Exosomes and Autism: What I Saw in 20 Patients Transcript

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Podcast Introduction and Exosome Training

Hey there, welcome to the Recharged Biomedical Podcast. I'm Dr. Edward Park, and if you're curious about regenerative medicine, you've come to The Right Place.

We're diving into the latest breakthroughs in telomeres activation, stem cell exosomes, all the cutting edge science that's shaping the future of healing and longevity.

Let's get started. Welcome, I am Dr Ed Park and I created an online training course based on the use of exasomes. How many of you have heard of Exosome?

Yay! So Dr. Arman is a esteemed colleague and he decided to take a leap of faith, took the course, and is an expert in internal medicine. His son is also affected by autism and has been experimenting and with great success the use of exosomes in his treatment population.

So explain why this works, whether it's something good for you. If you just want to learn how to treat your your mom's knees or your husband's shoulder.

I will give you the online training course here. But thank you for coming, and I'd like to start with a giveaway. This book I wrote about exosomes is free to the telepaths in the audience.

Dr. Nikagosha is an expert, by the way. He speaks at iLADS, MAPS. It's a chronic feature, even before it was the medical, what was it? Association of Pediatric Special Needs, yeah.

So he's Marquis name. And he is gonna teach you a lot, taught me a lots. For the Telepath, can anyone say where Dr Nikogoshan was born?

Speaker Introductions and Giveaway

No, no, we'll start with an easier one. Where was I born? Korea? No. Good guess. All right. The telepathy is low here. So mine is a Commonwealth country and Armens is?

A country that doesn't exist anymore. A Country that no longer exists. That's the clue. Shout it out. Good guess. They also called themselves sis for a while, right?

Yeah. Yeah, for little while. Well, okay. A lot of gold medals in the Olympics. Gold medals, a lot strong female athletes. Anyone? Bueller? No one. All right.

I'm a Commonwealth country, not so far from the US. There you go, Canada! Yes, I was born in Canada. Dr. Song, welcome! And no one's got the CIS, also known as…

What? Basilicas, onion-shaped domes. Big bears. Lots of snow. Yes. Snows. Residents of bushy eyebrows? Bushy eyebrows. Soviet Union. Close, but it's Russia.

Russia! Well, it was Soviet union back then. Alisa already has a book, so that's okay. There you go. All right. Without further ado, the brilliant and compassionate Dr.

Adamin Nikogoshian. Hello, everyone. Thank you for coming. So today we're going to talk about the use of MSC exosomes in the treatment of autism. This is something I've been doing over the past year and a half.

We're gonna kind of, well, let me go into the disclosures. I have a revenue sharing agreement with Recharged

Biomedical. More importantly, MSc exo zones are not non-FDA approved.

And the usage I'm talking about today is off label. Important to remember. So our objectives today, we're going to start off talking about what are exosomes.

We're gonna kind of dive a little bit into what they are, how they work, and what their benefits are over stem cells, over mesenchymal stem cell.

What Exosomes Are and How They Work

That's a common question that people have. In that vein, you'll learn about microRNA. that's an important aspect of their function. so we are going dive into a bit what micro RNA is and how it's distinguished from other forms of RNA and why it is so important.

We'll go into ASD. I'm sure many of you know about ASC and we'll kind of talk about really more of the, I would say, medical side of it in terms of neuro physiology and neuropathology that's going on and how things can help with that.

And then most importantly, how do exosomes help in ASDC? And there's a case series that I put together over the past year and will kind go in to what those results have been.

So what are MSCs? To know what exosomes are, we first have to know, what mesenchymal stem cells are or MSC's. So mesenchymal stem cell are their adult or perinatal stem, cells that are found in a variety of tissues, got some of the tissues listed there.

They self-renew, they differentiate into other cell types, and they actively secrete signaling molecules. Those signaling, molecules are the exo-zones.

And that those ex-osome are where a lot of, the clinical benefit that we'll see, The specific type of exosomes that I use in my patients come from placentas.

So there's differing opinions on which different sources to use. I like placentes, but there is a variety of sources. Some of the MSC functions, they will respond to inflammation and injury and they'll promote tissue repair and the immunomodulatory function really in the long term from my experience with it really is some of long-term benefit that you see.

So, a common misconception of MSCs is a lot of people think there's cell engraftment that happens, you know, the stem cell comes in there and turns into a neuron or something like that.

There might be some of that, but that's a minimal contribution overall. Really, it's the paracrine action, which is it is the exosomes that they produce, those signaling molecules where a of the benefit is coming from.

And the pericrine is important because it localized effect. So you really got to get the cells to the area that you want to help or heal. So exosomes are, micro vesicles are very, very small, 30 to 150 nanometers.

So we use that to our benefit. It's something that size can fairly readily cross the blood brain barrier. As I mentioned, they're harvested from mesenchymal stem cells and the exo zones just like the mesenchymel stem cell are paracrine effectors.

So the effect has to be localized. So, the contents are delivered directly into the target cells that you're looking at. Once they're in that target cell, they will mediate intracellular communication regulation and then they modulate the gene expression and the signaling pathways.

That's where the microRNA comes in. So the exosome has what's called an exozoom cargo. And because the x-some itself is just a very, very small little lipid bilayer ball.

The exozome cargo that comes with like placental mesocompostome cells will have different neurotrophic factors such as cytokines, sphingomyelin, BDNF.

But really a big therapeutic role is the microrna that's contained in there. So what is microRNA? As the name implies, it's a very small segment of RNA.

It's about 20 to 22 nucleotides. That's tiny in the world of of rRNA. But these little small nucleotype sequences have vast network effects throughout the system.

So they're non-coding RNAs. What they do is they regulate the gene expression after the transcription. They're post-transcriptional and they'll either turn on or suppress target messenger RNAs.

The picture I have here is like a light switch versus one of these sound boards like the AV guys in the back have over there. That's really the way I look at it.

A lot of things we tend to think of as on or off. The microRNAs work. They're really orchestrating it, they're modulating, fine-tuning this gene expression.

Single micro RNA can impact multiple gene locations and then one single gene location can have impact from multiple microRNAs. So if you do the math on that, you can see that there's just a lot of possibilities that can happen with that.

Why they matter biologically? is really about the link between heredity and environment. That's what I view as their primary function. They've classified over 2,000 microRNAs so far that they've been able to classify that are encoded.

And they regulate at least about a third of the human genome. So there's fine-tuning, there is network control, and there The microRNAs, the fine-tuning, they're not really silencing or turning it on.

Think of gene expression as being noisy. It's turning the noise off. it's cleaning up that gene-expression to make it more of a pertinent or targeted signal.

The network control, again, one microRNA can target multiple genes in the same pathway. And then that same transcript can actually be hit by multiple micro RNAs.

So it makes it complex in terms of, it's not like you can make a spreadsheet and say, this does this, you know, and so on. But we do get some ideas in term of how they work and then I'll get into that a little bit later.

And then there's timing. The timing is important in terms of gene expression. You know, you may have heard these sort of remarks about epigenetics where your epi genetics can be, a certain expression of breakfast and then be changed by lunchtime, depending on what happened throughout your day.

So, and that same timing as important, in particular with neurodevelopment. And that's why they play such a large role in those first couple of years where we see a lot of brain development happening.

So the therapeutic potential of microRNAs, and these are some ideas in terms of how they could work. I say could, work because this is something they're still working out.

There's a lot of literature on micro RNAs but there's still a lots to be done. In terms autism specifically, I think pathway level correction is really where it's at with the micro-RNA because it You know, it's not really doing like a single microRNA to single gene.

It's really hitting multiple downstream genes at once. So when you're looking at a disorder, this polygenic autism, that's exactly the kind of thing you want to do because it is hard to hit the individual targets if you have dozens and dozens or even hundreds of them.

But the microRNA also has mimics where it can actually restore, there's protective micro RNAs that need to be restored if they've been turned off. There's micro-RNA inhibitors and they call them entagomeres.

And these are antisense neutralizers. If you have a microRNA that's being overexpressed, this is gonna tone it down and turn it.

MicroRNA Basics and Therapeutic Potential

Then precision subgrouping, basically where you're subgrouping the disorder that you are talking about and then trying to find a microRNA sort of cocktail, I suppose, or a sequence that could help with that.

And again, in autism, that's very pertinent because autism although it's one monolithic diagnosis, it really is a lot of sub-groups in there. Dozens and dozens of subs groups.

So, with, here's a little bit of biochem here, it won't be a whole ton of it, I promise. So with microRNA, when they make it it's, there's precursor micro RNA and then there are some enzymes.

There's particular one called dicer, which makes it into mature micro-RNA. One of the ways that we found out the impact of micro rna in the exosomes was they created these mice called Dicer Argonaut 2 Knockdown Mice, where they essentially these poor mice, they impaired their microRNA function.

And then they harvested exosomes from them. Then they gave the normal mice exo zones to diseased mice. They gave them the knockdown exozones to disease mice that did not contain any micro RNA and they saw very little benefit from the exotomes that were given to those mice from knock down mice So that's kind of where the idea is where they microRNA has such a strong impact on the therapeutic effect.

So these are some micro RNAs that are listed in Some of these are the ones that we suspect may be having their impact in autism like microRNA 124 is involved in reducing neuroinflammation and it helps with neuro regeneration.

And all of them basically have to do with remodeling of the brain and then 181 reduces intestinal inflammation and permeability. So this is a field I think that is really has a lot of potential.

It's a whole new kind of world therapeutics that you can get into once we sort of learn more about how they're turned on and off. And right now, I'm using the exosomes as a delivery for these things.

I think an important point to also understand about it is, again, that these therapeutic effects are driven by what we call signatures, microRNA signatures.

So think of it as several, a dozen, possibly even hundreds, rather than, you know, 124 does this and 155 does. So they come in these sort of groups. And that's, I think what's made deciphering some of their effects a little bit more difficult.

So let's talk a little bit about autism. The DSM-IV has their diagnostic criteria. They have two domains, social communication and restricted repetitive behaviors.

And, you know, kids meet these criteria and they're diagnosed with autism Again, I do more of a biomedical approach with it, and this is certainly accurate, but this looking at it more from a subjective lens.

I think objectively, there's a lot of ways to diagnose it as well, just biochemically from looking their immune function and glutathione and methylation, lots of things.

Boys are about four times more commonly affected than girls. There are racial disparities, although that has been narrowing over the years. Prevalence has just exploded, as I'm sure most of you know.

It's now 1 in 31, which is 3 points. Back in 1980, it was 1 and 2,500. So we saw prevalence rise from 0.04% to

3.2% in 40 years. I mean, if that's not an epidemic, I don't know what is.

I know I've heard the arguments about better detection and how they broaden the diagnosis. That does have some role in that, but there's no doubt that there has been a large increase in these kids.

Just to be clear when I'm talking about autism, the kids in my clinic are all These are severe level three non-speakers that is severely disabled. I'm not disparaging people at the other end of the spectrum.

They have their own challenges, but that's not really who I am talking about. Talking about the kids that have severe disability from it. So there is a genetic component.

About 10% of autism cases are considered syndromic. And this one, it's pretty clear the cause is genetic. There are environmental factors with everything, of course, but a lion's share, a majority of the causes are genetic, and there's various distinct genetic syndroms that are associated with this.

About 90% of autism is non-syndromic, but that doesn't mean there's not a genetic component to it. There is. there is a genetics susceptibility for sure, But the environmental factors play a much larger role in these kids, which is the majority of the kids we're talking about.

But there has been about a little over a hundred genetic variants that have been associated with autism susceptability. Some of the common ones that I've seen are CHD8.

I won't go through all of them, but I can tell you, shank two very severe kids when I find them with that. CNT-NAP2 is present in almost every kid that i do genetic testing on.

Very, very common. And you can see that they're mostly involved in chromatin, synapse formation, neurolignans, postsynaptic scaffolding, BDNF. So essentially it's how the brain Puts together its structure during those kind of critical developmental years and that that's where the changes occur So environmental triggers this this is a big topic you could do a whole talk on this do whole conference on This I love this slide from the McCullough Foundation just came up in the last year because it was the first slide I saw that prominently featured vaccines on it.

Most of them didn't. Vaccines are not the only cause, of course, but there is a connection. And from what I've seen in my clinical experience, I mean, they make up about 50% of the known triggers.

I think that's accurate. But again, I also have kids in my clinic with severe autism that have never had a vaccine touch them. So there are absolutely other causes.

Some other notable triggers are in utero pharmaceutical exposure, environmental toxicant exposure. Eternal immune activation actually is a very large one.

And actually that's not really separate. That actually I think plays, it's a background factor. It can be a separate thing, but it also can't be background with all the other ones.

I actually think that is quite an important one And then of course, any sort of perinatal complications, prematurity certainly increases those risks. At the end of it though, when it's all said and done, the sequelae of these triggers really boils down to two big things with our kids, which is chronic neuroinflammation and immune dysfunction.

And this drives so much of the disease burden that we see with autism. So some of the neurodevelopmental effects, as I mentioned, you have the genetic susceptibility, the environmental trigger.

This leads to the chronic neuroinflammation and the immune dysfunction, which then leads the disruption of brain development and function. The developmental disruptions, they produce the structural and functional connectivity differences.

Autism Overview and Biological Subtypes

And some these differences can be seen actually on neuroimaging, but a lot of them really are at really a more granular level than that. So a lot of them you really can't see on an MRI or even sometimes a functional MRI.

So you have aberrant neurogenesis and neuronal migration. The neurons aren't positioned the way they should. You get synaptic defects. the synoptic connections are being disrupted in different ways.

EI imbalance, it means excitation inhibition that essentially has to do with glutamate and GABA balance. Most of our kids are very far on the glutamic side and not nearly enough on The GAB side.

This is why a lot of them are stuck in fight or flight all the time. When you have too much glutamate, it's a good thing for observation and whatnot, but too of a thing can actually destroy the synapse in the neuron.

So that also leads to axon and myelin dysfunction, which that impairs signal transmission and you'll see white matter connectivity in different brain regions.

And that connectivity is really what in the, once it's sort of established, it said those differences in connectivity that create so much of the kind of classic things that you'll see that a lot of autistic kids will exhibit and ultra dendritic growth as well.

So the immune dysregulation, which is the other piece, a lot of our kids, they lose their immune tolerance. They lose that self, non-self discrimination, and then they get an immune activation that's aberrant.

And, you know, I get this kind of question, is their system underactive, overactive? Well, there's aspects that are under and aspects over, it's just sort of confused.

That's really the best way to put it because it is not really exclusively under or over. The T and B reg cells, those are decreased. Those regs cells are what help that immune tolerance.

and help suppress excessive immune responses. CD4 T cells, those tend to be increased or hyper responsive. So you'll get this exaggerated adaptive immune response, which essentially turns to the inflammation never really turning off.

I see a lot of autoantibodies, particularly folate receptor auto-antiblodies. There are other auto antibodies as well. That's probably one of the most common ones.

They can help you when you're working with the child as far as helping to identify the sort of subtype. The subtypes aren't official, like there's not some diagnostic criteria for them, but they are a very practical thing in terms of how you approach the treatment with a child.

And then you get immunoglobulin shifts. You'll see a lot of immunoglobulins deficiency, particularly IgG subclasses, and also IgA and other types of immune globulas.

So the neuroinflammation is pivotal driver for that pathology. The immune dysfunction sets up the chronic inflammation, and then it gets settled into the neural inflammation.

And you get these pro-inflammatory activated microglia and astrocytes, or M1 and A1. They continually release these prone inflammatory cytokines, which then become neurotoxic.

But they create oxidative stress. These are predominant in autistic brain tissue. What we want to see is more of an anti-inflammatory picture. And usually there's a balance between this.

So we wanted that microglia and astrocyte to go into their neuroprotective and supportive roles, which would be your M2 and A2. These actually release antiinflammatories factors rather than being neurotoxic.

They are repairing tissue and remodeling tissue, and this is deficient. That balance is critical for any CNS homeostasis and restoring that balance, that's the brass ring in treatment.

If you can do that, the kid's gonna do a lot better. Some comorbidities that we see in autism, gut brain access, I mean, very high prevalence of GI symptoms in autistic kids, well over 70. It's even higher actually in your level three non-speakers, it's upwards of 80 to 90%. Gut dysbiosis, it's a lot more complex than this, but basically you get the high firmicutes, low bacteroides, overall low diversity.

But there's many, many sub-variants of this and Prevotella plays a role. There's, there is a whole lot going on there. You'll get intestinal barrier dysfunction or leaky gut as it is known.

And this persists well beyond the times when, you know, ther- there are times in development where that can actually be beneficial for training the immune system.

We're talking about this persisting well, beyond that time. That bi-directional gut-brain communication along the vagal nerve or microbial metabolites, immune complexes, there's a lot of that going on.

So a lots of times, if you want to get better behavior, you get their gut under control, and you see behavior improvements. Maybe not all the way to where you need them, but that's usually the foundation you start with.

Mitochondrial dysfunction, very prevalent as well, 30 to 50%. Again, in the severe subgroups, it's well over 50% impaired energy production and high demand tissues.

I'm sure many of you've seen talks on like cell danger response and the mitochondria essentially gets stuck in this inflammation producing state rather than an energy producing stage.

So another strategy is to try to get that to go back to producing energy. There can be carnitine metabolism abnormalities, and these are things you can measure.

You can them on organic acid tests, acylcarnitin profiles, so you don't have to guess on these things. you could get definitive lab results to see these.

Redox imbalance, reduced glutathione. If you ask me, what's the one thing to use to diagnose autism medically? low glutathione. It's just across the board.

In the Level 3 kids, in 12 years of doing this, I haven't seen an exception yet. They all have low gluteathion. Some have extremely low, some have mildly low but they're all depressed in their glutothione levels.

And glutathione is important. It's important for lots of things, not just for detox, but just maintaining that redox balance. Increased oxidative damage markers.

And these are things you can get on LabCorp requests. There's GFAP, NFL, NSC. So you don't necessarily have to use this. Especially labs can give you a little bit more in-depth look, But these things that are readily available through insurance.

Impaired methylation, transulphuration, and it creates this vicious cycle of neuroinflammation and cell injury that just keeps going and keeps goin', and you gotta break that vicious circle.

Folate metabolism, which, it's folate-metabolism methylation. A lot of our kids are poor methylaters. The foliate receptor autoantibodies don't help any, so restoring that can be important.

Some other common comorbidities, there's two that I underlined here. The reason I underlying them is they're classified as comorbitities because they are all seen as different diagnoses.

But these are, this is the core neurological problem here, is apraxia motor planning and sensory processing

disorder. To do a motor action, you need, it's a Motor Sensory Loop.

So if your sensory is off, which makes the motor off. That's the, that's core issue why so many of our kids are unable to speak. It's not a language issue.

they can't make the words. They have language, they have very in-depth language. If you find alternative ways of communicating like keyboarding or spelling or anything, They're very eloquent, and they write books and poems and all sorts of stuff, but they're stuck.

they are trapped in a body that doesn't listen to them. It's a comorbidity, but really it's the core neurological problem is what we're talking about there.

Sympathetic overdrive, this is very prevalent. Again, greater than 40% in the severe kids, it is 99%. It is all of them. They're stuck in fight or flight almost all the time.

And that's just how they live their lives. they think that's how everybody feels. I mean, they're just so used to it.

Triggers, Neuroinflammation, and Immune Dysfunction

So, sleep disorders, you know, which ties in with sympathetic overdrive. Seizure is very common, 25 to 30 percent lifetime prevalence, again, higher in the severe ones.

And then Pans-Pandas is commonly present as well. This is a challenging diagnosis to make in a severe autistic kid because there's a lot of overlap in symptoms.

You know, it's a clinical diagnosis, pants, pandas, but it is important because there are some key triggers involved there that until you address those, you're going to hit a ceiling on how far you are going get with these kids.

So the neuropathology, I've got this busy slide here, but again, it's just to reiterate, there's the M1. We want to shift it to the N2. So they're stuck in the one.

You lose the immune tolerance. The neuroinflammatory signaling is basically turned on. What does that all mean? In essence, it's neuronal network instability.

The network is unstable. And to stabilize the network, if you can stabilize a network you get better function. That's really what the goal is. So exosomes, where they come in, is the immunomodulation.

They can favor some of that polarization shift to that anti-inflammatory M2A2 state, which then promotes the tissue repair. It can promote immune tolerance through various neurotrophic factors, inhibit T-cell proliferation.

it will increase the Treg and Breg production so there's better tolerance. There's direct anti-inflammatory effects and tissue repair factors that are in the exosome cargo.

And then neuroregeneration, which again, I feel this is the brass ring with the Exosomes treatment in, in patients I've used it in. I have a limited toolbox for neuro regeneration as far as what I'm able to use.

So this where it's helped me a lot as, as the enhancing neuronal survival, improving the synaptic plasticity and the axonal regeneration. And there's some other things that can be done.

And again, I think anytime you're talking about neuro regeneration, it's important to note that, you know, your only going to wire what you are doing, right?

So if you doing all this stuff on a kid and he's on his tablet all day watching YouTube, that's what he is going wire. So, its important that you talk to the parents and you tell them that they have to engage them in the things you want them to wired as best you can as you do these sort of treatments.

So this, so the, yeah, actually the most important thing exosomes do is they turn the brain from red to blue. So that's the key aspect. It doesn't really do that.

I'm kidding. But the so this graphic kind of shows the autistic brain sort of inflamed. And then after post-exosome signaling, it's sort, this is a good sort summary slide where it shows different neurotrophic factors that change.

And on the bottom there, it has some of, let's see if I can do this. Yeah. You know, neuronal survival, synaptic plasticity, neural circuit, homeostasis.

So these are all the things that we're after with the treatment. So what do we have with studies with this? With exosomes and autism, there are no human studies.

I've looked all over the place. There's nothing there. in autism. So we have those. We know that the mesenchymal stem cells have a similar method of action like the exosomes do.

In the animal studies that they did, they had multiple studies and they used intranasal exasomes and showed improved social interaction, improved cognition, reduced repetitive behaviors.

The studies also attributed these results to the microRNA and the anti-inflammatory agents that were in the exosome cargo. And they did it via modulating the immune response and neural network functionality.

They saw it reduced neuroinflammation, increased synaptic capacity, enhanced neuronal survival and improved immunoregulation. So these were the things that when you look at the science, that's what we sort of predicted and that we saw in The stem cell studies that were out there, there's a lot of them actually, they were multiple small studies.

There's no robust like randomized control trials and they didn't exactly clarify the role of MSC exosomes in there exactly, but the exo zones are what's coming off of the stem cells.

So we're kind of using that in lack of that. This is one of things that I'm hoping to put something together as far as that because I have the patience for it and I think this would be something that would really be important to include in the literature.

So this is the case series that I did. This was from August 2025 to June 26. It was 46 exosome treatments that were done intranasally on 20 patients. The exozomes we used were placental derived.

They were screened, actually exceeding the levels of screening used by standard tissue banks. The placental source was pre-COVID. the sample tissue was collected in 2019, so COVID doesn't play a role in it.

It's a sterile preparation. And we determined the potency based on sort of age and weight. Ranged from one milliliter, which was three trillion microvesicles, all the way up to five milliliters, Which was 15 trillion, micro vesicles.

What I have pictured there is a nasal dart. It's basically an intranasal mucosal atomization device. So it sort of atomizes the fluid into a fine mist because the idea is that you're putting it into the sinuses here.

The exosomes are traveling through the cribriform plate. and then they should have about 80% penetration into the brain space. There was a study done in Israel where they radio tagged it and they used the same technique and got about eighty percent up there.

So it's actually superior to doing it IV, but not as good as if you did it intrathecally. But intratecaly in autism is a logistical nightmare. So that would be very difficult to do without knocking the kid out.

This is a painless method. There is no pain. The primary barrier really is overcoming the kids sympathetic overdrive because they're scared. And so it can be something as short as a five minute procedure.

It can take as long as 30 to 40 minutes. We tend to alternate nostrils. Getting them to calm down. I use a red light therapy while I'm doing it right on the cerebellum.

And I always put the kids in a hyperbaric chamber right afterwards. So we, the kids were 100% of the, uh, kids where level three autism. They all had varying degrees of global apraxia.

Uh, they were, none of them had speech. There were non-speakers, middle speakers or unreliable speakers, but none them, speech was not their primary method of communication.

It was two girls or two females and 18 males. And the age range was three to 23 years old. All 20 patients did report varying degrees of improvement in motor planning, regulation, attention span, and communication.

And communication was primarily on the younger end, of course. We did have three patients that had minimal to no improvement. Interestingly, this was, they actually did improve on first and second treatments.

By the third or fourth treatment, They actually seemed to plateau. They didn't come back. All of them were 19, 21, 23. So they're at the older end of the spectrum here.

So, again, the key improvements, most consistent improvement that I saw was improved motor planning. And again if you really look at what the neurological disability is in terms of how it's expressing, it is motor planting.

That's the issue. If that's what we're trying to get past. The secondary issue is regulation.

Gut, Mitochondria, and Methylation in Autism

That was the second most consistent thing we saw was improved autonomic regulation, they were better regulated. And again, we say improved speech in the younger end, three to 12, with again the, basically the youngest the patient was, the better the speech improvements were.

I used ATEC, The Autism Evaluation Checklist, that's, I use that a lot in my clinic. The largest drop we see on ATEK scores was 33 points. but I averaged them out by age group and it pretty much shows what you would expect.

Three to six, we saw the best results. And then it goes down seven to 12, 13 to 17, and then 18 and older. At 18 or older, on average, five point drops, which is not a big difference in A-Tech, but keep in mind that these are kids that are it's hard to move the needle in these kids.

It's to get any change. So I was happy, the parents were happy and most importantly the patient was with that. The 33 point drop was a patient that, again, when I did these, first group that I do were patients that had been seeing for years.

I had an opportunity to do all the foundational work. Fixed their gut and supported their Mito. Got there as immune modulated as possible. Some of those some of the better results were in the beginning.

Later on, as I'm doing this, we were getting new patients that were, well, I want to do this first consult. We didn't really do anything. I wanna schedule the exosomes tomorrow.

And I explained to them, oh my God, don't know if you'll get the best results, but it still can help. and we did do it and it's still did help, at that range when you haven't done the foundational work, you're looking probably at more anti-inflammatory effect.

So it did helped. you know, all the systems were still sort of dysregulated underneath, but it still was helpful. And I don't say no to any of the patients or families that want to do it, and you wanna do it in a way where you're getting the systems in line as best as possible beforehand.

As far as developmental plateau, clinical improvements were maintained even after this plateau was reached. What I mean by plateau is in terms of, we counted how many weeks the parents were reporting improvement.

So, and the exosomes last in your body about six weeks. They're gone after six week. And you can see in the three to six-year-old age group, And this is an average.

So we had some kids that were going much higher. They were improving well past 20 weeks. We're talking up to almost six months of improvement after the exosomes are long gone.

That microRNA, that regulation that they're doing, it's still working there as far as keeping things in line. And again, the older they got, less that effect was.

You know, this is something we're still working on in terms of how many treatments, how long, and how frequently. You can do the treatments every six weeks.

With stem cells, you're supposed to wait six months. This is every 6 weeks, so you certainly can't do it. I usually recommend that we wait until we feel the development is plateaued before we repeat the treatment.

There could be benefit to stacking it. These are questions I don't have answers for. This is why I'm doing these talks to get this out there. I am hoping more people can do this.

If we pool more numbers, we can get these answers. We need data, basically. This is my favorite slide. What I love about exosomes are after 46 treatments.

And I'm actually at this point now, as of the conference today, last treatment I did was on Wednesday. I am up to 88 treatments, still zero serious adverse events.

It's very, very safe. That's not to say there are no adverse reactions. We had two cases of transient hyperactivity. They both resolved in less than a week within a couple of days, and it was really, it wasn't severe.

Fairly common disease fatigue afterwards, which most parents did not view as an adverse reaction. They were okay with that, so we're not really counting that.

But again, it was transient, and it went away. There are some things in adults that are a little different, but at least in this population, those were really the only things that we saw.

So very low risk profile. Some emerging trends. You know, we saw that really the patients all reported improvement with the initial treatments. Some of the older patients, again, they kind of seemed to plateau around third or fourth treatment.

Interestingly, my son, he's 18 years old. He's a speller. Actually, had now had seven treatments and he actually didn't get a response on the third treatment, so he did nice sum of second, first, second.

Third, you didn' get anything. I did it again on fourth and fourth, fifth, sixth. I'm not even sure if that's a plateau versus I am not sure why sometimes I'll see a report or not.

Again, that is something we just need more data on. So we are continuing to go on with his and he is getting a treatment about every six to eight weeks.

Most consistent symptom improvement we saw was better motor planning, followed by improved autonomic regulation. And those are the two big things that I'm going after in all the treatments that do on my autistic kids.

We did see speech improvement. This was primarily in ages 12 and younger. we did some speech improvements in the older kids, but again, it would be speech that if someone's a parent of a non-speaker, they'd be very pleased with, But it wouldn't be a speech most people would consider you know, viable communications.

So you do still see some speech improvements in older kids, but it was mainly clustered in the younger ones. So some future directions, again, things to be determined on this or, you know, what are the optimal therapeutic interval?

Do we just do six weeks, even if they're still improving? Is that better? Does that stack it? do I wait till they plateau? I don't know. Hopefully I'll come back and tell you in a year or two.

I dunno. But these are things that I want answers for. We're going to get them hopefully soon. you know, in the older kids where they plateaued, was it a plateau?

How Exosomes May Help Autism

Was it just a variable treatment response? You know? We don't know. But overall, we definitely, you now, I think we had enough people in this where we saw the signals that this was moving them in a right direction and it was very safe treatment.

I'd think another potential future direction, this is kind of far in future, is engineered exosomes. And, yeah, the exasome is just the delivery mechanism, right?

you can put kind whatever you want in there. They are engineering them. kind of make the argument that COVID vaccine was an engineered exosome. So, you know, what you put in there, so I think there's a lot of therapeutic potential there as far as delivery.

I, think it's great delivery method. Just a quick word on regulation. There's sort of the good and the bad. I mean, on the good side, Bobby Kennedy, he's our new HHS grand poobah.

And, you know, He's definitely broadly friendlier to stem cell type regenerative therapies, which include exosomes. He is valid to end the FDA war on alternative medicine, and he has explicitly named stem-cell therapies and exasomes in this.

So he just created a more permissive enforcement environment for these regenerate interventions. The bad side of it is, there really hasn't been any clear formal alternative regulatory pathway that's been formed for these.

They haven't changed any laws. So, there's a patchwork of state laws and malpractice risks that I think if anyone that is interested in something like this, you really want to look into that beforehand to make sure.

That's where we're going. I hope he stays, but we'll see what happens. So we are Recharged Biomedical. We're actually at booth 13, not 16. we're looking out on the parking lot.

If this is something that you find interesting or you think could benefit your patients, there is an exosome master class, the clinical use of exasomes.

There's three modules on autism that I've put together and Dr. Park has put 20 modules actually on joint injections. And there's a lot of other really interventions that can be done in this outside of autism.

Autism is just sort of the area that I've been focusing on. And other than that, I'm open to questions if anybody has any questions. So thank you for that.

It was a great talk. My question is mainly around four kiddos who you are doing some diagnostic workup ahead of time. Are there certain markers that you're looking for, that would indicate that they would, besides the clinical appearance of the patient?

Are there certain markers that you would say, oh, this is a good candidate for exosomes? And then in terms of glutathione testing, you mentioned, are you doing RBC glutathiol or total glutathyone testing?

Yeah, I'll do both, actually. Usually, whichever one I look at, just look the sort of change, the interval change in it. As far as before we do the exo zones, Regardless of what the situation is, we've seen improvement.

Like if we see their inflammatory markers are really high, their NSEs high or their CRP or whatnot. Well, those kids actually we know are going to do better because the anti-inflammatory effect is pretty strong on it.

As far as that long-term effect where I showed the young kids where it's going 22, 23 weeks, that one I think is more of a neuroregenerative effect. So when I'm going for those longer term effects, ideally, I'd like to see all of that calm down.

You can make the argument that a little bit of inflammation is good as far it is supposed to.

Case Series Methods and Treatment Delivery

The exosomes seem to be drawn to it or I don't know how true or not true that is. But for me, the optimal effect would be the neuro regenerative affect.

But I will happily take the anti-inflammatory effect as well, if that's all that it's going to give me. But, I know that the neuroregenerative effect that is going have more legs, it is gonna give a lot more sustainable benefit on it.

So, due to the cost of these, this is something that you are, if resources are limited in the patient's family, you're saying, hey, we can do some other things first, or are you advocating for this relatively quickly in their presentation?

If resources limited, I don't advocate for it. I bring it up. But actually I tend to work on more things because they're not cheap. I mean, they are a lot cheaper than stem cells.

A lot cheap, but I wouldn't say they were inexpensive. But no, I would definitely focus more on just doing the groundwork. You know, balancing their functional systems and balancing nutrients, looking for auto-antibodies and put them on Leucovore and all those sort of things.

Okay. And last question, the levels of glutathione you're looking for optimally, do you have an idea? I mean, I like to see it at least over 800. Okay, for a total.

Yeah. But a lot of kids are coming in 250, 300. So sometimes I'll take 600. If they're around 200 and then get on 600, and I'm pretty happy. That's good.

Thanks. Hi, very interesting presentation. If somebody who's spent their career working with autism and Asperger's, what this presentation did for me was kind of highlight the difference of why we sort of arbitrarily split off autism aspergers.

Your presentation seemed much more like designed for Cantor's autism versus Hans Asparger Aspartger. And so the UC Davis Mind Institute that I've spent years dealing with, I don't know, have they looked into exosomes at all?

I haven't heard of that from them, like Rondi Hagerman and Bob Hendren. I'm not aware of it. I mean, I didn't see any of in the literature that I looked through.

So I don't believe so. Yeah. Thank you. Yep. childhood. Have you looked into that at all and the correlation with these children being born by c-section then having higher incidences of autism?

Well, I think the the c section babies have more higher incidence of like ear infection, probably dysbiosis, immune dysfunction. I know they did a study in China on vaginal swabbing a couple years ago where they took csection babies and they group they just delivered like normal, gave them to mom and that was that.

And then they had the group, they actually took vaginal secretions, swabbed them all over their mouth, put them in their eyes, nose, everything. Then they followed them for two years and they found they, had better immune

tolerance.

Results, Safety, and Future Directions

They had less ear infections. Their gut microbiome was, you know, quote unquote healthier. I don't know if they exactly know where normal is yet they're on that age group.

So, yeah, so, I mean, anything that's going to predispose to immune dysfunction or prime immune disfunction is going increase iris. I think there's more to the triggers than that, but certainly having that in the background, because it comes down to, you know, the straw breaks the camel's back, right?

So if that… maybe that's the straw in some kid, but yeah, I'd say keep all the straws to yourself as much as possible. I've just seen a high correlation there.

In Canada, there was a documentary done by David Suzuki called the Autism Enigma, and he was focusing just on the antibiotic use in early childhood. And I know it's not one thing, we all know that.

It is the strong that breaks. But there are many straws. Yeah. I think that's a straw. I agree. What genetic testing do you recommend for people that you suspect with ASD?

Well, I thinks it depends what you're looking for and what their finances are. If the finances aren't an issue, like the Intel X test, that expensive, but I it's good.

It's very thorough. But I also think there's also very good testing with neurobiologics, DNA mind. So I there are different options in terms of how deep that you want to look into it.

But I think that there's enough, the genes, greater than 100 genes for genetic susceptibility, they come up on so many panels. And some of it's like MTHFR.

You can get that through Quest or LabCorp, right? So some them you can even just get through insurance as well. So I I it depends on how thoroughly you want to look at that, get that list of genes so you can subtype the child.

Because that's at the end of the day, what you wanna do is you to sub-type them and you say, well, this is the Mito kid or the, you know, more of, the auto-antibody kid, because they do have sort of their biomedical classifications that fall in and then that helps sort-of direct treatment.

And do you expect to extend your work to less severe ASD patients? I'm sorry, I don't understand. With a less severe, you said you use stage three autism patients that have no speech, but do you expect to expand this study to include autism spectrum that's not as severe?

More high functioning? Yeah, possibly.

Audience Q&A on Testing and Candidate Selection

I do not have a lot of that. My clinic is all level three. Almost all the autistic kids in my clinic are non-speakers. they're all severe. So I mean, I have some that are milder, but yeah.

I wouldn't be opposed to it at all. Yeah. And just focusing now on the more severe, because I think the signals will be stronger as far as getting it.

But yeah, one day I'd love to do that. Thank you. Any other questions? If not, please join me in thanking. It was a wonderful talk. Thank you so much.

So there is another session in this room at 3.30. I just ask you kindly to collect yourselves and step out and back if that's what you need to do. Many clinicians are getting interested in exosome therapy and they hesitate for good

reason.

Questions like, does this work? What forms do I need? How much should I charge? how do i stay out of trouble? All these questions are addressed in my online course.

That's why I created it to help you get started. The online Course is your permanent turnkey resource to get start either treating yourself, friends and family or to expand your practice and help more people as well as increase your revenue.

Thanks for tuning in to the Recharge Biomedical Podcast. If today's episode got you thinking, you'll love my book, Exosomes, Songs of Healing. It's packed with cool analogies, full color illustrations, and all the science you need to understand how exosome are changing the game in aging and regenerative medicine.

Closing Remarks and Course Promotion

You can grab it in paperback, ebook, or audiobook, whatever works for you. Now head over to www.rechargebiomechanical.com to check it out. And don't forget to like and subscribe so you never miss another episode.

See you next time.