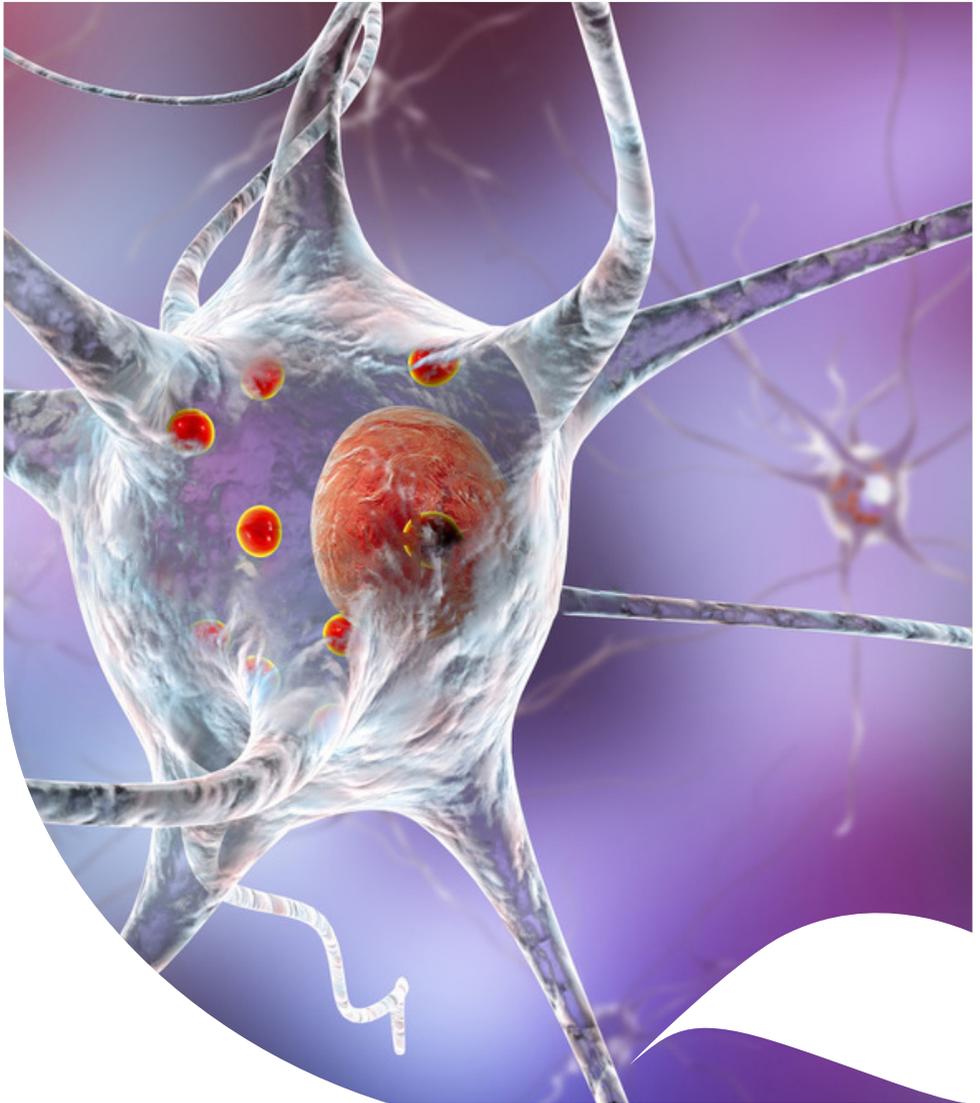


Biomedical Sciences

Preclinical Models of Neurodegenerative Diseases



The Woodruff Neuroinflammation Laboratory specialises in Parkinson's Disease, Motor Neuron Disease (Amyotrophic lateral sclerosis; ALS) and Neuroinflammation assays:

- Capacity for preclinical drug testing (efficacy) in disease models, or target validation studies.
- Range of state-of-the-art disease models accepted by funders and regulators as best-practice preclinical efficacy models.
- An extensive range of both standardised and novel behavioral and pathological analyses that can be customized to suit individual requirements.
- Ability to couple analyses with pharmacodynamic and pharmacokinetic data from the same animal to maximise data output and assay validation.
- Successful record in completing industry contracts, with two drugs undergoing human clinical trials after successful testing in models.
- Strong publication track record in preclinical neurodegenerative disease animal studies.
- Offer opportunities for leverage funding through non-diluting grant funding.
- Competitive rates for both fee-for-service or collaborative research projects.

In vivo models available

- Neuroinflammation endotoxin model (peripheral or central induction)
- *Parkinson's disease* 6-hydroxydopamine (6-OHDA) model
- *Parkinson's disease* synuclein pre-formed fibril (PFF-Syn) model
- *Motor neuron disease* SOD1G93A transgenic mouse model
- *Motor neuron disease* TDP43-transgenic mouse models
- *Huntington's disease* R6/1-transgenic mouse model

In vitro/ex vivo models available

- Primary *human* microglia neuroinflammation assays (derived from human blood)
- Primary *mouse* microglia neuroinflammation assays (derived from mice CNS tissue)

Publication examples

Inflammasome inhibition prevents α -synuclein pathology and dopaminergic neurodegeneration in mice. Gordon R, Albornoz EA, Christie DC, Langley MR, Kumar V, Mantovani S, Robertson AAB, Butler MS, Rowe DB, O'Neill LA, Kanthasamy AG, Schroder K, Cooper MA, **Woodruff TM**. *Sci Transl Med*. 2018 Oct 31;10(465). pii: eaah4066.

Pharmacological inhibition of complement C5a-C5a1 receptor signalling ameliorates disease pathology in the hSOD1G93A mouse model of amyotrophic lateral sclerosis. Lee JD, Kumar V, Fung JN, Ruitenberg MJ, Noakes PG, **Woodruff TM**. *Br J Pharmacol*. 2017 Apr;174(8):689-699.



UQ's School of Biomedical Sciences – mission statement:

By harnessing our diversity across the breadth of biomedical science, we will generate, disseminate and apply foundational biology underpinning health and disease to inspire and empower the next generation of leading researchers, educators, and healthcare professionals to innovate together for better health outcomes globally. Our innovative research encompasses basic discovery through translational pathways to medical solutions:

Cell architecture: We use sophisticated molecular and imaging techniques to explain how various cellular components and pathways contribute to building healthy bodies.

Receptors and signalling: We decipher the passage of external messages from the cell surface, through cytoplasmic signalling pathways, and ultimately to genetic regulatory circuits in the nucleus.

Chronic disease: We characterise the genetic, molecular and cellular microenvironments associated with diseases, such as Alzheimer's disease, cancer, MND and others.

Drug design and development: We identify critical biological targets and design drugs based on structural analyses to develop novel therapies.

Functional and comparative anatomy: Our interdisciplinary studies of structure and function across phylogenetically disparate species advance our understanding of the human body in healthy, aging and diseased states.

Injury and repair: We study fundamental mechanisms of cells in response to stress, consequences of repair processes and how these may be influenced for optimal outcomes.

Musculoskeletal and motor control: We develop and apply novel tools, to investigate muscle function and neural control of muscles in humans.

Neurobiology and brain function: We search for and discover genetic and environmental factors that lead to and maintain healthy nervous systems.

Reproduction: We investigate the genetic and molecular environment during early fetal development to advance reproductive technologies and facilitate healthy pregnancies.

Contact

Trent M. Woodruff, PhD

Associate Professor of
Pharmacology
Editor, Focus on Complement
Neuroinflammation Laboratory

T: +61 7 3365 2924

E: t.woodruff@uq.edu.au

W: biomedical-sciences.uq.edu.au/profile/561/trent-woodruff