UNIVERSITY of OTAGO

Te Whare Wānanga o Otāgo

Global move away from ineffective behavioural therapies for ME/CFS

Until recently Graded Exercise Therapy (GET) and Cognitive Behavioural Therapy (CBT) were proposed as the best treatments for Myalgic Encephalomyelitis /Chronic Fatigue Syndrome (ME/CFS). However, this has changed due to the now widely accepted finding that exertion aggravates the debilitating symptoms of ME/CFS - what we now know as Post Exertional Malaise.

Originally GET and CBT were promoted for ME/CFS by a small group of mainly UK and European psychiatrists who considered ME/CFS to be a somatic perception disorder where patients perceived their symptoms and their disability in an exaggerated manner, leading to avoidance of activity. This paradigm dominated thinking about the disease for over 30 years.

Recent key international trends see a move away from GET and CBT. This follows an extensive 2015 report from the Institute of Medicine (now the National Academy of Medicine, USA) which concluded that ME/CFS is a serious disease that deserves much more medical attention and social support and is not a psychiatric illness amenable to behavioural interventions. The 2020 National Institute for Health and Care Excellence (UK) evidence review's findings are also consistent with this latest thinking on ME/CFS.

Here in New Zealand, Professor Warren Tate's research group at the University of Otago has been actively researching the biological basis of ME/CFS together with an international expert clinician, Dr Rosamund Vallings, of the Howick Health and Medical Centre.

The research approach has been to study molecular changes in immune cells by precision (previously known as personalised) medicine. Multiple classes of molecules have been studied and many differences in physiology between ME/CFS patients and healthy age/gender matched controls, have been found; there are dysfunctions in the autonomic nervous system, immune regulation, inflammation, energy production, and a lowered general metabolism. Molecular signatures of ME/CFS have been deduced in the energy-producing machinery of cells, and in the DNA epigenetic code that controls the expression of all our information stored in our genes. These changes could directly explain some of the deficits in brain function.

Warren P Tate FRSNZ CNZM

Emeritus Professor of Biochemistry
University of Otago

August 31st 2021

Department of Biochemistry

PO Box 56, Dunedin, New Zealand
Tel 64 3 479 7863 • Fax 64 3 479 7866 • Email biochemistry@otago.ac.nz
www.otago.ac.nz/biochemistry

UNIVERSITY of OTAGO

Te Whare Wānanga o Otāgo

Appendix: **My recent International Publications on the Biological basis of ME/CFS**

Helliwell A., Stockwell P., Edgar C[1]., Chatterjee A., & **Tate W**. (2021) Dynamic Epigenetic Changes during a Relapse and Recovery Cycle in Myalgic Encephalomyelitis/Chronic Fatigue Syndrome -submitted to *Clinical Epigenetics* – August

Wood, E., Hall, K & **Tate W** (2021) Role of mitochondria, oxidative stress and the response to antioxidants in myalgic encephalomyelitis/chronic fatigue syndrome: A possible approach to SARS-CoV-2 'long-haulers'? *Chronic Disease and Translational Medicine* 7: 14- 26, doi.org/10.1016/j.cdtm.2020.11.02

Helliwell, A., Sweetman E., Stockwell, P., Edgar, C, Chatterjee, A & **Tate W**. (2020) Changes in DNA methylation profiles of myalgic encephalomyelitis/chronic fatigue syndrome patients reflect systemic dysfunctions. *Clinical Epigenetics* 12: 167, doi.org/10.1186/s13148-020-00960-z

Sweetman, E., Kleffmann, T., Edgar, C., deLange, M., Vallings, R. & **Tate W** (2020) A SWATH-MS analysis of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome peripheral blood mononuclear cell proteomes reveals mitochondrial dysfunction *Journal of Translational Medicine* 18:365, https://doi.org/10.1186/s12967-020-02533-3

Sweetman, E., Ryan, M., Edgar, C., MacKay, A., Vallings, R., & **Tate, W**. (2019). Changes in the transcriptome of circulating immune cells of a New Zealand cohort with myalgic encephalomyelitis/chronic fatigue syndrome. *International Journal of Immunopathology & Pharmacology*, *33*, 1-8. doi: 10.1177/2058738418820402

Department of Biochemistry

PO Box 56, Dunedin, New Zealand
Tel 64 3 479 7863 • Fax 64 3 479 7866 • Email *biochemistry@otago.ac.nz*
www.otago.ac.nz/biochemistry