A Special Interview with Dr. Duane Graveline
Cholesterol and Statin Drugs

DM: Dr. Joseph Mercola, DO
DG: Dr. Duane Graveline

DM: Welcome everyone. This is Dr. Mercola. Today, we're here with Dr. Graveline. We're going to talk about cholesterol and coenzyme Q10 and a lot of the implications of both of those. I'm sure you'll be enlightened with the discussion. Welcome.

DG: I'm glad to be here Joe. I'm always glad to talk about statin drugs, cholesterol, and CoQ10.

DM: You've had quite an experience with this. I'm wondering if you can share with our listeners how you came to develop your expertise in this area.

DG: This all started actually in 1999. At the time of my physical -- I get annual physicals at Johnson Space Center in Houston because I'm a former astronaut. In 1999, my cholesterol which had been progressively rising a bit, was around 280. They were pretty excited about it at that point. I was mildly concerned about it. When they suggested Lipitor 10 (mg), I went along with it because I had no reason to be particularly worried about statin drugs. I had used it a few years, like a year or so before my retirement but I wasn't a big user of it.

DM: These were the physicians with NASA?

DG: Yes, the cardiologists with NASA and the flight surgeons. Anyway, I accepted this. I was concerned slightly about my cholesterol just as they were, as a family doctor. I've been a family doctor for 23 years.

I went home on 10 mg of Lipitor. It was six weeks later when I experienced my first episode of what later was diagnosed as transient global amnesia. This is an unusual form of amnesia wherein you immediately, without the slightest warning, you are unable to formulate new memory and you can no longer communicate not because you cannot talk but you can't remember the last syllable that was spoken to you. So nothing you say is relevant anymore.

In addition, you retrograde. You have a retrograde loss of memory for sometimes decades into the past. In this particular episode, I seem to have jumped to 10 years into the past because my wife who saw me wandering about the yard with an unusual look on my face that morning. When she came out, I did not know who she was and when she invited me into the house, to me it was not a familiar home. I have never been there before. I had never met that blond woman before.
She did offer me cookies and milk and I took those outside on the steps but I would not go in the house. She was concerned about blood sugar for some reason. She plays nurse all the time. Anyway, she managed finally to get me into the car and off to the hospital and, the cookies did no good incidentally. I woke up getting to my senses, at any rate, six hours later in the office of a neurologist as he’s pronouncing the diagnosis of transient global amnesia. Just about that time, I’m normal. You become normal in a flash just as fast or rapid as you go into it, you come out of it.

DM: This occurred in the late 90s?

DG: This is in March of 1999. I say March because I get my physical at NASA every March. I stopped the drug on my own even though he counseled me that statins was not of concern. When I asked him about this drug, I said, “It’s the only medicine I’m on. Don’t you think you ought to give some consideration to this statin drug?” He said, “No, because statins don’t do this.” They’ve been using them for almost 10 years. Statins don’t do this.

So anyway I did not believe him. After 23 year of family practice, I decided to treat myself. I stopped the medicine and went home and was absolutely normal the following year. Now, I did have to go back to Johnson Space Center the following March and sure enough, my cholesterol was exactly what it was before around 280. They really strongly suggested that I go back on Lipitor because statins don’t do that. It was just a coincidence according to every doctor there.

So I did go back. I admit I was concerned but yet, I had to talk to maybe 30 doctors and a few pharmacologists during the interval -- every chance I got really -- and no one has given me any relief. They all said statins don’t do that. I allowed myself to go back on statins but this time I said -- just 5 mg.

So I went home on 5 (mg) and eight weeks later, I had my worst episode, my second and my last and my worst. In this one, I was a 13-year-old high school student. I was a high school student for 12 hours. When they told me I was a doctor and married. I laughed hysterically according to my wife, who was standing there to next to me listening to all this. I didn’t know who she was of course.

I was a 13-year-old kid and all of this is clearly impossible. I was 13, how could I be married? How could I be a doctor? I couldn’t remember anything. I couldn’t have doctored a mouse. This is what convinced me when I finally woke up that something was wrong with the statin drugs and yet the doctors there were, for years after that, still saying that this was just a remarkable coincidence. This took me out of retirement and I’ve been actively involved in researching statin drugs since that time.

DM: Very interesting. So you had a personal encounter of an adverse effect from the use of the statins. For the last 10 years, you’ve been researching that more carefully. I’m wondering what you learned in your journey.
DG: Step by step, I've been learning as I went along. I realized that I did need a book if I was going to be effective in giving this information back to the lay public and to the doctors. So my first book was titled Lipitor: Thief of Memory.

It only took a few more months before I realized that this wasn’t just Lipitor and it wasn’t just memory. It was soon evident to me that all of the statin drugs were involved and all of the full range of cognition was not only subject to adverse reactions but there were many other effects; personality change, and an unusual form of chronic muscle and a chronic peripheral neuropathy. Even in ALS type of thing was soon evident and people were reporting to me. My website very abruptly became a repository for statin users worldwide because of the media expression, the media support I had had from Joe Graedon of People’s Pharmacy.

In trying to reach an explanation, I had called Joe Graedon and asked him if he had ever heard of any unusual reactions associated with statins. Joe directed me to the statin study by Beatrice Golomb in San Diego, California. He also asked me if I minded if he put my story on his column that goes out on a syndicated newspaper column. I said, of course not, the more exposure the better.

Once that went out, over the next several months, tons of emails came in from people who just previously having no idea that all of these things that were happening to them could have been statin related. We got 22 more cases of transient global amnesia just in a matter of a few weeks. We got hundreds and hundreds of cases of cognitive damage, but anyway, my website grew to be a major repository. Ultimately, I got well over 10,000 people who have reported to me from everywhere.

DM: Has this amnesia been documented in the scientific literature now as an adverse side effect from the statin?

DG: Yes. It is now throughout the medical literature and scientific literature, all of these side effects. Things that I warned about 10 years ago are now completely endorsed by research. I have never come up with any blind alleys.

At first, I was very suspicious of this CoQ10 way back when. But then as I went on and realized that some of these chronic muscle cases and chronic nerve cases, if we postulated a CoQ10 deficiency what was our explanation for the fact that many of these people did not improve better than half of them. They stayed just the same regardless of how much CoQ10 you gave or whatever form you gave it in.

With that, it made me look for some other cause. This is where I realized that it was the mitochondrial damage and mutations that were the real endpoint of statin damage secondary to CoQ10 but once formed, all the CoQ10 in the world wouldn’t change things.

DM: Let’s step back a bit because the most common side effect from the statins doesn’t appear to be amnesia. I mean, it’s well documented prior to this that the breakdown of
the muscles is a really clear and potentially dangerous side effect from taking the statins. I think it’s called rhabdomyolysis. Maybe you review the typical side effects that people can receive. That clearly were not disputed and that were most likely also related to Coenzyme Q10 so if you can discuss that it would be great.

**DG:** There are three major classes of side effects on the statin drugs. You’re absolutely right that cognition, confusion, disorientation, forgetfulness, dementia are not the same mechanism. At least I don’t think they have the same mechanism. The cognitive appear to be a cholesterol deficiency. You’re just being given a statin and my cholesterol plummeted from 280 down to 155. I was overjoyed to get that first one back and then it was only the next day that I had my condition of transient global amnesia. This is due to cholesterol insufficiency. You’re going along at whatever your normal is suddenly after you take a statin, your cholesterol has dropped down, 40-50 mg. This is abnormal for your body.

This mechanism was sort of going around in my head and then it came out in research actually in the year 2001 how valuable cholesterol was in memory formation. This was something we didn’t know during the initial phase of statin marketing. We didn’t have the slightest idea what this would do to cholesterol and the brain because we didn’t understand the cholesterol value to the brain. So all of the cognitive problems are secondary, I say most of all the cognitive problems are secondary, to this lowering of cholesterol below normal for your body.

Then we have another whole group that has to do with dolichols. You recall when a statin is used statins block the mevalonate pathway to get at cholesterol inhibition. It works very beautifully. But in so doing, it’s inevitable that they will block CoQ10. Dolichols, as well as other major biochemicals, but these are the two I want to focus on -- dolichol is one that most doctors have never even heard of before. But it just so happens that dolichols are almost as important as CoQ10 and cholesterol in cell processing.

**DM:** Could you spell that for our listeners.

**DG:** D-O-L-I-C-H-O-L.

**DM:** I’ve actually not heard of that before either.

**DG:** Dolichols are absolutely vital to the process of glycoprotein synthesis. You and I are doctors, we never even heard of this in medical school. At least I didn’t. As I started poking around in the biochemistry books and I realized what is going on.

When you start reading on glycoprotein synthesis you read how valuable the dolichols are and you see it has to do with cell identification. It has to do with cell communication. It has to do with immunodefense. It has to do with the process of neurohormone formation.
All of the hormones that describe our mental condition; emotion, mood, that kind of thing, are all produced in the endoplasmic reticulum and passed to the (indiscernible 15:34). All of these are produced under the supervision or orchestration, I say, of the dolichols. If you don’t have sufficient dolichol that whole process of neurohormone production is going to be altered.

So all of these reports -- there are thousands of reports we’re getting now of aggressiveness and hostility and increased sensitivity and paranoia and depression and homicidal ideation. These suicidal ideations and the dozens and dozens of suicides that we’ve had where the surviving members of the family will write us and say, we know this can’t be proved but we’re absolutely sure that drug he started was the cause of our son’s suicide or daughter’s suicide or whatever.

All of this whole range of what I call personality or emotion and behavioral responses have to do with the dolichol deficiency that is brought of by the mevalonate blockade. It’s not just something that occurs on an occasional person. Everybody has the mevalonate blockade and most I suspect will have some element of dolichol because that’s the major route of production. That’s where our dolichol is synthesized.

You know we’re all the same and yet we’re all different. We all know that as doctors. You give one medicine to 10 people and you’re really lucky if six of them do what they’re supposed to do. That’s the way it is with this. I expect there are some people that won’t get any effects of dolichol suppression because they have alternative pathways. The same thing probably holds for CoQ10.

But anyway, the dolichols are associated with all of these neurohormonal things, the emotion and behavior. And then you get down to what we talked about originally, the mitochondrial dysfunction and yes, that’s the muscle. That’s the nerve. That’s the ALS.

I have ALS. I’m in the terminal phases now. I’m still able to walk but I supposedly use a walker all the time. This has happened in the last three years and it started coming on almost two, three years after my first exposure. And then I went to a phase of increasing muscular weakness and fatigue that I couldn’t explain. Suddenly, I was an old man. I couldn’t believe how have it occurred in months instead of years. But I have hundreds of others just like me that are now in wheelchairs.

I have over 500 people who have an ALS-like condition. It was brought on by this statin drug that are associated with statin drug intake and they’re now in wheelchairs so I consider myself lucky, I’m not quite there yet.

Now the World Health Organization reported on this in July of the year 2007 when Ralph Edwards who directs their Vigibase in Uppsala, Sweden. Vigibase is their FDA, basically, their MedWatch. He reported excess ALS-like conditions seen in statin users worldwide.
Now, our own FDA, “Who proved this?” They said, we haven’t seen a thing in our clinical studies. Maybe their clinical studies weren’t setup to see anything. I can’t explain that. All I know is I’ve got over 300 ALS cases that have recently been reported to me and I’ve gotten a few hundred more from Joe Graedon and his repository system.

We have forward all of ours to MedWatch so they will have the benefit of the full load. But still, FDA has not seen fit to concur with the World Health Organization and has not written a letter warning to the practicing physicians to watch out for this kind of a reaction because we have some anecdotal evidence that if you stop the statin drug early enough in the process, some of these cases regress. That’s why we thought it was important that FDA warned but they haven’t warned.

**DM:** So it’s your belief and conviction that the ALS you have was caused by the statins?

**DG:** Yes, there is no doubt in my mind.

**DM:** You also refer or implied that this is a terminal condition that there is essentially no reversal at this point.

**DG:** Well, I’m still alive. According to the good books on ALS I shouldn’t be alive but that’s why we’re calling this ALS-like. It doesn’t act quite like the basic. That ALS-like is a phrase coined by Ralph Edwards when he published it. He recognized that it wasn’t quite the same.

**DM:** It appears that if you are able to recognize this early on, after starting the statins and discontinue that you could avoid the progression. It sounds like you are on it for a few years before you stopped?

**DG:** Yes. If you stop the statins early on, but in my case, I had stopped the statins after only a few months, a total of five months of use. Two years before my first inkling so it still came on.

**DM:** So in your case, five months was enough to cause it. We may be talking about literally early enough maybe weeks.

**DG:** It seemed like it triggered something. I have to say that regarding FDA and the subject of transient global amnesia, right now, over 2000 cases of transient global amnesia associated with the use of the statin drug has been reported to FDA’s MedWatch just for the drug Lipitor and all of the other five drugs do the same the thing. They are all reductase inhibitors.

All statins work the same way so their range of side effects is bound to be very similar and they are. They have yet to report this observation, all of these cognitive dysfunction cases. They have yet to report this to their doctors so they can warn them that this is possible. So we have somewhere in this range of probably 80,000 reports that are in if
you added all the statins together in their MedWatch reporting and it’s greatly under reported.

DM: Sure. So there is loads of people who are affective by this. I’m wondering what your speculation as to the cause of actually the mechanism.

DG: There is inhibition of cholesterol and the memory apparatus requires cholesterol in abundant supplies to be processed, I mean, to function in the formation of memory. It even uses the glial cells as a factory for producing its cholesterol on demand.

Actually, these glial cells are just as subject to statins as the liver cells or any other cell in our body. So if you take a statin and they don’t function anymore so that on-demand cholesterol capability no longer exists for your brain function.

DM: This is not related to the mitochondrial dysfunction?

DG: I don’t think so and yet I know that in some of the cases, the brain is being damaged just like the skeletal muscles. They’re all the same. The brain is a major user of energy and I know it has to be due to that as well. But it’s clearly some of the brain symptoms I thought most would be due to this cholesterol inhibition. I could still be right but I have to admit, it has to have mitochondrial damage as well.

DM: Can you review the mechanism of how the statins cause damage to the mitochondria?

DG: Basically, think about mitochondria and realize that it has CoQ10. CoQ10 is going to be the key here. CoQ10 is the key to mitochondrial damage. We have CoQ10 involved first right in the structure of complex 1 and complex 2 of the electron transfer process.

So every mitochondrion contains CoQ10 in its very structure. That’s complex 1 and 2 of these four complexes that pass electrons along to form ATP. This is one of the roles by which CoQ10 affects function and the second role is that we constantly are producing free radicals. After every meal, we inevitably produce free radicals. CoQ10 is present right there and positioned right within the mitochondria to meet the needs of most of these free radicals.

The free radicals, you deserve a word of explanation there because they are made in the free radicals because they have just unwillingly given up an electron. Electrons have been stolen from these and they have become free radicals looking for an electron so they can achieve electrical neutrality again.

So that’s the reason that they’re free and they’re angry and they’re flying about and they’re snatching anything, an electron, from anywhere they can. Unfortunately, it’s very easy for them to snatch electrons from proteins and fats and whatever else exists in the mitochondrial DNA chain. It’s right there.
DM: And the DNA too.

DG: That’s mitochondrial DNA. So immediately, that’s where they all go. Any viable changes of CoQ10 will go up there. These free radicals, first of all, CoQ10 disables most of them. That’s its function. It disables most of them by giving them electrons but a lot of them get through.

In cases of CoQ10 deficiency, all of them get through to go into the mitochondrial DNA. That’s the only place they can achieve electrical neutrality is to steal an electron from a DNA chain and altering the chain immensely.

So we have these two roles of CoQ10: an ATP fuel production, an electron transfer, an ATP and this mitochondrial damage secondary to excess free radicals. We’re bringing excessive oxidation into mitochondrial DNA that’s really what’s happening.

So now you curtail that CoQ10 because you’re now preventing its synthesis at the mevalonate pathway. Because of the mevalonate blockade, you’re cutting out 50, 60, 70, 80 percent of your CoQ10. No longer does it function in this capacity which is why you’re getting all the weaknesses. Gradually, as the CoQ10 damage bails up, you’ll get mitochondrial mutations. As the mitochondrial mutations build up, in a matter of time, you lose your mitochondria. So soon, your mitochondria will begin to drop off.

So a cell, instead of having a thousand mitochondria working to produce for it, will now only have 500 or 250. It could be a muscle cell. It could be a nerve cell. It could be a heart muscle cell. It could be a brain cell. It all works the same way. Sooner or later, that cell is going to fail, and then another cell, and another cell. So pretty soon, we have a tissue response. That’s where your heart failure comes in and your muscle pain and your muscle failure. That’s the mechanism.

DM: Are there any other antioxidants that could substitute for Coenzyme Q10?

DG: Yes. All of the antioxidants we regularly take in supplements and then the body has some it regularly produces. Superoxide dismutase and glutathione are two standard things that the body produces in adequate amount. But, CoQ10 is still in that primary position to make a difference. It’s right in the mitochondria.

DM: Just to clear things up too -- so it’s really ideally suited for this. Coenzyme Q10 really isn’t a vitamin per se because it’s made by our liver. So by the strictest definition of a vitamin it’s something our body doesn’t make. So our liver makes this coenzyme Q10 and supplies it through the blood circulation to all the cells of our body. Is that the correct process or is that how it’s occurring from your understanding?

DG: I’m sorry. I’m not hearing everything. Are you talking about the synthesis of CoQ10?
DM: Right, because some people believe that they might be able to consume CoQ10 from the diet but this is really something that the liver is primarily responsible for producing.

DG: According to everything I have read, I don’t see that this is right. According to everything I have read, every cell produces CoQ10 and it produces it via the mevalonate pathway.

DM: So it’s not just the liver, it’s every cell that has that pathway.

DG: Every cell delivers its cholesterol, CoQ10, dolichols, any of that. Basically, it’s produced at the cellular level. I don’t think the liver is involved in all of this.

DM: Okay, so I was confused on that. There are other antioxidants you can substitute but primarily its something that your body is designed to produce. It’s not something you’re designed to consume, is a food or…

DG: That’s right. CoQ10, as you well know, it’s produced liberally when we’re young. By the time we get to be old people we produce none at all. This is kind of interesting to think about.

DM: Now, the other component that you allude to is this age related differences. My understanding of one of the challenges associated not only with the production of CoQ10 but the form of CoQ10. Typical CoQ10 is in the oxidized form. In other words, it cannot donate a free electron to serve as an antioxidant. But it’s actually the reduced form which I believe is called ubiquinol that can actually serve that antioxidant protection. Is that your understanding also?

DG: This is my understanding and of course these are ratios and different forms are extremely important. Being able to measure these is still a challenge for most laboratories. We’re just learning.

DM: I guess the general approach is that statins -- I guess I should give a disclaimer in that it’s my belief that there is virtually no use for statin drugs. In almost every single case with perhaps the rare exception of familial hypercholesterolemia which individuals with cholesterol, fasting cholesterol levels that far exceed 300 may benefit from these.

I would 99.9% of people should not be on these drugs who are taking them which is the vast majority. With that disclaimer, there are still some people who will choose to take them for whatever reason. If they choose to, it would seem the wise approach would be to take a supplement like Coenzyme Q10 or more ideally a reduced version which is ubiquinol.

But even doing that, I mean, you’re going to protect against some of the side effects as you mentioned. But clearly, just from its impact on lowering cholesterol, you’re going to have these neurological cognitive issues which you yourself experienced and have
documented reports from tens of thousands of people who have gone through this. There are still dangers to these that are unrelated to the administration of CoQ10.

I’m wondering, what percentage of the side effects in your review or understanding of the literature and experience could you prevent by taking coenzyme Q10 with a statin drug. Would it be the majority of them, would it be a third, would it be three-fourths, what do you think?

DG: This is something that I think would be -- I think it would come close to a half.

DM: About half.

DG: Yeah. We don’t realize how frequent, how common the cognitive side effects are and we really don’t realize how common the personality, behavioral, and emotional side effects are. Taken together, that comes pretty close to 50%.

DM: That’s a good answer. I was thinking it might be somewhere around there. Ideally, we would not take the statin drugs and implement lifestyle strategies which would include exercise and removing the grains and the sugars and fruits and juices from your diet because that’s a stimulus for insulin which causes your cholesterol to go up. And decrease the insulin levels which would decrease the actual liver’s ability to produce cholesterol and normalize it.

DG: I’ve got something I’d like to ride by you. It’s my philosophy, after 10 years of researching this, that cholesterol is not relevant to the cardiovascular disease atherosclerotic process. It’s just not a factor. I know that this is not widespread yet.

I based this on my growing awareness over the years as we realized that there is something else going on with these statin drugs than cholesterol reduction. We were seeing many cases where the cholesterol was not changed a bit and yet we saw improvements in terms of statistics after seven or eight years of use of statins. We realized there are other non-cholesterol mechanisms.

This is why we begin to think of how important is this cholesterol thing after all or is this a new feature of statin anti-inflammatory feature. This leads in to the Jupiter Study because it fits right in here. The Jupiter Study proved in just one little study, they prove the irrelevance of cholesterol as a screening tool…

DM: Let me respond there because I do believe that’s true in most part at least with respect to the total cholesterol. But, as we both know, cholesterol is subdivided into different fractions - the HDLs and LDLs.

DG: I say it doesn’t make any difference what fraction because they proved that CRP was a much better monitor of cardiovascular disease or future cardiovascular disease and they gave these people -- these are words I don’t want to say but they did gave them all a strong statin in a moderate dose and they got a remarkable change in
cardiovascular disease in the follow up over the next few years by using hard markers. They used heart attacks and they used actual strokes.

This is why I’m solid. I feel very comfortable now by telling people that cholesterol is not the issue. So when these folks with their 280s and 290s come to me I say, forget it. It’s the best thing that ever happened to you. It is a God given function.

**DM:** In my experience in taking care of large numbers of people, tens of thousands, and I’m sure you’ve seen that many yourself as a family physician that when I implement changes in a person who has a distorted cholesterol, less than ideal cholesterol pattern, and these changes would include dietary changes and exercise, you would see not only their weight drop but you would these ratios improve. It’s my belief that this cholesterol is not necessarily the cause but is a marker for changes that are occurring in the body whether that’s inflammation or some other mechanism. It still seems to…

**DG:** If you say ‘not the cause,’ I completely agree with you. That’s when it started out in 1955. It’s associated and all of them are talking, and in a couple of years it became causation. It’s horrible what we would treat it to.

**DM:** Which is why I don’t recommend any agents to lower it even niacin which is a vitamin. But still, I recommend that it useful index to follow and monitor because -- it’s the lifestyle changes, the exercise and the diet, that will tend to optimize these and when those are optimized -- because cholesterol maybe a reaction to endothelial injury within the blood vessels. So if it’s going up then it means you got damage somewhere…

**DG:** It’s part of the reaction. It’s part of the healing.

**DM:** Right. So if something is causing it then if you are able to change that and you see that improve then you know you’re making progress in the right direction.

**DG:** Okay. What I didn’t say about the Jupiter Study is all of these people in this study, the several thousands involved, they all had normal to low that is under 130 cholesterols.

**DM:** Which we know is dangerous. I have a congenital condition called thalassemia which results in very low red blood cell counts because there is a rapid turnover because one of the molecules of hemoglobin isn’t functioning properly, the chains. As a result, there seems to be an association with this that anyone who has thalassemia has very low cholesterol levels, the typical is below 150. So my whole life, I have had my cholesterol levels under 100 many times. That’s not a good thing. I mean, you need cholesterol. It’s part of what we require…

**DG:** Cholesterol is the most valuable biochemical in your body. I feel sorry for you having to worry about the levels. In my case, I’m happy.
DM: I’m actually eating somewhere between six and eight eggs a day now. I was actually able to get my cholesterol -- I just got it back this morning to 200. So that’s about the first, I think it’s the first time in my life ever that I have my cholesterol at 200 which is delightful for me.

DG: Wonderful. Mine is 280 and I would be seriously concerned if it started going down.

DM: That’s a good sense of reassurance.

DG: Don’t laugh. It’s got nothing to do with atherosclerosis. This is a wonderful feeling to be in. I wish I were not 80 years old because I’m getting too old for this stuff.

DM: But there still may be some value to seeing it normalized and optimized as I mentioned earlier with some lifestyle changes. There may be benefit to engaging in an exercise program designed to lower your insulin levels and also eating foods which would do that and as a result you may see some changes in the cholesterol patterns.

DG: When I talked about this Jupiter just a minute ago, my intent was to bring out the fact that even in this new almost modern day approach to inflammation being the cause of atherosclerosis, CRP being the measurement, that drug companies are still throwing statins at us as the logical treatment and they should approve this.

They’re started using a...they call them statin. Did it really (indiscernible 40:34) great job and we can’t deny it. It works via nuclear factor-kappa B inhibition. This is a different inhibition from that of any of our other anti-inflammatories. So a regular anti-inflammatory like ibuprofen and naproxen and all of the other NSAIDs that won’t work. Another (indiscernible 40:58) the anti-inflammatory, the one we have left, I forget its name, that won’t work.

None of the standard anti-inflammatories will work the same way as this nuclear factor-kappa B inhibition works and only the statin drugs do this. This makes it look really good for the use of statin drugs; however, statin drug side effects give us all these bad things. It gives us all of these effects on the mitochondrial mutation thing that we really have to worry about. So it’s going to be really interesting to see what happens in the future.

Why would a person for example use a cholesterol lowering dose in the future now when cholesterol is not the enemy? They’ve got to come up with an anti-inflammatory lowering dose and we don’t know yet what that dose is. Is it only 2 or 3 mg? I’m betting that it’s going to be that. Each of the directions that we’re really wondering about now in which way it’s going to go but it can’t stay the same. Now that we know inflammation is doing it, what is the anti-inflammatory dose, if we’re still going to use statin? What we’re really saying is that we need something that does the same thing statins do without the side effects.

DM: Normally, the typical approach that seems to make the most sense is to use natural products since our body is used to those and they’ve been exposed to them for
many generations and as a result, the likelihood for side effects is pretty minimal if non-existent for most of the cases.

**DG:** Right.

**DM:** When taken in normal doses. An example of this that’s commonly perverted I think is the administration of niacin in high doses, you know, hundreds of milligrams maybe even gram quantities.

**DG:** I was wondering what your philosophy is on supplements because this is where I’m turning now. There are certain supplements they seem to be the only thing that you can really do. Take mitochondria, there are about 10 or a dozen…I’ve selected 10 or a dozen. There must be 50 different supplements that are involved in mitochondrial maintenance but we can’t give them all.

**DM:** Sure. I would like to discuss that but just to answer your question, you know, I like to be a purist about it and believe that supplements are just what the word says, they’re a supplement. That means they are in addition to, not in place of normal lifestyle changes. It doesn’t eliminate the need for seeking to remove things from your diet which clearly are going to push your metabolism and biochemistry in the wrong direction.

The classic example would be the number one source of calories in the United States which is fructose. This sugar is just a terrible metabolic poison. You could take all the supplements you want and you’re not going to compensate for this. You’ve got to improve your diet and correspondingly, you also have to have an exercise program to give your body the demands that is required to have to optimize your health.

I use a fair number of supplements maybe half a dozen or so but it’s an addition to a healthy lifestyle which is I think is the foundation. If I can only do one or the other, I would definitely do the lifestyle, I would eliminate the supplements. But if you want to optimize…

**DG:** I completely agree. Everything you’ve said about lifestyle and diet, I completely agree with you Joe. I just sort of my (indiscernible 44:25) jump directly into this statin-cholesterol thing but you’re absolutely right. The diet I’m pushing is what I call caveman diet or at least it’s carbo-restricted diet. I’m very enthusiastic about carbo restriction. I think we’ve got to push that a lot more.

**DM:** Yeah. Actually, my first book was *The No-Grain Diet.*

**DG:** Oh really, okay.

**DM:** That was coming up 10 years now. I’ve been a long time believer of it too.

**DG:** Good man, to come up with that diet then. That’s impressive.
DM: I was mentored through Dr. Ron Rosedale who is really ahead of his time and really made a lot of the insulin-carbohydrate connection. And then I applied it clinically and I was seeing near miraculous results when you restrict people with elevated insulin levels with this approach. Really, there are life changes.

I’m curious as to what supplements specific -- so we’ve established that we were both in agreement that lifestyle changes are a priority. Anyone listening to this just needs to understand that. That’s the common basis. So to even think about a supplement without doing that is just really wasting your time. But assuming they are following those supplements or at least in process to optimizing that, what supplements have you found to be useful for optimizing mitochondrial dysfunction?

DG: CoQ10 is always right on top of my list. I am interested to learn that Russia -- there is word out that dolichol is now available in the supplement form. I have not used it but it’s available and it comes from the Arctic fur, the needles of the Arctic fur. It’s a dolichol that’s exactly like what we want. When that is available, I’ll actually be interested in that because after all, these are the two very important things that are inhibited by statin drugs because of the mevalonate blockade.

DM: That interesting. I haven’t heard that it was coming.

DG: Usually, we haven’t talked much about dolichols because you can’t replace it. There is no supplement for it but there actually is and it will soon be available.

DM: I’ll keep my eye on for that one. Before we go to the other ones, I don’t want to skip past CoQ10 because that’s an important one. You said it was the most important from your perspective. I’m wondering if you can comment on the need for the reduced version as ubiquinol as opposed to a simple CoQ10.

DG: I think you need the reduced versions.

DM: When you refer to CoQ10 you’re really referring to ubiquinol as a supplement.

DG: I would prefer everybody take ubiquinol, yes.

DM: I just wanted to establish that.

DG: Because it’s reduced. That’s the one is more available. Well, the body needs both oxidized and reduced. Part of it goes in the structure and part of it goes in the function but I would say mostly reduced.

DM: What are the other ones?

DG: Vitamin C is always cited as an antioxidant and it is. Vitamin D is up and coming as an antioxidant. We never dreamed of this. It started 25 years ago but that’s one of its new functions that I’m interested in. Vitamin E, there is a new form of it called
tocotrienol. This is so important because we, for 60 years, we’ve using tocopherol. Tocopherol is a good antioxidant but tocotrienol which is just the other half of the vitamin E molecule really, tocotrienol is 50 times more powerful antioxidant.

Tocotrienols, they not only give you 50 times greater antioxidation but it also enhances the anti-statin things anyway. It even helps to produce some cholesterol. It has a few other biochemical advantages that make it really attractive. So by all means, tocotrienol is the one.

For the elements there is only selenium and magnesium. Those are the two that are commonly involved as co-factors in various things. Selenium, we’re still learning about that. What we’ve learned about selenium has only been in the last few years. That is inhibited seriously by statins. So that’s one that’s gotten a lot more studied recently.

We have alpha-lipoic acid, I can’t help but be sort of interested in that. And then there is carnitine. L-carnitine is so important but it only has to be used initially because that is going to tell you if you are able to metabolize fats adequately. We have to remember that 70% of energy for our muscle function comes from fats. So it’s very important that we have the ability to burn fats.

If we take L-carnitine and find that suddenly we are much better then we have just proven that we need that the rest of our lives because we’re one of these people who have a dysfunction in this capability. We don’t have the means to properly burn fats at our muscle level. Instead of burning 70% fats, we’re only burning 50% and naturally we would get weak on exercise.

So carnitine is useful for making a diagnosis. If after three months of a good dose, nothing happens, I would say you forget your carnitine.

**DM:** That’s great. Also, with selenium too, that’s a co-factor for the production of glutathione, I mean, you can’t take glutathione supplements. It doesn’t work very well orally so you really need to do it intravenously and parenterally. It makes more sense for your body to make it than to take it as a supplement.

**DG:** There are so many of these supplements now. When you get up to a dozen or more and you threaten some poor patient with this, it’s going to cost them a lot of bucks. You wish you could be more specific. You wish you had the means of measuring all of these and treating only that ones that are low. This is where we are today trying to come up with packages that are meaningful.

I think that when you have a statin associated muscle or nerve or even brain dysfunction, this is where you’ve got to go because that’s where the trouble is. I think that’s where the trouble is of course if it’s cholesterol inhibition, you just take more eggs or take more cholesterol whatever.
I mean, I’m talking the truth there -- eating more eggs, I can’t believe I went 17 years and never ate an egg. I can’t believe how gullible I was. I was this medical young doctor. I will march to that band of the cholesterol causation people. I just marched to them so enthusiastically, talked at all the club meetings. I did everything I was supposed to do and it was all wrong. I can’t believe that I was led astray, maybe for 25 years of my practice, it was so bad. To have to look back and realize you’re treating cardiovascular disease erroneously because you’re doing what you’re asked to do.

DM: Well, you can only do the best you can. You modify your views as you go along and you uncover new information. That’s all you can do.

DG: All of us suffered from that. We all listened to what amounts to brainwashing. The brainwashing that we got from 1955 on to just recently, we were brainwashed. They have liberalized the diet stuff recently. So people are back to eating eggs and drinking whole milk and eating butter. I went around margarine so long and margarine is what’s causing disease.

DM: Absolutely.

DG: Butter is what’s helping to cure it. It’s incredible.

DM: Fortunately, most people understand that now. There is very few people who don’t realize that margarine is dangerous for you at least from my experience.

Let’s go and focus on CoQ10 for a bit. I am wondering if your studies have suggested an ideal dosage or range of doses for ubiquinol since you are in agreement that the ubiquinol would be the preferred version.

DG: That depends if you’re a person who is having symptoms of (indiscernible 53:47) muscle pain. You’re on a statin, you’ve been taking it for two months and your legs are hurting terrible. In a case like that, I would say anywhere from 200 to 500 mg. But in a case where you wanted to use it preventively, I would say not more than 200 should be sufficient.

DM: Are there any other conditions, clinical conditions other than addressing the side effects associated with statin drugs for coenzyme Q10. Do you recommend it as a general supplement to take?

DG: As a general supplement… I’m coming around now to just forgetting the statin thing and just thinking of the process of aging, realizing that really anybody over the age of 50 is probably not synthesizing CoQ10. The normal diets that we’re on will not provide more than one-twentieth of our needs of CoQ10 unless you eat a lot of beef heart.

Therefore, I’m getting to the point of recommending CoQ10 for everyone starting at a very young age, as much as 40 or 50. I just have to research it a little bit more and see what the levels are and see where in the fall off begins in our ability to synthesize. But it
comes certainly before 60. I’m thinking it’s probably present somewhere between 40 and 50 as the fall off gets really significant.

**DM:** My guess is it’s largely related to the lifestyle of the individual. If you have someone who is optimizing their health and is really leading a healthy lifestyle that may not occur until they’re 60. Where someone else who is smoking and drinking and eating junk food and processed foods, this may occur below 30.

**DG:** You’re right.

**DM:** I don’t think it’s age related just as long as you’ve been on the planet. It’s really a time related effect of your exposure to many of these toxic influences that eventually impairs the mitochondria.

**DG:** If you’re thinking that leading a toxic life is going to necessarily lower your CoQ10 synthesis, I don’t know if that happens. I just don’t know.

**DM:** It just makes sense.

**DG:** I’m not saying it doesn’t, but I just don’t know.

**DM:** I mean, you see that with all forms of aging. You can see people who are 60 years old and they look like they’re 40 and you see another 30 year old who looks like he’s 60. I mean, it’s pretty clear.

I’m wondering from your experience about coenzyme Q10 and other neurological problems something like Parkinson’s disease or even Alzheimer’s, do you think that there may be some protective benefit there?

**DG:** Yes, because I suspect that the cause is the same, mitochondrial. I would go for large doses in a person having persistent muscle pain despite stopping the statins. I would go for large doses because that means that it is mitochondrial and thus you’ve got to push. You have to push. It’s not an easy subject. I’m assuming that if you are on a statin drug and you are also on CoQ10 when you start I’m assuming that you probably would stay out of trouble.

**DM:** We talked about this earlier, that’s only 50% of the side effects. It has nothing to do with the cholesterol.

**DG:** (indiscernible 57:18) that type of side effect about 50% so that’s right. You’re absolutely right we did cover that.

**DM:** I believe CoQ10 has been recommended or used for certain types of cancers I’m thinking of breast cancer specifically. Are you familiar with its usage in this area?
DG: The most recent article I was reading was very important. It had 41 patients who were end treatment.

DM: End stage.

DG: Meaning that they’ve gotten all the treatments they could possibly have. Their cancers were of about seven different types. They were breast cancers, bowel cancers, brain cancers, everything you care to mention. They put them on supplements with CoQ10 and a few other antioxidants and followed them along and they gained some really remarkable longevity. Instead of dying in 12 months, which 50% of them were supposed to do, that same 50% lived for 28 months. This is basically because of CoQ10. I think we’re learning a lot about CoQ10 and Parkinsonism and dementia from Alzheimer’s probably justify large doses.

DM: It’s interesting too, I have recently became aware of one of the mechanism, at least purported mechanisms of the way that we age. That has to do with the telomeres and those are the end-caps on your genes somewhat similar to the hard piece of plastic on your shoelace. They are really relatively small.

DG: I know telomeres, yes.

DM: Just to explain it for our listeners. The theory is that if your telomere is progressed, I mean there are 15,000 telomeres, base pairs when you’re born and they rest down to the point where there is only 5000 at the time of death. Some researchers believe it’s the most accurate biological clock. So that any influence that will accelerate telomere shortening will shorten your lifespan. My guess is that this coenzyme Q10 because it’s such a potent antioxidant is able to sweep up many of these free radicals and as a result, probably prevent telomere shortening.

DG: The (indiscernible 59:42) theory.

DM: We don’t know. I don’t know if anyone has looked at it but it would seem to make sense.

DG: I have read this material and I agree with it. You have to agree with it because it’s pretty objective. What is the actual mechanism that makes these telomeres become shorter and shorter and shorter? What is the chemical equation that’s leading to this that’s what I mean?

DM: There is a certain intrinsic basal shortening that can occur but then there is also ways that it can be accelerated. We know certain toxins like smoking or lots of sugar, metabolic syndrome, those will accelerate telomere shortening. It’s the same type of process but there is a certain base. That’s why people don’t, at least at this point and time, don’t live over 120 because at some point you reach that threshold, you don’t enough telomeres and you’re dead. Some of the most exciting aspect of anti-aging medicine involves strategies to actually reverse this shortening.
**DG:** In my case, the more I’ve been reading on this statin thing that the CoQ10 appears to be primary in the aging process. If we forget the statin, the cause of aging appears to be increased mitochondrial mutations. Leading to decreased mitochondria in the various cells and then pretty soon, the cells fail and pretty soon, the organs fails hence we have aging. I’m saying now in my last two books, I’ve preached that statins enhance aging. That’s what they appear to do.

**DM:** I would tend to agree with you. You, mentioning it, that reminds me of a conversation I have with one of the scientists that’s working for the biggest manufacturer of ubiquinol, the reduced version of CoQ10. He was sharing some studies done on rats that really weren’t published but just showed. I mean, absolutely shocking extensions in lifespan with the use of CoQ10 strongly implicating that it has a very potent anti-aging benefit.

From my perspective, it doesn’t appear to have any downsides other than the cost. If that’s not an issue it would seem wise for someone who is interested in extending their lifespan and staying as young and healthy as long as they can that this might be a useful considerable tool to apply.

**DG:** I don’t know how many I have told, take as much as you can afford. That’s what I have told hundreds of people.

**DM:** So that’s your recommendation.

**DG:** It’s a shame at $25 a month or something like that. I try to buy mine two month’s supply for one (indiscernible 1:02:30). I agree with you.

**DM:** And you’re using ubiquinol.

**DG:** Yes.

**DM:** From your perspective there is really no downside from overdosing on this and taking a toxic dose or something that’s going to (indiscernible 1:02:44) too much CoQ10.

**DG:** I never heard of downside. I never heard of an overdosing. The only downside is cost like you have said. Actually, I’ve been trying to get the cheapest I could.

**DM:** Well, just be careful. Let me just enlighten the readers and perhaps yourself too that the reduced version which we both are in agreement is the one to take is only made by one company in the world. It’s Kaneka and they’re out at Japan. They have a patent on this. As long as it’s ubiquinol and it’s certified ubiquinol then you go for the lowest price because that’s the only one out there that has it.
DG: I know of Kaneka and I know that they’ve got the only patent and I begrudge that. Somebody is going to have to come back with a study to show there is no significant difference between the two.

DM: Between CoQ10 and the reduced version?

DG: Yeah. We need more studies along that line (indiscernible 1:03:41) what’s called the Ace card.

DM: I talked to Dr. Stephen Sinatra about this. I’m sure you’re familiar with him.

DG: I know Stephen.

DM: He’s a big believer in coenzyme Q10 not necessarily the ubiquinol version. I actually want to get him on the phone and review his (indiscernible 1:03:55). He actually did those studies and come up with some conflicting results. Apparently, that seems to be the best approach at this point is to take the reduced version.

I’m just wondering, you had mentioned earlier that as you age, your body’s ability to produce CoQ10 has decreased and since you interchanged CoQ10 and ubiquinol, I’m just wondering is it actually a total decrease in CoQ10 production or is it the fact that we’re making the CoQ10 and we just can’t reduce it?

DG: My understanding is we’re not able to synthesize CoQ10. Whatever that means, that’s what I read.

DM: It’s interesting because if that’s the case, then perhaps just taking it would be sufficient and your body’s internal recycling systems would do that. Isn’t that another function of CoQ10 is that it recycles these other antioxidants like vitamin E and vitamin C who can donate their electrons and then they are antioxidants but once they donate it then they are in the reduced version and they don’t work so well. Doesn’t CoQ10 tend to recycle them?

DG: That’s another one that does that. I’ll have to go back to my books to answer that question. I can’t answer that right now. But that would be a new one for me on CoQ10 is it really helps the others.

DM: I thought it did. I though it helped at recycling that. Any differences, since it is a -- there is fat soluble and oil soluble supplements and antioxidants specifically like vitamin C is water and vitamin E would be fat soluble. But CoQ10 is a fat soluble supplement so does it make any difference as to what you’re eating with the meal with respect to absorption.

DG: I don’t even want to answer that. I’m a very superficial biochemist. All I know is what I’ve been reading and I’m not even sure of most of what I read. So I don’t want to console anybody until I really have access to books.
DM: Fair enough.

DG: I function like a family doctor more. As we talk about this, it gets more and more in the role of the biochemist or at least the pharmacist.

DM: Sure. Well, or researcher who is really studying this in the lab and has direct experience with this. Your experience is somewhat like mine, I mean, you obviously are more extensive in that you have collected this information.

Why don’t we do that now. You have a website. This website serves as a tool that people can report complications from taking the statins and you’re collecting that data and then forwarding it on to the appropriate agencies. So that hopefully more conventional physicians will be aware of this and be alert to the fact that it could cause these side effects. What’s your website’s address?


DM: Interestingly, you’ve preceded me by a bit of course with respect to our age differences but when I was in high school, one of my ambitions was actually go to NASA and be an astronaut too but I wound up not doing that. I actually am very grateful because I actually wouldn’t accept payment to go on to space because of the exposure to ionizing radiation.

People are concerned about airport scanners and they don’t realize that even if you’re flying at 30,000 to 35,000 ft which is what most commercial jet liners fly, about two minutes up there is equal to the radiation dose you’re getting from one of these scanners but when you go further up into space -- what’s the altitude? It’s way beyond a hundred thousand feet. It’s what, two hundred thousand, three hundred thousand?

I mean, your exposure to radiation is just extraordinary. You’re really accelerating lots of damage. That’s one of the reasons why I wouldn’t do it. I’m just curious how you got into that from being a physician. Were you an astronaut first and then decided to go into…?

DG: I was an astronaut first. I didn’t go into medicine until later in life.

DM: So you went into the Air Force or the Navy?

DG: I was in the Air Force for 10 years. I was doing space medicine research then. It was during this time that I became fascinated with radiation to come. That right now is our most difficult challenge for the future in manned space flight is the galactic cosmic radiation which we call heavy primaries.

This is really a major concern because you cannot stop these. They are the nuclei. These are the nuclei of the atoms from hydrogen on up to iron. They’ve been stripped of
their electrons and they come at you at sub-light speed. So it’s almost the speed of light and they have incredible energies.

We first ran into these in Apollo 11 when we started with the light flashes that the astronauts are seeing and now, we know much more about them. We studied them at length and the reason that we can’t stay on the surface of the moon longer than a few days is because we’re being constantly peppered with these.

You cannot shield them with any existing technology. Even 12 feet of lead doesn’t work for these. These are amazing and when they hit, it’s not like ionizing radiation. That’s bad enough. When these hit, it’s like getting hit with a high powered rifle bullet. It leaves a hole of absolute destruction surrounded by ionized chaos is what we think of it because of all the damage it’s done. It’s so small. These lesions are so small. It’s only been recently that we can identify them. These are really amazing.

**DM:** That further supports my joy of having not been successful at being an astronaut. So I just avoid that damage.

**DG:** Now that we know about them, the guys are saying, “You mean you put me up on all that stuff out there?”

**DM:** That’s true.

**DG:** You’re absolutely right.

**DM:** I’m glad about that. I’m just curious because I hadn’t realized you had studied this. What is the source of most of those high reactive energy particles? Is it our sun or is it other suns in the solar system or universe?

**DG:** Actually, they call them galactic cosmic radiation so our sun contributes a little bit but these are from the galaxy. These have been everywhere else. All the explosions but most of them come from novas and supernovas way off that are surrounding us at all times. So anytime we go on the other side of the Earth’s magnetic field, now the Earth’s magnetic field is like a big umbrella, but once we get out to about 40,000 miles…

**DM:** Forty thousand, that’s where it starts.

**DG:** The umbrella goes from 400 to 40,000 miles. So the Earth is tucked away inside comfortably and you got this giant quite thick umbrella but once you get beyond 40, you get out to say 60 and that’s where they were on their way to the moon, heading for the moon which is about what, 240 but anyway, they are out at 60,000 and they started seeing light flashes.

So anytime from 60,000 miles away of the Earth is when deep space begins. That’s going to be with us all the way back to the moon and all the way to Mars and all the way to whatever else we do in the future. We’ve got the allow for the fact that we’re dealing
with extraordinarily hostile environments that requires probably some sort of electromagnetic shielding because these are all charged particles. If we can push them right or push them left a little bit, maybe we can secure a (indiscernible 1:12:07) compartment but that’s what we’re dealing with now.

DM: That would seem to make the most sense. When you flew as an astronaut, did you actually go to the moon or were you on orbital flight?

DG: No, I didn’t get any mission. Two of us did not get any mission out of the six of the scientists that were selected. I am the guy who did all the ground base stuff. I am the guy that did all the muscles and exercises and all that. They took my roommate but they didn’t take me. My roommate was a geologist, Harrison Schmitt and I know he was going to go. Why would they take a family doctor? Anyway they took Harrison Schmitt. He did a great job but that’s the only guy that ever went to the moon, the only scientist.

DM: Do you have any other closing comments you’d like to make on the topic?

DG: No, not really except I’m still very much anti-statin and yet I recognize that if we say that our heart disease, stroke is an inflammatory process I will offer that they do seem to give us a type of anti-inflammation that seems to work but the cost is terrible side effects. There are probably 500 people who have been put in wheelchairs because of their ALS-like conditions but there are tens of thousands of cases of permanent muscle and permanent nerve damage just walking about because they can’t get a lawyer to take their case.

DM: Hopefully, you’ve established a database that will provide the validation that this is in fact a real clinical entity and that would provide some of the plaintiff attorneys with the tools they need to successfully litigate these drug companies for the damage that they’re doing.

DG: That’s what I’m trying. I’m really close now. I’m very close. Especially now is the time to strike and to try to interest a group of lawyers especially since we know that this past 35 years of focus on cholesterol has been wrong. What better time to strike than now. The drug company has to realize that coming up with drugs that lower cholesterol was a bad idea. They have to do that now. This is the time to strike.

DM: We’ll hopefully expose your message to more people and provide you with some more ammunition to be effective in your battle with them.

Thank you for all your doing. I appreciate the time with you today.

DG: Thank you so much.