# 97| Medications for Memory Loss in Older Adults – With Dr. Matthew Growdon

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NAVIGATING NEUROPSYCHOLOGY

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**Speakers**: Matthew Growdon, Ryan Van Patten, John Bellone

**Intro Music** 00:00

**Ryan Van Patten** 00:17
Welcome, everyone, to Navigating Neuropsychology: A voyage into the depths of the brain and behavior. I'm Ryan Van Patten...

**John Bellone** 00:25
…and I'm John Bellone. Today we speak with Dr. Matthew Growdon once again. This episode focuses on medications for memory loss in older adults and pairs well

with the June 1st episode on polypharmacy with Matthew and Dr. Mike Steinman, so check out that episode if you haven't already. Matthew is a clinical geriatrician and research fellow, soon to be an assistant professor at the University of California, San Francisco. His research is in polypharmacy and deprescribing in older adults.

**Ryan Van Patten** 00:55

In this episode, we cover cholinesterase inhibitors for memory loss in older adults, their efficacy in Alzheimer's disease dementia as well as in other dementia etiologies, their efficacy in mild cognitive impairment, the NMDA-receptor antagonist memantine, combination therapies, how to talk to patients about these medications, how to write about them in neuropsych reports, and we end with a brief discussion of aducanumab. You'll notice that I was just coming out of a cold while we recorded this episode, so my voice is going to sound a little off. Sorry about that in advance but, you know, John always sounds froggy and you're used to that. So, hopefully, you can put up with my nasally voice for one episode. [laughs]

**John Bellone** 01:42

[laughs] Fair enough.

**Ryan Van Patten** 01:43

So now without further ado, we give you our conversation with Dr. Matthew Growdon.

**Transition Music** 01:47

**Ryan Van Patten** 01:57

All right, we're back with Matthew Growdon. Thanks for making the time.

**Matthew Growdon** 02:00

Thank you. It's good to see you both.

**Ryan Van Patten** 02:02

So today we'll start by talking about cholinesterase inhibitors. These medications increase the availability of acetylcholine at the synaptic cleft. They do this by blocking the acetylcholinesterase enzyme so that the neurotransmitter itself, acetylcholine, is not broken down quite as rapidly and then it's more available to

bind to receptors. So give us a brief summary of the evidence for these medications in dementia from Alzheimer's disease.

**Matthew Growdon** 02:31

I'd say cholinesterase inhibitors are a mainstay of treatment of dementia, particularly in dementia of Alzheimer's type. The top-line most important thing to know about these medications is that they are symptomatic therapies. They're not in and of themselves believed to be neuroprotective or to alter the disease of the underlying dementia. So, as Ryan mentioned, the theory underlying the use of cholinesterase inhibitors relies on this so-called cholinergic hypothesis of Alzheimer's disease from early studies in the understanding of Alzheimer's that showed that there was decreased acetylcholine, this essential neurotransmitter. So these cholinesterase inhibitors, by inhibiting the enzyme that breaks down the cholinesterase, increases the levels of the acetylcholine. The idea is to get more cholinergic transmission in the synaptic clefts in the central nervous system. These have been studied mostly in Alzheimer's disease and my feeling and the feeling of many geriatricians is that the benefit is modest at best. They are approved by the FDA in the use of all stages of dementia, so mild, moderate and severe dementia. Patients who are treated with these medications may have a small improvement in their cognition, potentially in neuropsychiatric symptoms, and potentially in activities of daily living.

There are some important side effects to talk about. I think we'll probably delve into those a bit more, but the big ones to think about are that because of these effects of having extra cholinergic transmission, we have to be careful about using these particularly in people who have a cardiac history or history of a slow heart rate because they can cause the heart to slow down even more or to have an entity called a heart block. They can have some pretty impressive GI side effects that are often limiting in terms of people's ability to take them - things like nausea, diarrhea. We can get into that a little bit more, but, in general, they are a first line. So if someone is newly diagnosed with dementia, particularly if it's felt to be amnestic maybe due to Alzheimer's disease, it is something that many clinicians will reach for to try to give their patients that benefit.

**John Bellone** 04:50

My understanding is that they're fairly well tolerated though, despite maybe some GI symptoms. Is that your experience?

**Matthew Growdon** 04:56

One good thing is that since they've been around for a long time, there are a number of different cholinesterase inhibitors. So there's donepezil, galantamine, rivastigmine. If someone develops some of those GI side effects I was talking about, clinicians can often try to either change the dose or come down a little bit on the dose, or sometimes switching between those different medications within the class can help for each person as an individual in terms of how they respond. If GI side effects are really a problem, there are patch formulations, transdermal ones, that people can wear. Those have the advantage of having less GI side effects. So if a family and a patient and the doctor decide that it's really important to have a trial on the cholinesterase inhibitor, there are definitely ways to get them onto one even if they run into some side effects. The cardiac side effects are more of something really to be careful about. In those cases, people will need an EKG and they may need to have conversations with their cardiologists about what's safe. That's more of an exception than the case, but something that's important to monitor for.

**Ryan Van Patten** 06:09

I've heard neurologists describe the utility of cholinesterase inhibitors to their patients by telling them, "These medications don't reverse or stop the disease, but they can turn back the clock on your memory decline. Your memory has been gradually getting worse over the last few years, which means it was better six months ago than it is now. So these medications, if they work for you, could turn back the clock on your memory so that it's as good as it was six months ago." I'm wondering if that's the language you would use? I've also heard "reduced worsening" as a way to describe it to patients. How do you talk to your patients about these medications?

**Matthew Growdon** 06:50

It's interesting to hear that because I've also encountered in my own training and now in practice different ways that these are presented to patients and their families. I think one thing just off the top of my head in hearing that is that it is a hard thing to explain to people, particularly people who may be newly diagnosed with an underlying cognitive disorder. I will often use the terminology that it “doesn't modify the underlying disease.” Unfortunately, this class of medication doesn't affect the process of something like Alzheimer's disease, but it may slow down some of the cognitive effects and potentially some of the functional changes. One study that I've referred to in counseling people showed that it might have the benefit of, for example, over the course of the year, of preventing a two month per year decline in a typical patient with Alzheimer's. Another thing I think it is important to tell patients is that there is a fair bit of variability in terms of patient to patient response. One of the hard things about this class of medication is that many

patients or families may not perceive the benefit because, like we're talking about, the disease unfortunately continues to progress. It may well be that it is actually helping very moderately. Some people will say they feel the benefits, some people won't. But I think, for me, the most important thing is to not give false premises or false hope. I think it can be helpful to frame it positively because it is very hard to get one of these diagnoses and it is positive to have symptomatic therapy. On the other hand, we don't want to mislead people into thinking that it's going to stop the disease in its tracks.

The other thing, just for your listeners since I'm a geriatrician, there's definitely a bias in terms of the types of people that I see. There was actually an interesting study some years ago that showed that geriatricians are actually less likely to prescribe older adults with dementia this class of medications than, for example, neurologists or psychiatrists and I think that's probably more than anything because of the different types of patients that we see. If I am seeing an older patient with frailty and polypharmacy, some of the things we talked about in our other podcast, or if they have other cardiac abnormalities or slow heart rate already, or if they're dizzy, all of these things may make it harder for me to reach for this medication. In fact, some of my conversations end up being more on the severe end of dementia whether to discontinue these medications because the symptomatic benefit may have run its course. Then we're more worried about maybe this is just an extra pill that has some side effects associated with it but isn't giving a lot of symptomatic benefit. That's not to say that other people - these are prescribed all the time and definitely, like I was saying, are workhorses in this category of medication, but it really depends on what kind of patient you have with you.

**John Bellone** 09:56

You mentioned the variability in response. I know that I've seen in the literature there might be responders versus non-responders and that maybe the small mean improvements on clinical scales [are] driven by a small percentage of patients deriving more benefit, while others don't benefit at all. What's your understanding of that literature?

**Matthew Growdon** 10:18

I think that's a great point. I don't want to get away from the citations you're thinking about but, in general, I agree with this notion that we make these decisions based on population studies that show, for example, maybe there will be a neuropsych inventory or let's say a cognitive battery and then there's a score. On average, people in the cholinesterase inhibitor arm in these earlier trials may have a few points of benefit on average compared to those who were given placebo. Clinically, that doesn't necessarily correlate in my mind with a huge benefit. Like, if you told

me someone has a two point difference on an overall cognitive test - I'm being kind of vague because they use different ones - but it may not clinically correlate with like, "Aha! That's a huge difference." But underlying that average, like you're saying, there could be people who are responding more and there could be people who are actually continuing to worsen. That's one of the challenges of all drug trials. In a sense, we're applying these averages to individuals. A lot of the cholinesterase literature is quite old. These studies were done many, many years ago, and a lot of them were funded by the pharmaceutical industry. There was a non-pharma funded study that looked at some other outcomes, things like delaying nursing home admission, again in the geriatrician world something that we think about, and the medications don't, unfortunately, at least in that study, show a benefit in terms of delaying nursing home admission. So the studies have been shown to change these smaller parameters around like, a few points on the cognitive test or in the neuropsych inventory or potentially on delaying some functional impairment, but not on those big life changing events, like in terms of where people need to live and how much help they need.

**Ryan Van Patten** 12:20

This idea of responders versus non-responders, some people benefiting and others not benefiting, brings me to thinking about precision medicine and hoping we can get there. Like, if we could take people's genetic profile and know, based on my individual genetics, if a cholinesterase inhibitor will work and/or which one will work better, like maybe rivastigmine works better for me than donepezil, we could prescribe the medication that will work best for my physiology. I think I've seen at least one paper on this related to cholinesterase inhibitors, but I honestly don't know if there's any really good progress being made on this front. Have you heard of anything?

**Matthew Growdon** 13:03

You may have to find and bring into a future podcast someone who's more versed in this area, but certainly I agree with you that there's a new frontier out there of more targeted medication use that would hopefully deliver benefits with fewer side effects in a precision medicine way. But, clinically, in my practice, unfortunately, I haven't seen that in this class. There's only a few examples I can think of, not just in neuropsych per se but across all current prescribing practices, where we really think of, "Oh, we're going to get a genotype, or look at this at a genomic level before giving a medication." But undoubtedly with the pace of research, hopefully, that'll be coming and will be really exciting and helpful because, like I was saying before, you really want to harness the benefits here particularly if you've just delivered a diagnosis and a family and a patient are looking for whatever they can do to slow the decline.

**John Bellone** 14:07

Do we know if the effects of these AChE inhibitors wear off over time? Obviously the neurodegenerative process persists, but do patients after a year or year and a half stop deriving benefits if they were deriving them? Do we know?

**Matthew Growdon** 14:22

I can't give you a great answer for that one. I would say in my clinical experience there's definitely some variability in terms of people who stay on them and feel that they're maintaining a benefit for longer than the time horizon you said, so on the order of many years. But there are also a number of people I've had who continue to progress. There are other medications like memantine or Namenda that are sometimes added or used in the more severe stages of dementia. That may become more the focal point. There are certainly a number of people, for example, in the nursing home population that I've taken care of at different points that the benefit of the acetylcholinesterase inhibitors is really small. So the conversation is a little bit more about whether there are side effects related to it and then it maybe a rationale to again pull people off of it.

**John Bellone** 15:20

I'd like to talk about memantine which has a completely different mechanism of action. It's an anti-NMDA agent, right? It blocks NMDA-receptors. I'm curious about the mechanism and your understanding of its efficacy for cognitive and behavioral symptoms.

**Matthew Growdon** 15:38

So memantine, which initially had the trade name Namenda, does have a different mechanism of action from the cholinesterase inhibitors. Glutamate is another important CNS neurotransmitter and it acts on this NMDA receptor that you were mentioning, which is important for cognitive function, particularly learning and memory. There's this theory of overly excited or over-NMDA-stimulation leading to damage at the cellular level in the CNS. So the theory of a medication like Namenda is that by antagonizing that receptor it can tamp down some of that excitotoxicity. The story on memantine, from my mind, is really that it's a medication that is approved in the more moderate stages of dementia. There's less benefit from studies in the early stages of something like Alzheimer's disease. The quality of evidence is not super strong. Again, this is not a wonder drug. This is a go-to medication in the management of this disease, but it also will not yield a disease modifying benefit for people who take it. There are small effects of unclear clinical significance, so things like cognition. Again, global assessments of the dementia severity may, in these smaller trials where it was approved, show some benefit.

The benefits, I think, are that it doesn't have those cardiac risks that I was talking about in terms of slowing down the heart rate nor does it have the same GI side effects that bedevil some people with cholinesterase inhibitors. Something that we run into with memantine is that it can be associated with dizziness. So if someone is having gait difficulty or falling it can be a medication to think about whether that's contributing. In some people it can have this worsening of agitation. It's been reported in some patients with Alzheimer's that when they go on memantine that it can have some worsening of those behaviors. So that's obviously not a good thing in those contexts. So, yeah. I think it's a different mechanism of action. It's something that is useful because it's approved in the more progressive forms of dementia. It can be used in combination with a cholinesterase inhibitor, but similar to the first class that we were talking about, it doesn't have a huge - we have to be, I think, modest in sharing what benefit people may have by going on it.

**John Bellone** 18:22
There's also Namzaric which combines donepezil cholinesterase inhibitor and memantine. I'm curious about your thoughts on that approach.

**Matthew Growdon** 18:32
I've actually never prescribed Namzaric, but, like you said, it is a combo of donepezil and memantine. Combo pills, in general, the benefit is from an adherence standpoint. It may be easier to keep track - many of these patients, as we talked about in the last podcast, are taking many medications. So they have this quandary of polypharmacy, which just from a logistical standpoint means that they may be managing a lot of pills and a lot of pill bottles. So it may be easier to have them combined into one so it's just one fewer pill to take. But I think the downsides are that these brand name medications are often much more expensive depending on the given individual's formulary and insurance. Some areas may not even cover it depending on what their formulary looks like because they say, "Hey, it's much cheaper to prescribe the generic donepezil and generic memantine than to give you this combo one."

Then the other thing, this is a bit of a theme you're getting from me, but if you combine things into one pill, it can be harder sometimes to disentangle if you have a side effect. Let's say you were worried about what is happening because of the cholinesterase inhibitor or someone is having too much bradycardia or slow heart rate and you want to stop that, you have to switch back to splitting them into two separate pills because you want to stop half of it. You can't just cut the pill in half or something. If you're tapering things or changing medications that can present its

own challenge. So, for the right person, I think if insurance covers it and it's very burdensome to take and keep track of pills, it might be helpful.

**Ryan Van Patten** 20:13

You talked about how memantine is often used in moderate to severe dementia. My understanding is that the first line treatment is typically cholinesterase inhibitors. I'm wondering, when you're going through the thought process of prescribing one or both of these medications to a particular patient, what are you thinking about? As we mentioned memantine [is] more often [used] in moderate to severe [cases], but they have different side effect profiles, different mechanisms of action. Potentially the biggest memory benefit could be from both, but then there's more side effects. Anything else to say about comparing them side by side?

**Matthew Growdon** 20:48

I think that those are the most important things. Having a good sense of the staging of dementia, and there are different tools and systems that people use, but that may be reliant on a combination of the latest neuropsych testing and sometimes that's as simple as looking like the MoCA or the Mini Mental. Keeping a track of people's cognitive deficits, if they're continuing to decline or if they've stabilized on the prior regimen. And then talking a lot with patients and family and caregivers about their function day to day to get a sense of how functional impairments are progressing. I think a lot comes down to who the patient is and the family, whether they are really looking for something else and trying to make a change, and that might precipitate a change. In the more frail population, as you alluded to, or people who have more multimorbidity, I think, attending to those side effects. So sometimes that is the matter of stopping a medication or tapering one to a lower dose. In general, with these CNS active medications, it's important to think about tapering them if you're going to stop them and with the cholinesterase inhibitors in particular, it's recommended that you don't just take someone off cold turkey. So that's something to know about because they can have a rebound or worsening of symptoms if you do that. So you can think about doing it over a couple of weeks with the cholinesterase inhibitors.

**Ryan Van Patten** 22:24

That makes sense. We've been talking mostly about Alzheimer's disease dementia thus far, but Parkinson's disease dementia, dementia with Lewy bodies, vascular dementia all can come with a cholinergic deficit as well. There's at least some evidence I've seen for cholinesterase inhibitors for cognitive functioning in these conditions, but Parkinson's and Lewy body come with this risk of extrapyramidal motor side effects with those medications. I believe rivastigmine is one cholinesterase inhibitor that is FDA approved for PD dementia. But, just generally,

how do you think about prescribing the cholinesterase inhibitors in the non-Alzheimer's disease dementias?

**Matthew Growdon** 23:06
I think this definitely is a great question and one also to share with behavioral neurologists, dementia specialists and geripsychiatrists that you may have on the show. In general, like you said, the indications and the studies have been done mostly for the cholinesterase inhibitors on patients that were felt to have underlying Alzheimer's disease type pathology - so plaques and tangles, the hallmarks of Alzheimer's disease. But certainly with Lewy body dementia, there's a feeling that these can offer a benefit, in some ways actually potentially more so than even in Alzheimer's. I do see people prescribing them in Lewy body for sure.

Another important detail that has become more prominent over the years is that many patients have a mixed pathology. So, for example, Alzheimer's disease and vascular dementia often are found together under the microscope in autopsy studies. So many experts and neurologists that I've learned from or practiced nearby, will often give trials of cholinesterase inhibitors in some of these other subtypes of dementia, including vascular dementia and Parkinson's disease dementia, as you mentioned. There's less consensus of whether there would be a benefit in some of the more rare diseases like frontotemporal dementia. My understanding is that there's less benefit and are not often used in that case. I think, in general, in Alzheimer's and Lewy Body dementias where there may be a mixed picture like mixed vascular and Alzheimer's picture it's reasonable and many people will give a trial of the cholinesterase inhibitors.

**Ryan Van Patten** 24:49
What about you and your fellow prescribers for mild cognitive impairment? Do you frequently prescribe these? Do you consider them?

**Matthew Growdon** 24:57
My understanding is that there's not great data, unfortunately, that the prescription of cholinesterase inhibitors in mild cognitive impairment prevents - you know, the goal would be to prevent progression from MCI to full blown dementia syndrome.

So my understanding is that that is not borne out. We can't prescribe someone the cholinesterase inhibitor and say this is going to prevent the decline that we're worried about. I don't prescribe it much in this area, personally. I think from an expert opinion standpoint and the guidelines that there are people who certainly will. If you have a patient who has very mild, like a mild cognitive impairment with an amnestic picture, that the memory is really bothersome to them, it's not

unreasonable to trial the cholinesterase inhibitor. But it's important, again, to make sure that the purported benefits in that case are outweighing and making sure that they don't have any of those troublesome side effects, because you certainly wouldn't want to precipitate problems for them if there's not a potentially huge benefit. So, yeah, I don't have much experience. I tend to follow the data on that one, but there's not, unfortunately, a strong benefit.

**Ryan Van Patten** 26:10

That makes sense. One space where I could see them being useful in MCI is - you know, MCI is a continuum, just like all these other stages. So if the patient is at the more severe end of the MCI continuum, then it resembles dementia in some way. The big difference between MCI and dementia is the lack of functional capacity with activities of daily living, [and] sometimes we just don't get great information there. So we're maybe conservatively diagnosing MCI, but we might think, especially like you said if it's amnestic, we might think that medication could help. It sounds like some prescribers will trial medications in MCI in certain circumstances.

**Matthew Growdon** 26:52

Definitely. I think particularly if you're in a memory clinic or dementia specialty clinic where there's a lot of knowledge, like you're saying, of this kind of deep phenotyping - because, like you said, these categories are very challenging clinically and they rely on the fidelity of the data that is going into them. Like you said, how good was the neuropsych testing? Do you have an informant? Do you have a trustworthy way of getting functional status? Just to reframe what you're saying, being humbled to the categories that we think we have people in whether it's MCI versus mild dementia. I think those things are getting much better, and you may have had other people on your podcast who know much more about this. But the diagnostic acumen that we have with dementia in terms of advanced imaging, even lumbar puncture and CSF studies, to really get a sense of what the underlying pathology is, all of that is changing very rapidly. So some of the information I've shared today may be outdated as the research comes in. But, yeah, I think it certainly is the practice that some people will use it as a trial in MCI.

The other thing, I would say that I'm not an expert on this, but my sense is that our understanding of MCI is also changing in that there are many subtypes of it and that sometimes it reverts in a large number of people without any treatment. That's great news and it's something that we flagged. We were worried about your cognition, but then over the years, thankfully, [you] don't convert to having Alzheimer's or some other form of dementia. So kind of like pre-diabetes. The diagnosis in and of itself doesn't mean, it's not a given that you're going to develop dementia.

**John Bellone** 28:43

Do you recommend that neuropsychologists discuss these medications with patients and prescribing clinicians? And, if so, how might we approach this in our report recommendations and our feedback sessions?

**Matthew Growdon** 28:57

I'll share, similarly to what I did last time we had the chance to speak, which is that I definitely hope that you and your colleagues will raise these issues up for treating clinicians. Just in the way that we were talking about looking for medications that could be causing harm to cognition, if someone is showing deficits in their neuropsych testing and [in] your interview with them [they] indicate to you that they may be a candidate for something like a cholinesterase inhibitor, and that's not on the radar yet, to bring that up in a pithy follow-up section that is separate from the very thorough notes that you guys write so it doesn't get lost. I think the other thing that can be helpful is assessing, if you have time during your evaluation, what the patient's or patient-plus-family's perspective is? Are they looking to try something? Have they tried something? Some of those factors are actually the most important and can be time consuming [to] get [an] understanding where they are on that journey. So I think it's definitely something worth discussing with your patients and clinicians.

**John Bellone** 30:10

I'm curious what your thoughts are on the controversy surrounding the FDA approval of aducanumab, Aduhelm.

**Matthew Growdon** 30:22

We, in my division, in the circles I'm often in conversations with, this has come up so much. I mean, it's actually kind of tapered off a little bit but, initially, it was just like we couldn't make it within a sentence or two without mentioning aducanumab. [laughs] I guess I should choose my words carefully, but I worried a lot with the approval of aducanumab that it was approved based on not the highest sterling or gold standard evidence. At the time when it was approved, we didn't even have published trials out there to really look at and dissect. It might have been motivated by a really strong desire to get a medication out there, but also recognizing that there's a huge market so there's potentially some financial incentives to get something improved. The thing I'm worried about is that it doesn't necessarily seem to provide very much benefit, even though it is in this new class of anti-amyloid antibodies that theoretically should have more of a disease modifying effects and have shown a lot of promise in mouse models, in the "mouse-zheimer's" that you may have talked to people about. The thing I was most worried about with the

aducanumab was that the trials showed actually a fair bit of side effects. And not like minor side effects, but things where people needed to be monitored very closely for brain swelling related to the anti-amyloid therapy. That's all well and good if you have someone in the trial protocol where you know and are watching them carefully, and you can make sure they come back for their MRI scans. But it scares me potentially in the leap into the real world that we just need to make sure that if these things are being used by a large number of people that there are really good systems to catch them and to make sure that they're getting the requisite follow up.

Then the other thing, and this is true of the aducanumab controversy as well as many drugs as they're approved, is that the sorts of patients who are involved in the trials are often quite different from the real world. A great example is this exclusion, for example, of people taking anticoagulants in the trial. So anticoagulants are extremely common in older adults and they would raise your risk of brain bleeding or swelling related to bleeding. So then to approve it across a wide indication of people who may be on those blood thinners, we don't really know in the real world what that's going to lead to and how we're going to manage the potential side effects of that. So, I think I said I'd choose my words carefully because there's obviously decades and decades of research and people much smarter than I am who thought about the amyloid hypothesis, but it really hasn't felt like aducanumab is a slam dunk. And, if anything, it's just maybe the very beginning of looking at this class of medications. I'm very happy that Medicare is basically linking coverage, it seems, to more discovery of whether it works in people who are enrolling in trials so that we can really get an answer. I think ethically we owe it to our patients to really only give them this medication if it is going to provide a true benefit.

The last thing is obviously a societal perspective, which is that it is a very expensive medication. It should be a really good medication from a purely financial standpoint if you were like, "Oh my gosh, we're going to really raid the piggy bank and everyone who pays taxes into Medicare is going to bear the burden of this." I think making that financial case and setting the price appropriately is for experts other than [me]. But, in general, I was not blown away and I'm pretty cautious. And happy, like I said, that we're going to get more more answers, hopefully.

**John Bellone** 34:16

That's all very consistent with what I've read and heard. I'm hopeful that, at some point in the near future, we'll have some better treatment for people going through cognitive decline. I really appreciate your overview of the different agents that we have now. It's been really informative and interesting.

**Matthew Growdon** 34:36

Great. Well, yeah. It's been a pleasure and, like you said, hopefully there will be more updates in the future of the pharmacotherapy in this area to provide a benefit for our patients.

**Ryan Van Patten** 34:47

Well, whenever there's a cure for Alzheimer's disease, we'll have you on to break the news. [laughs]

**Matthew Growdon** 34:52

[laughs] Yeah, yeah. I hope it's helpful for your listeners and keeps us posted.

**Ryan Van Patten** 34:58

All right.

**Transition Music** 34:58

**John Bellone** 35:02

Well, that does it for our conversation with Matthew. We hope you enjoyed it as much as we did. And, as always, thanks so much for listening, and join us next time as we continue to navigate the brain and behavior.

**Exit Music** 35:14

**John Bellone** 35:37

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**Ryan Van Patten** 35:49

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