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Examining the Correlation Between HIV & Neurocognitive Disorders

Dr. Lisk:
In an effort to examine the correlation between HIV-induced aging and Alzheimer's disease in the United States population, a team of researchers at the University of Tennessee Health Science Center are working to develop a novel drug delivery system.

Welcome to *NeuroFrontiers* on ReachMD. I'm Dr. Jerome Lisk, and I'm speaking with Dr. Santosh Kumar, who is a Professor in the Department of Pharmaceutical Science at the University of Tennessee. Today, we'll be discussing his current research on the relationship between HIV-induced aging and Alzheimer's disease.

Dr. Kumar, welcome to the program.

Dr. Kumar:
Thank you very much, Dr. Lisk.

Dr. Lisk:
So let's start with some background, Dr. Kumar. What are the underlying mechanisms between HIV and neurocognitive aging?

Dr. Kumar:
Well, I don't think we know the exact mechanism, but we do know some correlation between HIV and Alzheimer's, or aging, and what we know is that HIV-infected individuals age relatively faster than the non-HIV individuals. They show the plaque formation like beta-amyloid tau hyperphosphorylation much earlier than the normal population, so that's kind of the major correlation we know between HIV and aging. What we also know is that these HIV individuals, as they age, they start developing something called HIV-associated neurocognitive disorder. In short, we call it HAND, H-A-N-D, mainly the mild form of HAND, and that means they kind of start becoming demented like early, early phase of dementia. So that's the kind of correlation we have so far between HIV and HIV-associated basically cognitive disorder or aging.

Dr. Lisk:
Okay. So you may have already kind of touched on this in the question. So to expound upon this, what happens when HIV enters the brain?

Dr. Kumar:
So HIV enters the brain as early as in the second week of the infection. What it does is it enters into the brain using the mechanism called Trojan horse, so these packaged monocytes or macrophages, and then they enter into the brain and spread the virus in brain macrophages and brain macroglia, and once they are spread in these cells, which are some of the major type of cells in the brain, they hide there. They are persistent. Why? Because the anti-HIV drugs or antiretroviral drugs can't access the brain. So once they are spread, they start having viral proteins, oxidative stress agents, and inflammatory response, and all this together ultimately damage neurons, and once the neurons are damaged at different levels, they start showing neurodegeneration and neurocognitive impairments.

Dr. Lisk:
So you said the HIV drugs cannot enter the brain; is that right?

Dr. Kumar:
That's true.

Dr. Lisk:
Okay. Now does HIV induce tau pathology into the brain?

Dr. Kumar:
That is true. That's what they have shown, that HIV can induce tau hyperphosphorylation and also beta-amyloid and a plaque formation to some extent, yes. So there are some research, but again, the mechanism is still unknown.

Dr. Lisk:
That will be interesting because when we're looking at Alzheimer's disease in some of these tests, such as lumbar punctures to diagnose Alzheimer's disease looking for phosphorylated tau, it would be interesting if they could tell the difference between the HIV-induced aging or dementia versus Alzheimer's disease because with the phosphorylated tau 217 blood test that's coming out soon and the lumbar punctures, how are they going to tell the difference with these new diagnostic tests for Alzheimer's disease?

Dr. Kumar:
Well, I don't think at this point of time we have a specific biomarker—right?—which can actually tell you the difference, but what we can say is that these HIV-infected individuals start developing these characteristics much earlier in life.

Dr. Lisk:
For those just tuning in, you're listening to *NeuroFrontiers* on ReachMD. I'm Dr. Jerome Lisk, and I'm speaking with Dr. Santosh Kumar about HIV and neurocognitive aging.

So now let's dive into your team's research, Dr. Kumar. What is the current goal of your project?

Dr. Kumar:
So our current goal is 2-fold. The one is to find out a common mechanism or pathways by which HIV can lead to neurocognitive disorder as well as it can enhance aging and ultimately, again, cognitive disorder. So once we find the common pathways, our next goal would be to have a drug delivery system and with certain drugs which can actually attack that particular pathway. And we do have a drug. I can't tell you the name, but there's a drug which is already in the clinical phase II trial for Alzheimer's disease, and the same drug we have used in our HIV model, it can also show anti-HIV activity. And this drug is lipophilic; it can cross blood-brain barrier, and it can reach into the brain, so we have some light at the end of the tunnel for this drug for the common pathway.

Our lab is also working on a drug delivery system where we do have some kind of drug delivery system using artificial as well as natural nanoparticle where we can package our drug, either antiretroviral drug or this common drug for Alzheimer's or HIV, and even nutraceuticals. There are certain nutraceuticals which are common for both, so we can package those and use that for drug delivery. So we are kind of delving into some of these areas all together.

Dr. Lisk:
Interesting. And have you made any notable discoveries amidst your research?

Dr. Kumar:
Well, we have a common drug, or drug candidate I would say, which may target both Alzheimer's and HIV. We have a drug delivery system, both artificial and natural, which can package antiretroviral drug and nutraceuticals and deliver into the brain using intranasal delivery, so we are also working on intranasal delivery. It can bypass the blood-brain barrier at least 50 percent. We do have a target which we are trying to investigate and prove that this is the common target for the drug for both the diseases.

Dr. Lisk:
This has certainly been an insightful look at research on HIV-induced aging and Alzheimer's disease, and it's always a joy for MDs to talk to PhDs because there's things that you guys have researched and know that we don't, and then we know the clinical aspect, so it's been a joy talking to you, and I have learned quite a lot.

I'd like to thank my guest, Dr. Santosh Kumar, for joining me today to discuss his research and share his insights, and I am sure that with the rapid growth of this research you're going to have more to share with us in the future.

Dr. Kumar:
Thank you very much. I really appreciate talking with you, and I do agree that scientists need to collaborate with the clinician, MD doctor, so we can really understand these pathologies much better now with the help of clinician. Thank you very much.

Dr. Lisk:
So for ReachMD, I'm Dr. Jerome Lisk. To access this and other episodes in our series, visit ReachMD.com/NeuroFrontiers where you can Be Part of the Knowledge. Thanks for listening.