



## **Midlife Clarity with Dr. Tracy Page**

*"Clarity Changes Everything"*

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Hosted by Dr. Tracy Page

**Episode # 5 — The Five Cardiac Labs That Could Save Your Life**

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Hi, I'm Dr. Tracy Page, and welcome to the podcast. I'm so glad you're here. I'm a functional medicine physician, and today I'm going to share a case study with you because I feel like we're more the same than we are different, and I think that my patients and taking care of them is actually one of the ways I can help you as non-patients really learn and also learn from each other. You might be my patient listening to this podcast, but I still think you might identify with some of the information because, as I said, we're more alike than we are different. And all the topics I'm going to cover through cases apply to the patients I see and to the general population, especially women age 40 and above. But even women in their 30s should know this information. So if you're younger than 40, please listen, because it is pertinent. Today, I want to talk to you about a case of a person who looked perfectly healthy on paper, but just had major little blips in her LDL — just slightly elevated — and everyone thought she was just fine, but she was actually a cardiac ticking time bomb.

And I'm going to explain to you how we figured that out, and why, and how we're going to treat that, because you may, like I said, be like one of my patients, and so you need to know this information. Do you know that heart disease will kill more women than all cancers combined? Again, let me repeat that: heart disease will kill more women than all cancers combined. That's a lot of women. It's our number one risk factor, and especially once we stop producing estrogen. So we're going to talk about — I'm going to call her Claire; it's not really her name, and all the patients that I talk about, I'll never use their names, but all the facts are real. And this is a true story. Okay, she comes to the clinic. She looks healthy. She's the health — what I call a success story. She's 53 years old. She walks every morning. She plays pickleball three times a week. She eats mostly whole foods and maintains a healthy weight. I mean, you couldn't ask for a more pristine patient. Her standard blood work came back totally fine. Her doctor told her she looked great, but she had a little bit of elevation in her LDL, and her doctor said, "Keep doing what you're doing: exercise and eat well". So why did she end up in my clinic? Well, she came to see me because most of our patients come from other patients, and one of her friends referred her to our clinic because she said, you know, my friend told me that her LDL was elevated, but you guys did a much deeper dive, and she found out she had some other things that were concerning, and I just want to



make sure I don't have those too. And plus, she was struggling with sleep. She was struggling with being hungry all the time. She was struggling with not feeling her best, and some weight gain. So she had some other issues that she wanted us to address. She knew that she was going through the change as well. Her standard blood work came back—like I said, totally fine—from her previous doctor's office. But there were three numbers in her bloodstream that were never tested that made her, like I said, three biomarkers that placed her in a high category for cardiovascular risk, one of the highest in the country. Numbers that standard cholesterol panels, the ones most doctors run every single day, never check.

So today we're going to talk about those numbers, plus some more, and what they are, what they mean, why your doctor may never mention them, and the specific blood tests you need to ask for by name. I'm going to give you the tools to help you take care of yourself. So welcome back; I'm excited to share this information with you. If this is your first time on the podcast, I hope you leave here with some actionable items. That's my goal, in order to help you live healthy, well, with longevity and vibrancy. Okay, so let's meet my patient, Claire. As I said, she's 53 years old and menopausal. She came in with three main complaints: she can't sleep, she's been feeling off since her hormones started shifting, and she's gained some weight. Her doctor recently flagged a slightly high LDL, and she was told to keep exercising and eating well, which she was doing. So on the surface, she's doing everything right — her exercise, her nutrition — she takes care of herself. So why are we so concerned? The answer came when we ran something called a Cardio IQ panel. It's an advanced cardiac blood panel that goes far, far beyond what a standard lipid test shows us. And what came back was pretty significant. So I think of the lipid panel as what I call the preview to the movie. It's kind of like when you watch a trailer to a movie — you know who's in it, you don't really know much about the characters, but you can kind of figure out if it's a comedy or a drama. But when you watch the movie, you get to learn all the characters, you learn the plot, you know who's good or bad — you know the story. And that's what I consider Cardio IQ. It tells us everything.



So a full Cardio IQ picture — this is what we found in Claire's labs. It revealed LDL cholesterol of 146, elevated, and that's also elevated on a standard panel. Total cholesterol: 236, elevated — it should be less than 200. And by the way, LDL should be less than 100. She had a positive lipoprotein (a), or Lp(a), which was critical. It was high, 99.7. Optimal is below 30. So Claire's in the top 5% of risk. Her LDL particle size was small, and she had 874 of them. Optimal is below 527. These are dangerous, artery-penetrating particles — I'm going to talk more about them later. Apolipoprotein B was elevated, which confirms high particle burden — it's a little protein that's on the outside of LDL particles. She had a positive myeloperoxidase, otherwise known as MPO, and it was 79. Optimal is below 62. This is active arterial inflammation that's happening right now in her bloodstream. So when that number is elevated, she has active inflammation in her bloodstream. She also had elevated oxidized LDL, which is damaged cholesterol. And you've heard of oxidation — think about when things oxidize, like an avocado: it gets air exposed to it, turns brown, so it gets oxidized. So she has oxidized, what I call icky, rancid LDL in her body. Her fasting insulin was 9.3, which is creeping up, because we really want fasting insulin under 6. But hers was 9.3. Her estradiol was 19, which is low for untreated menopause. Her progesterone was low at 1.2, which again is suboptimal for treated menopause. Her testosterone was very low — it was 5. And her free testosterone, you couldn't even detect it — it was undetectable. Her cortisol was elevated, and we like to do a urinary four-point cortisol; her level was elevated, and, not surprisingly, her melatonin was low. Melatonin is what we release at night before we go to sleep that helps us wind down and signals it's time to get ready for bed, and it should be elevated at night.

So now, your standard lipid panel, the one most primary care doctors or internists run at your annual physical, would only show her LDL, her HDL, her triglycerides, and her total cholesterol. So none of those markers I just mentioned were even detected. And an LDL of 146 might raise an eyebrow; talk about diet and exercise — which are relevant 100% — but didn't really look at her true risk factors, because there's more to her risk than just getting a standard lipid panel. Lp(a) requires a specific test, and the Cardio IQ tests for that. The small LDL particle number, and also the MPO, oxidized LDL, and ApoB, those are all in a Cardio IQ or advanced lipid panel. So why do these three markers change



everything for Claire? Let me go a little deeper into them. I want you to understand these markers because they could truly change the trajectory of your health. Generally, they could change whether you have an event if you don't know these numbers.

Okay, let's take them one by one. LDL small particle numbers — you've all heard of LDL; it's the so-called bad cholesterol, and I always tell my patients, we don't really know if it's bad yet; we just know you have it. But what your standard panel tells you is just a concentration of cholesterol inside the LDL particles. It doesn't tell you how many LDL particles you have, or what size they are. And the distinction is truly everything. Think of it this way: imagine two trucks carrying the same amount of cargo. One is a big 18-wheeler, and the other is 874 — remember, her number is 874 — small pickup trucks. So all that cargo that's in an 18-wheeler is now divided among 874 small pickup trucks. Which scenario causes more accidents on the highway? One big 18-wheeler — you might say yes, but think about it. The chances of having an accident increase tremendously if you have 874 small pickup trucks all over the highways. One of those trucks, or two, or three, or maybe ten, might get in an accident versus that one 18-wheeler, because there are more of them, and they're harder to control, and they can squeeze into spaces the big truck could never get. So the chances of an accident are greater. That's exactly how small LDL particles work in your arteries: they're small enough to penetrate the arterial wall, lodge in the lining, oxidize, and trigger inflammation — the inflammatory cascade that builds plaque. Large, fluffy LDL particles don't do that; they're far less dangerous, but the small, dense LDL particles are very destructive. Claire's small LDL particle number was 874, and remember, optimal is 527 — she had 66% more of these dangerous particles than the upper threshold of normal, and her standard cholesterol test told us nothing about this.

Let me share some statistics about LDL particles and why they matter. LDL particle number is a stronger predictor of cardiovascular events than LDL cholesterol alone, and research studies support this. Small, dense LDL particles are 3 to 5 times more atherogenic — plaque-forming — than large LDL particles. Up to 35% of people with normal LDL cholesterol have elevated LDL particle numbers, so their standard test can give them a false sense of security. So you could have normal LDL but elevated LDL



particle numbers and never know it with a standard lipid panel. Small LDL particles are particularly elevated by insulin resistance, low estrogen, and high triglycerides — all three things Claire had. Optimal small LDL particle number should be below 527, and Claire's was 874, which is significantly above the threshold. So this puts her at greater risk. Claire could have walked around for another decade, 10 more years, being told her LDL is a little high but manageable, while 874 small, dense, artery-damaging particles were quietly doing their job. This is why the Cardio IQ test exists — because it's not just how much cholesterol you have, it's what kind, and how many particles are carrying it. So we have to know these numbers.

Let's move on to another marker, myeloperoxidase. Myeloperoxidase is an inflammatory marker—hers was elevated at 79. This is the one I want every woman listening to write down, because it reflects active inflammation. They're all important markers, but this one is really important, because if Lp(a) and small LDL particles tell you about your risk, MPO tells you whether the fire has already started. MPO is an enzyme — it's released by immune cells, specifically neutrophils and macrophages. And when there is active inflammation inside the arteries, it's not just a risk factor; it's a real-time signal. An elevated MPO means your artery walls are already under attack right now. Her MPO was 79. Now, remember, this is a healthy woman — she exercises, she eats well, she does all the right things. And hers is past the upper limit of optimal, which is normally 62 — she was higher than that. And it's not a borderline elevation — it's a meaningful signal that inflammatory damage is actively occurring in her vascular system right now, even as she walks to her pickleball game and eats her vegetables. She's having active inflammation in her body, and you're going to be amazed to understand why in someone who's so healthy.

So, elevated — let's talk about the research. Elevated MPO is an independent predictor of major cardiac events, even after adjusting for traditional risk markers. MPO greater than 62 nanomoles per liter is associated with a significantly higher risk of coronary disease and plaque instability. In a landmark New England Journal of Medicine study, elevated MPO in chest pain patients predicted heart attack with greater accuracy than troponin alone in the early hours — because if you go to the ER, they typically



measure your troponin level if they think you're having a heart attack, but actually MPO is a greater predictor of actually having an event. Elevated MPO is found at two to three times higher rates in menopausal women compared to premenopausal women. MPO directly oxidizes LDL, converting it to highly atherogenic oxidized LDL and creating a direct mechanism linking inflammation to plaque buildup. So when you have inflammation in the body, you're going to have plaque buildup — they go hand in hand. MPO plus an elevated Lp(a), that we talked about earlier, together represent a compounded risk: one creates the dangerous particle, the other ignites the inflammation that makes it destructive. So we have a damaged, dangerous particle, and inflammation that makes it even more destructive. This last point is critical, especially in Claire's case: she doesn't just have a genetic lipid problem; she has an active inflammatory process that is making that genetic problem exponentially worse. The Lp(a) is the fuel, the MPO elevation is the spark, and the small LDL particles — they're like the kindling. So MPO doesn't tell you what might happen; it tells you what is happening right now, because it's active inflammation.

Let's talk about her third marker, Lp(a). It was 99.7. We've talked about Lp(a) earlier, but let's bring it home in the context of everything else we're seeing in Claire's labs. So Lp(a), or lipoprotein A, is a genetically determined lipoprotein that is pro-atherogenic, pro-inflammatory, and pro-thrombotic — so it has three problems associated with just having that marker. Pro-atherogenic: you're more likely to make plaques. Pro-inflammatory: you're more likely to have inflammation, especially in the vasculature. And pro-thrombotic: you're more likely to have clots. It builds plaques, it drives inflammation, and it promotes clotting. It's like LDL's more dangerous cousin — kind of think of it that way. So Claire's Lp(a) is 99.7, which puts her well above the risk threshold. And unlike LDL, which you can meaningfully address through diet and exercise, 80 to 90% of your Lp(a) is written in your DNA — it's genetic. No amount of lifestyle can change it; it won't fix it. It requires awareness, monitoring, and often medical intervention. And I'll say, in my practice, I've had patients who have a positive Lp(a) — some of them are not on medication, and some of them are.



An Lp(a) greater than 30 is risk; anything above 50 equals high risk. So, above 30 is elevated risk, above 50 is high risk. And in Claire's case, she was 99.7 — that's what I consider the danger zone. About 20% of the population has an elevated Lp(a), and they have no clue. So you know how many people that is? That's approximately 1.4 billion people globally with elevated Lp(a), with those three increased risks—inflammation, atherogenic potential, and clot formation—and they have no clue. Think about the person that you know where someone said, oh, they had a heart attack, and nobody knows why — they were like the epitome of health, right? We had someone at our church who was a runner, and he and his wife were running, and they played tag team — he went out first to run, came back and tagged his wife, and she went out there getting the kids ready. He had a massive heart attack and passed. And, you know, no one knew he could have had that genetic marker and had no clue. So it's so important to get these tested. 80 to 90% of Lp(a) is genetically determined; lifestyle cannot meaningfully lower it. Most standard lipid panels don't test for Lp(a) — it requires a specific order. Lp(a) and small LDL particles are particularly dangerous in combination; their combination promotes inflammation that oxidizes the small, dense particles, making them even more atherogenic. So here's what makes Claire's case so striking: she has all three elevated at the same time—Lp(a), small LDL particles, and MPO. It's not one alarm bell; it's the whole system going off at once, and none of this showed up on a standard cholesterol panel. So now you understand the importance.

Let's talk about the hidden amplifiers — so how does her being in menopause, not sleeping, and having insulin resistance cause this situation to be even worse? Here's what makes this so important for women, especially the women I take care of, and why this conversation is different for a 53-year-old in menopause than it is for a 20-year-old. Estrogen is cardioprotective. It maintains vascular flexibility, helps clear LDL from the bloodstream, reduces arterial inflammation, and suppresses myeloperoxidase activity. When estrogen drops at menopause, all of these protections go away. This is why hormone replacement is so important — it's not just because of hot flashes and night sweats; it's actually cardioprotective. It keeps you from having a heart attack and stroke, and if you have Lp(a), even an aneurysm can happen. So how does low estrogen drive all three of these markers? Estrogen loss reduces LDL receptor activity in



the liver — LDL rises 10 to 15% in the first year when you're in menopause. Low estrogen shifts LDL particle distribution toward small, dense particles, directly worsening LDL numbers. Estrogen deficiency increases vascular oxidative stress, which actively activates MPO and amplifies inflammation. And menopausal women have two to three times higher MPO levels than perimenopausal women, independent of all other risk factors. So Claire's menopause didn't create her elevated Lp(a) — remember, that's genetic — but it absolutely worsened her small particle count, it amplified her MPO, which increased her vascular inflammation, and it removed the protective buffer that estrogen was providing against all of it.

Okay, so that's how estrogen plays a role. How does sleep play a role in her vascular inflammation? So she told us she wasn't sleeping, and we know that her cortisol was elevated from testing and her melatonin was low. So, I mean, it's bad when we don't sleep — of course, it's a comfort issue; we all want to sleep, and we feel horrible when we don't. But chronic elevated cortisol directly raises her MPO levels, promotes the production of small, dense LDL particles, and worsens insulin resistance, which in turn drives particle number even higher. So again, think back to someone you know who had a heart attack, who was working super hard, stressed all the time, wasn't sleeping — but they were a normal weight, they looked normal, you would never have known that they were driving their own cardiovascular disease. Chronic poor sleep, less than six hours a night, increases inflammatory markers, including MPO and CRP, by 25 to 40%. High cortisol directly promotes the formation of small, dense LDL particles over the large, buoyant ones. And sleep deprivation is linked to a 48% higher risk of coronary heart disease over time. So every night Claire can't sleep is another night her body generates more of those exact particles that drive the inflammation we're trying to control. So those particles keep building—it's like a factory.

Early insulin resistance — I call it the small particle factory — because Claire's fasting insulin was 9.3 and trending upward, and most people don't know that insulin resistance is one of the primary drivers of the shift from large, safe, buoyant LDL particles to small, dangerous ones. Insulin resistance is the number one lifestyle-driven cause of elevated small LDL particles. High insulin suppresses lipoprotein lipase, the enzyme that breaks



things down, leading to smaller, denser LDL particle formation. Insulin resistance also amplifies the inflammatory cascade that raises MPO, creating a direct biochemical link between her metabolic state and her vascular inflammation. Every one-point rise in fasting insulin — so every point above 6, above optimal — is associated with measurable increases in LDL particle concentration. So her menopause, her sleep, and her insulin resistance — every single one was turning up the dial on the same three dangerous cardiovascular markers we just talked about.

Okay, so what do we recommend, and why? So now you know what we look for, and you understand the full picture. So what do we actually do? Let me walk you through the priority-driven plan I made for Claire, and how each piece ties directly to these three markers.

The first thing I asked her to do was get a calcium score. It was a non-negotiable first step. When Lp(a) is that high, and small LDL particles are present, and she has active vascular inflammation, the single most important next step is a coronary artery calcium score, CAC — a CT scan, low radiation, that shows how much calcified plaque has already built up in the coronary arteries. If your score is zero, you have a low near-term risk, although your Lp(a) was elevated. So let me tell you something really quickly: the calcium score measures calcified plaque; it does not measure soft plaque. Most women have an event from soft plaque. A CT angiogram measures soft plaque. Who should go get a CT angiogram? Someone who has elevated Lp(a) and small, dense particles. And I have a patient who had a zero calcium score, sent her for a CT angiogram, and she had 80% occlusion by soft plaque. So don't be fooled, and make sure you know those previous numbers I just talked about — but you do need a calcium score regardless. A calcium score greater than zero means there's existing arterial disease, and that usually warrants a referral to cardiology.

And sometimes I start them on a statin. Statins get a bad rap, but I would say I have patients that are on statins who have no muscle pain — they take ubiquinol, which is CoQ10; we put them on the lowest effective dose, and they have no brain fog. And if it's warranted to prevent a cardiac event, they're more than willing to take it. So I would say every situation is different—of course, we always try supplements and other



modifiable lifestyle changes first. But in her case, she has a genetic risk marker, and as I said earlier, no exercise or nutrition is going to change that. She probably is going to need a PCSK9 inhibitor — she's going to need something like Repatha, an injectable cholesterol medication. It's the only thing that's been shown to lower Lp(a). But, you know, that would be a decision for the cardiologist, and usually you have to fail at least two statins before insurance companies—most insurance companies—will pay for it. A calcium scan is typically about \$50. You can call your local hospital and tell them that you need one. I recommend every patient who's 50 and over have one; if they have one at 50, I recommend it again at 60 to see if they've had any calcified plaque. Also, if you're younger and have a positive Lp(a) and small particles, you need a calcium score — I don't care how old you are; it's the best \$50 you'll ever spend. If her calcium score comes back elevated, then we are talking about a statin. Zetia is a cholesterol medication that works differently than a statin—sometimes we start that because there are emerging targeted therapies coming out too, and we really focus on Lp(a), so we want her to have the best options available. So I typically refer to cardiology, let them do the risk assessment, and decide the best next steps.

For her MPO and inflammation, we also look at targeted anti-inflammatory support alongside any pharmaceutical approach, so modifiable lifestyle changes are still important. To reduce her small particle count and MPO, we want to optimize her hormones—that's the first thing we can do. This is where functional medicine really changes the game for patients, because optimizing Claire's hormones isn't just about quality of life; it directly targets two of her three elevated markers. So transdermal estrogen has been shown to shift LDL particles from small, dense to large, buoyant ones, directly reducing dangerous particle count. Estrogen therapy reduces MPO activity and vascular oxidative stress, directly addressing her MPO reading. And testosterone replacement — and I know this is surprising to you — improves insulin sensitivity, which further reduces her small particle formation. So it's so important. The plan was to switch to transdermal estradiol for stable delivery, fix her progesterone delivery method for better sleep because she wasn't getting good sleep on the one that she was taking, and introduce low-dose testosterone. And here's the critical nuance: oral estrogen can actually raise Lp(a) in some women. So know if you have Lp(a), and then, if you are on



oral estrogen, please, please switch to transdermal — it is the preferred route for anyone who has elevated Lp(a). And honestly, in my practice, it's the route I prefer to give my patients.

So fixing her insulin resistance to reduce her particle formation was the next step. Bringing her fasting insulin down from 9.3 towards a more optimal level of under 6 is one of the most powerful things she can do to reduce her small LDL particle number over time. Resistance training two to three times a week is the single most effective intervention for improving insulin sensitivity and reducing small LDL particles—and remember, I said two to three times a week; we're talking 20 minutes. If you do twice a week for 20 minutes, I'd be happy. Post-meal walks—walking 10 minutes—reduce postprandial (after-eating) glucose spikes by 20 to 30%, directly reducing insulin demand. Berberine, 500 mg, 2 to 3 times a day with the meal, has been shown in studies to reduce small, dense LDL particles and improve insulin sensitivity. Omega-3 fatty acids, EPA and DHA, 2 to 4 grams a day specifically, have also been shown to reduce small LDL particle number and lower triglycerides that fuel this formation.

Okay, what was my priority with her? Lowering her MPO through targeted anti-inflammatory support. So Claire's MPO number, as I said, was 79 — that tells me she has active inflammation in her vasculature, which means we need to address it directly, not just wait for other numbers to improve. So I gave her high-dose omegas, EPA and DHA, two to four grams a day — it reduces MPO activity and vascular inflammation; it's one of the most evidence-backed interventions you can do. The other thing I gave her was vitamin C, one to two grams a day — it's shown to directly inhibit MPO activity and reduce oxidative stress in arterial tissue. I optimized her sleep by reducing inflammatory cytokines and MPO levels — sleep is anti-inflammatory medicine. So I'm telling you, sleep is so important; you have to get more than six hours a night. And I, you know, I can't tell you that I always do, and just even doing this podcast has made me realize, with all my research and everything, that we just have to emphasize sleep is the foundation of health — it really is. And then magnesium glycinate, 400 to 600 milligrams at night, reduces her cortisol, supports deep sleep, and has independent anti-inflammatory effects.



Okay, the panel that I want you to ask for — so this is for you, for you to go to your doctor. If you're not seeing me, I want to make sure you have everything you need to get the kind of care your heart deserves. So I want you to be very specific, because vague advice doesn't help. When you walk into your doctor's office, I want you to say these exact words: "Hey doc, I'd like to have you order a Cardio IQ Advanced Cardiac Panel — specifically, these markers" — and I'm going to give them to you — "can we add those for my next blood draw?" Okay, the Cardio IQ panel is offered by Quest Diagnostics and LabCorp, and you can get it at any major lab. Access does them as well — Access Labs is who we use at the clinic. It's increasingly available through functional medicine providers. It goes far beyond a standard lipid panel and includes markers that actually predict your cardiovascular risk, your true risk.

So what's in the Cardio IQ panel? If they don't know what a Cardio IQ is, you can ask for individual markers: Lp(a), lipoprotein A, that's your genetic cardiovascular risk marker; you have to know your number. LDL particle number and size, so LDL-P — how many particles you have, and are they dangerously small, dense ones. Apolipoprotein B, also called ApoB — total atherogenic particle burden, the best single predictor of cardiovascular risk. Myeloperoxidase, MPO — active arterial inflammation; it's the fire, so it's kind of like, is the fire already burning. Oxidized LDL — oxidized, damaged LDL directly accelerates plaque formation. High-sensitivity CRP is in that cardiac panel; it gives us a marker of systemic inflammation, also called high-sensitivity C-reactive protein. And then lipoprotein fractionation, which breaks down the LDL, the HDL, the VLDL into subclasses, which some of those cardiac tests will do as well.

This panel, combined with fasting insulin, a full hormone panel—estradiol, progesterone, testosterone (free and total) — and a calcium score if indicated, based on what I told you above, is a complete cardiovascular picture. For any woman over 40, and for some women even in their 30s if you have some of the things I talked about, this is what it means to actually know your risk. You have to know your risk. This panel, combined with fasting insulin and a full hormone panel, is a true indicator of your risk stratification.

Okay, step one: starting this week, ask for your Cardio IQ Advanced Cardiac Panel at your next appointment, and specifically request the ones I just mentioned. If your



doctor will not do it, find a functional medicine doctor — go to our website; we'll be happy to help. We do telehealth, but you need this cardiac panel run. Add fasting insulin to your annual labs—hemoglobin A1c alone isn't enough. Fasting insulin gives you a 5- to 10 year earlier warning about metabolic dysfunction and pre-diabetes, and metabolic dysfunction drives your small particle count, so don't forget that. Number three: if your Lp(a) is above 30, or your LDL particle small is elevated, ask for a calcium score — it's inexpensive, \$50 or less, it's low radiation, and could be the most important test you ever have. Number four: get a full hormone panel — estradiol, progesterone, testosterone, DHEA, and a cortisol urinary four-point test. Your hormones are directly linked to every marker we discussed today. Number five: add resistance training — even 20 minutes, twice a week, meaningfully reduces your small LDL particle formation, improves insulin sensitivity, and supports hormone balance. And please remember, exercise is medicine, even cardiovascular medicine. And number six: take sleep seriously — this is cardiovascular medicine as well. Poor sleep raises MPO, worsens particle size distribution, and elevates cortisol. Magnesium glycinate at night, morning light exposure, and no screens 60 minutes before bed are all starting points to help you live healthier, increase your longevity, and reduce your cardiovascular risk.

Remember, you are not at the mercy of your genetics; you are at the mercy of whether you know your numbers. So know your numbers. Okay, my final teaching pearls. I want to leave you with the single most important thing I took away from Claire's case: three numbers — Lp(a), lipoprotein A; small LDL particles; and myeloperoxidase. Three different mechanisms: genetic risk, particle danger, and active inflammation. They were all working against the same woman at the same time, and all of them were not visible in a standard cholesterol test. Claire did everything right — she exercised, she ate well, she showed up for appointments, she played pickleball. She was the epitome of health, and she was still walking around with what I call a cardiac time bomb that required urgent attention.

This isn't about fear, ladies; it's about information. When we have clarity and information, life is different, and we make better choices. The Cardio IQ panel exists; it's available; it's not exotic or experimental—it's factual, scientific data that will help



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reduce your cardiac risk. The women who are dying from heart disease in their 60s and 70s, sometimes in their 30s and 40s, are not the ones who knew their Lp(a), knew their LDL particle count, and knew their MPO. It's the ones who were told, "your cholesterol is a little high, watch your diet and exercise," and nothing else was done. Those are the unfortunate women who are having the events, and they didn't know they were coming. You deserve a full picture. Ask for the Cardio IQ panel. Know your numbers. Don't wait for the symptoms, because unfortunately cardiovascular disease is almost always silent — usually the symptoms come when you have an event.

If this episode helped you, which I hope it did — and I wasn't trying to be too heavy, but I'm very, I don't know, serious and passionate about this topic, because it's our number one killer, and I have the opportunity every day to hopefully change the trajectory of someone's life, and you do too. So if you know a woman, you should share it with her — especially the women you love, all women; it doesn't matter if you love them or not; share it with them, because if you have questions about your own labs, they probably do too, and they probably don't know this important information. Okay, ladies, I'll see you next week. Take care of your heart. Know it's working hard for you, so give it what it needs: proper testing.