MedicalXpress

# New research examines connection between inflammatory stimulus and Parkinson's disease

April 23 2013

Parkinson's disease (PD) is a progressive degenerative disease affecting a person's ability to coordinate and control their muscle movement. What starts out as a tremor in a finger will eventually lead to difficulty in writing and speaking, and ultimately the inability to walk without assistance. Since the 1950s research has shown that people with Parkinson's have decreased levels of the chemical dopamine in their brains, which is involved in sending messages to the part of the brain that controls coordination and movement. Subsequent research has found that dopamine-generating cells, known as dopaminergic neurons, are also absent in a specific area of the brain in those with PD.

The precise cause or causes of PD is unknown, but there is a consensus that an inflammatory event or episode is involved in the initiation of neurodegeneration, and that chronic neuroinflammation is a sustaining and exacerbating reason for the loss of the dopaminergic neurons. A new study conducted by a team of Texas researchers brings the understanding of inflammation's role a step further. They have found that a single, high-dose exposure of an experimental inflammatory agent in an animal model causes changes in brain tissue that are similar to those associated with the development of the disease.

The study was conducted by Roger Bick and his colleagues Marie-Francoise Doursout, Michael S. Schurdell, Lauren M. Young, Uzondu Osuagwu, Diana M. Hook, Brian J. Poindexter, Mya C. Schiess, and

Diane L. M. Bick, all at the University of Texas Health Science Center, Houston, Tex. Dr. Schiess will discuss the team's findings at the Experimental Biology 2013 meeting, being held April 20-24, 2013 at the Boston Convention and Exhibition Center, Boston, Mass.

The poster presentation is entitled, "Inflammatory cells and cytokines in the olfactory bulb of a rat model of neuroinflammation; Insights into neurodegeneration?" and is sponsored by the American Society for Investigative Pathology (ASIP), a co-sponsor of the meeting. The full study will appear this month in the online edition of the *Journal of Interferon & Cytokine Research*.

## Methodology

In the study, the researchers examined inflammatory cell and cytokine production in brain tissue from a lipopolysaccharide (LPS)-treated rat model that mimics many of the neuropathologic changes associated with PD. Concurrently, they monitored the appearance of glial cell line-derived neurotrophic factor (GDNF), a neuronal protective agent, and circulating nitric oxide (NO) levels. They also examined the immune system associated cells in the olfactory bulb of the brain. It is known that Parkinson's starts with this mechanism.

Twelve male Sprague-Dawley rats were treated with intravenous LPS in saline, 12 control rats were treated with saline, and all were maintained for up to 48 hours before euthanasia and brain removal. Brains were removed from both groups at defined times, blood and other tests were conducted, and images of various sections of the brain, including the olfactory bulb, cortex and cerebellum, were taken using fluorescent microscopy.

## Results and Conclusions

In general, the researchers found that a single injection of LPS elicited a systemic inflammatory response in the rats, as indicated by an elevation in certain circulatory cytokines. Tissue taken from the olfactory bulb showed the presence of immune associated cells. Individual cytokines within the olfactory bulb showed an increase in certain types of cytokines. Taken together, the complete analysis indicated that the single dose of LPS stimulated an inflammatory response that closely resembled the hallmarks of the development of the disease.

The results suggest an involvement of both the peripheral and the central nervous system immune components in response to inflammation and inflammatory episodes. As a result, the researchers suggest: (1) inflammation initiates an immune response; (2) the presence of continuing and increasing pro-inflammatory mechanisms results in a process whereby cellular protective mechanisms are overcome and the more susceptible cells, such as the dopaminergic neurons, enter into cell death pathways; and (3) this leads to a series of events that are a key part of the progression of PD.

## Next Steps

Neuroinflammation is a significant problem for those with PD, and it persists throughout the course of this debilitating illness. Understanding of the essential processes behind it is the best pathway to finding therapeutic approaches to address it. This study highlights an opportunity to better understand the role inflammation plays in the process.

Provided by Federation of American Societies for Experimental Biology

Citation: New research examines connection between inflammatory stimulus and Parkinson's disease (2013, April 23) retrieved 18 April 2024 from https://medicalxpress.com/news/2013-04-inflammatory-stimulus-parkinson-disease.html

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.