So a very big proportion of the cholesterol is being turned into the also protective hormones pregnenolone, progesterone and DHEA.

And that is a massive turnover of the cholesterol that's been produced. But it depends on thyroid and Vitamin A very largely, so the higher your thyroid function, the more cholesterol you're consuming. In the 1930s, someone noticed that when the thyroid gland had been removed, people's cholesterol immediately went up. So they would measure the cholesterol and give them a thyroid supplement, and immediately as the metabolic rate increased, the cholesterol would decrease. The blood cholesterol was a mirror image of their thyroid function. But the thyroid also supports the ability to make the cholesterol in the first place. But the level that a healthy person maintains depends on everything else that's happening. If you have, say, a 500mg of cholesterol blood level, that's considered very high and you can bring it down in a week to 200-180mg just by giving frequent small doses of a quick acting thyroid. That is protective, because it's what's needed for them to produce progesterone and pregnenolone, so they don't have to rely on the pure effects of the cholesterol for their anti-stress activity. If you lack something needed to turn energy into cholesterol…For example, sugar is needed if something is causing you to be overloaded with unsaturated fats; and too much starch and unsaturated fats, probably, are the main reason for not being able to produce enough cholesterol. The thyroid will also help you overcome some of the blocks to producing cholesterol. But a supplement of sugar sometimes is needed to bring up your production of cholesterol. So that you don't want your cholesterol to get below something like a 160mg, as a very minimum. In some studies after, they were seeing that some diseases such as cancers were associated with very low cholesterol levels. Instead of the cholesterol being consumed by the cancer, it turns out that the low cholesterol makes you susceptible to developing cancer. There's a Hungarian study about 45 years ago that found that when they chemically lowered the cholesterol, the mortality increased from all causes: accidents, homicides, suicide and cancer; a huge increase in cancer mortality, as they chemically lowered the blood cholesterol. The hormones that aren't being produced from the cholesterol are probably the reason for those increases in mortality.

JR: *Why after applying your recommendations some people experience higher cholesterol?*

RP: No one is really sure of what the optimal level of cholesterol is. For example, looking at old people in rest homes, they followed their cholesterol levels and saw that the ones that lived the longest had pretty high cholesterol. I think 270 was the number that corresponded to the longest survival. In the Framingham study they saw that people over the age of 50, who didn't have at least a 200 cholesterol level, were more likely to develop dementia. And in a big study of violence, they divided populations into those with at least 180 cholesterol and those below 180. The violent offenders were clearly connected with the below 180 cholesterol levels.

JR: *Can you elaborate on pregnenolone's effect on brain chemistry? Also elaborate on ACTH.*

RP: Several signals can tell your brain that you are suffering stress. For example low blood sugar is one of the basic things. The various things that amount to stress act on the stability regulators of brain cells. The GABA is a sleep-inducing peptide fragment of the glutamic amino acid. Glutamic acid excites the brain. If you take off the acid, it becomes gamma-amino butyric acid, which sedates the brain. And the stressed brain will activate enzymes to produce GABA and turn off the excitation. There are specific peptide signals that act parallel with glutamic acid to excite the brain and to interfere with GABA, the stabilizer. The Valium type of sedative, or tranquilizer, acts on the GABA stabilizing system of the brain. If something is

preventing the glutamic acid from being turned into sufficient amounts of GABA, valium will fill in and activate the GABA system. And activating the GABA system turns off the stress signals, and stops the pituitary from producing ACTH to activate the adrenal cortex. Progesterone and pregnenolone, and many other things, act on the GABA, the receptor system. Pregnenolone happens to be one that doesn't have many other hormone-like effects; it's basically a stabilizer at all levels of cells. In the brain, its effect is on the GABA system to imitate valium, to tell your pituitary that the stress is under control and to stop producing so much cortisol. So, in early experiments testing the toxicity of pregnenolone, someone gave a group of mice or rats 10g of powdered pregnenolone made into a slurry, injected into their stomachs. They didn't have any appetite for a few hours because being so full of powdered pregnenolone. But the only hormonal effect that they saw was that some of the animals that had been stressed, and had excessive cortisol or corticosterone before the dose of pregnenolone: when they were getting the huge dose, they were normalized. So, the animals were more normal after that big dose than they were before the dose. For a person, that would be like eating a pound of pregnenolone at once.

JR: *In people able to make this conversion, can good nutrition alone insure pregnenolone production ? Or should they supplement pregnenolone additionally?*

RP: It depends how far the system has deteriorated. Many years ago some Russians found, just with isolated mitochondria, that giving them pregnenolone would structurally restore damaged mitochondria, and allow them to begin producing more pregnenolone. And that's similar to what people saw with isolated progesterone on slices of adrenal gland. When they would give the adrenal slices progesterone, it would increase its production of progesterone. So in both cases there's a positive feedback, rather than the typical negative feedback; you give it some and it makes more. Which means that the organism is designed as if it wants as much as it can get. Ordinarily you can make enough from converting sugar to cholesterol, and with thyroid and Vitamin A converting cholesterol to the other hormones. But when you had been poisoned with not enough of the needed foods, or too much of the unsaturated oils, heavy metals, causing free radical reactions and so on, then it helps to use all of the supports possible: thyroid supplements, pregnenolone supplements, possibly DHEA and progesterone, saturated fats, sugar (everything that works in the same direction).

JR: *Why is growth hormone inflammatory ?*

RP: Most of the research is designed to praise its beneficial effects but there are some very basic observations that people who have been chronically deficient in it have a remarkable absence of atherosclerosis. The mice that are genetically deficient in the pituitary hormones, have no growth hormones, they aren't very big but they live 2-3 times longer as normal mice. One researcher removed the pituitaries from several species of animals and found that they lived much, much longer than the animals with their pituitaries, if they were given, basically, a thyroid supplement to keep their metabolism going. So when you have hypoglycemia, you increase your growth hormone and it tends to come with the other pituitary stress responsive hormones. And all of these tend to have their side effects, so they’re all beneficial in the right amount, in the right time. But they’re all harmful when they go on too long, or too high a level. People with gigantism, acromegaly (for example a tumor), or over activity of the pituitary gland; they have an extremely high incidence of atherosclerosis.

JR: *Talking about cholesterol, can you explain the basic differences between HDL and LDL ?*

RP: Both of them are able to participate in detoxification, the protein of it helps to carry the cholesterol in the bloodstream. Itself, has some very specific anti-stress, even anti-viral activity, so they are part of our ability to respond to stress (the protein as well as the cholesterol associated with the protein). They are both defensive, and both important. But the LDL is mostly the one that carries cholesterol into the places where it's needed; the brain, the ovaries, testicles and the adrenal glands, anywhere you're making steroids, and that includes the skin. Skin is probably our biggest endocrine gland, brain is the next, and then the ovaries, testicles and adrenals. The HDL has probably some more specific anti-toxic effects, whether the LDL is more a delivery system of cholesterol itself. Toxins will tend to increase the HDL, relative to the LDL; toxins of most types will increase both of them defensively. But the chlorinated hydrocarbons, radiation, estrogen and alcohol for example will increase the HDL relative to the LDL because it has some more specific anti-toxic effect.

JR: *You've mentioned in the past that DHEA can lower LDL; can you elaborate ?*

RP: Anything that is activating the turnover, increasing your metabolism; progesterone redirects your metabolism, DHEA intensifies some of the cellular turnover processes. Thyroid is the basic thing, increasing the turnover. The turnover of cholesterol, besides making more of the pregnenolone and progesterone… it's also important to keep it turning over so that it doesn't sit around and get oxidized in the presence of the unsaturated fat breakdown products, which everyone has exposure to. So, everything that increases your metabolic rate, such as thyroid, DHEA, coconut oil, sugar and salt; all of these things can potentially lower your cholesterol just by increasing its use. But it depends on where you're starting; they can potentially increase it if synthesis is the limiting factor.

JR: *How can a lack of light cause an increase in cholesterol ?*

RP: Someone took blood samples every 15 minutes during the night and recorded whether the person was awake or not. As soon as the lights went off, the cortisol began rising; and it continued pretty steadily through the night. And sleeping decreased the rate of increase in cortisol. It shows that the stress system recognizes very immediately (in less than 15 minutes) that darkness is stress in some way. And that seems to be that the intrinsic oxidation free radical production gets out of control, in proportion to the exposure to darkness. When you measure the cytochrome oxidase, which is what delivers food electrons to oxygen (it's the last step in the mitochondrial energy production), if you measure that during darkness, it degrades. And after several hours, like in the far North, where you have 15 hour nights, after about 12-15 hours, the mitochondria swell up, some of them explode and leak their content. Just from the 15 hours of darkness, fatal to a large proportion of the mitochondria (firstly, they produce more and more cortisol; but finally they are overstressed and simply die). So, that's part of the aging processes, being exposed to the prolonged stress of darkness. When they shined red light on the heads of experimental rats, the red light penetrates very easily, but it happens that the cytochrome oxidase enzyme contains copper in the blue state of copper oxidation, and the blue copper absorbs red light. And there isn't very much of it, so that the red light passes very freely through the tissues without being absorbed randomly. And so, it's delivered to the copper in the oxidative enzyme. And it restores the copper to its proper functional role. That's apparently what's lost during exposure to darkness.

The use of the cytochrome oxidase enzyme, in effect, wears it out in the dark; it needs to be recharged by light. You can see similar effects if you expose a bit of tissue to sunlight, the blue light and ultra-violet light will excite electrons (in the same way X-Ray excites electrons), in hard tissues especially. The excited electrons can last for years or decades in the hard tissues such as the tooth or bone. But in the soft tissues, these excited electrons can be detected in an electron spin resonance machine for hours after being exposed to sunlight. So you can tell the history of a seed, or bit of tissue, by measuring the state of its electrons, whether it was exposed to blue or ultra-violet light. But while you're measuring it in the machine, and expecting it to be able to show excited electrons for hours, if you shine just a burst of red light at the tissue while it's in the machine, it quenches those excited electrons and they go back to the resting state. That's apparently what's happening in the rat experiment, showing the copper by absorbing the red light, puts the electrons back in the proper arrangement.

JR: *Can you talk a little bit about circulating cholesterol, and correlate it to vitamin D and calcium absorption ? Is there a correlation ?*

RP: No correlation that I can think of. Toxic levels of vitamin D is one of the things that can injure the blood vessel wall. But, at physiological levels, the possibly interacting through parathyroid hormone would be an indirect way that they would relate.

JR: *It is possible to live entirely without eating fats, as the body can make all the unsaturated fats it needs ? Therefore, is there any importance in consuming saturated fats ?*

RP: One thing is that makes the food a lot pleasanter to eat. It makes it digest more efficiently and steadily. Experiments with a loop of intestine…they would put just proteins, or just carbohydrates, or just fats in at a time; they found that the digestion was very poor until you had all three types of food present at the same time. It was as if the intestine needed a complex stimulus before it would really effectively start absorbing and digesting the food. So it's partly a stimulus to your intestines to handle the protein and the carbohydrate effectively. It's a signal of satisfaction, that helps to lower stress, to have fat and sugar in your food.

JR: *Should, yes or no, a person live on a fat-free diet (because the body would be working more efficiently) ? Or should people simply increase their saturated fats intake ?*

RP: Yeah (the latter), largely because of the effect on the taste system, and the intestine reflexes; it helps to handle the other foods efficiently, and to make the whole body recognize that it's being fed properly. So it's part of the reflex nervous system that guides eating. And it helps to satisfy the appetite, so people feel more satisfied when they had fats, especially saturated fats. In the experiments with rats (they used a purified diet), when saturated fats where added, they had similar cancer free results; it's the very small amount of unsaturated fat that is responsible for the stress and cancer production. The equivalent of just about a teaspoonful of unsaturated fat per day is enough to show a threshold increase in the incidence of cancer. When we eat natural foods, where're always getting some of the unsaturated fats. On a normal diet it's hard to get down to that threshold of about 4g of fat per day. It's hard even eating coconut oil and butter fat, and beef fat, and so on (they only have about 2% of unsaturated fats). So, besides eating the most saturated type of fats, that's one of the arguments for using carbohydrates as a major part of your

energy supply. Because if we have some extra carbohydrates more than we need to burn at the moment, they'll turn into saturated fats and extend the proportions. So that in effect you can lower the unsaturated proportion below the threshold of carcinogenic fats.

Caller: *My friend has contracted AIDS, and I have recommended him aspirin and saturated fats, as you wrote. But his latest blood work showed decreased CD4 and CD8. What should he do ?*

RP: What about niacinamide? People with HIV often have a very high polyunsaturated fat circulating. And niacinamide is something that will help to lower the free fatty acids.

Caller: *If you lower the free fatty acids, will the CD4 and CD8 increase again? Because his doctor advised him to begin tri-therapy. I've told him to eat a lot of coconut oil, but that doesn't seem to help his CD count now.*

RP: I think so. Sugar, niacinamide and aspirine are all things that'll help to lower the stress and keep the immune system up.

Caller: *Would you suggest 15 aspirins per day?*

RP: Around that.

Caller: *Ok, I'll tell him. Thanks.*

JR: *Can you elaborate on why you're such a huge proponent of coconut oil ?*

RP: Any of the saturated fats have an anti-inflammatory, protective effect. A group studying liver disease has found that the fish oils and shorter seed oils (unsaturated forms) increase liver inflammation and tendency to become fibrotic and cirrhosis, and that can be blocked by the saturated fats. I think it was an Indian that noticed that alcoholics in India who lived in the areas where they had ghee or butter as their main fat, didn't develop liver cirrhosis despite being alcoholic. They began testing that, and saw that alcohol activates the unsaturated fats to react with iron to break down, and produce the liver damage. So, all of the saturated fats are protective when you have an inflamed situation. And that goes all the way up to the waxes, such as extracted from bee's wax, and sugar cane, and such, that are super long-chain saturated fats. Coconut oil is in the medium-chained lengths, that includes some of the very short-chain saturated fats; mostly it's 14 and 16 carbon chains. The shortness of the chain means that it's very mobile in your system. And the shorter saturated fats can be handled in the mitochondria without relying on the transport systems for handling 18 carbon chains for example. The 10 carbon chains can be oxidized as easily as glucose. And so, instead of interfering with glucose metabolism and switching the whole mitochondrial function, they can participate and even activate the glucose oxidation. They interfere with the anti-metabolic effects of the unsaturated fats. By interfering with the anti-metabolites, they let the mitochondria run at full speed; and that works as if you were giving a thyroid supplement. The unsaturated fats interfere with all of the effects of thyroid; all the way from the gland secreting the hormone, the proteins transporting thyroid hormone, and the cells responding to it. So, at all of those points, coconut oil is probably getting in the way of the suppressive effects of the polyunsaturated fats. But especially in the mitochondrion, where the coconut oil itself is being very quickly burned and used as energy.

JR: *So, saturated fats help to protect and detoxify the body from unsaturated fats; they help with glucose oxidation, and enable the liver to store glycogen and thus regulate blood glucose. They are pro-thyroid, and anti-inflammatory.*

RP: Yeah. Speeding the metabolic rate, that's the most important thing that thyroid does. And sugar and coconut oil work right with it to maximize the good metabolic oxygen consumption.

JR: *Working with our clients, in order to lower their cholesterol we've actually increased their saturated fats. It definitely works. Can you elaborate on Houssay's coconut's oil effects on the pancreas ?*

RP: He was experimenting at the time he was working; he was just discovering the multiple factors that regulate blood sugar. And he saw that the pituitary would activate the adrenals to increase cortisol and raise blood sugar, while the pancreas was making insulin to lower the blood sugar. The coconut oil and sugar work very similarly to keep the system working; to allow insulin, both to function and to be produced. He didn't know at that time, there were a lot of things about the interactions of the pituitary, sugar, adrenaline and so on that hadn't been discovered yet. But working with just those few elements he was one the right track.

JR: *You're a huge proponent of milk; most nutritionists say it's quite inflammatory, while you sustain it's actually anti-inflammatory.*

RP: I think the calcium content (besides, it contains such a broad spectrum of all of the nutrients)…this proportion of calcium is, I think, what makes it anti-inflammatory. And there are a lot of ways that the calcium is working; one is that it increases the oxygen-using ability of the mitochondria, the same way thyroid does. It specifically activates some enzymes (the uncoupling proteins in the mitochondria) that turn on a higher metabolic rate. But systemically, it's suppressing the parathyroid hormone. And the parathyroid hormone is another of the short-range protective adaptive hormones that, when it continues too long, becomes counter-productive and contributes to degenerative diseases. People on kidney dialysis tend to get very high parathyroid hormone; and because of an imbalance of phosphorus and calcium. And the parathyroid hormone is what eventually tends to kill them, causing all kinds of stress reactions, aging reactions, to be intensified. Having a chronically high calcium intake gives you a chronically low parathyroid level. So the parathyroid isn't taking calcium out of your bones and tending to put it into your arteries, and kidneys, and so on. Keeping the parathyroid hormone down protects your arteries and kidneys and bones, and allows your insulin to do what it should to keep the free fatty acids under control. And the whole system tends to function in a smoother way when you keep these stress hormones minimal.

JR: *Dairy being pro-thyroid, it helps with the conversion of cholesterol. Is that because of the progesterone in it?*

RP: That's just one minor factor. Human milk, at least contains enough progesterone and thyroid to meet the baby's needs. So that babies that are born without a thyroid gland, if they are being breast-fed, will not have any hypothyroid symptoms until they are weaned. Human milk has a full supportive endocrine system in it for the baby. Cow's milk isn't as appropriate for human use, but it still has some of the metabolism-supporting hormones, as well as the nutrients.

JR: *Can you elaborate on why eggs are an important staple in people's diet ? Dr Brewer talked about that as well.*

RP: With the industrialization of the egg industry, 40 years ago or so, the cholesterol content of eggs went down quite a bit and the polyunsaturated content went up. So, if you can get non-industrial eggs from chickens that have pastures and can eat varied food, the eggs are going to be a lot better. But the cholesterol in the egg seems to be one of its important nutritional values. Something like 600mg of cholesterol in your diet from some of the experiments, it increases the oxidative stability of your lipid particles in your blood stream, makes them less atherogenic.

Danny Roddy *(calls in) : A lot of people doing Paleo (high-protein, low-carb diet) are under the impression (from research) that free fatty acids in the blood is a good thing. Can Ray explain how these free fatty acids can actually harm the liver and thyroid ?*

RP: That's my main argument against eating a high meat diet. That's one of the important functions of sugar, I think, because my next newsletter in 4 months will be talking about free fatty acids. But, basically, one of the means by which stress causes it's damage… I've known people who were eating 2-3 pounds of meat a day and who were getting sicker and sicker as their free fatty acids and free amino acids increased. That started me reading more about the free state of fatty acids in the blood. Just about everything that goes wrong, involves free fatty acids increase. If they're totally saturated fatty acids, such as from coconut oil and butter, those are less harmful, but they still tend to shift the mitochondrial cellular metabolism away from using glucose and fructose, and turning on various stress-related things (by lowering the carbon dioxide production, I think, is the main mechanism).

Danny Roddy: *Amazing! Thank you, Dr Peat.*

JR: *What has cholesterol to do with mitochondrial respiration?*

RP: First of all, it stabilizes it. It protects against the free fatty acids that are one of the disruptive influences. Besides stabilizing it, it actually stimulates respiration, oxygen consumption (which might be just because of that stabilizing effect). But it functions as an antioxidant, partly by the way it interferes with the free fatty acids. Cholesterol associates with proteins and nucleic acids, as well as other lipid materials. I think the way it binds to proteins is responsible for its stabilizing effect in the mitochondrion. The proteins that make up the mitochondrion have a very lipid property, so that you can use an oily solvent, remove all of the oil-soluble materials and you still have a mitochondrion there. But that shows that the apparent mitochondrial membrane is really a protein-structure membrane, rather than a fat membrane system. And that protein structure is reinforced and stabilized by binding with cholesterol molecules. It stimulates the whole respiratory process, which includes conversion of some cholesterol to the hormones as well as producing carbon dioxide.

Caller: *Why many people have stomach aches when starting eating coconut oil ? Is it because the salicylates in it ? Should one use instead refined coconut oil ?*

RP: I recommend the completely deodorized kind. The research was always done with the filtered kind, that had no odor at all. Even though it tastes really good to make ice cream or cookies or something out of the very aromatic kind. For safety in

general use, because some people are allergic to the aromatic tasty things in the coconut, making it completely odorless I think is safest.

Caller: *Isn't that too much processing?*

RP: They just pass it thruough a diatomaceous earth or –inaudible-. So it's just a filtering process, no chemical treatments.

Caller: *When we had the regular virgin one, I always ended up feeling kind of achy or sick, like some inflammation from fermented foods, and didn't know what was going on.*

RP: It just takes a spoonful of the very odorous stuff to make me sick for about three days.

Caller: *Ha ha. How much a day should an average person take ?*

RP: A tablespoon with a meal was enough to make me breathe harder, get pinker, have a higher pulse rate and lose about I guess it was 1-2 pounds a week.

Caller: *Thanks. Have a great day.*

JR: *Some people aren't used in eating saturated fats. And they might have an overburdened liver, biliary insufficiency, etc… So, increasing saturated fat too fast…Could it be due to CCK release (Cholecystokinin), causing this nausea ?*

RP: Low thyroid people have very touchy gallbladders, and it just takes a little grease to trigger a gallbladder reaction. And you've got to get their gallbladder in good condition. Getting the estrogen down is a basic thing. But low thyroid people, a very high percentage of them have gallbladder disease.

JR: *Yeah. I remember reading that in Dr John Lee's work, a long time ago. He found a huge correlation between gallbladder surgery and estrogen dominance.*
Ok. Another caller's asking you on your views on Butyric and Propionic acids?

RP: Those are produced in the intestine by fermentative bacteria and they have such a protective effect against cancer that people are actually selling butyric acid pills for cancer treatment about 20 years ago. But the stuff smells so bad that some people prefer the cancer to the treatment. They sold tongs to pick up the pills before you swallowed them so you wouldn't have to touch such a stinky material. It's one of the components of milk, butter, coconut oil; the 4 carbon fatty acids metabolize into that when they are released from the glycerol in the triglyceride. So it's just a trace of it in that gives you the buttery smell when you eat butter. Butter gave butyric acid its name. But since it's produced by bacteria and since people with very good health have no bacterias in their small intestine… And the further up in your intestines that you have bacteria, it's the likelier you are to have a lot of digestive and other health symptoms. Low thyroid people have slow digestion and the bacteria tend to occupy most of their intestines. So, from the fact that the healthiest people don't have bacteria, and the butyric acid is produced by fermentative bacteria, it should be limited to the colon. And preferably the materials that feed the bacteria should've been digested and absorbed in the small intestine. I think, ideally we shouldn't be making any butyric acid in the intestine. Both butyric acid and propionic acid have some pro-inflammatory effect as well as their anti-cancer effect. Even though, they

are potential cancer therapies, ultimately for minimizing the intestinal production of them…

JR: *Ok. They are found mostly in the large intestine ?*

RP: No. When you have bacteria living up in your ileum, a lot of it is produced there.

JR: *Ok. Regarding the risks of lowering cholesterol, what are the risks ? And, most people don't know the implications of taking a statin.*

RP: The risks are tendency to become violent , suicidally depressed, and an increased tendency towards dementia. Also, making your red blood cells less stable. Everything becomes less stable. The cholesterol is an intrinsic part of chromosome and DNA function, and of cell division regulation. The first things people notice would be the emotional effects, probably. The statins, they have some beneficial anti-inflammatory effects. But at the same time, they do lower your cholesterol and they interfere with that whole chain of synthesis. So, they affect your coenzyme Q10 production, and anything in that system. And so, that probably is a factor in why they cause bones to rot and muscles to break down catastrophically; muscle pains and cramps are very common in people who lower their cholesterol very much with the statins. And that can cause what they call the rhabdomyolysis, where the muscle is basically dying. I think it's a similar process that causes what they call phossy jaw. The interference with our cholesterol; it affects everything.

JR: *Do you see a decrease in libido and hormonal function ?*

RP: Yes, I think that is one of the early things.

JR: *Ok. And it's interesting that some of the most common drugs are SSRI's and libido-enhancing drugs, as well as cholesterol lowering drugs. So it's kind of a little circle going on. So, to summarize, what are the things people can do to help regulate their cholesterol ? More specifically, to lower it, as most people complain of high cholesterol ?*

RP: A daily raw carrot will tend to lower your cortisol and estrogen. That, by stopping the stress, will let your thyroid work better and increase progesterone. That should accelerate the conversion of cholesterol into more progesterone and protective things. Avoiding the polyunsaturated fats is essential for your thyroid to recover. Everything that increases your metabolic rate, as such as calcium and sodium, are essential.

JR: *What has Vitamin C has to do with lowering cholesterol ?*

RP: It's part of the adrenal and ovarian systems. I don't think it has a direct synthetic relation. It's just part of the adrenal, probably having an antioxidant function when the cell is excited. I think it's like a general adaptogen, rather than a synthetic catalyst.

JR: *Anything you want to add about cholesterol and saturated fats?*

RP: The important thing I think is to not think of the unsaturated fats as anything essential. That has never been proved. What the Burrs helped proving was that the unsaturated fats lower the metabolism, so that the so called deficient animals had a

50% higher metabolic rate. That means that all of your nutritional requirements will increase when you shift to a pro-thyroid, saturated fat diet. The increased metabolic rate will increase your need for copper and selenium, the B vitamins. I'm not sure of what the effect on protein requirement is. The protein can contribute to that same intensifying metabolism effect, so I don't know what the ratio should be between the saturated fats, proteins and carbohydrates. But it probably varies with the individuals and stress level. For example, when you're at rest, your muscles can burn pure fatty acids. But as your level of activity increases, the proportion of glucose oxidation increases, decreasing the use of fats; so your activity level governs the amount of saturated fats or carbohydrates and protein that you need.

JR: *It's the opposite of what we've been hearing. People have to make a change in the way they've been eating up to now.*

RP: You probably have been hearing about the woman doctor who treated her husband's Alzheimer's disease with coconut oil and medium-chain triglycerides. That fits right in with the established evidence that it's the breakdown of the highly polyunsaturated fats that is contributing to the Alzheimer's disease. You can see the chemicals like isoprostanes, the spontaneous oxidation products of the polyunsaturated fats in the brains of Alzheimer's patients. One of the direct effects of the saturated medium-chain triglycerides or coconut oil is to provide the saturated fats to the brain, as well as the ketones and the energy supply.

JR: *Wrap up and closing.*