Sean Collins (00:06):
We are on the cusp of a game changing moment in medicine,

Dr. Radica Alicic (00:11):
However you take it. It is a game changer.

Sean Collins (00:18):
An evolving class of medical therapies originally prescribed for the treatment of type two diabetes is proving to be extraordinarily beneficial for people with chronic kidney disease and those with atherosclerotic heart disease.

Dr. Radica Alicic (00:36):
I think that patients calling game changers primarily because of weight loss, but providers and physicians are calling these medication gamechangers because of the organ protection and the effect on the risk of dying and progression of kidney and heart disease.

Sean Collins (00:53):
These agents are called glucagon-like peptide agonists, you'll see them referred to as GLP. And their potential to change how we treat two of the most deadly conditions is profound. These treatments are expensive, but so is the treatment of heart and kidney disease that we're doing now.

Dr. Katherine Tuttle (01:14):
We've got to have changes in health policy and the type of healthcare we prioritize in our healthcare systems. We focus way too long on disaster relief, right? Whether it's an acute MI or a car accident that frankly is not very cost-effective. And honestly, if we actually truly walked the talk of improving the health of the population, it would be cost effective

Sean Collins (01:42):
On today's program discussion of a game-changing moment in medicine with two research physicians on the front lines. Stay with us. It is the Hear Me Now podcast. I'm Sean Collins. Thanks for listening. Sometimes you just have to marvel at how the body works. We all grow up learning about blood circulation and how red blood cells deliver oxygen to tissues throughout the body. And we later learned that the same bloodstream collects waste material that then gets filtered by the kidneys or metabolized by the liver. We learn about the nervous system and how electrical chemical signals get sent back and forth between different parts of the body and your spinal cord and your brain and all of that information gets taught to school kids in one form or another. And we walk away thinking that we've got a pretty good sense of how things are interconnected and interrelated in our bodies.

(03:00):
Then they throw in hormones. They're like the secret undercover agents of the body being released by a gland in one part of the body, traveling through the bloodstream and having an effect in a distant part of your body, sending a crucial message without a nerve signal, but just as powerful to turn on some function or to down-regulate something else. These hormones are constantly sending messages throughout our bodies that are picked up by receptors and acted upon, and our life depends on them. Someone should make a movie about hormones. A relatively new class of drug therapies are available now that involve agents that look enough like human hormones, that they function in many of the same ways. One class is called GLP one agonists more on what that means in a few minutes. But these GLP one agonists are being used to treat type two diabetes to reduce cardiovascular risk, to help people with chronic kidney disease and to assist with weight loss for people with obesity.

(04:18):
You've probably heard some of the brand names of these drugs, Ozempic and Mogo and Mandu. They've all made headlines this year. They get talked about on talk shows, patients and doctors call them game changers. Today we're going to talk about them

too, to try to understand how they're being used, how they work, how they're being paid for, and what they might mean not only for people with type two diabetes, but people who are obese, people with heart disease and with kidney disease. My guests are both involved in research at Providence. Dr. Catherine Tuttle is the executive director for research at Providence Healthcare in Spokane. Dr. Tuttle, welcome.

Dr. Katherine Tuttle (05:04):
Hello, great to join you today

Sean Collins (05:06):
And also with us from Spokane, her colleague Dr. Radica Alicic, who's the associate director for research. Dr. sic, thank you for being with us today.

Dr. Katherine Tuttle (05:17):
Thank you, Sean.

Sean Collins (05:18):
I keep saying that lots of people are calling the GLP one agonists game changers. Am I overstating things?

Dr. Katherine Tuttle (05:26):
No, I think that is a very true statement. I think that patients are calling game changers primarily because of weight loss, but providers of physicians are calling these medications game changes because of the organ protection and the effect on the risk of dying and progression of kidney and heart disease. So however you take it, it is a game changer.

Sean Collins (05:53):
So you're

(05:53):

Saying that the weight loss element that gets so many headlines is a game changer for a lot of people who are interested in losing weight, people who with obesity and also people who have a sort of cosmetic interest in losing weight, but there's also organ protection that is accompanying the use of these drugs that in some ways is much more important and will have a bigger impact on the overall population health because heart disease and kidney disease are so deadly.

Dr. Radica Alicic (06:26):
Yeah, Sean, I'm happy to weigh in on that one. The drugs do cause a remarkable weight loss, which in and of itself is beneficial, especially for people who are living with obesity, and there are many health benefits for weight loss in people who are obese. So I want to acknowledge that upfront. But the effects of the GLP ones and the GLP one GI agonists, the dual agonists like ura, go beyond the effect on weight. And there have even been studies we call mediation analysis done, where we look at what are the contributors to the improved outcomes and the outcomes we're talking about here are survival alive or dead and whether or not there's been a kidney or heart event. And in those mediation analyses, well, the drugs do provide benefit through weight loss and improved glycemic control. That only accounts for about 25% of the benefit indicating that 75% of the benefit is independent of weight or blood glucose, and based on an abundance of preclinical science, what we know is that these agents have direct effects on the heart and kidney, which are probably dominating for organ protection.

Sean Collins (07:47):
It sounds like we're on the cusp of things changing in a pretty profound way with disease processes that are responsible for a lot of deaths across the population.

Dr. Radica Alicic (07:59):
Right, and that's why they truly are game changers. There are ongoing studies, but we already know enough about at least the GLP one class that both the American Heart Association, the European Society of Cardiology, recommend them as first line treatments for people with atherosclerotic cardiovascular disease to reduce risks of

major cardiovascular events, and at least one agent in the class liraglutide and the leader study reduction in cardiovascular death and actually also Semaglutide and Pioneer six. And then with regard to the kidney studies, they are still ongoing. Although the big flow trial, the kidney disease outcome trial made a decision, the executive committee made a decision to stop the trial on October 11th, 2023 because the Data Safety Monitoring Board said that there was clear evidence of efficacy and that we should not continue. This is the first trial stopped for early efficacy. So we don't know what the outcomes will be yet, but that's a very high bar. And so with regard to kidney disease, they're recommended in people with kidney disease for weight management, glycemic control and atherosclerotic risk reduction. But based on stopping the trial early, it is very highly likely that GLP ones will be elevated to first-line therapy for kidney protection in the near future.

Sean Collins (09:34):
And if I understand it correctly, the reason that a trial would be stopped for ethical reasons is because it's considered unethical to continue delivering a placebo to a population of people who would clearly benefit from the active drug. Is that right?

Dr. Katherine Tuttle (09:53):
That's a great way to summarize it, Sean? Yeah.

Sean Collins (09:57):
Dr. Alicic, do you have anything to add to that?

Dr. Radica Alicic (10:00):
Yeah, I think that change could not come soon enough. Right now, American Association of Endocrinologist actually has two pathways for treatment of people with diabetes. So they pointed out that use of glucagon-like or GLP ones you're talking about, semaglutide should be used in everyone who has kidney or sclerotic heart disease independent of their need for blood sugar control. The recommendations from some other professional societies a little bit different, but just to keep in mind that

these are not just blood sugar lowering agents, but that we are on the path of approaching these medications as a organ protective therapies independent of their blood glucose or blood weight lowering properties.

Sean Collins (10:52):
I'd like to talk about that organ protection and what we know about what's going on there. And I hesitate to ask in any sort of listed way in order, because obviously these conditions are sort of linked together in a network in a lot of ways in real life. So to talk about kidney disease without also acknowledging diabetes or without acknowledging heart disease is a little artificial. But let's climb into some of those silos for a moment and talk about atherosclerotic heart disease. What do we know about how these agents are functioning in that environment?

Dr. Katherine Tuttle (11:37):
Well, I'm happy to jump in on that one. And both Dr. Alicic and I have been involved in these studies for a number of years now. It's very gratifying to see the therapies translating to true clinical benefit, but we really think that the GLP ones and like agents like the GIP GLP one agonist and actually a future triple agonist to come predominantly are working in an anti-inflammatory mechanism. And while there are LP receptors in both intrinsic heart and kidney cells, the real action may be on invading immune cells in those organs that are injured. So macrophages and T lymphocytes, which have an abundance of the receptors, and basically these agonists when they engage that receptor at the cell level, shut down a number of the pathways to inflammation and then fibrosis, which is the scar that forms, and whether we're talking about an atherosclerotic plaque or the kidney, I think that those are likely shared mechanisms.

(12:48):
And the other thing I want to bring up with regard to the heart disease is that, well, the main benefit to date for these agents has been on atherosclerotic disease at the European Society of Cardiology this fall in end of August 23, they were reported the step heart failure trial, which was in obese individuals without diabetes who had heart

failure preserved ejection fraction, and they showed a clear benefit on this form of heart failure and preserved ejection fraction. Heart failure is a form of heart failure, where until recently, we have not had effective therapies. And so the common link between preserved ejection fraction heart failure, chronic kidney disease, and atherosclerosis is likely to be the inflammatory component. And these very profound anti-inflammatory agents, which I think is largely unrecognized by the clinical community.

(13:49):
And we even have data out of our own study called Ward Seven, where we looked at blood and urine markers of inflammation and fibrosis in people who received, in this case, dulaglutide versus placebo or insulin. And in the people who get active treatment, there's a marked reduction in biomarkers of inflammation and fibrosis, whether it measured in the blood or the urine. And that's important because if it's in the blood, it's everywhere. Right? Right. So it's likely that a reduction in the pro-inflammatory processes that are driven by obesity and diabetes contribute to the organ damage, and these agents basically interrupt that inflammatory pathway that causes injury

Sean Collins (14:33):
For participants in your study who are getting active treatment, can you characterize in any way for me what their lived experience is? I mean, there's one thing for you to notice a change on the numbers on a spreadsheet, but for the patients, are they noticing a difference?

Dr. Radica Alicic (14:56):
Oh, yes. Yes, they do. Thank you. That is actually excellent question. So the first study I actually worked with Dr TA on was on lucine quite few years ago. And those studies are randomized. That means that you have 50 50 chance to be on an active treatment, and it's blinded. So we don't know who is on treatment, but few months into the study, if you look at the blood work on the patients, you can clearly see improvement. I had to repeat one of the blood draws two times because I couldn't believe that kidney

function improved to that degree. And what does that mean? This is associated with fuel kilograms, like about between three and four kilograms or six to eight pounds of weight loss and improve blood pressure control. So these people do feel better. So you are blinded in a sense, but because the fact is so profound, you see the changes.

Dr. Katherine Tuttle (16:01):
The main side effect of the drug is gastrointestinal, upset, nausea, and interestingly enough, in some cases, diarrhea, others constipation. So I do want to acknowledge that there can be some unpleasant side effects, but they are most apparent at the beginning of treatment. So it turns out if you start low and go slow, start at the lowest dose of whatever agent you've chosen and you titrate up to the next step, usually wait about four weeks. People adapt to it over time. So upfront, there are some side effects that people find unpleasant, but what we do is we coach them through it and then like Dr. Lys said on the other side of it, they feel so much better

Dr. Radica Alicic (16:49):
And more energized.

Sean Collins (16:51):
Well, in the interest of full disclosure, I take one of these drugs myself and have noticed as the dose has been titrated up, I've noticed incremental periods of nausea and GI upset, which can be a real problem for days on end. And then it seems to resolve each time it has resolved each time, every time the dose has changed.

Dr. Radica Alicic (17:19):
And usually if you go slowly in advance, slowly dose, the effects will subside and even you can even go on lower dose until you adjust and then increase gradually.

Dr. Katherine Tuttle (17:35):
Another interesting aspect of this is if the field is really moving to dual agonists. So I mentioned the GLP one receptor agonist combined with something called AGIP

agonist that stands for glucagon intestinal peptide. And it turns out that when we give the dual agonist, there's more therapeutic efficacy for both weight and glycemia, but there's actually less nausea. And it's thought that the gyp component actually ameliorates some of the nausea. So as the field evolves, I really do think we're going to be moving to dual agonist therapy, both for efficacy and tolerability.

Sean Collins (18:25):
Any J school professor who's listening will scold me for doing this 20 minutes into an interview, but I think it's probably useful to explain what GLP means and the mechanism that it's mimicking. These agents are mimicking from real life, and I'm sorry that we didn't do this earlier because we've been throwing around GLP one and as if everyone knows what that means. And frankly, the word agonist, I think most people are more familiar with the term antagonist, which is the absolute opposite of an So could you walk us through the lingo here so that people understand what's being talked about?

Dr. Radica Alicic (19:15):
So I will, I think we'll share this question. And actually I have to say that this is very good moment to introduce biology of these medications because you mentioned the feeling nauseous intermittently and having some GI upset. So that is actually biological action of these medications. So GLP means glucagon-like peptide, and the receptor agonist. Agonist means that stimulates the receptor. So works as So GLP one is actually produced in our

(19:55):
Organism

(19:56):
In parts of enteroendocrine system that is the nervous system specific to our gut. So once when you eat biological function of this GLP one is to stimulate secretion of insulin. At same time, it slows downing of the stomach and sends signals to brain to

part of our brain to say you are full. But one of the parts that, so it works, the GLP one is the one that is naturally produced in our organism secreted by enteroendocrine cells, lasts for only few minutes, three to four minutes because the enzyme called DP will make it inactive. So the medications we are using today chemically engineered to last much longer, as you know, some last for a week. So the side effects we see so frequently are actually part of biological effect of this cause of medications.

Dr. Katherine Tuttle (21:14):
And I just want to add that with the dual agonist we add the glucose dependent insulinotropic, polypeptide, gyp. And it's very interesting too because it has similar effects except that it actually increases glucagon secretion, whereas GLP one decreases glucagon. Glucagon is another glucose regulating hormone that we've traditionally viewed as opposing insulin by raising glucose. But it really turns out that that's probably the magic bullet in therapeutic development as we move forward. Because glucagon also has a thermogenic effect, that means it makes people burn more calories. And that is thought to be a major reason why the dual agonist TIR peptide that has both GLP and GYP agonism causes more weight loss. And in fact, that led to the triplet, which is now adding glucagon per se. And in fact, this year at the American Diabetes Association to Scientific sessions of June of 2023, professor Mattias TCH from Germany gave the Banting lecture, which is the highest award given by the ADA for development of the triple agonist along with the medicinal chemist at Eli Lilly.

(22:37):
And he really told the story of originally the concern that adding glucagon would abrogate the effects. It turned out that they amplified the effects, and that was the nugget that caused them to move on to adding glucagon agonism. And it's really a remarkable story because it's a single drug that hits three receptors, and that was the magic of medicinal chemistry to make a small molecule that could hit three different receptors simultaneously. And so they got the Banting award this year, the highest award from the American Diabetes Association for introducing first the twin, which is

TIR peptide. And now moving on to the triplett, which will be GLP one, GIP one and glucagon agonism in an agent called ide, which is now going into trials. Wow.

(23:33):
And Sean, can I just add one more thing? Sure. I think it's good to understand the anti-inflammatory effect that the tattle was talking about a few moments ago, which we think is the major of action of organ protection is if you think the GLP one is secreted by our gut when we eat, but actually it is also stimulated by certain bacteria, and you think it actually works as an interface between what we eat and metabolism and inflammatory or our immune system because that's how we protect ourselves. We absorb food and can be toxin from bacteria, it can be food, and that stimulates secretion, different peptides and stimulation of the immune cells.

Sean Collins (24:28):
As a layman, I go back to the idea the germ of the idea that I began the program with, which is I do find hormonal regulation of our lives to be really fascinating because there is something just mysterious about something being released at a remote part of the body having an effect someplace else. And I listened to the two of you talk in awe of what appears to be about to happen with the treatment of disease. And I want to throw a question at you that is more philosophical, which is for a long time people with obesity have felt morally responsible for their disease process and have been treated as somehow lacking willpower or lacking some moral fiber that would better regulate their metabolism. And what I hear you both saying is that this is a very complex system,

Dr. Katherine Tuttle (25:43):
Sean. There's an entire biology of obesity, and I'm glad you brought that up because separate from the cosmetic effects that get so much attention, obesity really is a disease and there's a strong biology around it. And it turns out that with weight gain, basically the hypothalamus, the central gland that controls multiple outputs as you set hormonal outputs, resets itself to a higher weight threshold. So you could call it a fat

moat, but once you gain weight to a new set point, it will fight you every step of the way. Which is why weight loss maintenance has heretofore largely been unsuccessful because once a person loses a certain amount of weight, they are driven biologically to eat. The hypothalamus says you're starving, get your weight back up. And what these agents do, and actually GIP one agonism more than GLP one agonism actually has receptors in the hypothalamus that basically reset the fat masta, they shut down stimuli to appetite and they promote stimuli of satiety.

(26:59):
So if you look at, I think the real, again, back to the term game changer for obesity will really be the twins and the triplets because of the GP one. But adding glucagon is really incredible. So if you look at the obesity studies, even without diabetes for tze peptide, you're looking at mean or average weight loss of 40 to 50 pounds per year, not the three to four kilos that we see with semaglutide, which is in the range of seven to 10 pounds. But on the red aide studies, they're getting mean weight loss of 60 pounds in a year.

Sean Collins (27:41):
That's amazing.

Dr. Katherine Tuttle (27:42):
These agents are really likely to relegate bariatric surgery to a niche procedure for very unique people who can't take these agents. And the other thing is bariatric surgery changes a person for life. And as a nephrologist, I will tell you over time it is very problematic. The bariatric surgery causes the gut to absorb a compound called oxalate forming kidney stones. And we see terrible problems with kidney stones, even kidney failure

(28:14):
Some

(28:14):
Two decades after bariatric surgery. And obesity is really the cause of not only diabetes, but the excess risk of kidney and cardiovascular complications. So if we deal with obesity upfront, we will take care of many of the downstream effects. The other thing about the medications is if it's not working for you, you can stop it and you're not changed forever. Whereas surgery to my test of reason, ruins a person's gut, nothing will ever be normal again. It's really draconian approach to weight loss. And now we have agents that actually change central nervous system biology to where you can reset the fat masta and get weight loss in approaches or even matches bariatric surgery without the side effects.

(29:05):
So

(29:05):
Honestly, I think if we dealt with obesity upfront, we would have much less diabetes, heart and kidney disease. And I think these agents are poised to really solve a major public health problem.

Dr. Radica Alicic (29:19):
So Sean, just to illustrate what Dr Tuttle was talking, so in the study, ongoing study with ide, she's talking about GLP one GPP patient, by the way, she's probably on medications, but she somehow did not miss a dose. It's a weekly injection, also missed a dose. And so she was without medications for two weeks, and she came back almost in tears explaining how she was slave of food and her appetite. Again, she did not feel she's in control. She had horrible food cravings to eat, salty and fatty. And so it really talks about the central nervous system or hypothalamus role in the obesity.

Sean Collins (30:17):
Does that mean that if you begin therapy and are benefiting from this, that you have to continue it for the rest of your life?

Dr. Radica Alicic (30:27):
Well, I guess we don't know that yet, Sean, but the studies obviously don't go lifelong. They go two or three years. But it is true that when people stop the agents, they do regain the weight. So at this point, we're considering them chronic therapies like we would give for diabetes or high blood pressure or high cholesterol. But the truth is, it fundamentally changes the biology that drives so much of those other risk factors. So I do think even in the beginning we talked about weight loss, but from a cosmetic standpoint, I'm not really interested in that. But from the standpoint of obesity as a disease, I am interested in it. And one of the things that I think the audience should know is we're not trying to give people more diagnoses than they need, but truthfully, people who are living with obesity haven't been able to access therapies because when it's not classified as a disease, then they can't get approval for these transformative therapies.

(31:27):
So you talked about the stigma and shaming of obesity. There is a biological basis for it. It does run in families, and however it starts, once it gets going, it's very hard to ever turn around. And this really is the bedrock of so many of these other conditions that come later, diabetes, kidney disease, heart disease. So I think from the standpoint of trying to reduce the public health burden of the most common causes of death and disability in modern society, we really have to deal with obesity. And now we have the most powerful tools ever to do that.

Sean Collins (32:05):
That's remarkable. And yet you have to wonder whether there are financial pressures by people who are making a lot of money, treating disease processes that stem from obesity or weight loss devices or therapies that aren't going to be as effective.

Dr. Radica Alicic (32:23):
Yeah,

(32:24):
Sean, we are saying that yes, there is always that effect, but we are taking care of patients who are on 15 supplements, never shown to do anything, and someone is making money out of that.

Dr. Katherine Tuttle (32:38):
Well, I think you've both hit on an important point. I think it really, we've got to have changes in health policy and the type of healthcare we prioritize in our healthcare systems. We focus way too long on disaster relief, right? Whether it's an acute MI or a car accident that frankly is not very cost effective. And honestly, if we actually truly walk the talk of improving the health of the population with approaches that we now have, there are cost-effectiveness ly that have been done, it would be cost effective. But our healthcare systems by health policy are incentivized to treat failure and disasters not to keep people healthy. And to give you an example of that, just to be honest with you, if we go back to the flow trial, which is semaglutide for people with type two diabetes and chronic kidney disease, the day it was announced that that trial would be stopped early for efficacy, the stock for dialysis companies plummeted and the stock for Novo Nordis who makes semaglutide sword. So to your point, there are economic incentives all around this, and I think we just have to acknowledge that. But I think since we're in the United States, it's a global problem. But let's focus on America. I think we have the American people and the people in Congress really need to ask fundamentally, what do we want to pay for?

Dr. Radica Alicic (34:14):
Yeah, do you want to pay for dialysis or you want to pay for open hearts or you want to pay for patients staying in good health active, which will of course improve quality of life, but also help with the cost of care delivery

Dr. Katherine Tuttle (34:33):
And maybe keep people healthy enough that they stay working and paying taxes.

Sean Collins (34:37):
There is an approach that will catch people's attention.

Dr. Radica Alicic (34:40):
So I work as a hospitalist, so I'm acute care internist, and we actually really have to fight with the different systems in place to start patients who had acute stroke or acute heart attack or worsening of kidney function start on these medications because they are expensive and not everyone will approve them, which I think clearly from previous discussion is at this point, unethical. There's no absolutely good moral justification to keep someone on allegria or just metformin if they have complications, if there are medications that prevent dying and protect the organs.

Sean Collins (35:30):
I'm of Irish heritage, which explains why I get weepy at times when I think of ancestors, and I think of people who have struggled with heart disease, kidney disease, obesity in my family, and how early death accompanied that family history. And I think, boy, this is right on the edge of changing things so that families can have one another around for a longer period of time. I mean, it must be amazing to be involved in that research and that work to see that family life is going to change for people in the generations to come.

Dr. Katherine Tuttle (36:20):
It's transformative. And I know our focus today has been on GLP one and like agents, but we have the SGLT two inhibitors that just came before them over the past five years, remarkable reduction. People with chronic kidney disease all cause mortality reduced by 30%, and one of the most fatal yet unrecognized conditions in our society. Those patients die at a rate of 10 to 20% per year. It outpaces death to many of the common forms of cancer. And I'm not competing them, I'm just benchmarking them. But we would not tolerate a highly fatal condition like some form of cancer without treatment when we have a drug that reduces death by 30%. So start right there. And

then among living reduces kidney failure by 40% and heart failure by 25%. So that's why Radia said it's a moral and ethical obligation if you have these tools in hand to deliver them to the people who can benefit.

(37:21):
We wouldn't do this for other conditions. We've let this go, especially diabetes and chronic kidney disease. And so we have the SGLT twos, we now have the GLP ones and the series of the twins and triplets. And then to be honest with you, we have new agents that block something called aldosterone, which are providing additional therapy. And what we see is when these drugs are used in combination, the effects are additive. So you're exactly right. When you walk back to a family like yours, if we actually had enough awareness and detection to treat people when we could save them, it would change lives of millions of people all over the world. And I think we've been silent too long. I applaud the groups that have had wonderful advocacy and been able to get resources and funding to treat other fatal conditions, but we need to do the same for these people. And these are some of the most common becking conditions, and not only the United States, but in the modernization, if you will, of cultures worldwide. We got to get this right now that we have the tools we cannot stand by idly and let people continue to die like this.

Sean Collins (38:37):
And from a population health point of view, I think it's probably fair to point out that chronic kidney disease, the incidence of it is higher among African-Americans than among Caucasians. So the fact that there is a lack of treatment and concern for kidney disease has a cultural

Dr. Radica Alicic (39:03):
Implications implication. Absolutely. Native Americans, Alaskan, native American Pacific eye is very high.

Dr. Katherine Tuttle (39:11):

Kidney disease is the poster child for disparities. So to your point, a third of the people in this country on dialysis identify as black or African-American, yet it's only 14% of the general population. And this is not driven by genetics. This is a social determinant of health. And then like Radsa said, out of our large cure CKD registry on the Providence system with 4 million unique patients, we published a paper in the New England Journal last year and asked who gets kidney disease and diabetes incidents? So that's new onset disease. It it's every non-white group other than people who identify as Asian. But to her point, people who identify as Pacific Island or Hawaiian, 66% higher risk,

Sean Collins (40:00):
That's astounding.

Dr. Katherine Tuttle (40:01):
People who identify black or African American, 40% American Indian, Alaskan native, 33% and Hispanic or Latinx, 25%. So you see it's across very diverse groups. This has much more to do with social determinants than genetics. I'm not saying that in some groups there aren't genetic predispositions. So let's acknowledge in African-Americans, there are the AOL one gene variants that predispose to certain forms of kidney disease and accelerate progression of others, but that's not responsible for this huge excess risk. It is mostly things related to education, opportunity, income, neighborhood, and frankly racism.

Dr. Radica Alicic (40:51):
So at the end, Sean, we do have moral obligation to adopt this therapist. And just to add, because I think we are at the close to the end of interview, you ask, is it exciting? So I had a very good fortune to come to Spokane and find Dr. Tattle, so she's my mentor and shining star and allow me to be part of this project. But I can tell you that we have nine coordinators and the whole team, research team is very excited and driven because we are part of studies that do make these changes and change lives.

And you can see patients who have their lives are changed. So everyone is very motivated, and we all take great pride.

(41:48):
And I couldn't have done all this without Dr. Alicic and a number of other partners who've worked together with me over all these years. But you're right, what really drives us is we want to make a difference. We want to use science to solve big problems for health and community. And of course there are lots of different problems, but this is one where we had the skills to do it and where the condition had been under-recognized and undertreated. So we really could make a difference here. And now is the time. And like you said, cost is an issue, but that really needs to be dealt with at the policy level because we wouldn't let this happen to other people with equally fatal conditions where we have a treatment that truly has, I mean, this is a moment in medicine, the effect on mortality. This is right up there with the major advances that have occurred in the past century. I mean, this is opening an artery with an acute mi, the mortality benefit, we cannot ignore it, and especially because the populations who could benefit most often have the least access.

Sean Collins (42:58):
Well, I'm glad that the two of you took the time to talk with me today. And I think it's important to remind the audience that in the next couple of weeks you're going to see headlines about some of these issues, and I'm glad that we're able to sort of fill in some of those gaps in the background. Dr. ic, Dr. Tuttle, I'm so grateful for the work you're doing, but also for you taking the time to talk with us today. Thank you.

Dr. Radica Alicic (43:24):
Thank you.

Sean Collins (43:27):
Dr. Catherine Tuttle is executive director for research at Providence Healthcare, and her colleague Dr. Radica Alicic is the associate director for research. They spoke with me

from Spokane. If you want to do more reading about their research, visit our website, hear me now, podcast.org. The Hear Me Now podcast is a production of the Providence Health System and its family of organizations. Subscribe at Hear Me now, podcast.org. The program is produced by Scott Acod and Melody Faucet. We have research help from medical library staff Basha, Delawska- Elliot, Carrie Grinstead, Sarah Viscusso, and Heather Martin. Our theme music was written by Roger Neil, the executive producer is Michael Drummond. I'm Sean Collins. Thanks so much for listening today. Be well.