Lupus Society of Illinois
Moderator: Sonya Loynachan
May 15, 2014
7:30 PM ET

Sonya Loynachan:

Thank you for joining us tonight for our educational teleconference. I'm Sonya. I'm the Health Promotion Manager at the Lupus Society of Illinois. This evening, Dr. Robert Katz is going to present on the latest in lupus research.

Dr. Robert Katz is an Associate Professor of Medicine at Rush University Medical Center, a graduate of Columbia University and the University of Maryland Medical School. Dr. Katz completed his internal medicine training at Washington University Medical Center and his fellowship training in rheumatology at Johns Hopkins Medical School.

Currently, Dr. Katz serves in differing capacities on various local boards, including Chairman of the Medical Advisory Board of the Lupus Society of Illinois and the Board of the Arthritis Foundation of Illinois. Nationally, Dr. Katz serves on the board of the Lupus Research Institute. Dr. Katz has been the recipient of multiple awards and has been named one of the best doctors in America numerous times.

Thank you, Dr. Katz, for speaking to us tonight.

Robert Katz: Thank you for introducing me, Sonya.

Robert Katz: When lupus was first described, it was considered to be a potentially fatal disease. We didn't have much treatment available. And some of the worst cases were those that came to the attention of doctors because we didn't have blood testing, we didn't have any way of determining early mild cases, so the most severe cases often came to people's attention and we had no really effective treatment. And then people would try different things and then finally steroids -- and the steroid used these days is prednisone -- and that turned out to be incredibly effective but (inaudible) potential side effects and probably didn't really completely quiet down the disease going forward. They realized after a while that lupus was an immune disorder -- considered to be auto-immune, auto being self. So instead of the immune system just defending us, it seemed to be attacking the self -- auto-immune. And so then there was a push to try medicines for auto-immunity -- self-immunity. And some of those were to dampen down the immune system misdirection overactivity -- overactivity of the immune system but misdirected. And so some of the immune suppressants started being used because they were used in cancer chemotherapy to try to quiet down cancer. They turned out to be suppressing the immune system which was otherwise defending us, and so then the immune suppressants began to be used, like Imuran, also called azothioprine -- like methotrexate and others started to be used to suppress the overactive but misdirected immune system. At the same time, medicines that were used for other purposes, especially Plaquenil, or hydroxychloroquine, that were originally used for malaria but found to have a new home in treating lupus -- seemed to be helpful, especially for lupus rashes and lupus joint pain.

So you had steroids. You have immune suppressants. You have Plaquenil. And then a few other things like non-steroidal anti-inflammatory drugs, like Motrin or Celebrex or things like that which are mildly helpful for joint pain -- not extremely potent.

As more immune suppressants began to be used, then it was determined that the strongest one being used in lupus was Cytoxan, or cyclophosphamide, and that's still used today, especially to try to induce remission in bad active lupus kidney disease or major lupus problems, like central nervous system lupus. Otherwise, people try to stay away from Citoxan and cyclophosphamide because of the side effects of infection, of rare cases of tumors and leukemia going forward, and also in young women of causing infertility. Now Cytoxan was used both intravenously -- which is used more common by rheumatologists these days -- and orally as a pill. After Cytoxan proved to be effective but perilous, then Cellcept came along as a competition for cyclophosphamide, and it proved to be either equally effective or slightly less than equally effective -- maybe Cytoxan-light, in the sense that it's effective and it's an immune suppressant. It's almost equivalent to Cytoxan. It doesn't cause the same array of side effects. It's usually better-tolerated. Hair loss can occur with Cytoxan. It doesn't occur with Cellcept, for example. And the infections can occur with both, but more commonly -- as well as blood count reductions like white blood cells and red blood cells -- more commonly with Cytoxan. So Cellcept came to be a very frequently used drug for organ lupus where there was more significant involvement and Cytoxan for the sickest patients, along with prednisone to suppress the inflammation caused by auto immune disease. And inflammation is like getting a splinter in your finger, the finger gets red, tender and swollen. That's inflammation, whether it's in the joints or it's around the lungs -- pleurisy, around the heart -- pericarditis. It's the same kind of inflammation -- pain, swelling, tenderness -- but can't see it because it's internal, but there's still pain, swelling and tenderness.

So between Cytoxan, Cellcept -- which is also -- has a long generic name called mycophenolate mofetil -- methotrexate, cyclosprine, tacrolimus, imuran azathioprine -- there are a number of immune-suppressing drugs out there that are used to try to quiet lupus down.

Now many people with lupus have concomitant osteoarthritis, concomitant fibromyalgia with widespread pain, and some of those patients need to be treated both for lupus and for the other pain problem that seems to occur in some patients concomitantly.

The era that we're into today is is the day of biologics coming? So biologics are medicines that are made in living organisms and then duplicated by growing these organisms in tissue culture, having them produce the substance that we're looking at and maybe antagonizing that substance with a protein antibody and producing large amounts of it. So not like in a chemical factory, but in a biological lab with living organisms do they make this new array of medications. The first one that made any sense was Embrel, used for rheumatoid arthritis. Then Humira, which -- and Remicade, used for rheumatoid arthritis, and sometimes other kind of arthritis -- ankylosing spondylitis, Crohn's disease, arthritis and psoriatric arthritis. And then the Orencia, Actemra and others. And so more recently, there's been a push to look at biologics made from living organisms in lupus. Is this the day of biologics? And so the most recent interesting research in lupus has to do with the onset of clinical trials for biologic therapy in lupus. The first significant one was Rituxan, which was approved for treatment of lymphoma and some other disorders. It proved to be somewhat helpful in people you sometimes give it for lupus patients, but it was never approved by the FDA because it didn't meet the endpoints in terms of effectiveness. But it's good and still used.

Then there were various trials of Benlysta, belimumab. Belimumab -- umab means a monochronal antibody -- MAB -- directed, in this case, against a product that makes antibodies. There are lymphocytes -- white blood cells -- that make antibodies, and they're called B-lymphocytes -- B as in Bob. And Benlysta antagonizes one of the

products made by these B-lymphocytes -- B-lymphocyte stimulating factor. And it's found to be moderately effective for lupus -- not used in the most severe cases yet. But it helps people get off steroids. Many people are very happy with it. It helps the skin problems, the joint problems of lupus, and maybe -- especially at a higher dose -- it might be helpful for more serious lupus. That remains to be seen.

Recently, there have been some clinical trials ongoing on a medicine called Orencia, which didn't quite succeed in meeting endpoints for efficacy for lupus. And Eli Lilly is testing antibody against a product called CD-22. That's on the surface of these B-lymphocytes that make the antibodies. And the antibodies, as I said, in lupus are misdirected.

So there are other studies out there looking at other clinical products for lupus. Whether they'll prove to be effective enough, safe enough and receive FDA approval to be able to give it to lupus patients remains to be seen. But at least there's a lot more interest in using these biologics in lupus patients.

And so that's sort of where it stands. We've already identified a lot of antibodies in the blood associated with lupus and tried to understand them better. We're aware of the increased cardiovascular risks of active lupus and lupus in general, and we're trying to reduce those cardiovascular risks. We're aware of the antibodies that predispose to blood clots in lupus, and we're trying to deal with that like any cardio (inaudible) antibodies and the lupus anticoagulant. And so there is much better and more active treatment and more active research in attempting to treat lupus and identify lupus and treat lupus better.

So that's an overview of where we stand.

Sonya Loynachan: Do we have any questions?

Unidentified Participant: I have a question.

Robert Katz: Yes.

Unidentified Participant: I've recently heard something about biomarkers. Can you explain a little bit of that for me?

Robert Katz: Yes. I'll try to. Rheumatoid arthritis -- there's been a development of CCP antibodies -- cyclic citrullinated peptide antibodies. That's an example of a biomarker. It helps to identify patients who truly have rheumatoid arthritis. And it's hoped that, by following this antibody level over time, it may lead to an index of how active and severe the disease is. Biomarkers in lupus are usually blood products that tell you it's lupus, tell you what -- how severe it is, and are measures that can be looked at in blood testing that will -- can be followed to determine the success of treatment. So they're generally blood tests that are markers for severity, the diagnosis and treatment effects.

Unidentified Participant: Okay.

Unidentified Participant: I have a question that follows that question.

Robert Katz: Yes.

Unidentified Participant: In your experience with your patients, I know that a lot of times the blood markers are not very telling in terms of disease activity, or there isn't a real strong correlation between

blood markers and efficacy of drug treatment or when do we use drugs again or that sort of thing. Have you found that? My daughter has very high levels of CCP auto-antibodies and they've never changed throughout the course of her treatment, regardless of what drugs have been used, and they're not really using that as a biomarker. I'm just curious in -- as a clinician treating patients with lupus, what do you tend to follow in labs and so forth to make the hard decisions? For example, should we use Rituxan again or we should try this course of therapy? To me, it just seems like there aren't any hard and fast protocols right now in lupus treatment, as far as I can see anyway, and that's one of the things that seems to be so frustrating.

Robert Katz: Good question. Well, lupus is not a one-size-fits-all disease. So when a patient comes to me, I try to identify which clinical features -- it could be pleurisy, it could be joint pain, it could be a rash, it could be something else -- and what laboratory features -- it could be a sed rate, it could be complement, it could be DNA antibodies, it could be C-reactive protein -- are most abnormal or elevated, and those are the -- the clinical factors and the blood factors are what I'm going to follow in that Patient X. I'm not going to follow kidney function terribly closely if there's no involvement in the kidney. I'm not going to follow CCP antibodies, which is more rheumatoid arthritis antibody, because generally they don't change too much anyway, but I'm going to try to identify in your daughter or anybody what correlates with either more serious disease in that person or more active disease in that particular patient. Is it a low platelet count? Is it a low white blood cell count? Is it a high -- again -- DNA or other antibodies? And try to tailor that so that when I start them on treatment, if those -- if they get better clinically -- their symptoms are better, joint pain's better, their rash and all that stuff -- and some of those features we talked about before -- lab work stuff -- improves, then I look at it as a valuable index in terms of how that person is doing and will continue to follow it. But when you start reading about lupus, you read about a million different biomarkers, clinical markers. You can't follow everything. It won't make -- you'll just get lost. So you have to identify for that particular person the fingerprint of what seems to correlate with active or more serious disease and try to modify those things and follow those things, in particular rather than the whole (inaudible) -- the whole thing, which will just get everybody confused.

Unidentified Participant: To follow along with that question -- and this is probably a more difficult area and it's -- there's probably less known about it -- but more specifically, in CNS lupus, other than doing spinal taps every so often, what markers would you look for in CNS lupus? I know that spectography and MRI scans aren't really sensitive enough in that disease to really give you much information. I -- we had a spinal tap done at the very beginning that showed some oligoclonal bands. She responded very positively to Rituxan after it was given. Then she also had Cytoxan. But about a year after the Rituxan was given, she started to show signs of slipping. And again, it's not like you can just plunk her in an MRI machine and look to see if there's any changes going on in the brain.

Robert Katz: Well, generally speaking -- not every time, central nervous system lupus occurs in the context of active general lupus. So in other words, if the person also has -- I don't know -- joint pain or has a rash or has pleurisy or has fever or something like that, those are the things you follow clinical. If the lab tests show a high sed rate, high C-reactive protein, low complements, high anti-DNA, at the time that they're sick, those are the things you follow clinically. You're right. Treating the brain is challenging, especially if it's not within the context of active lupus, because you really can't get in there. It's bordered by the skull. MRIs aren't that sensitive. Spinal taps can be helpful, but who wants to keep doing spinal taps? So in a way, you're looking at the lupus globally. You have to be sure, if nothing else is active -- only the brain -- that it's truly central nervous system lupus and isn't masquerading as that because -- so that's tricky. But generally speaking, it

occurs in the context of active systemic lupus where there are other features that are easier to follow.

Unidentified Participant: Yes. I understand what you're saying. That makes sense. Thank you.

Robert Katz: Sure.

Unidentified Participant: You mentioned something about replacing prednisone with something. Is there something out there – have there been studies recently? Because I have been trying to get off of prednisone for 15 months now, and every time I do -- my lupus seems to be affecting my lungs only, and the only symptoms I have is I have shortness of breath, I have trouble breathing, my heart rate goes up. Is there anything out there that I can talk to my doctor about?

Robert Katz: Yes. So in other words, prednisone was the first effective treatment and then --

Unidentified Participant: Yes. And I've been on it for 15 months, and every time they try to taper me down, I get down to 5 or 4 and I start having chest pains again. And for CT -- I've had 15 CTs in my 10 years and -- with this. Well, actually, I was only diagnosed 15 months ago, but they claimed I had rheumatoid arthritis 20-some-odd years ago, which now they're thinking may have been lupus all along. Who knows? But that is my only issue. I don't have any rashes. I'm not sensitive to the sun. I have no problem sleeping. I have no aches and pains. It's just my lungs. Which my other question I guess would be can there be something mimicking lupus, that could be completely something different? I don't feel like I have it. I don't have a bad case, compared to so many people that I've been reading about. I have energy. I can sleep (all these things, where a lot of people don't have those things).

Robert Katz: So originally, prednisone was the most effective treatment and then we started using the immune-suppressing drugs because lupus has an overactive but misdirected immune system.

Unidentified Participant: Right.

Robert Katz: And so whether you're talking about methotrexate, Cellcept -- mycophenolate mofetil -- you're talking about azathioprine -- Imuran -- you're talking about -- immunosuppressant -- most people -- if people are able to get down to a low enough dose of prednisone but can't get off of it, I don't think that's so bad because a low dose of prednisone isn't really so bad. But if somebody can't get down from higher doses on prednisone, then usually one would go to the immune suppressants next -- one of the ones I mentioned or something others -- and try to then quiet down the lupus and reduce the dependency maybe not to zero, but (inaudible). Whether you have lupus or something else involving your lungs, if you're going to rheumatologists and pulmonologists, you're getting the best shot at trying to define what it is --

Unidentified Participant: Yes.

Robert Katz: But if you're not -- can't get to a satisfactorily low dose of prednisone -- usually 10 mg or less --

Unidentified Participant: Okay.

Robert Katz: That's not so bad. But otherwise, it would be immune-suppressing medicine and then at the end -- I don't know if you heard me. We were talking about the coming age of biologics, including Benlysta --

Unidentified Participant: Yes.

Robert Katz: But others are coming out. And then someday it might be biologic therapy that would be the go-to drug to try to reduce or be able to allow you to discontinue prednisone. So it depends on what dose of prednisone. If you're not able to get down to a low dose like 5mg or 10 mg, I don't know if you want to consider adding another drug. That's an individual decision.

Unidentified Participant: I'm on prednisone, as well as Plaquenil and Cellcept. Cellcept doesn't seem to be doing anything either, to get me off of prednisone. I have also read that Cellcept combined with prednisone increases your risk of cancer. So that's what my naturopathic doctor suggested, and that scares me to death. You hear a lot of higher increases of cardiovascular disease and obviously bone issues. Preferably, I'd like to be on no medicine, but I know that's virtually impossible at this point. But my goal is to at least, at minimum, get off of prednisone and it doesn't seem to be happening. I guess I just have to deal with it. I'm trying every other avenue to get off of it and I just started some other stuff through the naturopathic doctor, as I mentioned, but -- okay. So you're saying as long as -- 10 and under, that's considered low-dose.

Robert Katz: 10 and under is your low -- I don't think there's any concrete evidence, unlike with Cytoxan, that Cellcept causes cancer, or prednisone causes cancer. There's a question of lymphoma, but lymphoma is probably increased in lupus to begin with. That's a lymph node --

Unidentified Participant: Okay.

Robert Katz: And most of them -- I don't -- I wouldn't worry about cancer, except for Cytoxan where there is some concern.

Unidentified Participant: Okay.

Robert Katz: I think if you're down to 10 mg or less and you're okay, I would say that's not so bad. Other immune suppressants might work better or worse than the Cellcept. I don't know. But usually it's prednisone, immune suppressants, Plaquenil and then the coming age of biologics.

Unidentified Participant: How far out are the biologics? Are we talking years or decades?

Robert Katz: I think at this point, in terms of getting more on the market, we're talking years, and in terms of -- lupus -- the financial incentives for drug companies to develop expensive lupus biologics isn't quite there yet, as opposed to more common illnesses like rheumatoid arthritis. So it's slower than it is for more common illnesses, but it's still there and I think it's -- it'll be -- it's coming out gradually.

Unidentified Participant: Okay.

Robert Katz: Okay?

Unidentified Participant: Okay. Thank you.

Robert Katz: Sure. Thank you.

Unidentified Participant: Hi. I have a question.

Robert Katz: Sure.

Unidentified Participant: I was diagnosed when I was 35 with rheumatoid arthritis. So I've been taking Plaquenil all of these years for that. And in 2008, I was diagnosed with lupus. I've been on prednisone and methotrexate. Okay. So I moved to another state last year and I've gone to three different doctors here, and each one of them is telling me now, "Oh, you don't have lupus. You have Sjogren's. You've been misdiagnosed." And I've had three doctors in the other state tell me that it definitely was lupus, but when I moved to this state, they're telling me that I was misdiagnosed. But they're still letting me take the Plaquenil. Why? I don't know. They said they weren't going to take me off of it. But they're saying that I just have maybe arthritis because back in the day, they are saying that now -- it was -- the test that was used to determined rheumatoid arthritis -- they don't use those anymore and even the test that they used in 2008 was outdated. He asked me, "What did they do?" I said, "They took blood." And I gave them all of my symptoms and he says, "Well, I don't think that you have it." So at this point, I really don't know what to do. I don't know what to think. And I ache constantly. I'll be in excruciating pain, all of my joints, and I'm just -- I get the rash on my face. I do have the Sjogren's -- the dry mouth -- but they're saying that I don't have it. So I really don't know what to do at this point.

Robert Katz: Yes. Lupus is sometimes very easy to diagnose, but it has cousins, like Sjogren's syndrome and rheumatoid arthritis. And then sometimes you see people with widespread fibromyalgia pain who also have lupus or Sjogren's syndrome. So the complex of related illnesses makes it sometimes imprecise. Sometimes it's obvious. It's lupus rash. You biopsy it and it shows lupus on the biopsy. A kidney biopsy shows lupus. Sometimes it's clear-cut. But there's an art to it, and sometimes it's not so clear-cut and you can get different opinions about precisely what it is. And when I go to rheumatology scientific conferences and we present a case, there's sometimes quite a bit of discussion about is it lupus or is it not lupus. So yes, those illnesses -- whether it's lupus or Sjogren's or rheumatoid arthritis or fibromyalgia -- can be -- there can be overlaps. There can be -- sometimes when we have more information, you change your opinion. So sometimes lupus is clear-cut as anything and sometimes it's a bit muddy -- murky. I don't know what else to say to give you peace of mind on it. It's just that you have to choose a doctor who you feel comfortable with and go with that because you might still be getting different opinions.

Unidentified Participant: Well, I went to three different doctors that were referred to me. And you won't believe it. All three of these doctors studied together at the same school and they know each other. So what they're doing -- the last two -- they're basically looking at -- my papers -- my medical records are transferred to them. They're looking at the very first doctor and telling me, "Well, this is what she found." So they're not really listening to me and I'm afraid that if they take me off the Plaquenil, I do not know what I will do.

Robert Katz: Yes. Well, again, there's a comfort level in dealing with your medical people and that you have to get to some -- one of those doctors or another doctor who is going to try his/her best to help you and stick with that one person. Sometimes it does get confusing when you're seeing multiple doctors, especially if they don't all agree. Some agree Sjogren's, some lupus. But you've got to choose somebody who's going to be your -- whether rheumatologist or anything -- pulmonologist, nephrologist, cardiologist --

because it's going to totally confuse you like it's doing -- you're doing now -- it's doing now to get these similar but different opinions.

Unidentified Participant: Well, let me explain something. The reason -- I would have just gone to different doctors. The reason I did that -- the first two -- they were really rude. It was like a joke. They started laughing. And I told them that I was diagnosed at Rush Presbyterian at St. Luke with RA in Chicago and then I told them that I was diagnosed with lupus and they laughed, like it was a joke. They were laughing. That's why I didn't feel comfortable with them, so that's why I went to the other doctors.

Robert Katz: Okay. I understand. But you've got to find somebody that you do feel comfortable with that seems to be on your side and stick with that person.

Unidentified Participant: Yes, of course. I agree.

Robert Katz: It's important for many reasons to trust the person you're dealing with and to have a positive therapeutic relationship. You can't feel that anybody is, like, unkind or laughs behind your back or -- that's not going to work. So you do have to get that accomplished with somebody who you trust.

Unidentified Participant: Okay. Well, I guess what I was really asking was can that happen when you know you have people that you really trust that you've been diagnosed by a team of doctors, and then all of a sudden you go to another state and then the other doctors are, like, ridiculing those doctors and they don't know who they're talking about.

Robert Katz: Well, that's pretty annoying but -- and I think it's inappropriate, but I don't know. Lupus and Sjogren's syndrome are similar enough that you could have the diagnosis change sometimes from one to the other, but I can't tell you which would fit you better. I'm just telling you that they are similar.

Unidentified Participant: Alright. Okay.

Robert Katz: Okay? Good luck.

Unidentified Participant: Thanks.

Unidentified Participant: Dr. Katz, I do have a question.

Robert Katz: Yes.

Unidentified Participant: I was admitted about 5 years ago several times with pleurisy and pericarditis. I was finally diagnosed with lupus approximately 3 years ago. My problem is like every lupus patient, I guess. They get this cornucopia of drugs and try to figure out rolling the dice. The new one -- the latest combination I have is the Cellcept, Plaquenil, prednisone and something new that I have never heard of called Arava. Can you tell me a little bit more about Arava?

Robert Katz: Sure. So among the immune-suppressing drugs -- cyclophosphomide, also called Cytoxan, mycophenolate mofetil, also called Cellcept, methotrexate, azothioprine, Imuran, and others like cyclosporine, tacrolimus -- there's one that was used for transplantation called Arava. And so you have -- obviously you have to suppress the immune system if you're getting somebody else's liver or kidney or heart. And so Arava was used for that. It's also called leflunomide. And so because it was an immune

suppressant and lupus is an autoimmune disease, there was a natural interest in studying it and it proved to be somewhat effective. So leflunomide Arava, an immune suppressant, is used for lupus and it can be effective in many patients.

Unidentified Participant: Do you have a group of meds that you find most successful with the majority of your patients? I understand that everyone's different. But in your experience thus far, have you found one set of drugs that have been most successful with limiting the number of flare-ups with lupus? And I guess I'm talking systemic lupus, no kidney, because that's me. I'm usually impacted at the lungs and heart, having trouble breathing, loads of problems. Not as much pain. But I'm just looking for a combination that might work for me and I'm on low dosages of prednisone right now at 5 mg. I was just wondering if maybe there's a combination that you've seen be most successful with many of your patients.

Robert Katz: Well, I'm going to tell you what I personally have seen, but it's going to be different than what your doctor has seen and different than the next doctor, because that's why you do clinical trials and bigger studies is to see, in a whole array of patients, what seems to work. I've found in the past that methotrexate is quite effective. I've found Arava is quite effective. Those are two drugs that I kind of like to use. I think Cellcept is effective. Cytoxan is the most effective but it's more toxic. I would say methotrexate and Arava -- I really like. I like Tacrolimus, but I hardly ever use it these days. So it depends obviously. Every patient is different. But that's been my experience.

Unidentified Participant: Okay. Would you be using Arava and methotrexate at the same time?

Robert Katz: Without knowing the details, I don't want to tell you what to take, but I do use them together sometimes, yes. I don't usually use methotrexate and Arava and Cellcept together, because that's three immunosuppressants. But Arava and methotrexate, I do use together.

Unidentified Participant: Okay. So with a combination of prednisone, Arava, Plaquenil -- do you think that's a decent combination? I know you don't me yet and you're not my doctor and I'm not going to go to my doctor and say, "Dr. Katz says do this." That's the combination I'm on.

Robert Katz: I like that --

Unidentified Participant: Would you suggest adding methotrexate to those three?

Robert Katz: Well, I like that combination. Some people would be hesitant to add methotrexate because of liver toxicity or other issues. It depends how sick you are and how high a dose of prednisone you are on. You've got to individualize it. But I would say I like Arava. I like prednisone and Plaquenil. Whether to add methotrexate -- that would be somewhat -- be hesitant to do that and others wouldn't. It depends.

Unidentified Participant: Okay. Wonderful. Thank you.

Robert Katz: Sure.

Unidentified Participant: So the combination of methotrexate and Arava -- would that be both combined -- maybe a prednisone-sparing combination or no? I know, again, everyone's different but (inaudible)

Robert Katz: I would -- in a very small percentage of patients do I use methotrexate and Arava together.

Unidentified Participant: Oh, okay.

Robert Katz: It's not -- I think it's a good combination. I do use it. But it's not very common because I don't want to expose my patients to increased risk. But people -- the first thing out of their mouth is, especially when they hear these TV ads about --

Unidentified Participant: Yes.

Robert Katz: All of these medicines. The first thing out of their mouth is, "I really am nervous about taking these medicines because of side effects."

Unidentified Participant: Of course.

Robert Katz: So to expose them to multiple medicines is not what I like to do. I don't want to see complications. So I do it sort of cautiously and reluctantly in patients not responding to prednisone, or those who can't get down to a lower dosage of prednisone. I'm referring to 10 mg or less. I'm happy with 10 mg.

Unidentified Participant: Okay.

Robert Katz: And those -- and in individual cases, you have to, change the recipe, but not for everybody.

Unidentified Participant: Got it. Okay.

Operator: Do we have any other questions?

Unidentified Participant: I'm just curious about something. I've never really understood this with lupus. There are so many different drugs with so many different mechanisms of action, and it is kind of a recipe, as you said, in trying to find the right recipe for the right person. Are the mechanisms by which people have lupus different or does everybody have the basic same underlying disease? Do you understand what I'm saying? Is it just affecting a lot of different things in each individual patient and that's why the recipe is so drastic? If somebody has hypertension, they go to the doctor. You have step A, you have step B, you have step C. With lupus -- I was a pharmaceutical rep for years and I worked in a lot of different areas. I never worked in autoimmune but I'm learning an awful lot about it because of my daughter. It's just so -- it just seems like such a dice toss to figure out what the right combo is for treating patients in this disease.

Robert Katz: Yes. Well, you have to rely on clinical trials. You have to rely on experience. You have to rely on national meetings and reading about this to try to get a sense of which drugs to go to under which circumstances. Autoimmunity is complicated. Because it's a multi-organ kind of problem, it becomes complicated. Blood pressure -- you're looking at pretty much 2 numbers -- the systolic and the diastolic. Also, you want to be very careful about side effects. So with a combination of medicine that includes prednisone, Plaquenil, non-steroidal anti-inflammatory drugs, that includes all the immune suppressants we talked about, that includes Benlysta, Rituxan and some of the newer ones coming up, to tailor the treatment to get a good response is tricky because every patient's just not the same. I don't know. But lupus is definitely a complicated illness. Most doctors are wary of attempting to treat it because it's -- there are a lot of variables.

Unidentified Participant: Are there genetic links between the different autoimmune diseases? We had 4 different generations in my family of women with 4 different autoimmune diseases.

Robert Katz: I've seen a ton of people who have antinuclear antibodies and they sometimes want to know if their symptoms are due to those, and a lot of times they're not. They're just familial. And then sometimes people -- in one study, if people would develop lupus -- it was done in the military -- people with antinuclear antibodies without lupus were followed. And the people with antinuclear antibodies without lupus who slowly over a 10-year period acquired more autoimmune antibodies -- Smith antibody, DNA antibody, Sjogren's A antibodies, ribonuclear protein antibodies, chromatin antibodies and others -- people that acquired more of them were the ones who developed lupus. So there's a sort of basic level of autoimmunity that can exist in families, a susceptibility. And then over time, there are people who, for whatever reason which we're not sure, march on and develop lupus from that susceptibility. And that's always hard to predict. So there's an autoimmune -- autoimmune findings often in many relatives who don't actually progress to develop the disease. And sometimes people develop a different array of antibodies and often different symptoms and clinical manifestations. So it's complicated, but most of us in the field can recognize lupus. They realize the cases are different. We try to follow markers to see whether the patient is improving because when they were sick, these particular markers were abnormal, and trying different strategies until the patient is better or in remission. I don't know. It's complicated. That's true. How's your daughter doing?

Unidentified Participant: She was good for about 11 months. She went through Cytoxan and also Rituxan. She has CNS lupus. They originally diagnosed her with rheumatoid arthritis when she was 12. And within months of being diagnosed with rheumatoid arthritis, she started to develop signs of CNS lupus and so they had to kind of pull out the cannons. And she was good for about 10 or 11 months and then slowly started to show signs of -- well, lupus exacerbations and then the CNS manifestations as well. And for me, it seemed kind of coincidental. I don't know -- I know Rituxan can last for 12, 13, 14 months, but it was kind of coincidental that around that timeframe is when she started to show signs of starting to slip again. She's in the process of going through another series of Rituxan infusions and high-dose pulse steroid infusions. But she's got just about every blood marker you could have, both for very high CCP auto-antibodies and RF factor and had multi-joint involvement in the beginning as well. So she's kind of got the -- I guess they call it rupus -- but she had a seizure disorder. And just to complicate things even more, they -- she had immune system issues when she was young so they're thinking that the -- her body -- that might be correlated with the autoimmune issues that she's having now. Like I said, 4 generations. We've had MS, lupus, Sjogren's -- just about everything you can say as far as autoimmune issues with the women in my family. She's going through another tough time right now with treatments.

Robert Katz: Well, good luck.

Unidentified Participant: Thank you. I think that Rituxan, even though I know the FDA didn't approve it for lupus -- I felt like that was the one drug that really helped her the most.

Robert Katz: Well, I like that drug in that sense because it's not terribly toxic --

Unidentified Participant: Right.

Robert Katz: If it works -- but -- yes.

Unidentified Participant: I'd rather use that again than Cytoxan.

Robert Katz: Cytoxan is --

Unidentified Participant: Yes.

Robert Katz: Stay away from that if you can.

Unidentified Participant: Yes.

Robert Katz: Even though -- I don't want to be -- in any individual case --

Unidentified Participant: I get it. Believe me.

Robert Katz: Yes.

Sonya Loynachan: Do we have any other questions?

Unidentified Participant: I have kind of a general question, I guess. Once you have lupus -- and like for my instance, it's affecting my lungs primarily, a little bit to the heart -- could it develop to be in the kidney or anywhere else? Or does it -- once it attacks one organ, it pretty much sticks with that?

Robert Katz: Well, because of the antibodies being different, the markers being different, usually the lupus that you have will stay the same. Not every time and lupus can surprise But I don't see too many people who have skin and joint lupus who, 5 years from now, develop kidney lupus. It can happen but usually it doesn't happen. So most patients, early on, develop a pattern to their lupus -- not everyone though.

Unidentified Participant: Got it. Okay.

Sonya Loynachan: Any other questions?

Robert Katz: I thank you. Let me just tell you about the Lupus Foundation changing its name to the Lupus Society of Illinois because we've sort of become independent and we're kind of going out and doing the same education and supportive research and things like that, but in a slightly different way, and we hope this new era is going to be a successful one. So just to say that I'm pleased to be able to speak on behalf of the Lupus Society of Illinois.

Sonya Loynachan: Thank you, Dr. Katz.

Robert Katz: Thank you, Sonya.

Sonya Loynachan: This educational teleconference is recorded, so it will be posted on our website within the next few weeks. Everyone can take a look at the transcript. Thank you!