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AMD: IN CONVERSATION

Speakers: Morgan Ineson; Dr. Larissa Moniz, PhD; Dr. David Ta Kim; Dr. Sheldon Rowan

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Welcome

Morgan Ineson: Good morning, everyone, and welcome. Thank you so much for joining us for ViewPoint. My name is Morgan Ineson, and I'm the Senior Manager of Education and Engagement at Fighting Blindness Canada.

For today's session, we're going to explore the latest in age-related macular degeneration, or AMD, care and research, including current approaches to treatment, emerging developments, and the role of nutrition and other lifestyle factors in supporting eye health. We have two fantastic experts who will bring complementary perspectives to the conversation: Dr. David Ta Kim, who brings expertise in the clinical care and treatment of AMD, and Dr. Sheldon Rowan, whose research focuses on nutrition, metabolism, and renal health.

I'd now like to introduce my colleague, Larissa Moniz, Director of Mission and Research at Fighting Blindness Canada, and our esteemed speakers, Dr. Ta Kim and Dr. Rowan. I'll turn it over to you, Larissa.



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Introductions

Dr. Larissa Moniz: Thank you very much, Morgan, and welcome, Dr. Ta Kim and Dr. Rowan. We're so happy to have you here. Quick introductions before we jump in. Dr. David Ta Kim is a retina specialist and a clinical instructor at the University of British Columbia. Dr. Sheldon Rowan is a scientist and professor at Tufts University whose research explores how diet and nutrition influence age-related eye disease. Welcome, both.

Dr. David Ta Kim: Thank you so much.

What Is AMD? Wet vs. Dry

Dr. Larissa Moniz: To start, I'm going to ask Dr. Ta Kim to set the scene for us. What is age-related macular degeneration, or AMD, as we'll be calling it? And what is the difference between the wet and dry forms?

Dr. David Ta Kim: Absolutely. First of all, I want to thank Fighting Blindness Canada for organizing this. I think it's a great initiative, and thank you for inviting me.

I explain macular degeneration to my patients probably 20 times a day, so I break it down simply. With age, our macula, the center portion of our retina, loses cells and loses function over time. That's macular degeneration.

The next question is always, "What's the difference between the dry form and the wet form?" I tell patients that everyone has the dry form. You gradually lose these cells. First, you might hear the word drusen, which is just an accumulation of waste products. Eventually you lose cells, and that's the atrophy, or geographic atrophy, that you may hear about. That is a slow loss of vision over time.

On top of that, you can also have the wet form, which I explain as abnormal blood vessels that grow underneath the retina and cause leaking and bleeding. That happens acutely. Within a week or two, you'll notice your vision drop quite quickly if treatment is not started.



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When people have the wet form, they actually have both the wet and the dry. The injections work really well for the wet form, but behind all that, there is a slow, progressive loss of tissue. We are not changing the dry aspect, the atrophy that is evolving, and so patients can lose some vision over time. They always ask me, "If I'm getting these injections, why am I losing vision?" Well, that's because you have both the dry and the wet.

We have a great treatment for the wet form that has revolutionized our field and the lives of many people. The next frontier is to have a great, sustainable treatment for dry macular degeneration.

Geographic Atrophy and Complement Inhibitors

Dr. Larissa Moniz: Thank you. You mentioned the wet form, which has the anti-VEGF injections that many people on the call know well, and the dry form. In the last decade, maybe five or six years, we've also started to hear about geographic atrophy. Where does that fit into the conversation?

Dr. David Ta Kim: Atrophy means tissue loss. It's called geographic because it starts somewhere around the center of the vision and grows in a shape like a country on a map. Sometimes it grows around the center and eventually encompasses it, and sometimes it goes straight into the center.

We're hearing about it more and more because previously there was no treatment for it at all. In the last few years, there are now FDA-approved treatments for geographic atrophy. There are no Health Canada-approved treatments yet, and we can talk about the nuances behind that.

The first treatment for anything is never the big solution. Does it work? Yes, but all that means is that it slows things down. It does not stop the tissue loss, and it does not reverse it. My personal opinion is that it slows it down too little to be worth the injections, which is probably why Health Canada, and the European equivalent, have been hesitant to approve it. The FDA approved it around 2023, so my American colleagues have a lot of experience with it. But it is an injection every month or every other month, and each injection comes with certain risks. It's not like anti-VEGF, not yet. Maybe one



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day we'll find something that works as well. Anti-VEGF is magical. The anti-complement injections approved for geographic atrophy are not there yet.

Dr. Larissa Moniz: We get a lot of questions about the complement inhibitors, the newer treatment for geographic atrophy. It's approved in the United States and a couple of other countries like Australia, but not in the majority of countries, and not in Europe. One of the complement inhibitors is under Health Canada review right now, so we're waiting to see the outcome.

Your point is well taken. The anti-VEGFs were a real game changer, whereas the complement inhibitors are a first generation. It's amazing that these drugs are out there, but people generally see a small change in progression, and at least in the clinical trials, it hasn't affected visual function. The atrophy may progress at a slower rate, but it hasn't been shown to change how well people can see. Is that a fair summary?

Dr. David Ta Kim: Yes. To add some color on the background of anti-VEGF: before anti-VEGF, we had photodynamic therapy, and before that, we had macular laser. With macular laser, when you found abnormal blood vessels growing, you would blast them with a laser and burn them off. You can imagine how destructive that was to the macula. The center of your vision is the fovea, and the wet form can start right in the center or off to the side, but if you shoot laser there, you're going to lose some vision. You were basically telling patients, "I'm going to shoot this laser in your eye, and you'll lose some vision, but you'll thank me because in five years your vision would have been much worse." Even for me, that was hard to stomach.

Then we had photodynamic therapy, where you inject a medicine into a vein, it travels to the eye, and you use a laser to activate it. The medication goes into the abnormal blood vessels and closes them off. It was less disruptive, but you weren't really making people's vision better. That's where we are with the complement inhibitors: we slow down progression, but we don't really make vision better.

Anti-VEGF, though, is pretty incredible. You start a patient with macular degeneration on anti-VEGF, and when you see them a month later, many have not only had the disease arrested, but their vision is actually better. Many will gain one, two, or three lines on the eye chart. They notice it, we notice it, and it's a magical moment. It's one of the best parts of my job, and it's thanks to the revolution of anti-VEGF medication.



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Choosing and Using Anti-VEGF Treatment

Dr. Larissa Moniz: I have one quick question before I move to Dr. Rowan. There are a lot of different anti-VEGFs on the market, first generation, second generation, and so on. I know every province funds these medicines a little differently. When you're deciding on an anti-VEGF with a patient, how do you pick the right one?

Dr. David Ta Kim: I'm currently in BC, where we usually start with Avastin, which is a great medication, and then there are some newer ones. I did my vitreoretinal surgery training in Toronto, so I'm very familiar with Ontario, and I did my residency in Quebec, so I've been across the country. The funding is different in each province.

Each retina specialist is a little different, but this is how I do it, and I think most people do it this way. We treat every four weeks until all the fluid and bleeding are gone, which we can see on the scans. I tell my patients we'll treat regularly until I don't see any more difference, and then we know the disease has been arrested.

Then we start what's called treat and extend. Every time you see the patient, you treat them, and if they look good, you extend the interval from four weeks to six, eight, ten, twelve weeks, slowly spacing them out. Sometimes with the newer medications you can jump up four weeks at a time. That's my algorithm regardless of the medication: we always go in a stepwise fashion.

For anti-VEGF in macular degeneration, I try to keep patients on it as long as possible, even if we stretch to 12, 16, 18, or 20 weeks, which I have some patients on. We know macular degeneration is forever. The medication treats the fluid, but we don't cure people of macular degeneration. There are other, longer-lasting and perhaps more curative treatments coming, but anti-VEGF today is like Tylenol: it's symptomatic and temporary. It lasts a month, two, three, or four, depending on the agent and the patient.

So I tell my patients they're going to be on it long term. Sometimes I use the word "forever," although that's a little scary. If we want to keep your vision as good as it is, we have to do these injections long term. Even if patients don't want the injections anymore, I try to keep following up, because in almost every case, the disease comes back, and every time it comes back, we lose vision that we don't get back. Unfortunately, some people have difficulty getting to the office. It's not an emergency,



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but you do have to be seen within a week or two. The sooner you get in, the more vision we have to save.

Dr. Larissa Moniz: Thank you, that was really informative. We also had a question about how to spell anti-VEGF, which my colleague Ushany has answered in the chat. VEGF stands for vascular endothelial growth factor, which causes blood vessels to grow, so this type of medicine works to stop it.

Nutrition and AMD

Dr. Larissa Moniz: I'm going to turn to Dr. Rowan now. This is a topic we get a lot of questions about from our community: diet and health, and what we can do with our diet. Right now, we're thinking about AMD. What does the evidence tell us about the role nutrition plays in the development or progression of AMD?

Dr. Sheldon Rowan: I'm happy to be here, thanks to Fighting Blindness Canada. I'm originally from Toronto, so I wish I could be there in person, but I'll make do remotely. We know a lot about the role of diet and nutrition from epidemiology, which is the study of large groups of patients. We follow people who develop macular degeneration, assess which one's progress to advanced forms of the disease, and then look backwards to find the environmental factors that may have contributed.

For example, it was through epidemiology that we realized cigarette smoking is a large risk factor. People who smoke cigarettes have at least a two-fold greater risk of developing advanced macular degeneration. We used the same approach to look at people's diets, and those studies revealed that certain food groups seem to be protective.

Generally, these studies looked at patients who already had early or intermediate macular degeneration, because they were being followed by clinicians and were eligible to enroll. Multiple classes of foods seemed protective. Increased intake of green leafy vegetables was protective. So was increased intake of fruits and vegetables with lots of color, which comes from carotenoids, bioactive compounds in those foods. Intake of fish was protective, and decreased intake of red meat protected against macular degeneration.



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Most of what I'll talk about has to do with progressing from early or intermediate to late forms of the disease. We have a little data on prevention of new macular degeneration, but it's not as strong, because it's harder to follow patients who start disease-free.

What was really revealing is that even though many of these foods had protective effects, they didn't work well on their own. People who ate more vegetables, but not the colored fruits or fish, didn't have as strong an association. What helped put it together was studying dietary patterns, the foods people eat together.

The best-understood healthful dietary pattern is the Mediterranean diet. It involves a higher intake of fruits and vegetables, usually nuts and legumes, and whole grains and less processed carbohydrate-containing foods. Seafood and fish are also associated with it, as are certain fats. We think of olive oil as the classic one, but we've learned from other studies it doesn't have to be olive oil. Generally, it's higher intake of vegetable oils, those that are monounsaturated or polyunsaturated fatty acids, compared to animal fats, which are usually saturated fatty acids.

People who adhere to a Mediterranean diet have increased protection against progression of macular degeneration to multiple advanced forms, both wet and dry. There's also evidence that it can prevent progression from early to intermediate macular degeneration.

On the converse side is the Western dietary pattern: more refined carbohydrates and ultra-processed foods with a very high glycemic index, higher intake of animal proteins and animal fats, lower intake of fruits, vegetables, and dietary fiber, and less consumption of nuts and seeds. That pattern carries an increased risk of macular degeneration and its progression, almost on the same level as cigarette smoking. A poor diet is a distinct risk factor for macular degeneration.

Fats and oils

Dr. Larissa Moniz: That was really well laid out. I have a few follow-up questions. First, vegetable oils. I use canola oil. Is that a good vegetable oil, or a bad one? What are some of the other good ones?



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Dr. Sheldon Rowan: Canola is fantastic, because it's a rich source of both omega-3 and omega-6 polyunsaturated fatty acids. Both are essential in our diets.

Our bodies need omega-3 fatty acids. The ones most people know are DHA and EPA, and the major dietary sources are seafood, especially fatty fish, like salmon and tuna. If you don't have a high intake of those, you can get precursors from plant-based sources, which is useful for vegans. Flaxseed oil is a great source, and so is canola oil. Omega-6 fatty acids are also protective against multiple forms of cardiovascular disease, and they're a good substitution as well.

Dr. Larissa Moniz: We don't hear as much about omega-6. I feel like you always hear about omega-3.

Dr. Sheldon Rowan: We need both. It may also be more about what you're not having. We know the eye needs a lot of omega-3, and the retina is one of the most enriched sources of omega-3 fatty acids, but omega-6 is important too. Every time you choose an omega-6-containing fat, you're reducing your intake of saturated fats.

The other class of fats we don't talk about as much now, because they're bad, is trans fats. Most people who now have macular degeneration grew up when trans fats were common, especially in margarine and Crisco, the more solid vegetable-based fats. Trans fats are a really high-risk factor for macular degeneration, so cutting them down is just as important as having the healthy, essential fatty acids.

Dr. Larissa Moniz: So, unsaturated and polyunsaturated fats are basically fat sources that are liquid at room temperature. Is that a fair statement?

Dr. Sheldon Rowan: That's a good way to think about it.

Dr. Larissa Moniz: And fats that are solid at room temperature, like Crisco, butter, margarine, and animal fats, would be more of a trans fat or a saturated fat. So unsaturated and polyunsaturated are good, and the others should be eaten in moderation or minimally.

Dr. Sheldon Rowan: Correct. I know someone asked about butter. Butter in itself is probably neutral. It contains some less common saturated fats that may not be bad for



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you. The ones we know for sure are bad are those you get from eating things like red meat. Not all saturated fats are equal.

Dr. Larissa Moniz: The Mediterranean diet comes up for many diseases, and people say to eat it to be heart healthy. Is there anything specific about the diet you're describing that relates to AMD or retinal disease, or would a generally heart-healthy diet also be healthy from an AMD perspective?

Dr. Sheldon Rowan: That's a great question, and it's one of the reasons I love the Mediterranean diet. It's really protective against cardiovascular disease, and I've never seen a study where people with higher intake of a Mediterranean diet are at increased risk for any specific disease. In some cases we can separate its benefits in macular degeneration from those in cardiovascular disease. The original Mediterranean diet randomized controlled study had two arms, one with olive oil and one with nuts, and both were equally cardioprotective. But for macular degeneration, nuts and seeds end up being a little more beneficial than olive oil itself. We don't have a strong signal for olive oil and macular degeneration.

Dr. Larissa Moniz: Fascinating. We have a lot of questions coming in. Don't fear: Dr. Rowan will be here for the Q&A, and he'll also be in one of the breakout rooms afterwards, so there will be lots of time to ask questions.

AREDS2 and Supplements

Dr. Larissa Moniz: We've talked about diet a lot, so let's move to supplements. We know AREDS2 in particular is recommended as a supplement for slowing the progression of age-related macular degeneration. Dr. Rowan, I'll get your input first, and then Dr. Ta Kim's. What is the evidence around supplements and AMD?

Dr. Sheldon Rowan: I was hoping we'd talk about AREDS. I wanted to start with the nutritional epidemiology, because that's how we got to AREDS. The original AREDS formulation contained vitamin C, vitamin E, beta-carotene, zinc, and copper. It combined what were thought to be antioxidant vitamins and mineral-based compounds that epidemiology suggested could be protective. What changed in AREDS2 was replacing the beta-carotene with two other carotenoids, lutein and zeaxanthin.



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The AREDS2 study was a large randomized controlled trial, and it reduced progression from intermediate to advanced macular degeneration by about 25% compared to placebo. In AREDS2, everyone was already taking AREDS, so we don't have as strong a sense of whether replacing the beta-carotene with lutein and zeaxanthin offers additional benefit.

There are a few nuances compared with the Mediterranean diet. They may not work through exactly the same mechanisms, and in some cases they may have additive effects, so taking one doesn't mean you shouldn't take the other. In terms of preventing progression, AREDS2 works really well for preventing progression to the wet form, but it doesn't have as strong an effect on preventing progression to the dry form. The Mediterranean diet seems to work better at preventing progression to the dry form. So the best advice for someone with intermediate macular degeneration is to do both: take AREDS2, and stick as close to a Mediterranean diet as you can, based on your food preferences and availability.

AREDS2 has also not been shown to prevent development of AMD in people who are originally disease-free. There may be hints in participants who had AMD in one eye but not the other, suggesting it slowed development in the disease-free eye, but that's not what the trial was designed for, and the numbers were too small to know. We can't say for certain that AREDS2 prevents development of macular degeneration. There is a little data suggesting the Mediterranean diet may be protective against that, but we know a lot more about how these work in progression from intermediate to advanced macular degeneration.

What patients ask

Dr. Larissa Moniz: Dr. Ta Kim, I'd like to pull you into this. What are common questions or misconceptions your patients have about diet or supplements? For instance, if there's a family history of AMD, do patients ask whether they should be taking AREDS, and what do you say?

Dr. David Ta Kim: Absolutely. Many of the patients I inject are in their late 70s, 80s, or 90s, and often it's their children, in their 50s or early 60s, who bring them in. They ask, "My mom or dad has wet macular degeneration. What can I do?"



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Number one, anyone over the age of 50 should be screened for macular degeneration. If you don't check, you don't know, and waiting until you have symptoms is not ideal. I'll echo what Dr. Rowan said: AREDS2 has great evidence. These studies were done in the 90s, with a huge amount of work and money behind them, and they might never be replicated, but they gave us excellent data. We can say with a lot of confidence that AREDS2 vitamins work really well at slowing progression from intermediate to advanced macular degeneration, specifically wet macular degeneration.

Clinically, when the children of my patients ask, "Should I be taking AREDS2?", the answer is simple. If you have intermediate macular degeneration, yes. If you have early macular degeneration or none at all, we don't have evidence that it benefits you. So, to simplify, the answer is no. Maybe it helps and maybe it doesn't, but I can't tell you with conviction that it will. I have that conversation dozens of times a day.

Dr. Larissa Moniz: Are there other common questions or misconceptions that come up, about diet or anything else in your practice?

Dr. David Ta Kim: People ask, "What else can we do?" I echo the statement about smoking. Smoking is very harmful for macular degeneration, and really for anything in medicine. Luckily, smoking is much less prevalent these days. I tell patients to stop smoking all the time, and they often tell me they stopped 30 years ago, which is great. The Mediterranean diet is good for your eyes, your heart, and your brain, including stroke prevention.

People also ask about sunglasses, and it makes sense, because UV light is a source of damage over time. But starting to wear sunglasses in your 70s probably won't change all that much, and before recommending something that changes someone's lifestyle, you need a lot more evidence. If it's sunny outside and you want to wear sunglasses, great. But don't wear sunglasses outdoors and indoors thinking it will decrease your risk of AMD progression, because we don't have the evidence. And what if you trip and fall because you can't see? That's not something I recommend.

The other thing about vitamins is that people bring in bottles of brands I've never heard of, so I Google them in front of them to see what's in them. A lot have lutein, zeaxanthin, vitamin E, vitamin C, and zinc, very similar to AREDS2. But the AREDS2 studies are the ones with the most evidence. If it says AREDS2 on it, you can be sure there's evidence



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for it. If it says "additional this or that," it has not really been proven. If you're going to take something, it might as well have a lot of evidence behind it.

Dr. Larissa Moniz: That preempted my next question beautifully: how can you tell the difference between promising research and what has been shown to work? Studies tell you whether there is an impact or not. If there isn't, sometimes the supplement doesn't do anything, but sometimes it just means we don't have enough evidence. We can't recommend something if the evidence isn't there. It might work, and future studies may show it, but from what I'm hearing, AREDS2 is the only one with really strong evidence that it can reduce progression, specifically from intermediate to advanced.

Dr. David Ta Kim: That's right. It's been around a long time, since the studies in the 90s. People always want the latest and greatest, like the new shiny iPhone, but those studies took a lot of money and effort, and I highly doubt any vitamin supplement will ever replicate the amount of data and the power of the AREDS2 conclusions. I don't expect my patients to sift through the data, so I say, ask your eye doctor. My patients bring in their supplements all the time and ask whether they're worth taking. If it says AREDS2 on it, yes. If it doesn't, maybe it's just as good, maybe a little better, maybe a little worse. No one really knows.

Dr. Sheldon Rowan: I want to add something about fish oils. I mentioned the very strong protective association with fish intake, and there was an assumption that the fish oils must be the healthful part. It turns out it's probably more complicated. One branch of the AREDS2 study tested whether fish oil supplements containing DHA and EPA would have an additional protective effect, and they didn't. Maybe they weren't in the right form, or the right dose, but supplements don't always replicate what the foods do. Fish oil supplements are probably not harmful, but I don't know that they're helpful either, and they shouldn't be viewed as a replacement for eating fish or seafood if those are part of your diet.

Dr. Larissa Moniz: What about eggs that are enriched with omega-3? Are they as good as getting omega-3 from fish?

Dr. Sheldon Rowan: They won't be quite as available to the eye as omega-3 from fish and seafood.



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Dr. Larissa Moniz: That's unfortunate, because I'm allergic to a lot of fish.

Looking Ahead: What Is Exciting in AMD Research

Dr. Larissa Moniz: One final question for both of you. We always like to end by asking what in AMD research and treatment excites you most as you look to the future. Dr. Rowan, you first, and then Dr. Ta Kim.

Dr. Sheldon Rowan: What excites me is understanding how things work. Beyond knowing that certain foods or vitamins are associated with protection, I want to know the mechanism, how it works, and whether we can make it work better. What's exciting is the understanding that the effects of many nutrients and vitamins may work through the gut microbiome. Part of my research is understanding how those foods get translated from the gut into protection in the eye. We've made some very interesting discoveries, and we even think it's possible that AREDS2 works through changes in the gut microbiome. Once we work out those mechanisms, we could find ways to make them work better, or find new drugs, foods, or nutrients to complement what we already have.

Dr. Larissa Moniz: That's really exciting. The gut microbiome is a hot topic lately, and many people are drinking kefir. To simplify, is kefir part of having a good gut microbiome?

Dr. Sheldon Rowan: It's not too simplistic. Fermented foods can contribute to usually positive changes in the gut microbiome. The only thing is that you have to keep eating them. If they're foods that are part of your diet, keep eating them, and it's probably good. A one-shot deal won't do much. The microbiome is very resilient.

Dr. Larissa Moniz: Thank you. And Dr. Ta Kim, ending with you before we move on to the audience Q&A: what excites you about the research and clinical trials coming up for your patients?

Dr. David Ta Kim: I can speak to both wet and dry macular degeneration. My practice is involved in a lot of this exciting work, including phase 3 clinical trials, but also phase 1 and 2.



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Let's start with the wet form. We have injections that, like I said, revolutionized the practice, and they work really well. The downside is that you need them forever. It's a lot of burden for patients to come in, especially at this age, and their families have to bring them. Every injection also carries a small risk of complications, like infection. So what excites me most is gene therapy. We're involved in two phase 3 studies of gene therapy. The idea is that you inject a gene into someone's eye, either with surgery or with a simple injection, and it turns your eye into a biofactory. Say you're getting Eylea or Lucentis: the gene therapy transforms some of the cells in your eye into producing that medication. So instead of getting an injection, you produce the medication yourself.

The results so far in some of these trials have been very promising, and hopefully we'll get more data in the next year or two. I'll be realistic, though. This isn't going to happen tomorrow or even next year. Getting this into routine use will take many years, and there are many challenges. But it's very exciting, because it's tough for patients to come in for these injections all the time.

What about the dry form, which doesn't have any great treatments right now? We're also several years away, possibly many, from something routine. But we'll be involved in trials of injections, stem cells, and gene therapy. There's a lot coming down the pipeline. I want to reassure everyone that tons of people are working very hard on this, at both the basic science and clinical levels, and we're working together to offer something to our patients. I'll end on that high note.

Dr. Larissa Moniz: Thank you both. Please stay where you are, because I'm going to hand it back to Morgan, who will move us through the audience Q&A. Thank you for that wonderful discussion.

Audience Q&A

Morgan Ineson: Wow, we have so many questions, which is fantastic. Dr. Rowan is going to stay with us for Community Connect, so I may skew some of these questions toward Dr. Ta Kim while we still have him. Rest assured, we'll have lots of time for questions in the breakouts as well.



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The macula and its function

Morgan Ineson: Let's go back to the beginning. Can you talk about the normal function of the macula, and how it's affected by AMD?

Dr. David Ta Kim: The macula is the center part of the retina. Think of the retina as the film in a camera, and the macula as its center. It's important for any central vision function: reading, or looking at people's faces, or making out any detail. The side, or peripheral, vision is more for movement. When you're driving and see something coming from the side, that's your peripheral vision.

The macula has mostly cones, the cells responsible for seeing fine detail. When you get macular degeneration, you get a preferential loss of central vision. Patients sometimes come to me a little late, after a lot of vision loss. The one good thing I can tell them is that, assuming you don't have any other eye conditions, you will never go completely blind from macular degeneration. Worst case, you lose the entire function of your macula, which is very unfortunate, but you won't lose your peripheral vision. You'll still be able to walk around. It won't be pitch black, if that's any consolation. The function of the macula is central vision: reading, details, recognizing faces. It's super important for our lives.

Anti-VEGF for wet vs. dry AMD

Morgan Ineson: Can you clarify that anti-VEGF medications work for wet AMD but not for dry AMD?

Dr. David Ta Kim: That's correct. Vascular endothelial growth factor is a chemical inside the eye that gets released and causes abnormal blood vessels to grow and leak, producing fluid and blood. The medication stops that molecule, so the blood vessels basically wither and die and stop leaking and bleeding. In a nutshell, that's how anti-VEGF works.

Genetics and screening

Morgan Ineson: A couple of questions about the genetics of AMD. Is it hereditary? What is the evidence that it's genetic?



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Dr. David Ta Kim: We have quite a bit of evidence of a genetic link. In my practice, when I ask whether anyone else in the family has this, patients often say, "Six of my ten brothers and sisters have it," or "My mother had it, my grandmother had it." So there is definitely a genetic component. But the "A" in AMD is age. It's both your genetic predisposition and age, and no one gets AMD at age 20. It's usually 50 and above, so I think age is the biggest component. Every year we all get older, and there's no treatment for aging, at least not much yet.

Morgan Ineson: A question from someone in their 70s with wet and dry AMD: should their children, who are in their 40s, be tested as a preventative measure?

Dr. David Ta Kim: The guidelines may have changed, but I usually recommend starting at age 50. With a family history it can sometimes start in the late 40s. If you're in your 30s, there's no point screening for it. But in general, forgetting macular degeneration for a second, I recommend that everyone get their eyes and vision tested every one to two years, whether you're 18, 30, 55, or 80. There are other things that can happen, or sometimes glasses can fix it. At least every two years, for sure.

Morgan Ineson: Our discussion today is focused on AMD, but there are other conditions, like glaucoma, cataracts, and diabetes, that show up in the eye earlier too.

Dr. David Ta Kim: Many, many.

Red light therapy and electrostimulation

Morgan Ineson: Before we get to the nutrition and vitamin questions, there are several questions about MacuMira, or red light therapy, also called photobiomodulation. We're hearing more about these treatments in the last couple of years. Can you explain how they're supposed to work, and what the advice is at this time?

Dr. David Ta Kim: There are basically two classes of these treatments. One is electrostimulation, and the other uses red light. The mechanics make sense on paper: you're stimulating the cells to keep working and ward off degeneration. Both MacuMira and Valeda are Health Canada and FDA approved. What does that mean? I get this question a lot. The Canadian Retina Society, an association for retina specialists across



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Canada, in conjunction with the Canadian Ophthalmological Society, has come out with a position paper explaining this in detail, but I'll sum it up.

Both treatments are Health Canada and FDA approved, but there's not good evidence that they work. How does that make sense? Medications, like the complement inhibitors I mentioned earlier, which have FDA approval but not Health Canada approval, must be shown to be both safe and effective. For devices, you only have to show they're safe. For both Valeda and MacuMira, we can say with relative confidence that they are safe. You're not going to be harmed. They had studies to try to show effectiveness, but most of my colleagues and I don't find that data strong enough to recommend them.

The take-home message I give my patients is that you can go ahead and try them, and I don't think they'll harm your eye. Will they work? I don't have the answer right now. They're trying to gather more data, and if something worked, I would love, love, love to offer it to my patients, because right now there's really nothing. But in medicine, the first rule is to do no harm, and even if a treatment is proven not to do harm, it takes a lot of time and a lot of money, because it's not covered. For that to be worth it, I'd need a lot more data before I start recommending either of these treatments. I wish them well, but right now I cannot recommend them.

Morgan Ineson: So, waiting for more evidence, because as you said, there's a personal cost associated with these treatments, not just a cost to the system.

Dr. David Ta Kim: Yes.

Exercise and alcohol

Morgan Ineson: Now some questions on lifestyle. Is regular exercise protective against AMD? Is alcohol harmful? Dr. Rowan, I'll start with you, then Dr. Ta Kim.

Dr. Sheldon Rowan: The evidence does suggest exercise is beneficial. I think there's stronger evidence that exercise may protect against the initial development of macular degeneration, and less about progression. There's no downside to exercise. It's beneficial for many aspects of health and well-being, and it could even make having macular degeneration a more workable condition. When people look at lifestyle factors



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overall, with exercise as one of them, they see a positive signal for exercise as part of a healthy lifestyle.

For alcohol, there's some data suggesting heavy alcohol intake could be harmful, but it's hard to disentangle that from cigarette smoking. It may be the lifestyles associated with heavy alcohol intake that are harmful. There's no good data suggesting that very low to moderate intake is beneficial. Even though the Mediterranean diet may include red wine, that part hasn't been associated with macular degeneration.

Dr. David Ta Kim: I agree with Dr. Rowan. Exercise is always good. I don't just treat eyes, I treat the whole patient, and it's important for heart health and brain health. For low to moderate alcohol consumption, more data is coming out. For the eyes, I'm not aware of anything specific, but for your general health, it's probably best to limit alcohol.

Vitamin brands and side effects

Morgan Ineson: Someone asked whether MacuHealth Plus is better than AREDS2. There are lots of vitamin brands out there. Dr. Rowan, I'll start with you.

Dr. Sheldon Rowan: At a recent ARVO meeting where I was presenting research in this area, one group measured how much lutein and zeaxanthin were in some of these off-brand supplements, and the numbers were all over the place. They weren't what was reported on the bottle. So I'd be cautious, and make sure you're working with a reputable source. As for anything being better than AREDS2, we have absolutely no data for that.

Morgan Ineson: And supplements aren't regulated in the same way as drugs, so you want to be careful and choose a reputable source.

Dr. David Ta Kim: I get this all the time, people bringing in their off-brand vitamins. AREDS2 is old data, but it's the best data we have, and we're probably never going to get better. I tell my patients it's very simple: if it says AREDS2 on it, I approve. I don't care what brand, but it has to say AREDS2. That's it.

Morgan Ineson: Some people report that AREDS2 upsets their stomach or has other effects. Someone in the Q&A said their optometrist told them they couldn't take AREDS



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because of the zinc, and asked about saffron. Are there alternatives for people who have difficulty taking AREDS2?

Dr. Sheldon Rowan: There are probably some components that are not as important as others. The AREDS2 study tested a lower dose of zinc. I think the original was 80 milligrams, and they found that 25 milligrams worked just as well. It's very possible that you could take the parts you tolerate better and still get some benefit, as long as you're getting high-quality versions.

Dr. David Ta Kim: Absolutely. In my experience, most of the side effects from AREDS2 are due to the zinc. A couple of my patients really can't take it at all. In that case, they could take a version without zinc. It wouldn't be AREDS2 at that point, but if they have intermediate macular degeneration or more, I think it's reasonable. I tailor it to the patient. I always say to try AREDS2 first, but zinc is known to cause GI intolerance, so if you're someone who suffers from that, don't power through it. You can derive some benefit from the other components, like Dr. Rowan said.

Dr. Sheldon Rowan: The lutein and zeaxanthin are probably especially important. Some studies show that those on their own can improve visual acuity in patients with macular degeneration. If those are tolerated, try to get them, because I think they have a lot of benefit.

Morgan Ineson: And make sure you talk with your eye doctor about what you're taking before you start new supplements, and get their advice on what works for you. We're not all built the same, so different things affect different people in different ways.

Implants and port delivery

Morgan Ineson: We'll move to Community Connect in a minute, and there are still lots of questions about nutrition, which we'll take there. One last question for Dr. Ta Kim. Someone saw a news article about implant surgery for AMD. Is that happening, and what are the trials? I guess it could be a port delivery system.

Dr. David Ta Kim: There are many different things. Port delivery is a little older technology. You install a depot in someone's eye and refill it, like filling a gas tank, maybe every 6 to 12 months instead of coming in for injections every month. Honestly, I



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don't foresee that coming to market in any meaningful way, because it has some issues. With a device in your eye, there are long-term concerns about infection and wound problems, especially now that we have more promising avenues, like the gene therapy I described, where you don't have a foreign body in your eye. Of course, gene therapy is so new that other issues may come up, and we need more time to study it.

There are also other devices. Second Sight was a company that implanted devices in people's eyes when I was in training, a little microchip that goes onto the macula. That was more for inherited retinal diseases than macular degeneration, though it could have worked for that. It's not so much at the forefront anymore, because of the issues with foreign bodies and the big wounds that come with it.

Morgan Ineson: That would have been a retinal prosthesis, like the bionic eye. And there's now the PRIMA implant, which is also under investigation and showing some positive results.

Dr. David Ta Kim: Yes, something to watch. There are a lot of different things we can do, and we're trying to figure out which has the least harm and the best efficacy.

Closing

Morgan Ineson: Thank you so much to both of you. That was great, and I think we could keep going for hours. We're going to let Dr. Ta Kim get to his clinic, and we're going to move into our Community Connect session. Thank you both for your fantastic answers.

Dr. David Ta Kim: Thank you for organizing and for inviting us. It's been a pleasure.

Dr. Sheldon Rowan: Thank you. This has been fun.