

Anti-Inflammatory Drug Reduces Nerve Death in Mouse Model of Parkinson's Disease

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Using a mouse model which mimics neuronal cell death in the part of the brain associated with Parkinson's disease (PD), scientists at the Buck Institute have shown for the first time that these neurons die due to inflammation. The study also shows that treatment with the antibiotic/anti-inflammatory drug minocycline, started before symptoms began to appear, reduced neuronal death in these mice.

The findings, published in November 8 edition of the *Journal of Neuroscience*, provide a glimpse at potential treatment options for humans and highlight the need for pre-symptomatic tests that would identify those at risk for the disease that affects one out of every 100 people over the age of 65.

The research, led by Buck Institute faculty member Julie Andersen, PhD, involved a mutant mouse known as weaver, so named because it exhibits a loss of balance and motor control early in life. In addition to suffering from a genetically programmed loss of neurons in the cerebellum (the portion of the brain attached to the brain stem) that plays an essential role in coordinating movement), the mice also suffer from nerve death in the substantia nigra, a region in the mid-brain which produces the chemical dopamine that regulates motor control. In humans, PD develops when dopamine levels become deficient, resulting in symptoms which include tremor, slowness of movement, rigidity and problems with balance.



After examining the midbrain of the animals for alterations in gene expression, researchers discovered that weaver mice "over-express" particular genes associated with inflammation compared to normal control littermates. Andersen's team began treating the mice beginning at birth with minocycline, an antibiotic that also has anti-inflammatory properties. At three weeks of age, the treated mice showed a 30% loss of dopamine-producing neurons in the substantia nigra, as compared to a 50% loss commonly seen in mice not treated with the drug.

"It is satisfying to be able to show that inflammation contributes to dopaminergic cell death in the weaver mouse since neuroinflammation is a commonly observed feature in Parkinson's disease itself. This suggests that the weaver mouse constitutes a good model to explore potential antiinflammatory treatments for the human disorder," said Andersen. She added that the findings also highlight the urgent need to develop tests that would identify at an early age, those at risk for PD. "Currently, by the time humans are diagnosed with Parkinson's disease they have already lost 60% of their dopamine-producing neurons," said Andersen, "Anti-inflammatory agents would likely be maximally effective if taken before symptoms appear in order to halt disease progression."

Others joining Andersen in the study include Buck Institute researchers, Jun Peng, Lin Xie, Fang Fen Stevenson and Simon Melov, along with Dino DiMonte of the Parkinson's Institute. The work was supported by grants from the National Institutes of Health.

Source: Buck Institute for Age Research

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