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Cough medicine fights dyskinesias in Parkinson's

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A cough suppressant and a drug tested as a schizophrenia therapy curb the involuntary movements that are disabling side effects of taking the Parkinson's disease medication levodopa, Portland scientists have found.

Dextromethorphan, used in such cold and flu medications as Robitussin, Sucrets, Triaminic and Vicks, suppresses dyskinesias in rats, researchers at Oregon Health & Science University and the Portland Veterans Affairs Medical Center found. Dyskinesias are the spastic or repetitive motions that result from taking levodopa, or L-dopa, over long periods.

The researchers also found that BMY-14802, a drug previously tested in people with schizophrenia and found to be safe – although not effective in treating schizophrenia symptoms – suppressed dyskinesias in rats more effectively than dextromethorphan did, suggesting that BMY-14802 might work to block dyskinesias in people with Parkinson's.

"These results were unexpected, but very exciting," said the study's lead author, Melanie A. Paquette, Ph.D., postdoctoral fellow in the Department of Behavioral Neuroscience, OHSU School of Medicine, and the PVAMC. "We have filed a patent for the use of BMY-14802 for dyskinesias and we hope to get funding to begin human trials very soon."

The study, titled "Differential effects of NMDA antagonists and sigma ligands on L-dopa-induced behavior in the hemiparkinson rat," is being presented during a poster session today at Neuroscience 2007, the 37th

annual Society for Neuroscience conference in San Diego.

The results also affirm the value of the rat model for dyskinesias that Paquette's team used in the study. Previous studies by other researchers have shown the drug amantadine already is effective in treating dyskinesias in both humans and rats, and dextromethorphan's effectiveness against the condition in rats provides more data supporting the use of the model.

"Basically, these two drugs work to block dyskinesias in both humans and rats, and that means the rats are a good model to screen potential drug treatments for humans with dyskinesias," Paquette said.

But BMY-14802, which is an antagonist at sigma-1 receptor sites in the brain, "worked much better than dextromethorphan," an antagonist at N-methyl-D-aspartate (NMDA) receptors.

"There's something special about BMY-14802," Paquette explained. "The effect on dyskinesias is really striking and I've repeated it several times, so it's a reliable finding. It's a very exciting result."

Source: Oregon Health & Science University

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