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# PRESS RELEASE

## COMMON HEART MEDICATIONS MAY ALSO PROTECT AGAINST PARKINSON'S DISEASE

In the first large-scale population-based study of its kind, researchers at the University of California, Los Angeles and the Danish Cancer Society have found that a specific type of medication used in the treatment of cardiovascular conditions is associated with a 26-30% decrease in risk of developing Parkinson's disease. Building on prior research in animal and cellular models indicating that calcium channel blocker medications – used in humans to treat hypertension, angina, and abnormal heart rhythms – might also protect certain brain cells from damage, this research asked the question: Are calcium channel blocker medications associated with protection from Parkinson's disease in humans, and if so, do all calcium channel blocker medications provide the same protection, or is it just one class of medication?

Using detailed medical history data available through Denmark's National Health Service record system, Dr. Beate Ritz, a professor at UCLA's School of Public Health, in collaboration with Drs. Soren Friis and Jorgen Olsen of the Danish Cancer Society, evaluated medical history and medication usage data from 1,931 Parkinson's patients and 9,651 unaffected subjects, for a period up to 12 years prior to the diagnosis of disease. By separately evaluating different classes of drugs prescribed for hypertension, researchers found that only centrally acting calcium channel blockers of the dihydropyridine class were associated with significantly lower risk of developing Parkinson's disease. Other classes of anti-hypertension medications and the peripherally acting dihydropyridine amlodipine were not associated with a lower risk of Parkinson's disease.

Parkinson's disease, the second most common neurodegenerative disorder in the US, is primarily characterized by a loss of voluntary movement resulting from the death of neurons in the substantia nigra of the brain, an area involved in movement control. How is it, then, that a medication used to treat heart conditions and hypertension might protect these specific neurons from death in Parkinson's disease? The key was to consider the mode of action and the brain availability for each class of these drugs. In particular, the dihydropyridine class of medications acts on a specific type of calcium channel in the heart known as the L-type calcium channel – a similar type of channel is present on the neurons that degenerate in Parkinson's disease. Additionally, only a subset of dihydropyridine class drugs cross into the brain where they might be able to act on the calcium channels of neurons and provide a protective effect; these include felodipine (Plendil), isradipine (Dynacirc), nicardipine (cardene), nifedipine (Adalat, Procardia),

nimodipine (Nimotop), nitrendipine, lacidipine (Lacipil, Moten), and lercandipine (Lastolic, Zanidip). Interestingly, amlodipine (Norvasc), the one dihydropyridine which does not cross in to the brain and non-dihydropyridine drugs (such as verapamil and diltiazem) that do not act on L-type calcium channels, were not associated with a lower risk of Parkinson's disease. This supports the idea that mode of action and brain availability are important factors when studying drugs for neuroprotection.

Although the results are intriguing, the authors caution that more detailed studies and a more complete understanding of the biology underlying the action of these medications in the brain are warranted, particularly as some Parkinson's patients can suffer from too low of blood pressure, a condition which could be worsened by taking calcium channel blockers inappropriately. Furthermore, these results should be confirmed in other studies of human populations with detailed medication data capable of differentiating between peripherally acting and centrally acting dihydropyridine calcium channel blockers.

The study appears in the upcoming issue of Annals of Neurology. In addition to Ritz, Friis, and Olsen, study authors included Dr. Shannon Rhodes and Lei Qian of UCLA's School of Public Health and Dr. Eva Schernahmmer of Harvard Medical School. The authors declare no conflict of interest.

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