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Research examines environmental triggers altering gene function in Chronic Fatigue Syndrome patients

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A University of Toronto Scarborough (UTSC) researcher is examining how environmental triggers might alter gene function in people with Chronic Fatigue Syndrome. The research could lead to better insights into the disease and eventually to new treatments.

Patrick O. McGowan, professor in the department of biological sciences and director of UTSC's Laboratory for Epigenetic Neuroscience, is looking at how environmentally triggered changes to gene expression might alter immune function and stress response in ways that contribute to the disease.

McGowan's field of study is epigenetics – long-term changes in gene function that do not change the underlying DNA sequence. An epigenetic change wouldn't change a gene itself, but would influence whether, when and how the gene is turned off. Epigenetic changes can be caused by environmental triggers such as infections, toxins, stress, nutrition, and even the social environment.

McGowan will conduct the study into the epigenetics of CFS with a grant from the CFIDS Association of America. As part of the grant, he will have access to the SolveCFS Biobank, a collection of biological samples from CFS patients.

CFS, also known as Chronic Fatigue and Immune Dysfunction

Syndrome (CFIDS), is a complex and debilitating chronic illness that affects the brain and multiple body systems. Symptoms include incapacitating fatigue and problems with concentration and short-term memory. Millions of North Americans are thought to suffer from the disease.

McGowan will look specifically at the relationship between a system called the hypothalamic-pituitary-adrenal (HPA) axis and immune function. The HPA axis is involved in the regulation of the stress response, and has effects on immune function and inflammation through cellular signaling mechanisms involving the steroid glucocorticoid.

McGowan's hypothesis is that there is an epigenetic mechanism in CFS that disrupts glucocorticoid signaling in white blood cells called lymphocytes. By studying tissue samples from the SolveCFS Biobank he hopes to pinpoint the mechanism.

Provided by University of Toronto Scarborough