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AUTOIMMUNE DISEASES AND MOVEMENT DISORDERS

2012, Politics & Science with John Barkhausen

JB - John Barkhausen

RP - Ray Peat

JB: Today we're going to talk about some autoimmune diseases and some movement disorder dysfunctions. I'm intrigued by them, not only because I have friends suffering from these, but also because the medical world offers so little knowledge about the diseases and so little hope or practical knowledge in terms of treatment. I'm very happy that Dr. Raymond Peat is joining me again today. There are many theories as to the causes of the diseases and one common factor that is present in all of them and that most people agree on is inflammation. Ray, why is so little understood about these diseases?

RP: Well, I think there's a lot more known than the public is aware of. The medical journals aren't a good place to look, if you're just wanting to find out how much is known. But if you read widely, in not only medical journals but also general science journals, you see that people have discovered really interesting things about all of them, and that there are patterns that show up across the various diseases...that I think, really, things could be put into practice more than they are. And there are people demonstrating improvement in the degenerative diseases with very simple antioxidant supplements and creatine supplements and such that you just don't hear about in the New York Times stories about advances in health. I think part of it is that when a generic substance looks like it might prevent or cure one of these horrible conditions, the drug industry isn't interested, and so there's no advertising money to be made by running publicity about it.

JB: So nobody pursues it. Ray, can you explain where you're finding out this knowledge of hopeful techniques to combat these diseases?

RP: A lot of it you can find right in PubMed and Google; some of it in more obscure journals, that there's enough to keep a person busy for years, just putting pieces together. Like making a meaningful puzzle out of scraps from the various lines of thinking. Like, if you follow one disease over 20 or 30 years, like Alzheimer's, you'll see there are styles, focusing on the cholinergic nerve death or the accumulating fibrils, amyloid and such, and explaining that as the toxin that causes the disease. And the various different diseases, each one goes through its styles of what they think is interesting, but the pressure on funding research and such pushes generally towards a genetic explanation that makes a simple drug solution conceivable, like something to stop that one genetic defect from taking its effect.

JB: So, when you say "styles", are you're talking about there are certain fads in research that are prevalent at certain times?

RP: Yes, definitely, fads. But the basic, big fad, has lasted for a hundred years: it's a genetic explanation. Like in Huntington's disease, it's a certain repeat that causes a series of glutamine amino acids in the protein to increase. And that creates a protein that shows up as if it's doing damage. And, so, the framework idea is that the gene expresses itself in a protein, and the protein causes the symptoms of the disease. And

so, it's the idea that the gene is causing the disease. But there are several ways of approaching that; one is that something is causing this repeat to be formed in the gene itself. For example, notice that generations, even though typically, Huntington's is thought to set in at the age of 40 or so, they noticed that the children of those people had developed it about 8 years earlier. So, each generation anticipates and starts the condition earlier. So, there's something causing that repeat in protein to increase in each generation. And, that's something that's slow to sink into the genetic causality, that things are happening right now, each generation creating a tendency to mutate in a certain direction. There were many genetic theories that said that mutations do have a directionality. And they used to explain the growth of horn antlers on elks and so on as getting bigger and bigger because of some tendency in the organism to go in a certain direction. Orthogenesis, they called it. That was sort of vaguely anti-Darwinian and inclined towards Lamarckism. And so, it brought out the idea of the defect in the gene that causes it to get worse and worse quickly with each generation. It's more acceptable, because in immunology, that was a solution to how antibodies can adapt so quickly to any conceivable infection or antigen. They said they have to adapt by mutating so fast, that it can evolve in just a few days to match whatever antigen they're exposed to. So, this idea of almost directed mutation got put into genetics by way of immunology. And there are the trailings in a few places: John Cairns and Ted Steele are the people known to be working on the idea of directed mutations in a constructive way, not just destructive.

JB: I'm awfully confused by this, because I think of geneticists as saying, in the past at least, that genes were a permanent thing, that were passed down from generation to generation and could not be changed easily.

RP: Yeah. They have been open to the idea — they're still not seeing it as anything deliberate or constructive, just a way the defect can develop. But it's a way to save the genetic causality, rather than seeing that the same thing causing the symptoms of the disease, might also be causing the genes to change in the same direction. That's what they don't want to see is a link between the way the gene changes, and the function of the protein in the life of the individual organism — that's where it implies Lamarckism.

JB: You mean that the organism is directing the gene mutation?

RP: Yeah. Or, that something is causing the organism, on the cellular, not genetic level, to change at the same time that the gene that regulates that cellular function is changing in the same direction. They shouldn't be coordinated that way, so that the function and the gene change simultaneously, or even with the information going from the function into the gene and then back.

JB: So that would mean that the organism is a purposive being on an evolutionary level?

RP: Yeah, exactly, that's the whole point. Ever since the anti-Darwinians in the 19th century, Weismann in particular, they hated the idea that things could be changing meaningfully or purposefully, and wanted to say that there is no real change, and genes were the way of proving that you might get a different mixture of traits but the traits are eternal, and the gene is what causes that. One of the people questioning this, James Shapiro, was working along in ordinary bacterial genetics, and he noticed that individuals exposed to an antibiotic could become resistant to it, and that they could

pass that information on, very intentionally, to their neighbors. And it could even cross a variety of one bacteria to another, and spread it through whole systems. That got him thinking about this idea of purposive change, and he's proposed that the organism does genetic engineering along the lines of what Barbara McClintock was talking about. But he says this is the general way genetics works in the organism, that the organism is its own genetic engineer, doing changes for its own benefit.

JB: Yeah. I can believe it, because I was looking today at a physiology book, trying to understand the nervous system, because a lot of the diseases we started off talking about are diseases of the nervous system, and it's pretty phenomenal — if you open up an encyclopedia and look at how the nervous system is laid out, it's an awe inspiring system. The idea that some scientists and philosophers think that happened by random evolutionary trial and error seems impossible to my mind.

RP: Yeah, the establishment genetics biology system, including most of medicine, are attacking James Shapiro, with his application of Barbara McClintock's way of thinking. What was your point?

JB: The complexity, the organization of it...

RP: The complexity, yes. Randomness is such a deep part of their way of thinking that they are accusing Shapiro of being a Creationist. He says, well, the Creationists sometimes speak very reasonably, and sometimes the so-called neo-Darwinians don't speak so scientifically and reasonably. So he isn't attacking the science, invoking Creationists, because sometimes their arguments are plausible.

JB: Yeah, I mean I don't personally buy into the father figure in the sky, looking down on us all, but...

RP: No, but he is saying the organism itself, is creating something...

JB: Yeah, that life has intelligence, you've said before, and...

professors were afraid to say they were an atheist or agnostic.

RP: Yeah.

JB: That to me has the ring of truth to it, because as you look around the world and see, basically, as you say, the world organizing itself — it's a good example of the intelligence all around us. And one of the things that has sort of interested me, that Carl Lindegren said in his book "Cold War and Biology", was that in order to practice science back in the 40's and 50's in the United States, it was very helpful to profess some kind of belief in a God, in order to keep your job. He said that

RP: Yeah, all of my professors were church-goers, which used to be....In the 19th century, they tended to be agnostics, biologists specifically. But they really did get a religious boost in the 1940's, with the anti-...they considered it anti-materialist, but what it was, was a different kind of materialism — randomness based materialism — rather than the idea that material is part of the purposive, intelligent, life process.

JB: Bringing this back to our topic today, in talking about the inflammation that appears to be present in all of these diseases we're talking about, whether we're talking about amyotrophic lateral sclerosis, which is ALS or Lou Gehrig's disease, or

multiple sclerosis, which you have written about quite a bit, or rheumatoid arthritis—all these diseases, which they don't really have any known cause or cure for, involve inflammation. Maybe you could outline for us how science has perceived inflammation over the years?

RP: In the middle of the 20th century, there was a heavy concentration on inflammation as a reaction to infection, to the extent that about 40 years ago, 35 years ago, when I mentioned to a recent graduate something about sterile inflammation, she wouldn't let me continue and said "There's no such thing as sterile inflammation". But, in 1900 and before, people were demonstrating that you could extract something from infectious organisms, that would create inflammation, even if it was sterile. And with radiation experiments, they found that radiation ???preceded??? inflammation. Or trauma, completely sterile burns, shutting off the blood supply, creates inflammation. I think the only way to approach inflammation is to think of it as the gap that shouldn't exist between the demands made on the cells or the tissue, and the resources to meet those demands. If you traumatize or overstimulate a tissue, or if you don't provide enough sugar and oxygen and carbon dioxide to meet that stimulation, to hold the stimulation under control, then things go wrong — and the tissue becomes edematous, and chain reactions happen that can kill the tissue. Or, if the organism can manage to recruit enough systems to provide sugar, for example, to stop the excitation, then it can heal — a little inflammation rouses the organism to cause regeneration, otherwise it can lead to fibrosis and atrophy.

JB: So, let's use an example of one of these diseases, like multiple sclerosis: the myelin sheath for some unknown reason, according to the medical authorities, becomes worn away or taken away and the nerves stop functioning and people start having trouble with motor control; they say it's caused by inflammation. How does that relate to your idea?

RP: Anything that causes a lack of energy will cause tissue to swell up, take up water and as it swells up the tissue tries to renew itself. Cells are always renewing themselves in a tremendous churning process of taking down the old stuff and putting up new stuff. For example, someone said that during the night, I think it was 60% of our molecules in our brain, the fat substance, that consists a big part of the brain, 60% of them are totally resynthesized every night. And just an hour after death, a massive amount of the brain substance has decomposed because it isn't constantly being reconstituted. So you have to think in terms of a healthy stable organism as being in extremely intense turnover processes. So if you cut off the energy supply, the first thing that happens is the cell takes up water and that excites the restorative process to run faster. But if it takes up more and more water, that shifts the whole direction and the cells at a certain stage of excitation will de-differentiate and try to turn into a stem cell, to grow new tissue as a healing process. If there's even less energy, then that process stops. When you have just a chronic, slight energy deprivation, you get a chronic slight edema and that edema, one of the things that happens is that the myelin swells up, and while it's being taken down it isn't being resynthesized efficiently. Thyroid, progesterone, pregnenolone and saturated fatty acids are things that support the reforming of the myelin. And when the energy is down — for example, thyroid is low — then you can't make the pregnenolone and progesterone and so you just can't synthesize it as fast as it's being taken down.

JB: So it's really, it's a condition when you're put under stress and you don't have the energy resources to keep rebuilding yourself under that stress.

RP: Yeah, and it's just remarkably similar in the processes in the degenerative brain diseases of aging, or the development of cancer, or of deforming arthritis. Inflammation, chronic inflammation like pancreatitis, hepatitis, chronic kidney disease and so on, all the same processes are involved, just in different proportions of energy supply and irritation or stimulation.

JB: Is that what Szent-Gyorgyi referred to as the condition of being sick?

RP: Yeah. No...that was Hans Seyle.

JB: Oh it was Hans Seyle, thank you, and that's just a shortage of energy, basically.

RP: Yeah, I think that's the gap between stimulation and energy resources. It's used to define excitotoxicity that kills brain cells, but it's really the same process in your pancreas or kidney or skin...anywhere exactly the same energy systems, slight differences in the particular proteins — like in Huntingdon's disease, there's that polyglutamine repeat that accumulates — but it's really just a symptom of an inflammatory state with a particular history that leads to that being a problem. But the fact that it usually waits until you are 40 years old means that — the same with rheumatoid arthritis or Crohn's disease or any of these chronic inflammatory things — they almost never develop in little kids; it takes a while, being exposed to certain environments for each kind of thing to develop. But there are a few common factors in the organism and its environment that are involved in almost all of these: Alzheimers, Parkinson's disease, Lou Gehrig's disease, Huntington's, various nervous-dementia diseases.

JB: MS?

RP: Yeah, and the skeletal, nervous, inflammatory...diabetes even, involves inflammation and the failure to regenerate properly the beta cells. [Beta cells] are being killed in the same way as brain cells are being killed basically. Instead of making insulin as the cells are renewed, the cells are killed as fast as they are renewed so they stop making insulin. But if you stop killing them they can start making insulin again. The same with the brain, if you stop killing the brain it's always in a process of repair and regeneration. I might have mentioned a man with ALS that I talked to about 8 or 10 years ago; 70 years old and he'd had all the best neurologists examine him and he absolutely was convinced he had Lou Gehrig's disease. He was declining the same as other people he met in the neurology offices. He decided to start doing things to stop inflammation and support repair, and he did them consistently for a few months while still declining, but then he stopped declining and within a few months was repaired. It was less than a year of the whole process, and people he had met in the neurology offices when they had the same rate of decline were totally disabled by the time he was totally well.

JB: That's impressive. So, one of the things you've talked about in terms of helping your body rebuild from conditions like this is basically a very simple thing, just keeping your blood sugar up; maybe you can describe what happens when somebody is low on blood sugar — reading you talking about what happens, basically this catabolic effect that happens in your body just from low blood sugar, to my mind is pretty convincing about how important it is to keep your blood sugar up.

RP: Yes. The first thing when your blood sugar falls because your liver hasn't stored enough glycogen to turn into glucose, the first reaction is for adrenaline to increase to try to squeeze more glycogen into your circulation, for your brain primarily. And when the glycogen is absolutely gone, the adrenaline keeps activating the breakdown of fat and provides increased amounts of circulating fats to make up for the lack of sugar. But, after the fat becomes a source of energy, your cells still need some sugar to maintain their basic processes, and so they turn protein into sugar. And to do that, they increase cortisol, which breaks down muscle, skin, thymus gland (thymus is the first to go). And the cortisol will eat up your muscle and skin and immune system pretty quickly to feed your heart, lungs and brain, to keep them alive. So every time your blood sugar falls, you're shifting over to fat metabolism and breaking down protein, so that your muscles are one of the places that store glycogen. So as your muscles get smaller, then more burden is put on your liver to keep your blood sugar steady and that makes your liver progressively suffer, and eventually it gets to the point that your brain isn't getting either the right energy or the right kind of energy. One of the things that happens with aging, is that we progressively, from the time we are born, at birth, we're very highly saturated in our fats, because they've been formed from glucose in utero. And we can only make saturated, mono-unsaturated and omega-9 unsaturated fats when we're supplied with either sugar or protein. But once we start eating in the ordinary environment, our tissues start loading up on the polyunsaturateds from the environment. By the time a person is 40, the brain is pretty full of either the arachadonic acid series or, if they have eaten a lot of fish, there will be mostly the long highly unsaturated fats, mostly the DHA type of fish-oil derived omega-3 fats. And even with a pretty average diet, the old person's brain is very highly biased towards the DHA type fats. And if you look at Parkinson's Disease, their favorite genetic protein — that some people like to say is the cause of Parkinson's Disease, synuclein — is the Parkinson's equivalent of the glutamine repeat of Huntington's, or the amyloid, or tau fibrils of Alzheimer's Disease. Each disease tends to have its own protein that goes haywire. In the case of Parkinson's, it's the alpha-synuclein. And DHA, the fish type of unsaturated fat, causes the synuclein protein to change to its toxic form that appears in Parkinson's Disease. And saturated fats can protect against that. So, very clearly, in Parkinson's you can see the role of fat, inclining the brain towards that degenerative change in the protein. And since pretty much everyone in the environment accumulates these highly unsaturated fats, especially in their brain, but in all tissues with aging, by the time you're 30 or 40, you become more and more susceptible to all of the degenerative, inflammatory diseases, very much in proportion to the unsaturated fats. And you can find the breakdown products corresponding to the seriousness of Alzheimer's Disease or Huntington's or Multiple Sclerosis. The specific breakdown products, such as acrolein, which comes largely from the omega-3 fats, the various reactive break-down products show that these unstable fats are breaking down at an increased rate in the degenerative brain conditions.

JB: I see, and also, in that cascade of bad effects from low blood sugar, after the free fatty acids are released, you said that it actually pulls down your whole thyroid system, maybe you could talk about that.

RP: Yes, a series of studies in France about 30 years ago...25 to 30...showed that exactly in proportion to the number of double bonds in the fat, increasing from a purely saturated fat such as stearic acid or palmitic acid, through oleic acid, up increasing with linoleic, even more with linolenic, and greatly with the 5 and 6 double bonds, each increased double bond impairs the thyroid function at the level of

secretion, transport and response. They looked at 4 different systems, different kinds of response in the cell. But every one of these was impaired in proportion to the degree of unsaturation of the free fatty acids in the blood.

JB: When was that study done, Ray?

RP: In the 80s. In Annals of Endocrinology, a French journal.

JB: Well, they are traditionally the, and up into recent history, the "kings" of using saturated fat in their cooking; French cuisine is known for its use of butter.

RP: Well, the French have fallen for the propaganda against saturated fats, and cholesterol and so on, to the extent that some of their famous fat researchers were convinced that giving a fish oil supplement to pregnant women would make their babies smarter, even though animal studies show that in proportion to the unsaturation of the fat in the pregnant animal's diet, the babies' brains were smaller and less able to learn. But anyway, the French fed some pregnant women the unsaturated fats while measuring the fetus' ability to react to sounds applied to the abdomen. And they found that, contrary to what they believed would happen, the learning was impaired by the diet with more of the highly unsaturated fats. And when the babies were born in line with the animal experiments, their growth was retarded.

JB: Well that seems really immoral, to be testing that theory out on infants and their mothers.

RP: Well, the publicity of the animal studies has pretty much suppressed the fact that these fats didn't have consistently good effects on brain and eye development, but what got publicized was the few studies showing what were interpreted to be "good" studies, and on the basis of that, the baby food industry was allowed to add these things to their powdered milk for making baby formula. But even in the powdered milk, they're so unstable that the breakdown products — toxic oxidation fragments — are just tremendously increased in these baby food additives, but still, the publicity is such that they're promoted as protective.

JB: So currently, as it stands today, baby formula that people are using has the DHA oils in them?

RP: A lot of them do. I don't know if there are some without it.

JB: Yeah. That's a little discouraging. Yeah, I can see adults going along with different fads and trying things out for themselves, but when you start experimenting with infants, it seems like not a very good idea.

RP: One of those things that happens at the same time these unsaturated fats are accumulating in the body, is that the ratio of estrogen to progesterone in the body is increasing, so that by the time a woman is 40, she has...in an absolute sense, her estrogen is even higher than it was when she was 20, but even worse is that her progesterone has decreased so the ratio is shifted very powerfully in the direction of estrogen. And estrogen happens to synergize with the polyunsaturated fats, so that women have more DHA circulating in their blood, and these polyunsaturateds activate the action of a given amount of estrogen, and at the same time, interfere with the production of progesterone and suppress thyroid, which has the same bias; lower

thyroid increases estrogen, decreases progesterone. But the estrogen industry has convinced most doctors that estrogen is good for the brain, and for preventing heart disease and strokes and so on, so that when the Women's Health Initiative pointed out that estrogen supplements increased dementia, heart attacks and strokes — things that reinforced what animal studies had shown — the medical establishment took two or three years to come back and respond and come back and say what must have been wrong with that Women's Health Initiative study to incriminate estrogen in dementia and heart disease.

JB: That's what they're saying now, that the study was wrong.

RP: Yeah, the very heavy propaganda to improve the sales of estrogen which dropped off drastically when that study came out.

JB: That was a very convincing study.

RP: Yeah, especially because it absolutely corroborated, in a not too strong way, but it was absolutely in line with the animal research going back 50 years before that.

JB: And these diseases we're talking about today, they affect women way more than they affect men. I think MS is 10 to 1, and I forget what ALS is, but...

RP: Even Alzheimer's, in the 90's it was already well documented that women had two or two and a half times the incidence of Alzheimer's disease as men. And in spite of that, people who wanted to sell estrogen said, "Well, that's because women's estrogen declines with aging." But, in fact, by the age of 40, it has increased tremendously, and that's when the brain damage is being done by the bad ratio of estrogen to progesterone.

JB: I know you've covered this before, Ray, but explain how it is that people think estrogen is declining when it actually isn't. It's increasing and the tests just don't pick it up.

RP: Well, one thing is that the estrogen is stuck. When it's in the cells working, it's bound to the things they call estrogen receptors. And progesterone's effect...it should rise right after ovulation. There should be this huge excess of progesterone. Progesterone destroys and decomposes the estrogen-binding proteins and activates enzymes that inactivate estrogen, getting it out of the cells. So, if you're deficient in progesterone, you can't get estrogen out of the cells, and it not only stays there, but it even, with its own action, it tends to activate enzymes that create more estrogen, so that your aromatase in your fat tissue and fibrous tissue, and various tissues increases, making fat outside of the ovaries as you age. Even in a young monkey, they were studying the estrogen output in the ovary, and as a control, they used the blood coming from the arm veins, and found that the monkey's arm was producing more estrogen than its ovary was. And that process increases with age. But most of the estrogen, when you're deficient in progesterone, most of the estrogen stays inside cells, working, affecting the cells, rather than getting out into the blood, where it could be excreted, and so the only way you can really tell how much estrogen influence a person has when they're 50 years old, is to take a snip of tissue and analyze its estrogen content.

JB: And on top of our bodies producing more estrogen as we age, we have a huge

environmental load coming down on us because many of the chemicals we use in our modern lifestyle are estrogenic, and plus pollution is estrogenic and...

RP: In the 1930s, before the estrogen industry took off, people were studying what estrogen is and does, and they found that soot is estrogenic, and the same things that produce the estrogen effect produce inflammation and cancer. And that basically it's a process of cell excitation, followed or accompanied by blocking oxidative energy production. And that was pretty much covered up when the estrogen industry convinced doctors that estrogen was the pineal hormone that would prevent infertility and aging and so on.

JB: Yeah. Good for their business and bad for everybody else.

RP: The interaction of estrogen as an excitatory thing, with the polyunsaturated fats, which are excitatory things...these, besides producing inflammation and blocking energy production, they activate other systems. For example, the glutamate; glutamic acid...it's why monosodium glutamate produces brain injury. Because that excites cells to the point that if there's not enough energy supply, the cells will die. Estrogen and the unsaturated fats both activate this glutamate excitatory system, and those interact, all of them, to increase a set of enzymes that the transglutaminase is the enzyme that's involved in celiac disease, the gluten sensitivity disease. And this enzyme is normally involved in maturing cells that are under the influence of stress, as in the surface of the skin when it's maturing into a hardened, keratinized layer. Or in the uterus, as estrogen is causing the lining of the uterus to mature and cause keratinized cells to form. But in the brain, this excitation from unsaturated fats lipid peroxidation breakdown and estrogen and the glutamic acid system — these excite the formation of the transglutaminase. And transglutaminase happens to form polymers and fibrils and deposits of these various enzymes that are known to accumulate in Huntington's, Alzheimer's, Parkinson's, multiple sclerosis and so on. The tau protein, for example, in Alzhehimer's disease, transglutaminase activates a reaction at the end of the tau protein, or in various places with all of these other proteins that accumulate and form fibrils. And this enzyme works on amino groups which, when the metabolism is healthy and producing energy by use of oxidative metabolism, it's producing a constant supply of carbon dioxide, and carbon dioxide spontaneously combines with amino groups. All kinds of amino groups, every protein in the body should have a supply of carbon dioxide, preventing the action of enzymes such as transglutaminase, which would bind them and cause them to condense and form fibrils. And I suspect that in places where estrogen is dominant, or in the skin, where the cells are exposed to purer air, the carbon dioxide is displaced either by the effect of estrogen, or just by the high saturation of oxygen. And the absence of the carbon dioxide allows this transglutaminase to crosslink, inactivate, and harden the protein. When it happens inside your brain, you get these abnormal deposits of protein that should only happen in cells that are terminally differentiating and getting ready to slough off.

JB: And your body's getting rid off...

RP: Yeah.

JB: So CO2 is very important to the health of the body.

RP: There was a survey in Nepal at very high altitudes and they found lots of sick

people, but they didn't find the degenerative brain diseases that you would expect at lower altitudes and poor populations. I suspect it's because when you adapt to high altitude your body retains a much higher level of carbon dioxide. Your blood is more in the carbamino state. Probably all your proteins are.

JB: You've said that reduced thyroid function, not having enough of the active thyroid hormone T3, that's when you start having trouble, not only with energy levels, but also the myelin sheath of your nerves can't regenerate without T3. Is that right?

RP: Yeah. The first five people that I met who had a diagnosis of multiple sclerosis — I was starting to believe there wasn't such a thing as multiple sclerosis, because all of those first five people had such classical symptoms of hypothyroidism. I pointed that out and when they took thyroid their symptoms totally disappeared. It's very easy to confuse hypothyroidism with multiple sclerosis.

JB: So in the few minutes we have left, maybe you could just run through some of the simple things that people could do to alleviate their symptoms or even avoid them entirely.

RP: It's known that aspirin prevents most of these degenerative conditions. People who have chronically used some aspirin are much less likely to have Parkinson's or Alzheimer's. And caffeine is another generally protective thing against inflammation, fibrosis and degeneration. And avoidance of the polyunsaturated fats I think is the basic and most important thing — and avoid the anti-thyroid foods. The worst anti-thyroid foods are these polyunsaturated fats.

JB: And protein levels. Keeping your protein level up.

RP: Yeah. And gelatin seems to have a therapeutic, anti-inflammatory effect. So, eating the cheapest cuts of meat — bones and skin. In the US, few people eat the skin and since the fat avoidance fad that people tend to eat skinless chicken and so on. But when animals are fed a good diet, the skin fat is more saturated. So pork rinds are a very good source of gelatin. And if you boil a chicken, even without the feet and other parts, the skin and bones would release a lot of gelatin, which is an anti-inflammatory, protective protein.

JB: And you mentioned how important it is to keep your blood sugar up to handle stresses. How should one do that? I have a feeling that a lot of problems today, at least in some people, is that they are avoiding sugar, because it's gotten such a bad rap.

RP: Yeah. Every day I hear at least one or two people saying what happened when they started eating sugar. One guy this morning said his hair stopped falling out a couple of days after he started eating sugar. A kid was having seizures and dropped down to 112 pounds in just two or three weeks was back up to his normal weight for keeping something like 8 or 9 ounces of sugar added to his other foods. In crisis, sugar in itself, just a simple kind of honey or sugar, can be very therapeutic. But, in general, you want to shift your diet towards fruit, rather than grains and starchy vegetables.

JB: Just to finish up, you were saying that starches were a bad way to get sugar, like wheat is bad and...

RP: Yeah, partly because of the other things they are associated with, but they...one of the things they do harmfully, is to support bacterial growth. The poorly cooked starches or the more undercooked vegetables and the complex forms of starch support bacterial growth and the bacteria produce endotoxin and endotoxin works with these other pro-inflammatory things. Some of the structural changes of the degenerative proteins are very similar to the structure that is defensive against endotoxin so some of them might be provoked by the presence of endotoxin as a defensive reaction. It's well known that the polyunsaturated fats activate the prion formation and so on but the unsaturated fats are probably biologically analogous to the endotoxin produced by the bacteria.

JB: Yes, and you've written a lot about the relationship of the sort of "mal-digestion" of food creating a lot of the problems that we've been talking about or helping to exacerbate them.

RP: Yeah, and the unsaturated fats contribute even at the digestive level because they interfere with the protein digestive enzymes as well as after they get into the blood stream.

JB: All right. Well, we've filled up the hour Ray, it's gone very fast and I really appreciate you coming on Politics and Science again. And perhaps at some point we can follow up and get into some of the details of how digestion does relate to disease. 'Cause I think that's something that the medical world doesn't cover at all.

RP: Yeah, if you go back to the mid 19th century, you see that medicine was really making progress and then into the 20th century the Russians were continuing digestive physiology. But after about 1920s in the US, it was pretty much ignored.

JB: All right, well thanks so much for sharing your knowledge with us and I'll give out your contact information when we're done.

RP: OK. Thanks.

JB: All right, thanks a lot, Ray. Bye.

RP: Bye.