Ubiquinol:
A Special Interview with Risa Schulman, Ph.D.
By Dr. Joseph Mercola

JM: Dr. Joseph Mercola
RS: Risa Schulman, Ph.D.

JM: Coenzyme Q10 and ubiquinol, two vitally important nutrients that you need to know about. Hi, this is Dr. Mercola, helping you take control of your health. Today we are joined by Dr. Risa Schulman. She is a biologist, has a Ph.D. in that field, and has studied this area for the last 20 years. She’s passionate about it. She just devours the literature. She’s going to enlighten us about the subtleties and some of the details that you and I probably aren’t aware of. Welcome and thank you for joining us today, Dr. Schulman.

RS: Thank you so much. It’s a pleasure to be here.

JM: I gave a brief introduction to your background and history. But why don’t you provide us with some of the highlights of your academic experience, so that we could provide a frame, so that people would have a context of what you’re sharing.

RS: Sure. I’m sort of the quintessential science head, the kid who wondered what the ants are doing scattering around and how the dew got onto the grass. I figured out later in life that I was actually a biology nut and studied all kinds of biology in various universities.

I kind of put together both my love of human physiology, plant physiology, and the environment, and pulled them together in a career that has now been my lifelong career, looking at how compounds and plants and various natural products can help us to keep our wellness, keep our bodies working optimally. I’ve been doing that now for almost 20 years.

JM: That’s great. That’s useful, because one of the principles that we teach on the site is to absolutely use that, the food choice as the primary source, but then also natural compounds as a far safer alternative to potentially dangerous toxic and certainly in most cases, very expensive drug solutions that is typically provided by conventional therapy recommendations.

RS: Absolutely. I love what I do. I’m very happy to be able to unearth the truth. That’s really been my mission in my career. It’s to dig into the science and separate the wheat from the chaff, help the science to become better and better over the years as it has, and then to get the word out to the public as to what the health benefits are, how they can be used, and what things are useful.

JM: Let’s focus on what the topic of the discussion is today: coenzyme Q10 or ubiquinol, primarily made in the liver, of course. The challenge, of course, which we will discuss shortly, are some of the common drugs that are used to suppress its formation. But before we start there, why don’t we discuss the differences between coenzyme Q10 and ubiquinol, the reduced version?

RS: Sure. I love talking about this, because I also love bringing science down to a language that anyone can understand. Ubiquinone or coenzyme Q10 (they’re the same; it’s two names of the same thing), or
ubiquinol are actually, they’re the same molecule. The difference is two electrons, plus two protons, and two hydrogen molecules.

When ubiquinone, coQ10, for example, is reduced, it takes on two electrons, then it turns into ubiquinol. We call it ubiquinol. In the body, this happens hundreds and thousands of times every second, where this is flipping back and forth between coQ10 and ubiquinol. Where does this happen and why does it happen? It’s in the mitochondria, which is a little machine inside of every single cell and which is really the engine of the cell.

The reason it does all this flipping back and forth between these two forms of the molecule is this is part of the process that helps us to change our food into energy. This is really very core, very important to our healthy functioning, health and well-being, and obviously important for all muscles, in particular the heart muscle, which works hardest of all the muscles.

**JM:** It has the highest concentration of the mitochondria, too.

**RS:** That’s right. As would be expected for a muscle that’s beating all the time. Other muscles like, for example, I’m sitting right now. My leg muscles are not doing as much as my heart is to keep me going.

Beyond just these flipping back and forth and their function of converting our food into energy, ubiquinol actually has a number of additional functions compared to coQ10. Ubiquinol is an antioxidant. It’s a very special one. There are many, many antioxidants that we hear about, that we talk about. Why is ubiquinol so special? It’s a lipid-soluble antioxidant, meaning that it works in the lipid portions of our body. That includes cell membranes, the bag so to speak that encloses every cell. It’s always made up out of fat lipids. Ubiquinol kind of lodges itself (fat can dissolve into fat) into that membrane and it acts there. It’s one of the very few antioxidants in fact that is lipid soluble.

Vitamin E is one of the other ones that is very well known. But ubiquinol is actually more powerful than vitamin E, because vitamin E can’t really completely lodge itself inside the membrane where all the oxidative activity is happening; whereas ubiquinol can. It’s really sort of the first one to fire and vitamin E is kind of the backup.

The second thing that makes ubiquinol a really super antioxidant is that it can regenerate itself. Antioxidants get used up. They do their job and then they’re finished, so to speak. The battery is dead, so to speak. But ubiquinol can regenerate itself; whereas vitamin E cannot. In fact, vitamin E is regenerated by ubiquinol.

For those two reasons, ubiquinol is just a very important antioxidant for the body. It’s also the only lipid-soluble antioxidant that’s actually generated within the body and doesn’t have to be ingested from your food.

**JM:** Excellent. I guess, maybe you can discuss at this point, the requirements for those. Because you mentioned, it’s really difficult to obtain it from the food supply. But there’s a tendency also to have your body’s ability to convert that ubiquinone to ubiquinol decrease as you age. Maybe you can discuss those points.

**RS:** Yes. That’s a very important point. Because when we think about things in the body working less well and working less efficient as we age, we generally think about maybe 65 years old, 75 years old, 85 and up. But ubiquinone and ubiquinol, it’s actually been shown that there’s sort of an upside down U-shaped distribution curve over a person’s lifetime.
Ubiquinol production within the body ramps up from early childhood up until about, let’s say, the mid20s to late 20s. Once you hit that 30-year-old mark, it actually starts to decline at that point. We don’t really think of 30 as sort of it’s all downhill from here. But in the case of ubiquinol, that is true. It just does go downhill from there.

It’s a very important supplement for even younger people. They can take coQ10. Young people can take coQ10. We say that ubiquinol is better to help replenish those levels as people age, because it’s better absorbed, much more highly absorbed.

But also, here’s something that I think would be very interesting to folks: some people cannot actually convert coQ10 to ubiquinol on their own in their bodies. That’s what generally happens. If someone takes a coQ10 supplement, the body very quickly will convert it to ubiquinol, because that’s the preferred form. It will transport that coQ10, now ubiquinol, through the blood as ubiquinol, into the tissues, and eventually into the mitochondria.

But there are some people who lack the enzyme that helps to convert the coQ10 to ubiquinol. That could be partly an aging thing, but it’s actually also a genetic thing. There’s something called a SNP, a single nucleotide polymorphism. We see that now more and more. It’s in the news with the Human Genome Project (HGP) and various things like this. Most of those testing kits, those gene testing kits…

JM: 23andMe primarily.

RS: Exactly, things like that, all of them are looking for SNPs. There’s a particular SNP that’s called NQO1. When a person has either one or two copies of this SNP, their ability to convert coQ10 is either slightly compromised or really compromised.

What that means practically is that if that kind of a person is taking a coQ10 supplement, it’s pretty much almost useless for them, because they can’t use it. The body can’t convert it in a way that makes it usable. Those people in particular can benefit very much from taking ubiquinol, instead of ubiquinone.

How do you know whether or not you have this? There are maybe ways to get tested. But it’s also known that various ethnic groups, some have more, some have less prevalence of this. Research has shown that Hispanic and Chinese populations have actually double digit frequency of having this SNP. That’s just sort of one guideline that people can use.

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JM: A significant motivation to get genetic testing to confirm that you are not possessing this single nucleotide polymorphism.

RS: Correct.

JM: Perfect. There’s an interesting study, which was recently published, that showed a biological way to improve this conversion that almost all of us have access to. That is eating lots of green leafy vegetables full of chlorophyll. Why? Because once chlorophyll is consumed, it gets transported into your blood, and then you expose significant amounts of your skin to sunshine, that chlorophyll actually absorbs the solar radiation and facilitates the conversion of coQ10 to ubiquinol.

RS: I’ve actually read about that.

JM: It’s a recent study which was published. It’s somewhat magical almost. It makes common sense. What’s the downside? I don’t think there’s any expert, a rational expert, who would disagree that we need to eat plenty of green leafy vegetables.
**RS:** That’s for sure.

**JM:** There are some irrational dermatologists who believe that we shouldn’t be exposed to the sun. I think there’s a lot of science to refute that. But just combining those two could be a simple way… I’m not sure how it would affect this if you had this genetic mutation that prevented that. But it certainly would be harmless and there’s essentially no cost to it.

**RS:** There’s no cost. That’s for sure.

**JM:** Yeah. No danger.

**RS:** I would love to see what science is saying and what the mechanism is there.

**JM:** I’ll send you a copy of the study. It was really interesting.

**RS:** Thank you.

**JM:** I should have sent you a copy before we record. It just occurred to me as you were discussing this issue.

**RS:** Interesting.

**JM:** You mentioned the point of absorption, which is a key concern. Now, there are supplements of coQ10 that address this quite well. They are much better absorbed than others. But if you choose to take coQ10 for reasons primarily related to cost, because it’s significantly less expensive, you need to seriously look at the absorption of that and be careful to choose the lowest-costing one, because you may not be getting essentially any of it.

**RS:** Yes. That’s right. A lot of different coQ10 supplements out there have manufactured the coQ10 in various ways to increase the bioavailability. That’s something to look at.

**JM:** One of the simple things you can do to, because it is fat soluble, as you mentioned, is that ideally, this is one of the supplements where you want to eat a meal where that has the most fat to facilitate the absorption.

**RS:** That’s right.

**JM:** There are actually emulsifiers. Sometimes people who have had their gall bladder removed, they could use things like ox bile, which helps digest and breakdown the fat. They form these little [inaudible 13:03] that bind into the nutrients, such as coQ10, and helps absorb it. Just some little tricks, you know. Frequently, the devil’s in the details.

You mentioned that it’s useful for generating energy ATP, the energy currency of the body. It recycles quite well. How many times does ubiquinol recycle? Is it like 20,000 times? I might have mixed it up with PQQ.

**RS:** I don’t know the actual numbers, but it’s got to be. Its main function is flipping back and forth.

**JM:** It’s very important. If you’re deficient in this, it can radically reduce a variety of functions. But one of the ones – the disease that almost, not almost, but every single person watching this has… Do you know what that disease is? Everyone watching this has it.

**RS:** Aging.
JM: Aging. You guessed it right.

RS: You can call that a disease.

JM: Yes, you got it. It’s aging. Why don’t we discuss some of the molecular mechanisms of how ubiquinol might facilitate putting the break on the aging process?

RS: Right. Absolutely. When we talk about aging, the next thing that always comes out of your mouth is oxidative stress, because that’s one of the major causes of aging in every way around in the body. We spent a little time just now talking about the antioxidant properties of ubiquinol. There’s another special thing about it. A lot of the reactive oxygen species are produced in the mitochondria.

JM: Actually most of them. I would say it’s over 90 percent.

RS: It very well could be. If you think about the mitochondria as an engine, we’ll extend the analogy to say that every engine has exhaust, just like a car has exhaust. If you breathe that stuff, that’s terrible for you, because it’s full of free radicals. It’s packed in reactive oxygen species and other kinds of free radicals. The same is true in the body. Every kind of combustion is incomplete and there are byproducts, just as well in our cells.

One of the functions of ubiquinol is to mop up all that exhaust, so to speak. If ubiquinol is not there, the exhaust stays around and it starts tearing up the cell. It can tear up the mitochondria in the cell. It can tear up other organelles, the cell membrane, and then outside into the tissues. Whatever tissues are around, that’s just going to get attacked. Clearly, this is a very close relationship between ubiquinol’s ability to prevent that kind of oxidative damage throughout the body.

JM: What’s the mechanism there? Has it got something to do with cyclic adenosine monophosphate (AMP)?

RS: There are studies that have shown… Cyclic AMP is part of the signal transduction cascade. What is that? That means that this is how molecules from without the cell give signals to the nucleus, which is all the way very deep inside the cell.

It’s a pretty complicated domino – I don’t want to say effect – game. You push the first domino, and a hundred dominos later, something else happens. There are all those dominos in a row because you want to break this process pretty carefully. You don’t want to be just like a hair-trigger process to turn on various genes. Cyclic AMP is one of the dominos so to speak. Ubiquinol, when it’s around, has been shown to upregulate various signal transduction cascades that turn on various genes.

There’s actually been some really interesting research that goes into several different areas for a little bit, which is ubiquinol’s benefit for heart health in particular. It’s actually a brand new research in the last year or two. We have all different markers of heart health like C-reactive protein. If it’s elevated, it’s indicated as a risk for heart disease and various kinds of cardiovascular disease.

JM: Because it shows inflammation. It’s a marker for inflammation.

RS: A marker for inflammation. Right. There are two other molecules that have recently been shown to also be markers like gamma-glutamyl transferase (GGT) (which has actually been known for a while to be an early and pretty sensitive marker of heart failure) and then also NT-proBNP. It’s a mouthful. NT-proBNP. It’s been shown that there’s an association between the levels of these markers and ubiquinol. When ubiquinol is supplemented, both these markers actually go down and genes associated with them go down.
That’s where I’m getting back around to the signal transduction cascade. Because when you ask, how does happen, if the genes associated with these markers go down, that’s where the cyclic AMP comes in. The question is does ubiquinol have an effect through these cyclic AMPs getting into the nucleus and turning the genes off?

JM: Great. I appreciate your comments on this topic. One of the items that you mentioned was this suppression of free radical formation from reactive oxygen species.

RS: Yes.

JM: That was the commonly held view 20, 30 years ago with respect to the free radical theory of aging. But there seems to have been a morphing of that in the last 10 or 15 years, and there’s a recognition that these reactive oxygen species in fact are quite important signaling molecules.

If you indiscriminately suppress them, you can actually run in some very serious complications. It would seem that the wiser strategy would be to actually use a cleaner fuel. To refine your analogy somewhat that you’re preventing pollution, to use a fuel that generates less pollution.

RS: Interesting.

JM: That fuel I think is becoming more recently appreciated for burning fat, because burning carbohydrates is associated with about a 30 to 40 percent increased production of reactive oxygen species as opposed to burning fat. It makes a lot more sense to produce less reactive oxygen species to begin with, then you don’t have to be as careful about these external antioxidants. I have some concern over indiscriminate use of antioxidants, but not necessarily ubiquinol.

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This is where I have not studied the science that much to know, but perhaps you would be able to shed some light on this. Is it a blanket suppression? Or is it significant throttling and feedback loops that occur, so that it doesn’t non-discriminately suppress these free radicals? The reactive oxygen species and then secondary, the free radicals.

RS: Right. I know exactly what you’re talking about. Like you were saying, counter to how we’ve all been kind of trained to think in the last years, regarding the antioxidant free radical theory of aging, you don’t want to suppress it all the time.

In fact, free radicals actually play a very critical positive role in the body, because they turn on various very important functions. Nitric oxide, for example, has free radical properties. It’s a critical signaling molecule and is also critical for the health of the arteries. Yes, that’s a very important and good point.

I haven’t read anything that’s already been published in the literature regarding whether there’s a discriminating or non-discriminating suppression of reactive oxygen species by ubiquinol in the mitochondria. But my understanding of the biochemistry and the bioenergetics that are happening there leads me to believe that it’s more of a random process.

Because this flipping back and forth between the antioxidant in a reduced state is being driven by what’s being fed into the engine, so to speak, like you alluded to. Whichever form it’s in, whether it’s in the coQ10 form or it’s the ubiquinol form, it’s kind of floating around a little bit in there, in the middle range between complex I and complex III. There’s complex II in the middle. These are all parts of the engine, so to speak.
If it sort of bumps into a free radical in its travels, it’s going to become oxidized at that point and do its thing on the free radicals. I’m really kind of speculating here, but it seems to me that it’s just part of the kinetics of what’s going on within the mitochondria, of what’s bumping into what and where’s the most demand. Does that make sense?

**JM:** Sure. You know another strategy, it just occurred to me, that you could use to reduce the production of excess reactive oxygen species would be the timing of your meals. Many, many people make the mistake of eating a large meal before they go to bed. What happens when you do that is that you have this large fuel supply that’s going and generating these ATPs as they’re designed to. That’s part of the process.

Then your metabolism sort of radically reduces because you’re sleeping and you get this back up. This back up reduces reactive oxygen species that you have to take care of. It’d be far wiser to eat the biggest meal before your biggest movement of the day or the most caloric requirement. It seems to be a far more effective long-term strategy. Sometimes it’s not possible to do that. But for the majority of the time, that would seem to me a wise strategy to stay healthy.

**RS:** Right. It makes total sense.

**JM:** The devil’s frequently in the details as I’m sure you’ve learned to appreciate it over all these years.

**RS:** Most definitely.

**JM:** I mentioned the use of statins, which in my view, and I suspect yours, too, a pernicious and inappropriate medication used in tens of millions of patients, probably more than that worldwide. But in the United States, I know the numbers are at least 1 in 4, working to 1 in 3 adults over the age of 40, which is just extraordinary. You go to a meeting with people over 40, adults. You look around and you realize that 1 in 3 people around here are taking a statin drug. That’s the truth.

Why don’t we talk about [statins]? It’s an enzyme inhibitor, a very effective enzyme inhibitor, the HMG-CoA reductase. Why don’t you talk a little bit about that and how it impairs the production of coenzyme Q10 and some of the complications of that impairment?

**RS:** This is a very important topic. It’s actually been in the news just even the last couple of days, because of a new study that’s come out. Many people who take statins have the side effects of muscle pain, fatigue, memory loss. We’re going to stick to the muscular benefits and what we’re going to discuss here. To such a point that compliance becomes an issue; people don’t want to be on it anymore, on the statin anymore.

It’s been documented now and recognized medically that these are real effects and that they’re due to the statins. What’s actually happening? The way a statin works is that it blocks the body’s production of cholesterol. We’re always thinking about cholesterol from the diet, because anyone who’s over 25 or 30 years old is conditioned that cholesterol is bad. “Don’t eat eggs. Don’t eat that.” Bad cholesterol, good cholesterol, and all that.

**JM:** It’s actually changing.

**RS:** Interesting.

**JM:** The most recent dietary guideline, which is from the conservative US Department of Agriculture (USDA), actually suggest that it’s not an issue anymore and that you don’t have to limit cholesterol. Finally, they got some common sense into their recommendations.
RS: Right. Which is very, very good news. But at the same time, most people don’t realize that cholesterol in the body comes from two places: it comes from the diet and it also comes from your internal production of cholesterol.

Cholesterol actually is quite important to the body, because cholesterol is one of the major components of cell membranes. It’s also the precursor for all the sex hormones. It’s not all bad. It’s just bad when there’s too much and that depends on what kind of too much as well.

When someone takes a statin, you’re generally doing that because their dietary modifications have not brought the cholesterol down to the satisfaction of their doctor, and the doctor feels that the cholesterol that the body’s producing should be curtailed. That’s what a statin does. It shuts down, like you said, this particular enzyme HMG-CoA reductase, which is one of the facilitators of the body’s production of cholesterol.

Now, like all things in the body, nothing is an isolation. This HMG-CoA reductase not only facilitate the pathway that produces cholesterol, but it also facilitates the pathway that produces coenzyme Q10 or ubiquinol. When you’re taking a statin, you’re shutting down the body’s ability to produce ubiquinol.

As I mentioned earlier, ubiquinol is made in the body. It’s one of the few lipid-soluble antioxidants, an enzyme in its own right, a [inaudible 27:37] co-factor in its own right that is produced by the body. By the way, those aren’t the only two things it shuts down. It also shuts down the conversion of vitamin K1 to vitamin K2, which is critically important in many body functions, including heart health.

JM: Interesting.

RS: Yes. There’s actually a consequence to shutting down all three of these pathways. That is that it has adverse effects on both the production of energy, but also on cardiovascular health. I’ll explain why. When you cut down on ubiquinol, there’s not as much around playing its role in the engine, and therefore, the conversion of your food to energy is less efficient.

That obviously leads to lower energy. That’s particularly important in the muscles and particularly important in the heart. That’s where your fatigue comes in. That’s where your muscle pain comes in. It generally comes in with long-term use of statins, and statins, of course, are prescribed long term. People are on it for years and years.

JM: Until they kill them.

RS: Yeah.

JM: Prematurely.

RS: The longer you’re on it obviously, the more ubiquinol-starved your body becomes and the greater the side effects become. One of the implications is on energy levels. The other implication is on cardiovascular health. This area is somewhat controversial, more than somewhat controversial.

There are definitely some published articles that come out in the last year talking about all of the repercussions for the cardiovascular system that are in fact causing a lot of the disease endpoints that statins otherwise promised to prevent. We can get into more detail with that as well if you like.

JM: Yeah, we can. Let me just interject a point here. In addition to those three pathways that you suggested were impaired – the production of cholesterol (which is important), the coenzyme Q10, and then the vitamin K1 to K2 conversion – there’s another really vitally important pathway that I wasn’t aware of until I started scouring the molecular biology literature.
You may not be aware of it either, but I had alluded to earlier. To me, the most rational strategy to reduce reactive oxygen species is to burn clean fuel. Ultimately, that results from using the high-fat diet, high-quality fat. When you convert or metabolize the fat, the end product is a ketone, a fat soluble molecule that is burned readily in the mitochondria. But you have to produce these ketones. The ketones are produced in the liver. Guess which enzyme produces those ketones? Do you have an idea?

**RS:** Is it HMG-CoA reductase?

**JM:** Yes, it is. That’s the important one. If you are taking a statin drug, you essentially have shut down your liver’s ability to make ketones and essentially absolutely compromise your ability to benefit from a clean fuel. It’s just like putting handcuffs and weights, 100-pound weights, and your system trying to move around. You just can’t do it. You’re metabolically compromised.

**RS:** Right.

**JM:** Even if you’re taking vitamin K2 and ubiquinol, you still have to address the fact that you cannot make ketones. You can’t take a ketone supplement. It’s not going to be as effective as burning the fat yourself. You can’t. You’re metabolically screwed when you’re on a statin. Bad news. Which ultimately has consequences in cardiovascular health, too.

**RS:** Yes.

**JM:** Because, as we mentioned earlier, the most mitochondrial-dense tissue in the entire body is in the heart, the cardiac tissue. If you’re depriving them of their primary fuel, you’re impairing cardiovascular health by definition.

**RS:** Yeah. When there’s a buildup of ketones in the body, there’s a condition called ketoacidosis, which is not a good thing. There needs to be a balance there between the high-fat diet…

**JM:** Usually that condition, ketoacidosis occurs when you have type 1 diabetes and there’s an insulin deficiency. The millimoles of ketones go up to greater than 20. When you have nutritional ketosis, which is a completely different animal, because it’s modulated by insulin as a hormone, it is almost physiologically impossible to go over 8 or 9 millimoles. You cannot get into a diabetic state. It’s like physiologically impossible. The only way you get into ketoacidosis is to have an insulin deficiency.

**RS:** OK. You have more knowledge about that than I am.

**JM:** That’s the only way. There’s a lot of confusion on that related to Atkins. Many of the high-fat diet suggesting this is so useful. There’s a lot of confusion that’s related to Atkins because he had a fatally flawed implementation of the program. He was recommending far too high proteins and was never paying attention to the quality of the food. Two just prominent critical and vital issues to successful implementation. But if you have the right amounts of proteins, high amounts of fiber carbohydrates, low amounts of non-fiber, net carbs, and lots of good fat, you just create these ketones in the ideal level.

Typically if we’re burning fat efficiently, it’s really difficult to go over one or two millimoles of ketones, which is not going to cause any problem at all. It’s actually beneficial because ketones are, just like the reactive oxygen species, really vitally important signaling molecules metabolically. If you don’t have them, you are messing up your system on steroids.

**RS:** Just to play devil’s advocate, I want to say that the reason that statins have been in the news again in the last couple of days is, because of a new study that was published in the *New England Journal of
Medicine, which shows what the party line has been all along which is that statin use does in fact lowers the frequency of certain kinds of cardiovascular events, disease events. It is a very controversial topic. There’s a lot of ammunition on both sides. Frankly, I think it’s hard to weigh and balance it.

JM: I don’t think it’s that hard. We’ve interviewed a lot of experts in this area who actually go into these studies. We’ve got previous interviewees who really expand on that in great detail. But some of the primary principles – and I haven’t reviewed the most recent New England Journal of Medicine study that you referenced, but my guess is it is confused absolute risk with relative risk. That is the primary issue. When you look at that more carefully, you realize… This is done intentionally, by design by the drug companies.

One of the former editors of the New England Journal of Medicine, Marsha Angell, she was the editor for over 10 years I believe. She wrote a book almost 10 years ago now that exposed this fraud and this massive corruption between industry and the journals. She was the chief editor of the journal. She went into all these topics. It’s a magnificent read. I’ve never been able to interview her but she really wrote a phenomenal book on it.

I would be very cautious about any study promoting that without looking into the details. There may be some benefit. But you just got to look at common sense rational approach. We were never designed to be taking statin drugs. Never. Now, does that mean it doesn’t help anyone? Probably not. Probably some rare individuals could benefit, but certainly not 1 in 3 Americans over 40.

RS: Yes.

JM: That makes no sense at all. That’s only benefitting the drug companies.

RS: If you want to talk another point in ubiquinol’s corner, we can talk about the research that’s been published about its benefits for heart failure, heart failure patients.

JM: Sure. I don’t have access to the current statistics, but I do know that heart failure is almost an epidemic. There’s a specific physiological condition called diastolic dysfunction where essentially your ventricle becomes really hardened as you age and it can’t really relax and contract completely and diastole as it doesn’t refill efficiently. That continues to worsen.

There’s sort of an epidemic of this in people who exercise quite a bit, especially endurance exercises for many decades. That hits them as they get older. Eventually, it can progress to heart failure. There’s a large number of people who have it. My guess is, somewhat like diabetes and hypertension, the majority of people who have this condition don’t even know it. You mentioned some of the markers for it before, those blood hormones – NT-proBNP [and GGT] – that can effectively screen for that.

RS: Right. There are a couple of papers out there now that is talking about actual physician experience with patients with heart failure. This is what they do day in, day out. They had some of these patients on coQ10, and then they end up switching to ubiquinol because of the better absorption. But bottomline is that they were able to see a reversal in the New York Heart Association (NYHA) class.

That’s the New York Heart Association’s way of rating the severity of the disease. They see reductions in the severity of the disease. They see improvement in the ejection fraction, which is a measure of how well the heart is working in patients for whom they had nothing else to do. They were really going downhill. This is one of the other fantastic benefits of ubiquinol and something that both doctors and patients should know about.

JM: Absolutely. To me, I don’t believe it is accepted as such yet, but it should be standard of care. Any heart failure patient should be on ubiquinol. To me, it’s medical negligence, malpractice not to put a
person who has heart failure on this supplement. Of course, we’re both in agreement that ubiquinol is the preferred form. Even though it’s more expensive, it’s the one that essentially you don’t need to worry about absorption or the conversion to the active form, the reduced form which it’s already in.

RS: That’s right.

JM: It’s clearly worth the extra resources to use that as if you’re going to supplement with coenzyme Q10.

RS: One of the other heart health benefits, which is just a more general nature, is it hooks it to its antioxidant capacity. Whereas it’s not just an antioxidant to the mitochondria but also in the blood where it prevents the oxidation of LDL cholesterol, which is one of the primary steps that leads atherosclerosis. This is another important heart health function of ubiquinol.

JM: Most of the damage that occurs from reactive oxygen species occurs where they’re mostly produced, which is within the inner mitochondrial membrane. But they’re certainly occurring in other places and tissues in the body, as you mentioned. These proteins and cell membrane structures can be damaged by these free radicals, which can be mitigated by the ubiquinol.

I’m wondering, I have a point here to discuss about insulin resistance. To your knowledge, is there any influence of ubiquinol on insulin resistance?

RS: There’s a recent paper that’s on my desk to be read. I haven’t read it yet.

JM: OK.

RS: I can’t talk about that right now. Is there something specifically that you’re referring to?

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JM: No. I just thought there may have been some. I’m not aware of any, but it doesn’t mean it doesn’t exist. I just haven’t seen it. I thought that might be a function.

RS: There is some recent information that is sort of speculating on the possible contribution of people who are on statin drugs who eventually developed diabetes, and there being a connection there. I’m trying to remember exactly what the mechanism was, of why the long-term statin use was causing the diabetes. I can’t recall it right now. But I’m not sure if that has to do with insulin resistance in particular or not.

JM: I know that association, too. That’s a relatively recent association, only recognized within the last few years as far as I remember. We didn’t understand that but it’s not surprising. I’ve seen that pattern so long, so frequently as I’ve been in medicine.

The most significant one that comes in mind is the use beta blockers. That was the greatest thing of all strategies to lower your blood pressure and anxiety. Then we realized, oh, it increases your risk for diabetes. If you have diabetes, you shouldn’t be taking beta blockers. OK.

I call it the law of unintended consequences, which is why you really want to restrict yours strategies to natural therapies if at all possible. They can have them, too. No question about it, especially if poor-quality ones are used, used excessively, or incorrectly. But the likelihood and the consequences are going to be radically reduced. I think that’s a good strategy to use natural approaches like ubiquinol.

In your review of the literature and study of this topic, what would you say are the biggest pearls you’ve learned that we haven’t already discussed?
RS: That we haven’t already discussed… Let’s see.

JM: Or reinforce maybe ones that you already have from a different perspective that we didn’t mention.

RS: I’m a big fan of ubiquinol just because we don’t know, any given person doesn’t know whether or not they have the ability to convert the coQ10 to ubiquinol. If it’s not working, switch. Before you say it’s not working because it’s not working for me, switch to ubiquinol and see. Maybe it’ll work now. I think that’s very important.

I think the idea that our total coQ10 levels start to decrease even by the age of 30 is an important pearl. To recognize that this should be on your radar even if you’re pretty young and still feel pretty good, and that there’s a potential to ramp up your energy levels, your performance – whether it’s exercise performance or mental performance, and whatnot. What else? I think we’ve covered a lot of it.

JM: Let’s talk about the doses.

RS: OK.

JM: Let’s focus on three different groups: 1) seeking the use for anti-aging possibilities, just really integrating it into their program, because they understand that at 30, these levels start to go down. What dose would that individual take versus 2) someone who is already starting to have signs of heart failure (actually four groups), then 3) the group that’s taking statins, and 4) those who are in significant heart failure? Class III or Class IV.

RS: Right. For those people who are just looking for anti-aging or to make sure they have optimal levels in their body but they are healthy people, 100 to 300 milligrams per day is what was used in studies. There’s actually something very interesting that was shown in the study where the blood levels of ubiquinol where looked at over the first, let’s say, (I can’t remember exactly how long it was) a month or two months beginning supplementation.

What they saw was that for the first three weeks, it was ramped up until you got the blood levels to the highest levels they were going to be, until they plateau. Then you can actually take a little bit lower and you would maintain the levels that you have gotten to in just the first few weeks. What we often recommend is 200 to 300, even as high as 300 milligrams, for a healthy person for the first few weeks to ramp up. Then you could drop them to 200 as a maintenance level.

As far as people who have heart disease, clearly they are going to need to do this in conjunction with a physician. But what’s been published by doctors who deal with heart patients is that on average their patients are taking about 350 milligrams, sometimes more, to bring up their blood levels. Their blood levels to start with generally are very, very low. Heart disease is definitely associated with much lower blood levels of total coQ10, which is the ubiquinone and the ubiquinol together.

JM: Is there a blood test one could take if one was seeking to measure this?

RS: Yeah. A physician could take that for you.

JM: The commercial laboratories like Quest or LabCorp would be able to provide that? It doesn’t need a special lab?

RS: That I don’t know. I’m not a clinician. I don’t work with it at that level. I don’t know the answer to that question. I know it’s tested for scientific purposes all the time, but that’s a different kind of lab that one would be using to process the samples.

JM: Sure.
RS: But in any case, higher levels are required for people who have chronic disease. It’s not just heart disease. There are lots of papers that show that many different kinds of chronic diseases are associated with lower levels of total coQ10 – including diabetes, amyotrophic lateral sclerosis (ALS), autism, and syndrome – where supplementation with ubiquinol brings those blood levels back up into the normal range.

JM: Great. The high end would be maybe 1,000 milligrams, 1,200 milligrams?

RS: I don’t know if that much has been used in studies and maybe it’s been used clinically. But I think the highest amount I’ve ever seen used that’s published is about 600 milligrams, and that was for sick people.

JM: OK. Great. Thank you for all the information you provided. It’s been a pleasure to connect with you and to share your insights on this important topic.

RS: It’s been a pleasure to be able to talk with you. Thank you for the opportunity.

[END]