



# **FastForward**<sup>SM</sup>

## **General Fund**

This project is funded by Fast Forward, LLC, a nonprofit organization established by the National Multiple Sclerosis Society to accelerate the development of treatments for MS. Fast Forward will accomplish its mission by connecting university-based MS research with private-sector drug development and by funding small biotechnology/pharmaceutical companies to develop innovative new MS therapies and repurpose FDA-approved drugs as new treatments for MS.

<i>Primary Investigator</i> David Selwood, PhD Medicinal Chemistry Wolfson Inst. for Biomedical Res. University College London London, UK	<i>Project Title</i> Development of a selective cyclophilin D (CyP-D) blocker as a new MS drug	<i>Amount to be Committed</i> \$285,000 Term – 12 months
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## *About the Investigator*

Dr. David Selwood earned his PhD in insect chemistry at the University of Southampton and completed postdoctoral studies in onchocerciasis (river blindness) at Wellcome Foundation, Beckenham, Kent, UK, funded by the World Health Organization. He then served as Senior Research Scientist at the Wellcome Research Laboratories, directing science for a group of 17 medicinal chemists. Dr. Selwood is Head of Biological and Medicinal Chemistry, Wolfson Institute for Biomedical Research, University College London, and Founder and director of Carbex Ltd. His laboratory conducts research on the cutting edge of chemistry and biology using the latest techniques and developing new ones for the study of biological systems. Dr. Selwood is a Fellow of the Royal Society of Chemistry.



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### ***Project Background & Goals***

Multiple sclerosis causes the loss of nerve fibers (axons) and nerve cells (neurons) in the brain and spinal cord. Protecting axons and neurons from damage, or repairing this damage, is a crucial, unmet need for people with MS. It is this damage that contributes to the progression of long-term disability.

Dr. David Selwood and colleagues are investigating an exciting possibility for neuroprotection. Inhibiting the molecule cyclophilin D may be an important part of this process; cyclophilin D controls the flow of molecules into neurons and may allow a toxic excess of calcium to flow into the cell. A commonly used immune system-suppressing drug, cyclosporin A, has been shown to inhibit cyclophilin D, but its side effects prohibit its use for neuroprotection. Dr. Selwood's team has found a way to chemically engineer cyclosporine to remove its immune-suppressing effects.

In this project, the team is synthesizing and optimizing a variety of chemicals similar to cyclosporin A. They are testing them in cells isolated in the lab to optimize delivery to the brain and to test for safety. Dr. Selwood's group is then testing the best candidates in an animal model of MS for their nerve-protecting effects.

This project may yield a new strategy for stopping nerve damage in its tracks in people with MS.