CBD/THC & Beta-Caryophyllene: What You Need To Know Transcript

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Summit Introduction and Speaker Welcome

Welcome to the Parkinson's Solutions Summit. I'm your host, Dr. Ken Sharlin. I hope you've been enjoying the many wonderful interviews we've had so far.

This happens to be your first day or the first day of the summit. Well, we've got a lot in store for you today. I'm interviewing or visiting with Colonel Dr.

Philip Blair. He's on the West Coast in North Pacific Northwest. He is a family physician and a consultant and an expert in cannabis medicine. I know he's going to tell you more about what he does and his background.

And today's presentation is a little more formal. We're going to show some slides. I know you're going to enjoy them. And then hopefully toward the end, he and I will have an opportunity for a little bit of a discussion.

So, Dr. Blair, welcome to the Parkinson's Solutions Summit and take it away. It's a pleasure and an honor to be associated with you and with this group and this summit taking place.

I've got some impressive slide presentations I'd like to share. So I want to get to that right away. So this is the title of my talk. I'm just an endocannabinoid modulation and treatment of Parkinson's disease.

I'm focusing on the endocannabinoid system as the modulator for this problem. So my history goes back to graduation from medical school. In 1978, I became a family physician.

I was a U. S. Army colonel. I retired in 1996.

Dr. Blair Background and Talk Overview

I entered a disease management program in 2001, and I learned about cannabidiol in 2014. Since then, I've been working with patients and developing other products, including B-Caryophyllene which I'll tell youabout a little bit later on.

I've also written a book, Medicinal Cannabis and CBD and Mental Health Care, and that's available as well. It's a focus on the cannabinoid therapies for treatment for mental illness and I think it's a stellar work.

It really dug me into a great deal of the science behind mental health issues and CBD as well as Caryophyllene So when we're talking about Parkinson's disease, I know there's a number of causes, but I've broken it down into these major categories of protein degradation, neuroinflammation, auto autophagy, endoplasmic reticulum, stress, and this is where the stress is most focused on, on the body and in the cells mitochondrial function, oxidative stress and exosomes.

Exosomes are kind of a peculiar thing, and I'll tell you about that a little bit later. Now first off, in terms of inflammatory modulation, one of the things that we know is the NLRP3 inflammasome seems to be a key factor for inflammation leads to Parkinson's.

I've noted a number of different studies here that the NLRP is a protein that plays a vital role in the activation of the inflammatory processes in the body and in Parkinson's.

The activation triggers neuroinflammation and a degeneration of dopamine producing neurons in the brain. And in addition, there's a cb2 receptor that is also present that has low levels of presence in the normal brain, but it becomes highly activated in a disordered brain with neurodegeneration.

Now, one of the processes is, is this molecule, this protein molecule that gets misfolded along the way. And part of that process, this is alpha synuclein.

And so and this particular molecule goes through a process of improper folding. And that that process may be generated by a glycation element. In this particular slide, I'm demonstrating the glycol defense system for methyl glycol as, um, as an unavoidable byproduct of the capability processes.

And this particular monarch molecule generates a significant amount of glycosylation, and it forms the advanced glycation products, end products. And that leads to problems with misfolding.

Parkinson's Disease Mechanisms and Inflammation

And then once this forms, then it has problems with the release of this particular molecule. It has the ubiquitination and the protein that are Proteus degradation and autophagy.

And these are all powerful product processes for the elimination. But in fact the glycation of alpha synuclein actually impairs the clearance of these particular pathways, and so it impairs the release, the proteasome degradation and the autophagy.

And this has been shown that advanced glycation end products have been increased in us since looking brains, and they are detected in the periphery of a Lewy body.

So it's an active ongoing function and it's worthy of note. Now this particular study was done looking at the Alpha synuclein and they found that with the mechanism was actually through the gut.

So the so when the gut formed different types of molecules, alpha synuclein nuclei would form in the gut and would be passed up through the vagus nerve.

Now, this is a unique pathway and it leads to the assumption that there is like a prion particle that actually causes some seeding of the gut of the brain in terms of the degradation products.

And so in this particular study, they did a vague automate and they they got to me in the clients. And so the the image on the left is a normal individual and then the one in the center is where they have induced a Parkinson's like syndrome.

And in the right is shows that the disruption of that by doing of they got to me so the point is that the they got to me limits the exposure to the molecule by way of blocking the pathways and the transference of this molecule all the way up the chain from the enteric nervous system.

Now, this process is quite involved and it occurs through the exi ptosis of alpha synuclein from the enteric and sympathetic neurons into the extracellular space and the psychosis from the neighboring neurons, retrograde transport into other neurons.

That alpha synuclein acts as a seed for the aggregation of these molecules in the brain and in other tissues. It now is function as a misfolded protein impairs mitochondrial complex one.

So it's impairing the mitochondrial function at all levels and it causes oxidative stress. Now it is particularly sensitive the areas of the brain, the substantia nigra and the dopamine receptors are particularly susceptible to the sensitivity of these aggregates.

So the conventional treatment of Parkinson's has to do with dopa stimulation, antidepressants, cognitive enhancement and substances and awareness and anti tremor type of medications.

And, and now I they haven't stopped the progress of this disease at all. It tends to be relentless in terms of its progression and leading to demise. And many of the physical effects and quality of life effects that are

encompassed.

Parkinson's disease. Now, what about the endocannabinoid system? The endocannabinoid system could be an answer and could be a target for this particular disease entity.

Now, the endocannabinoid system is a are a a complex system of regulation of different neurotransmitters, as well as metabolic and immune functions in the body.

It relies on a number of receptors and endocannabinoid substances, ligands as well as enzymes and transport molecules that deliver these particular molecules.

It's involved with almost all of our processes, whether it's a gut or neurologic or cognitive or mood or diet and nutrition. It has a lot to do with inflammation and cell growth and proliferation.

So it is a master controller of these this complex systems we have within our body. And we've just getting into the depth of learning about it. But this could be an answer for many of the problems that we're facing with Parkinson's disease.

Now I'm going to talk about the major dysfunctions in the endocannabinoid system with Parkinson's disease, as well as other neurodegenerative problems.

The CB1 and CB2 receptor, the GPR55, which is a receptor that is now considered part of the endocannabinoid system and the TRP, the R one, which is another important receptor that handles a great deal of pain and neurologic function.

Mm hmm. And we're going to talk about restoring the endocannabinoid system as a whole. So we know that the endocannabinoid system by directly interacts with the doping in the glutamic and and the GABA energetic systems.

We we are well aware of it. So with this incredible integration, then there's got to be connections here with the endocannabinoid ligands and with phyto cannabinoids as well and some of the receptor systems that are involved.

We know that there's a doubling of the primary endocannabinoid, which is AA levels, and we know that there are compensatory mechanisms to alleviate dopamine depletion that go through this system.

We also know that dopamine lesions are reversed by R by dopamine too, with x receptors and X inhibitors.

Endocannabinoid System and Parkinson's

And we know that CB1 ah receptor is is downregulated early on, but it's upregulated later on. And we've got CB2 upregulation occurring with this situation.

Now what about the endocannabinoid role in Parkinson's? Well, we know that there's a crosstalk between the CB1 and CB2 and the TR v one receptor systems, and we know that there is a complex with the GRP 55 and it potentiate the neuroprotective capability in Parkinson's disease.

We also know about TRP, V1, GRP 55 and other connections, and then we also know about p PA proximal with the p PA receptors and their nick and their neuroprotective effects in Parkinson's disease.

And we know that has a lot to do with this pathology. We also know that cb2 receptors are very key in this rectification of this disorder, and many of the symptoms that go along with it.

Now, first off, the CB2 receptor is it controls every one of our immune cells. Every every immune cell has a CB2 receptor and it controls the inflammatory signals that that cell gives off.

It has a purpose in shifting the metabolism, the immune immune system to the M2, the healing phase of the

inflammatory system from the pro-inflammatory state.

So it's going from a switching the system from an inflammatory stage to an inflammatory stage. And the healing of that Cb2 receptors are located on all our inflammatory cells and all our immune cells, including macrophages, neutrophils, t cells and dendritic cells everywhere in the body.

But oftentimes it's left after the CB2 receptor has a great deal of connections within the neurologic system, the central nervous system where it orchestrates the ion currents, transmitter release, postsynaptic reception expression and postsynaptic currents that have to do with excitability as well as membrane potential.

Now another key factor here is the P PA alpha receptor and the P four excuse me, P PA gamma receptor, the p PA Gamma. And I'm going to focus on the immune system.

It's a nuclear receptor system on each of our nuclear membranes that controls in detail the immunologic system in a cascade fashion. So it's not just one function, it has a host of different functions, and it's key and important in the inflammation cycles that go on within the body.

What are cbd's? And I'm going to jump to cannabidiol right away and I'm talking about cannabidiol is actions on the endocannabinoid system. This is a potential for a major benefit in the body in that cannabidiol blocks the CB1 receptor and blocks the tr v one and the R 55, and that has major therapeutic events in Parkinson's.

It increases arachidonic ethanol mean, which is the AA, which is a the key ingredient in our endocannabinoid system. It activates the p pa receptors and it actually includes an exon exosome inhibitor.

And I'll get to the EXOSOME inhibition in a moment because what we talked about earlier was a little bit of the migration of the the molecule alpha synuclein into the brain, and it goes by an existing mechanism and this is a pathway that CBD can block and it as a weak modulation of the cb2 receptors.

So adding anti-inflammatory effects in specific. So there have been a number of clinical trials with cannabidiol, but they haven't been terribly successful.

It Now this is contrast between a an uncontrolled randomized well, an uncontrolled study based on clinical practice and data collected from patient charge to a randomized controlled trial.

And the randomized controlled trial did not see any specific benefits. But in the trial with medical charting and the Non-Randomized fashion, they had an 87% improvement in some symptoms related to Parkinson's disease.

Now, this makes it very difficult to get to the detailed science of what's going on and is it a real benefit? Now, this was a quality of life study that was done that actually showed none of the movement disorder had any statistical benefit.

But look at the quality of life improvements here, where you had mobility increasing by four times, emotional well-being increasing considerably stigma, social support, cognition, communication, physical discomfort, all these things improved immeasurably with the cannabidiol in this particular group.

So quality of life issues appear to be very, very significant. And a short term study like this may not see the end results of correcting for or reversing some end stage Parkinson's disease symptoms.

Next, I want to get into Carrie offline. Will Carrie offline is a endo.

CBD Evidence and Quality of Life

It's a phyto cannabinoid. So it actually is a substance that connects up to the endocannabinoid system very directly and hitting all the targets that we have already talked about with CBD.

It's an essential oil common in many spices. It alleviates pain, inflammation, anxiety and metabolic disorders. It's actually found in hundreds of plants and it's got high concentrations in 36 and has over 3000 articles that have been published on it.

But there haven't been very many human studies. In fact, it is FDA approved as a food flavoring and it has no toxicity and no significant drug interactions.

Now, how does body work? PCP work, What works across this this range of targets and I'm talking about cytokines, enzymes, genetics signaling molecules all the way over to the receptors, the cb2 receptors and the opioid receptors, as well as the key part to keep our people are gamma and the people are alpha and the tumor, the TLR receptors as well.

Not only that, it's actually signaling on the nucleic Factor Kappa Beta. It's a signaling for autophagy in the mTOR and that whole list of combined connections there.

So it's got over 75 different connections on it all for the benefit of the individual and it's got huge potential for everything. Now, because of this potential, we've only been able to target a number of different substances and connections to disease.

But across the board it has been effective for many different problems in and pre-clinical studies for ulcerative colitis, chemo genic, pain, dyslipidemia nephropathy.

And so the list goes on, but includes pain and cerebral ischemia as well as neurodegenerative conditions. So the focus here is on Parkinson's disease would be to care offline and I want to get into that right away.

These are the human studies that have been done on IBD care offline. They don't amount to much. So there's only there's less than ten human studies that have been done on beta care offline, and yet we've known about it for for 20, 30, 40 years.

And we haven't really entered into any insignificant dialog with it. So you can't rely on the human studies. We can only rely on the pre-clinical work on animal models and cell culture programs that have been going on.

And based on these data care offline has magnificent effects in regulating the endocannabinoid system. It promotes neurogenesis, it shifts the micro glia from the M2 to the to healing type of immune function that restores the blood brain barrier.

It's anti-inflammatory. It regulates the Proteus status and autophagy, and it prevents tau tangles and neuron degenerative amyloid beta. But we don't know about synuclein, and it increases the mitochondrial formation and function.

And that's that's very important that it actually restores mitochondrial function in all of these neurodegenerative diseases. We're dealing with a mitochondrial failure in many, in many cases.

I mentioned before that Cb2 actually has two avenues of approach. It's working on the glial cells for the inflammatory signals and reducing inflammation, but it's also reducing it's also coordinating for neurotransmitters and ion channels, ligand receptors and synaptic functions, as well as on neural networks in neural functions.

All of these bear a great deal of I have a great deal of bearing on the neuropsychiatric and the neurologic diseases and neurodegenerative in particular.

Now, this is very interesting. The the there's a dysregulated micro glial polarization in alpha synuclein and induced synaptic pruning and this is related to the CB2 receptor.

If the CB2 receptor is actually removed from the body and becomes incapacitated, then there are increases the amount of synaptic pruning that occurs, and that's going to be leading to a pro-inflammatory phase and a a major

functional problem within the body and within Parkinson's disease, specifically.

So the I get to the point of what actions does beta Carrier actually have on neurologic and pain syndromes. First of all, it's attacking it's targeting the CB2 receptor, activating, activating it, It's affecting the microglia and the astrocytes, energy reducing neuropathic pain.

B-Caryophyllene and Neuroprotection

It's also reducing neuroinflammation and it's promoting brain derived neurotrophic factor for improved memory and function. And over on the left, it actually is working on an impressive effect on mast cells.

And I've seen this time and again where it has an anti-allergy effect in mast cells that tend to have a lot of connections with our neuropathy type of functions and our many other function malfunctions as well.

And I want to keep that in as a key factor. And then on the amyloid beta, it's it's handling this misfolding mechanisms that are going on. So it's hopeful that it could prevent the misfolding of the alpha synuclein and inflammation.

So the key factor here is a CB2 receptor, but it also connects up with to also a endocannabinoid so palmitic ethanol made and all the all f enormously abbreviated PGA and okay and they activate the p par alpha receptors and this has a great deal of reduction of pain and inflammation and it targets the tumor necrosis factor alpha and interleukins and IAV and gamma.

And it's it's doing this very effectively and it's going through p pa gamma and it's also affecting the immune and the vascular inflammation by interfering with the nitric oxide synthase and promoting that as a mechanism for decreasing vascular inflammation sort of has its effects on the heart, on the brain, on the gut and in the airways and the enteric circulation.

So with regard to the inflammation, I want to point out once again that the the the action of beta core offline is to switch the body from the M1 type of activated microglia to the M2 healing like glia, and it compensates by reducing all those inflammatory markers that I have listed there.

So what's the evidence in Parkinson's? Well, again, it's it's not human evidence, it's animal models. But BCP attenuates, oxidative stress, inflammation, glial activation and salvages, dopaminergic neurons in a rat model of Parkinson's disease that generates neuroprotective effects against the neuro, the dopaminergic neuron injury.

And it ameliorates the a model of of beta of Parkinson's disease. In this MP cytotoxic city model and upregulation of cb2 it up to regulates the regulation of it upregulates the amount of cb2 in Parkinson's disease patients specifically now cb2 agonists have reduced the L-dopa dyskinesia as ineffectively as Amantadine in some cases, and basically offline inhibits.

And LRP three the inflammation and the new nucleic factor Kappa beta signaling pathway. So extremely important factors for inflammation and disease entities.

Now I assure you this picture of Parkinson's, but I want to emphasize the quality of life issues here with these types of problems. There is all kinds of social and lifestyle problems that occur, and the emphasis here on these use of cannabinoids is really going to be on the quality of life.

As we saw from that earlier study that I mentioned, anxiety, depression and beta carry off lean and cannabidiol can reverse significantly anxiety, stress and depression.

Now, a particular point I want to make here is that this particular study was evaluating obesity associated airway responsiveness, but it gives you some mechanisms that are involved with better care often and why it works this way.

It has a it converts macrophage repolarization to the M2 phase. That's understood, but it also reduces food intake by decreasing appetite and it upregulates GLP one.

And that's an important factor that we found that has a great deal of to do with neurodegenerative problems as well as diabetes and obesity and upregulates the uncoupling proteins, the uncoupling proteins and actually switches the body from a white fat to a brown fat by energy conservation.

It increases mitochondria, but it decreases reactive oxygen species. And that's a unique mechanism where it increases the mitochondrial function, but it actually decreases react to an oxygen species probably from its anti oxidant nature and it downregulate inflammatory nuclear receptors.

Now, furthermore, it protects the gut magnificently. And this is important, particularly in Parkinson's, because there is up to an 80% rate of constipation that occurs.

And there's probably issues about the the leaky gut and the problems that can occur and the inflammatory molecules that can arise from the gut into the brain and sleep better.

Care offline has been fabulous for improving the sleep quality. I've got a couple of testimonials there where these people have slept better and more effectively without without being having any ill effects on the next day.

And what about CBD versus BCP? Am I really am I comparing them? How am I comparing them, and is it one or the other? Well, actually, it turns out that they both work in combination and it's a most effective arrangement is in combination where in this particular study there was a comparison between CBD and BCP and they almost do the same, almost the same dose.

But when they were combined there was a synergy that occurred and so that there is for analgesia, that's certainly the case.

CBD and BCP Synergy, Practical Use

But it's also true in ischemia disease, and this is a stroke of a model that the combined effect actually reduced the, the infarct area significantly and with a narrower and it shallower range of damage and both directional defects or effects are protective changes mediated by different mechanisms.

So they they work together and they work in a blended fashion. And so that's my recommendation for you is that we should combine the CBD with Beta Kappa Alpha and to reach the maximum potential of stimulating and balancing the endocannabinoid system.

Now Carrie offline is a natural herbal food substance. It's all around us. We just don't get enough of it and we don't get it delivered in a bioavailable fashion.

It's a broad spectrum cannabinoid that has not been really recognized by the medical profession or the naturopathic for professionals for that matter.

It's FDA accepted it's low dosing, it's got few adverse effects. It's synergistic with CBD and it's an alternative to cannabis product. So if a person can't take or is reluctant to take a cannabis product like CBD, then they can take better care offline.

It's just a food, natural food herb that they can use as effectively. And for people who are have or at risk for urinary drug testing like athletes, police, military or medical personnel, you can use this product and don't have to worry about coming up positive on a drug test because it comes from hops or cloves and that's the main source of the beta carry off in the product.

So it's got an established, effective liposomal format and it's very, very effective for many types of problems. So I'm really trying to create a balance and I'm trying to compliment health through this endocannabinoid system, and I

want to restore people's health with this system, not only with these phyto cannabinoids, but with diet, exercise, proper sleep, rest and social interactions.

Those are all important elements for encouraging and enhancing the endocannabinoid system. But when we're faced with serious diseases, we may have to go to using phyto cannabinoids to help restore our balance.

Thank you. That was excellent and really informative. So important for folks to understand where we are today with Parkinson's disease and that right now there is no available or commercially available disease modifying therapy for Parkinson's.

We treat symptoms. There are some papers on neuroprotection and potentially particularly with a family of monoamine oxidase inhibitors such as Selegiline But these are very theoretical and they may or may not be appropriate for a lot of people because the enzyme system that they inhibit.

So we have very promising compound that is currently available for folks to sample and experience for themselves and see how it works for them. Dr. Blair, If somebody wants to get B-Caryophyllene I my apologies.

How can they how can they do that? Well, they can they can go to the website and that's my prominent position. We've got BlairMedicalgroup.com or BlairMedicalgroup.shop and you can buy four different products are available and Caryophyllene form there is the liposomal oral form there is a tincture which is a topical and a gel that's a topical and there's a crunch that is a small cookie, it's an edible form and there the dosing is highly variable because people have such a variable response to the cannabinoid effects.

So I generally tell people start low and move slowly upward, but half a milliliter or 15 milligrams of Caryophyllene is an ideal starting dose for an average person.

If you're particularly sensitive to substances, I would start you off with five drops and or really down to a quarter of a mole liter. But I've had people take two and three milliliters and four milliliters for serious problems and they get tremendous benefits from that.

Q&A, Product Access, and Closing Remarks

Now, in case someone wants to try this, are there any side effects or sensitivities that have been reported that they should at least be aware of before they try it?

Well, Ken, this is the most magnificent part of it. There haven't been any significant side effects with it. There's no drug interactions. I haven't encountered any drug interactions with.

It all doesn't have any toxicity. And I offer samples. The website actually offers five milliliters samples to get you started because this is so new, nobody has any experience with it and they won't believe their response.

And typically the response occurs within 10 minutes of you. So you get a significant response after taking out of 15 drops or 30 drops, you can have amazing response within 10 minutes and you can that you are really getting the benefits that you're looking for.

Excellent And so again the website is www. BlairMedicalgroup.shop. Practice exactly right. And you have a code for folks who would like to try it have a little discount on the cost.

Right and we have a code that is THANKYOU23 THANKYOU23 I also have one is it's an okay to use BCPARK23 Yes, that's probably the ideal code to use because that will give us a little bit more insight as to who's ordering it and where they're coming from.

Wonderful. Dr. Philip Blair, thank you so much for joining us today on the Parkinson's Solutions Summit and sharing your unique experience with this emerging compound they hope will help thousands, if not ultimately, over a million people suffering from Parkinson's disease.

This is very important information. And once again, thank you for your time, your effort. I know what it takes to put together a slide presentation. Folks, this is hours worth of work that we are bringing to you.

Complimentary of DrTalks Sharlin Health Neurology and Dr. Philip Blair with the Blair Medical Group. Thank you again.