ME/CFS
The biomedical basis, diagnosis, treatment and management

International Research Symposium

PROGRAMME
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Welcome

I am so thrilled to be welcoming everyone - keynote speakers, guests and delegates - to Geelong, Victoria for the 2019 Emerge Australia International Research Symposium. Firstly I’d like to thank my team at Emerge Australia for their dedication and hard work in, not only giving information and support to so many people with ME/CFS everyday, but in addition to all the many things that we do every day - for stepping up and managing to organise this conference.

I need to extend a special thanks to Danielle Griffin - COO of Emerge Australia - who worked tirelessly to get everything from finalised abstracts, web registration systems, conference food through to live streaming & video production, organised so that we can be joined by delegates around the world.

I would also like to extend my thanks to the scientific advisory committee established to select the keynote speakers and to consider abstracts. Associate Professor Brett Lidbury from ANU and Professor Paul Fisher from La Trobe University deserve a special thank you for going above and beyond throughout the process of organising the conference.

Finally, I’d like to thank the Dept of Health for supporting this conference with a generous grant. It has enabled us to put together a truly international conference which, in conjunction with the recent NHMRC Advisory Committee into ME/CFS, will genuinely drive progress in this field. By doing this we, and the scientific community, offer hope to everyone in Australia and around the world struggling with ME/CFS.

From,
Dr Heidi Nicholl
CEO of Emerge Australia
General Information

Registration

The registration desk will be manned 1 hour prior to the commencement of each day’s Program.

When you register you will be provided with a lanyard and a name badge. We ask that you wear your lanyard at all times during the conference to assist with us identifying delegates and assisting with you getting to know each other.

Streaming & Social Media

@EmergeAustralia
#MEcfsconference
https://www.facebook.com/EmergeAustraliaInc

Online Symposium Delegates

To support members of the ME/CFS community who are unable to attend the Symposium, the proceedings of Day 1, 2 and 3 will be streamed live via Emerge Australia’s facebook and through GoLive at:


Following the conference videos of presentations will be uploaded to the Emerge Australia Vimeo Channel.

What if I have questions?

Unfortunately, we could not make it logistically possible for our online audience to have their questions answered during the broadcast. However we encourage people to post Questions & Comments as they watch. This content will be reviewed and every effort will be taken to address questions through the Symposium Summary Reports on Day 3.
Social Events

We encourage you to take this great opportunity to get to know your fellow delegates and the event speakers. Please find more information on social activities below.

TUESDAY 12TH MARCH
Welcome Reception

Join speakers and fellow delegates as we informally welcome everyone to the Symposium with drinks and a BBQ.

Schedule:
- 6 00 PM Bar Open
- 6 15 PM Welcome Toast
- 6 30 PM BBQ Dinner Service

Location:
Limeburners Room

THURSDAY 14TH MARCH
Symposium Dinner

At the end of Day 2 the Symposium Dinner is a great opportunity to compare notes. We will also have awards for ‘best in conference’.

Schedule:
- 7 00 PM Start
- 11 00 PM End

Location:
Peninsula Room
**Excursion: Wine, Surf & Dine**

This excursion will provide an opportunity to taste and view a few delights of the Great Ocean Road and Bellarine Peninsula region. The excursion begins after the conclusion of the Wednesday program. The cost is $25. Places are limited so please let us know as soon as possible if you will be joining us, payment can be made at the registration desk.

### Schedule:

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
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<tbody>
<tr>
<td>3:30 PM</td>
<td>Tour Bus departs Novotel</td>
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<tr>
<td>4:10 PM</td>
<td>Arrive at Bell Brae Estate for wine tasting &amp; “Tour of the Vines“</td>
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<tr>
<td>5:00 PM</td>
<td>Departure</td>
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<tr>
<td>5:20 PM</td>
<td>Arrive at Bells Beach</td>
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<td>“<strong>Bells Beach is an iconic international surf beach, located near Torquay on the southern coast of Victoria in the Great Ocean Road region. High cliffs provide a dramatic backdrop to the natural amphitheatre of the beach and large swells from the Southern Ocean, which slow down and steepen over the reef-strewn shallows, create the outstanding surf. Bells Beach is a popular spot with great vantage points along the cliff. The beach is an exposed reef and point break with excellent right hand breaks, at their best during autumn and winter.”</strong>”</td>
</tr>
<tr>
<td>5:50 PM</td>
<td>Depart Bells Beach</td>
</tr>
<tr>
<td>6:30 PM</td>
<td>Arrive ‘At The Heads’, Barwon Heads</td>
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<td>“<strong>The holiday town of Barwon Heads is located on the southern coast of the Bellarine Peninsula. Barwon Heads is a popular seaside community, offering a diverse variety of coastal attractions from the shallow and sandy shoreline of the Barwon River to the windswept surf beaches which front Bass Strait. You will dine ‘At The Heads’ restaurant, iconically located on the jetty at Barwon Heads, it has unparalleled views of Barwon Heads through to the Heads.”</strong>”</td>
</tr>
<tr>
<td>8:30 PM</td>
<td>Bus departs to return to Novotel</td>
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## Day 1 Agenda

### Symposium Opening w/ Emerge Australia CEO Dr Heidi Nicholl
8:30 AM - 8:40 AM

### SESSION 1

**Mitochondrial Function & Signalling**
Chair: Prof. Sonya Marshall-Gradisnik and Prof. Donald Staines

- "Specific mitochondrial respiratory defects and compensatory changes in immortalized ME/CFS patient lymphocytes."
  8:40 AM - 9:30 AM
- "Cellular Bioenergetics in ME/CFS."
  9:30 AM - 10:00 AM

**Prof. Paul Fisher**, LaTrobe University, Australia

**Dr Cara Thomas**, Newcastle University, United Kingdom

### Morning Tea
10:00 AM - 10:20 AM

### SESSION 2

**Immunology**
Chair: Prof. Paul Fisher

- "Immunological and calcium signalling for defining Chronic Fatigue Syndrome/ Myalgic Encephalomyelitis."
  10:25 AM - 11:25 AM
- "From Gene Expression to Multisystem Regulation: Identifying Optimal Treatment Courses for Complex Chronic Illnesses."
  11:25 AM - 12:05 PM

**Prof. Sonya Marshall-Gradisnik & Prof. Donald Staines**, National Centre for Neuroimmunology and Emerging Diseases at Griffith University, QLD

**Dr Travis Craddock**, Nova South Eastern University, Florida
“Abstract Presentation - Differences between immunophenotype of B-cells from healthy controls and patients with ME/CFS persist following in vitro culture and correlate with energy metabolisms.”

Prof. Geraldine Cambridge, University College of London, UK

Lunch 12:25 PM - 1:10 PM

SESSION 3  Neuroimaging  Chair: Dr Paul Gooley

“Brain stem myelination and MRI changes in CFS/ME.”

Dr Leighton Barnden, National Centre for Neuroimmunology and Emerging Diseases, Griffith University QLD

“Mapping fatigue in the brain in paediatric chronic fatigue syndrome.”

Dr Elisha Josev, Murdoch Children’s Institute of Research

SESSION 4  Biobanking & Clinical Data  Chair: Dr Elisha Josev

Awaiting Title

Dr Luis Nacul, London School of Hygiene & Tropical Medicine

“Rethinking Diagnostic Reference Intervals for ME/CFS via Machine Learning, and the Utility of Activin B to Assess Symptom Severity”

Assoc. Prof. Brett Lidbury, The Australian National University, Canberra
Day 2 Agenda

**Day 2 Welcome** w/ Emerge Australia CEO Dr Heidi Nicholl  
8:30 AM - 8:40 AM

**SESSION 1**  
**Research Innovation, Big Data, Bioinformatics**  
Chair: Dr Alice Richardson

“Establishing new mechanistic and diagnostic paradigms for ME/CFS”  
8:40 AM - 9:30 AM  
Dr Ron Davis, Director of the Stanford Genome Technology Centre.

“The Severely Ill Patient Study of ME/CFS”  
9:30 AM - 10:10 AM  
Dr Wenzhong Xiao, Assistant Prof. of Bioinformatics at Harvard Medical School

“Metabolic Traps in ME/CFS”  
10:10 AM - 11:00 AM  
Dr Robert Phair, Integrative Bioinformatics Inc

**Morning Tea**  
11:00 AM - 11:25 AM

**SESSION 2**  
**Metabolomics and Transcriptomics**  
Chair: Dr Neil McGregor

“Longitudinal metabolomics research”  
11:25 AM - 12:05 PM  
Dr Christopher Armstrong, Open Medicine Foundation

“Transcriptome analysis of QFS and CFS in the Netherlands.”  
12:05 PM - 12:45 PM  
Ruud Raijmakers, M.D., PhD Candidate Radboud University Medical Center
<table>
<thead>
<tr>
<th>Session</th>
<th>Topic</th>
<th>Time</th>
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<tbody>
<tr>
<td>Lunch</td>
<td>12:45PM - 1:30PM</td>
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<tr>
<td>SESSION 3</td>
<td><strong>Genomics &amp; Proteomics</strong></td>
<td>1:30PM - 2:10PM</td>
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<tr>
<td></td>
<td>&quot;An Omic Analysis of ME/CFS: An assessment of potential mechanisms&quot;</td>
<td>Dr Neil McGregor, Melbourne University</td>
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<td>&quot;Role of Stress-Activated Circulating MicroRNAs in ME/CFS Pathophysiology&quot;</td>
<td>Dr Alain Moreau, Université de Montréal, Québec, Canada</td>
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<td></td>
<td>Awaiting Title</td>
<td>Dr Jonas Bergquist, Uppsala University, Sweden</td>
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<tr>
<td>Afternoon Tea</td>
<td>3:40PM - 4:00PM</td>
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<tr>
<td>SESSION 4</td>
<td><strong>ME/CFS Methodology, Computer Interaction &amp; Collaboration</strong></td>
<td>4:00PM - 4:15PM</td>
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<td>&quot;Using N-of-1 methods to identify patterns and predictors of ME/CFS symptom exacerbations&quot;</td>
<td>Dr Suzanne McDonald, The University of Queensland</td>
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<td>&quot;ME/CFS Research in Human-Computer Interaction&quot;</td>
<td>Dr Ryan Kelly &amp; Melissa Rogerson, Interaction Design Lab, School of Computing and Information Systems, The University of Melbourne</td>
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<td></td>
<td>The European Network on Myalgic Encephalomyelitis/Chronic Fatigue Syndrome - how best to connect research and healthcare across Europe</td>
<td>Dr Eliana Lacerda</td>
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# Day 3 Agenda

## Welcome with Emerge Australia CEO Dr Heidi Nicholl

**9:50 AM - 10:00 AM**

### SESSION 1

**Clinical Experience**

*Chair: Dr Heidi Nicholl*

<table>
<thead>
<tr>
<th>Event</th>
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<tbody>
<tr>
<td>Awaiting Title</td>
<td>10:00 AM</td>
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<tr>
<td></td>
<td>10:40 AM</td>
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<tr>
<td><strong>Dr Mark Donohoe</strong>, Mosman Integrative Medicine, NSW</td>
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<tr>
<td>Awaiting Title</td>
<td>10:40 AM</td>
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<tr>
<td></td>
<td>11:20 AM</td>
</tr>
<tr>
<td><strong>Dr Bruce Wauchope</strong>, Bedford Medical Clinic, SA</td>
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<tr>
<td><strong>Clinical Q&amp;A</strong> w/ Dr Donohoe, Dr Bruce Wauchope, Dr Don Lewis, Dr Lisa McLendon-Smith &amp; Dr Nicole Phillips</td>
<td>11:20 AM</td>
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<td>12:00 PM</td>
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### Coffee Break

**12:00 PM - 12:15 PM**

## SESSION 2

**Day 1 & 2 Symposia Report Back**

<table>
<thead>
<tr>
<th>Event</th>
<th>Time</th>
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<tbody>
<tr>
<td>&quot;Day 1 Report&quot;</td>
<td>12:15 PM</td>
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<tr>
<td>12:35 PM</td>
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<tr>
<td><strong>Daniel Missalidis</strong>, Latrobe University</td>
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<tr>
<td>&quot;Day 2 Report&quot;</td>
<td>12:35 PM</td>
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<tr>
<td>1:00 PM</td>
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<tr>
<td><strong>Assoc. Prof. Brett Lidbury</strong>, ANU</td>
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<tr>
<td>Time</td>
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<tr>
<td>1:00 PM</td>
<td>Lunch</td>
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<tr>
<td>1:00 PM</td>
<td><strong>SESSION 3</strong> Patient &amp; Researcher Workshop</td>
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<tr>
<td>1:40 PM</td>
<td>&quot;Establishing new mechanistic and diagnostic paradigms for ME/CFS&quot;</td>
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<tr>
<td>2:20 PM</td>
<td>Dr Ron Davis, Director of the Stanford Genome Technology Center</td>
</tr>
<tr>
<td>2:20 PM</td>
<td>&quot;Research Update&quot; Video Screening</td>
</tr>
<tr>
<td>2:20 PM</td>
<td>Dr Jarred Younger</td>
</tr>
<tr>
<td>2:20 PM</td>
<td>&quot;Empathic design and ME/CFS: A conversation&quot;</td>
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<tr>
<td>2:20 PM</td>
<td>Anna Kerr, Patient &amp; Anthony Bloxas, Architect, Bloxas Design</td>
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<tr>
<td>2:20 PM</td>
<td>&quot;2 x Breakout Sessions with tables of researchers and patients,</td>
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<tr>
<td></td>
<td>facilitated discussions and Q&amp;A.&quot;</td>
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<tr>
<td>3:50 PM</td>
<td><strong>Symposium Close w/ Sally Missing</strong></td>
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<td></td>
<td>Coffee &amp; tea will be available until 5:00 PM</td>
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Keynote Speaker Bios

Thanks to the guidance of Australia’s leading ME/CFS researchers, Emerge Australia has confirmed an impressive line up of local and international keynote speakers including Dr Ron Davis, Director of the Stanford Genome Technology Center and ME/CFS Scientific Advisory Board director of the Open Medicine Foundation, and Dr Luis Nacul, who leads the ME/CFS Biobank UK and CureME Team at the London School of Hygiene and Tropical Medicine, UK.

We will also be joined by leaders in the field of ME/CFS research from Australia including scientists from the National Centre for Neuroimmunology and Emerging Diseases, Bio 21 in Melbourne, La Trobe University, the Murdoch Children's Research Institute and the ANU, amongst others.

Special Guest:
Dr Ron Davis
Professor of Biochemistry & Genetics
Director of the Stanford Genome Technology Center and ME/CFS Scientific Advisory Board Director of Open Medicine Foundation

Dr Ron Davis is Director of the Stanford Genome Technology Center and a Professor of Biochemistry and of Genetics at the Stanford School of Medicine in Stanford, California. Dr Davis is considered to be a world leader in biotechnology, and in the development and application of recombinant DNA and genomic methodology to biological systems. His laboratory has developed many of the techniques currently used in academic and industrial biotechnology laboratories, in particular, he was instrumental in the development of DNA microarray technology.

He is also considered to be a world expert in the electron microscopy of nucleic acids and has developed many of the mapping methods. For this, he has received the Eli Lilly Award in Microbiology in 1976. His laboratory was also instrumental in the development of lambda vectors, which were commonly used for the primary cloning of DNA molecules in E. coli. He has
also developed many of the yeast vectors and helped to develop yeast as a host for recombinant DNA. For this, he received the United States Steel Award in 1981, presented by the National Academy of Sciences.

In 1983, Dr Davis became a member of the National Academy of Sciences. He has also helped advance the United States’ policy on recombinant DNA and was a co-signer of the 1973 letter, alerting researchers to the potential hazards of recombinant DNA.

Dr Davis participated in writing the NIH guidelines for recombinant DNA and was very active in the downgrading of these guidelines. He participated in the dissemination of recombinant DNA techniques by teaching a course in Bacterial Genetics at Cold Spring Harbor from 1976 to 1981 and wrote the first manual on genetic engineering techniques, published by the Cold Spring Harbor Lab Press. Dr. Davis was a co-author on a publication that first described a new approach for conducting human genetics and for the construction of a human genetic linkage map. For this, he received the Rosentiel Award for Work in Basic Medical Research.

Dr Davis and his research team are developing new technologies for the genetic, genomic, and molecular analysis of model organisms and humans with a focus on clinical medicine for which he received the 2004 Sober Award from the American Society for Biochemistry and Molecular Biology (ASBMB/IUBMB). In addition, the last nine years he has had the opportunity to participate as Principal Investigator and a member of the steering and advisory committee in one of the largest collaborative projects funded by the National Institute of General Medical Sciences (NIGMS): “Inflammation and the Host Response to Injury,” a project that drove Dr Davis to become very familiar with clinical approaches and actively participate in the decision process of bridging basic science technology development and implementation of novel methodologies to the biomedical field.
Christopher Armstrong, PhD, is most well-known for his research using metabolomics to observe biochemical alterations in ME/CFS patients. Chris published the first ME/CFS metabolomics study on blood and urine in 2015. These studies recognised an alteration in energy, amino acid, purine and oxidative metabolism in ME/CFS patients. In 2017, this work was followed up by observing how these alterations in metabolism were related to changes in gut bacteria and their metabolites. Since then Chris has set up collaborative efforts to apply metabolomic experiments to immunological experiments on ME/CFS, observing how the metabolome may relate to immune cell alteration. He is also focused on cell metabolism and longitudinal research in ME/CFS while looking to extend metabolic capabilities across the field of ME/CFS to help collate different groups researching ME/CFS. Dr Armstrong is a member of the Working Group, which offers their expertise and resources to the ME/CFS Collaborative Research Center at Stanford University.

Dr Barnden started his career in Nuclear Medicine, but for the last 25 years has specialised in medical image processing. His publications on brain 'structural' MRI imaging in CFS reported abnormal autonomic nervous system function and depleted brain stem myelin levels.

Prof. Bergquist leads a research group focused on the development of analytical tools for screening and discovery of biomarkers for various pathological states. Prof. Bergquist’s group works to increase understanding of what initiates the disease process to enable early diagnosis. Prof. Bergquist is a member of the Working Group, which offers their expertise and resources to the ME/CFS Collaborative Research Center at Stanford University.
Dr Travis Craddock

Nova South Eastern University, Florida

Travis J.A. Craddock, Ph.D. is an Associate Professor in the Departments of Psychology & Neuroscience, Computer Science and Clinical Immunology at Nova Southeastern University (NSU) in Fort Lauderdale, Florida. He serves as the Director of the Clinical Systems Biology group at NSU’s Institute for Neuro-Immune Medicine where he applies computational systems biology and biophysics methods towards the purpose of identifying novel treatments for complex chronic illness involving neuroinflammation. Dr. Craddock received his PhD in the field of biophysics at the University of Alberta where his graduate research activities focused on subneural biomolecular information processing, and nanoscale neuroscience descriptions of memory, consciousness and cognitive dysfunction in neurodegenerative disorders. His current research activities are focused on using a theory driven approach to understand the underlying molecular regulation of chronic illness resulting from exposure to neurotoxins, such as anesthesia and nerve agents, or viral infection in order to improve diagnosis and putative treatment strategies. This work is primarily funded by the U.S. Department of Defense and the National Institutes of Health.

Prof. Paul Fisher

Microbiologist
BSc (Hons), MSc, PhD, Head of Microbiology, Department of Physiology, Anatomy and Microbiology, La Trobe University, Melbourne.

Prof. Fisher studies neurodegenerative disease, mitochondrial biology and the roles of mitochondria in disease, using 2 model systems to understand the cytopathological pathways involved. He has advanced expertise in mitochondrial & molecular biology, biochemistry and cell physiology, including the complete array of modern molecular techniques as well as in the diverse statistical methods in the data analysis.

Dr Elisha Josev

PhD M.Psych (Clinical Neuropsychology), Murdoch Children’s Research Institute

Dr Elisha Josev is an early career postdoctoral researcher at the Murdoch Children’s Research Institute. She holds a PhD and a Masters of Psychology (Clinical Neuropsychology) from the University of Melbourne, and is a registered psychologist. She has a
Dr Brett Lidbury

Associate Professor
National Centre for Epidemiology and Population Health, Research School of Population Health, The Australian National University (ANU) College of Health and Medicine.

Dr Lidbury came to ME/CFS research via fundamental experimental investigations into arboviral pathogenesis, particularly for Ross River Virus (RRV), a long-suspected pathogen in Australian cases of ME/CFS. These fundamental studies revealed several mechanisms of immune manipulation and evasion associated with host macrophages, including RRV persistence strategies, and the ablation of early pro-inflammatory activity post antibody-dependent enhancement (ADE). A relationship with CFS Discovery (Melbourne) commenced in 2010 – 11, and led to the completion of two projects – the first a pilot study funded by the Alison Hunter Memorial Foundation, and a recently completed project funded by the Judith J. Mason Foundation. Together, the results of these projects revealed activin B as a serum biomarker of significant potential, as well as the development of the weighted standing time (WST) as a simple proxy for ME/CFS symptom severity. Additionally, important observations were made on co-morbid POTS, and via machine learning and statistical analyses, redefinition of routine pathology markers as screening tools.

Prof. Sonya M. Marshall-Gradisnik

BS (Hons), PhD, co-director of the National Centre for Neuroimmunology and Emerging Diseases (NCNED), Menzies Health Institute QLD, Griffith University, Queensland, Australia

Professor Marshall-Gradisnik’s team and expertise is in CFS/ME with particular focus on Natural Killer Cell, cell signalling pathways, immune cell functions and signalling, gene expression and ion channel function.

Dr Neil McGregor

(BDS, MDSc, PhD), Periodontist
Honorary Senior Fellow, University of Melbourne, Faculty of Medicine, Dentistry and Health Sciences

Dr McGregor is a highly experienced ME/CFS researcher, having co-authored more than 40 papers in ME/CFS, over a period of more than 20 years. He has been involved in research both in Australia and in the U.S. including chronic pain aetiology.

special interest in the neuropsychological effects of early disruption to normal brain development, particularly in children born preterm. She also investigates the impact of chronic health conditions on child development, and currently coordinates a paediatric chronic fatigue syndrome research program at MCRI.
Dr Alain Moreau
PhD, is a Professor in both the Department of Stomatology, Faculty of Dentistry and Dept of Biochemistry and Molecular Medicine, Faculty of Medicine, at Université de Montréal, Québec, Canada

Dr Moreau’s chief interests of study are pediatric scoliosis, osteoarthritis, osteoporosis, and Myalgic Encephalomyelitis. Dr Moreau is a member of the Working Group, which offers their expertise and resources to the ME/CFS Collaborative Research Center at Stanford University. Professor Moreau’s research team has developed an innovative test to reproduce post-exertional malaise, which led to a molecular stratification of ME/CFS patients using a panel of circulating microRNAs. He is investigating genetic and epigenetic determinants of ME/CFS.

Dr Luis Nacul
Clinical Associate Professor
London School of Hygiene and Tropical Medicine, UK

Dr Nacul leads the ME/CFS Biobank UK and CureME Team. The team is driving research for the recognition, diagnosis and treatment of ME and CFS. He serves as a Member Substitute for EUROMENE, an international not for profit organization for ME/CFS research. In 2018 Dr Nacul was appointed to the review committee for the NICE guidelines for ME/CFS used by the British NHS.

Dr Robert D. Phair
PhD, Co-founder and Chief Science Officer, Integrative Bioinformatics, Inc

Dr Phair is an MIT electrical engineer with a PhD in (cardiovascular) physiology. After post-docs in computer modelling and cellular endocrinology, he was a professor of physiology and of biomedical engineering at The Johns Hopkins School of Medicine for 16 years. In 2001, he co-founded Integrative Bioinformatics Inc, a scientific consulting and software development firm in Silicon Valley where he is Chief Science Officer. Dr Phair has built mechanistic computer models and performed experiments at levels of biological organization from physiology to biochemistry to...
molecular cell biology. About 3 years ago he met Dr Ron Davis and joined the quest to cure ME/CFS. Dr Phair is the originator of the metabolic trap concept and will tell us about Metabolic Traps in ME/CFS.

**Prof. Donald R. Staines**

**MBBS, MPH, FAFPHM, FAFOEM**

is a Clinical Professor at Menzies Health Institute Queensland and the co-director (alongside Professor Sonya Marshall-Gradosnik) of the National Centre for Neuroimmunology and Emerging Diseases (NCNED), Menzies Health Institute Queensland, Griffith University, Queensland, Australia

Professor Staines’ research team is working towards discovery of diagnostic tools and treatments for chronic fatigue syndrome.

**Dr Cara Tomas**

Research Associate Newcastle University, UK

Dr Cara Tomas completed a master’s degree looking at the role of pyruvate dehydrogenase in ME/CFS before completing her PhD in 2018 researching the role of cellular bioenergetics in ME/CFS. Cara is also a ME/CFS patient and therefore can approach the subject from both a researcher and from a patient perspective. Her publications in ME/CFS cover various aspects of the disease including HPA axis dysfunction, cardiac abnormalities, mitochondrial function, and biomarkers.

**Dr Wenzhong Xiao**

Ph.D. Assistant Professor
Bioinformatics at Harvard Medical School and director of the Inflammation & Metabolism Computational Center at Massachusetts General Hospital.

As a world expert in computational genomics, Dr Xiao develops bioinformatic and statistical tools for use in understanding human diseases, especially in studies of immuno-metabolic response. He focuses on integrative analysis and interpretation of multi-dimensional molecular, cellular, and clinical data of many types of patients, including those with ME/CFS.

**Dr Lisa McLindon-Smith**

Clinician

Dr McLindon-Smith is a qualified Medical Practitioner and Teacher. After postgraduate years in hospital medicine, Dr Lisa then went on to train in Naprotechnology, and became Melbourne’s first Medical Consultant in this field. Her areas of expertise in private practice have been recurrent miscarriage, subfertility, infertility and women’s health. Further areas of long term interest are ME/CFS, adrenal fatigue, chronic pain, & the treatment of the secondary anxiety and depression often associated with these conditions.
Dr Lisa has suffered herself from ME/CFS since the age of seventeen. Having had to reduce her clinical hours of recent years she is now busy perfecting both the art & science of managing ME/CFS, and a busy family life. She is a wife to Christopher, and mother of their five beautiful children.

Dr Nicole Phillips

Clinician

Dr Phillips is a psychiatrist in private practice in Armadale, Victoria. She graduated from Auckland Medical School, then completed diplomas in family planning and obstetrics in Melbourne before entering psychiatry specialty training. She has been a fellow of the Royal Australian & N.Z. College of Psychiatrists since 1994. Nicole’s main professional interests are women’s mental health, medicolegal assessments and ME/CFS.

In the 1980s & 1990s she established and ran an innovative psychiatric referral and consultation service at the Mercy Hospital for Women whilst working on the Mother-Baby Unit for post-natal depression. She was thrown into the world of ME/CFS in 1989, when she developed the illness, and after her recovery some years later, vowed to help others with the illness.

From 2001-2013, she was medical advisor to ME/CFS Australia (now Emerge Australia), and was medical editor of The Emerge Journal. Nicole has spoken at many meetings and conferences, addressed a parliamentary forum, and has been interviewed by the media many times, including ABC’s Mind Matters. She has a number of CFS patients in her practice. Her approach to the management of all her patients, no matter what the diagnosis, is holistic.

Dr Don Lewis

Clinician

Dr Lewis’s interest in ME/CFS began in 1985 and has steadily progressed since that time. In 2001, the CFS Discovery clinic was established, being a facility where attention is directed solely to the management of people with this illness. In addition to establishing a protocol leading to diagnosis and relevant investigations to uncover the mechanism of this illness, patients attending also became ready subjects for many research projects that are covered in the past than currently in conjunction with five universities. This has led to the formation of ME/CFS Discovery Research Network (MDRN), which is forming the harbour supporting these tertiary institutes in their current and foreseeable research, and providing information of the advanced research work conducted by various bodies in Melbourne and Australia.
DAY 1
Session 1: Mitochondrial function and signalling

“Specific mitochondrial respiratory defects and compensatory changes in immortalized ME/CFS patient lymphocytes.”

Speaker: Professor Paul Fisher, Latrobe University, VIC, Australia

Specific mitochondrial respiratory defects and compensatory changes in immortalized ME/CFS patient lymphocytes.

Daniel Missailidis1, Oana Sanislav1, Claire Y. Allan1, Brett A. Lidbury2, Don Lewis3, Sarah J. Annesley1 and Paul R. Fisher1.

1Discipline of Microbiology, Department of Physiology Anatomy and Microbiology, La Trobe University, Melbourne, Australia; 2National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australia; 3CFS Discovery, Melbourne, Australia.

Mitochondrial dysfunction has long been suspected as an underlying cytopathological agent in ME/CFS, but the nature of the suspected defect remains unclear. We used Seahorse respirometry of immortalized patient lymphocytes (lymphoblasts) to show that in intact, metabolically active ME/CFS cells, mitochondrial ATP synthesis by Complex V is inefficient, representing a significantly lower proportion of the basal mitochondrial respiratory activity. However, absolute ATP synthesis rates (pmol/min) were not significantly lower than in control cells, while glycolysis rates and steady state ATP levels were unchanged. In order to achieve this, our results suggest that ME/CFS cells compensate for the reduced efficiency of ATP synthesis by upregulating mitochondrial respiratory capacity and shifting the pattern of use of oxidizable substrates, consistent with increased utilization of fatty acids and amino acids, as suggested by previous metabolomics studies. Thus, we found significant increases in maximum respiratory capacity, including Complex I activity, and the use of the proton motive force in processes other than ATP synthesis (the “proton leak”) as well as the expression of mitochondrial respiratory proteins and proteins involved in mitochondrial amino acid and fatty acid oxidation. “Nonmitochondrial” O2 consumption by other cellular enzymes was also elevated. The compensatory upregulation of these various respiratory functions could be driven by either or both cellular stress-sensing kinases, TORC1 (whose activity was increased) and AMPK (whose expression was elevated). However, the mitochondrial membrane “mass” and genome copy number were unchanged. Thus ME/CFS cells do not have “more” mitochondria, but their mitochondria have greater respiratory capacity. This increased capacity is underutilized because of the Complex V defect, so that the respiratory “spare capacity” was increased and the mitochondrial membrane potential was elevated. The altered respiratory and TORC1 activities in ME/CFS lymphoblasts were correlated with the disease severity in the patients, as measured by Richardson and Lidbury’s Weighted Standing Time.

“Cellular Bioenergetics in ME/CFS”

Speaker: Dr Cara Thomas, Newcastle University, UK

Abnormalities in bioenergetic function have been cited as one possible cause for ME/CFS. One hypothesis to explain this suggests that ME/CFS may be caused, at least in part, by an acquired mitochondrial dysfunction.

We used the Seahorse XF®96 analyser to assess the two major cellular energy pathways (OXPHOS and glycolysis) in real-time with live cells. A series of assays were conducted using peripheral blood mononuclear cells (PBMCs) from healthy controls and both moderately and severely affected ME/CFS patients to investigate different aspects of cellular bioenergetic function. We look at the effect of disease severity, cell freezing, and glucose concentration on PBMC energy production.

CFS PBMCs were shown to have significantly lower mitochondrial respiratory rates than control cells for key parameters such as basal respiration, maximal respiration, and reserve capacity. These results were consistent in all experimental conditions used. Importantly, these results suggest that CFS PBMCs perform closer to their maximum under normal conditions. This means that when CFS PBMCs come under stress they are less able to increase their respiration rate to compensate for the increase in stress. No differences in terms of glycolytic function were detected between the control and ME/CFS cohorts.
Investigations were then conducted into the validity of the standard calculations used for some of the glycolytic and OXPHOS parameters.

The metabolic differences discovered highlight the inability of CFS patient PBMCs to fulfil cellular energetic demands both under normal conditions and when mitochondria are stressed. Results suggest that abnormalities in the bioenergetic function of PBMCs may be the cause, or a consequence, of CFS. These findings provide an interesting starting point for investigations into cellular bioenergetics in CFS.

Session 2: Immunology

"Immunological and calcium signalling for defining Chronic Fatigue Syndrome/Myalgic Encephalomyelitis"

Speakers: Sonya Marshall – Gradisnik & Donald Staines, National Centre for Neuroimmunology and Emerging Diseases at Griffith University QLD

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Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME) is a disorder with hallmarks of varying changes in immune cells. Investigations of natural killer (NK) phenotypes, dendritic cells (DCs), neutrophils, B cells, T cells, γδT cells and Tregs as well as cytotoxic activity, expression of cell surface receptors, adhesion molecules, intracellular proteins and cytokine secretion have been reported for CFS/ME patients. MicroRNA investigations have also provided potential insight into additional immunological and genetic markers for CFS/ME. However, cytokine profiles and immune cell phenotypes have produced equivocal results.

More recently calcium signalling and ion channel function have provided significant direction in understanding the pathology of CFS/ME. Collectively these studies are reviewed, suggesting comprehensive dysregulation of the immunological response in CFS/ME, suggesting impaired ion channel function in the pathology of this illness.

“From Gene Expression to Multisystem Regulation: Identifying Optimal Treatment Courses for Complex Chronic Illnesses”

Speaker: Dr Travis Craddock

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Discovering novel treatment strategies for complex chronic illnesses through traditional discovery pipelines is extremely expensive, carries a high probability of failure, and a lengthy cycle time. Furthermore, it is becoming clear that “one target, one treatment” solutions may not be capable of addressing difficult conditions. Repurposing Food and Drug Administration approved drugs offers a cost-effective solution with a significantly abbreviated timeline. Furthermore, combining multi-system modeling with these bioinformatics techniques can harness the regulatory dynamics of the human body to identify robust treatment courses that might produce lasting remission. Here it will be discussed how differentially expressed gene modules cross-referenced with drug atlas and pharmacogenomic databases can be used to identify targetable systems and agents. Based on these results it will be discussed how to construct a discrete ternary logic representation of signaling networks from physiological and biochemical literature to provide a qualitative description of multi-system behavior. By exploiting the regulatory dynamics of the resulting model through the application of a combinatorial optimization scheme and Monte Carlo simulation, it will be discussed how to predict treatment courses that might produce lasting disease remission. While the methods presented here can be applied generally, they will be discussed in the context of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome, a debilitating chronic multi-symptom disorder for which there is no known treatment.
“Differences between immunophenotype of B-cells from healthy controls and patients with ME/CFS persist following in vitro culture and correlate with energy metabolism”

Abstract Presenter: Professor Geraldine (Jo) Cambridge

Authors: Fane F.K Mensah, Christopher M. Armstrong, Geraldine Cambridge

Infection or other insult is generally assumed to be a major factor contributing to the pathophysiology of ME/CFS. We have described alterations in markers on peripheral blood (PB) B-cells from patients with ME/CFS which suggested abnormal differentiation of IgD+B-cells which we further linked with metabolic function [1; 2].

The aim was to test whether the immunophenotype and metabolic changes suggested by our studies of circulating B-cells persisted following in vitro activation by agonists inducing proliferation and differentiation.

PB B-cells were isolated from 8 patients with moderate ME/CFS and 6 age-matched Healthy Controls (HC) and cultured with either T-cell-dependent (TD: anti-CD40, Anti-IgM, IL2) or Toll-like-receptor agonists (TLR9D: CpG plus IL2, anti-IgM). Immunophenotype was determined on cells sampled at days 1, 3 and 6. Mitochondrial mass (MM) over 4 cycles of proliferation was calculated using Mitotracker-Red. Supernatants were tested for levels of glucose and lactate (1H NMR spectroscopy).

There was a highly significant persistence of CD24+B-cells from ME/CFS patients compared to HC, most evident in TD stimulated cultures. Significant correlations were found between CD24+B-cell frequencies and usage of glucose and production of lactate in vitro. MM was significantly lower (P<0.05) in ME/CFS B-cells over 4 cycles of proliferation induced by TLR9D-stimulation.

Immunophenotype and metabolite profiles of B-cells from HC and ME/CFS patients revealed different dynamics in response to interventions in vitro. Persistently lower mitochondrial mass of ME/CFS B-cells suggested decreased ability to respond to increased energy demands. Correlations between CD24+ B-cell frequencies and the usage of glucose and production of lactate confirmed a link between CD24 positivity of B-cells and energy metabolism. The dynamics of in vitro B-cell differentiation may therefore provide a platform for more focused research and possibly development into a diagnostic tool.


Session 3: Neuroimaging

“Brain stem myelination and MRI changes in CFS/ME”

Speaker: Dr Leighton Barnden, National Centre for Neuroimmunology and Emerging Diseases, Griffith Uni, Gold Coast, QLD

The brainstem lies between the brain proper and the body and is pivotal in regulating the unconscious functions of our bodies, including levels of arousal and cognitive performance, transition to sleep and duration and intensity of pain. It is also the seat of the autonomic nervous system which regulates heart rate, blood pressure, body temperature, breathing, gut and immune function, and muscle metabolism. We report CFS/ME brain MRI studies in which severity, heart rate and blood pressure data were also collected. Correlations of the MRI with these clinical measures in CFS/ME and healthy controls revealed abnormal dependences in CFS/ME and implicated impaired myelination and nerve conduction within the brainstem. This was also seen with a different approach and more recently connectivity between brainstem sites has been confirmed. Brainstem deficits may contribute to many of the symptoms of CFS/ME.

“Mapping fatigue in the brain in paediatric chronic fatigue syndrome”

Speaker: Dr Elisha Josev, Murdoch Children’s Research Institute, Royal Children’s Hospital

Cognitive dysfunction and fatigue following mental exertion is commonly reported in Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME). Emerging evidence in adults suggests that central nervous system dysfunction may underlie these core CFS/ME symptoms, yet this has rarely been measured objectively or longitudinally in the paediatric population. This talk will present the findings of a longitudinal research study which used resting-state functional MRI in a novel repeated-measures design to examine the impact of mental exertion on brain functioning and paediatric CFS/ME symptomatology. Intrinsic connectivity, cognitive function, and subjective fatigue were evaluated immediately before and after a period of cognitive exertion in adolescents with CFS/ME and healthy controls, at the time of diagnosis and again at 2-year follow-up. The findings and clinical implications to be discussed will be important for clinicians, patients, and school staff to
consider in understanding and managing this syndrome

Session 4: Biobanking & Clinical Data

Awaiting abstract

Speaker: Dr Luis Nacul, London School of Hygiene & Tropical Medicine

“Rethinking Diagnostic Reference Intervals for ME/CFS via Machine Learning, and the Utility of Activin B to Assess Symptom Severity”

Speaker: Assoc Prof. Brett Lidbury, The Australian National University, Canberra

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A paradox for ME/CFS is that while patients present with obvious physical disability, serum/blood/urine pathology markers fall within the reference intervals, and are classified as “normal”, encouraging some to suggest psychological aetiology, and assumptions that there are no biomedical explanations. Deeper analysis of pathology data via machine learning proposed a new method to interpret results, provide insights into biomedical mechanisms, and assist the screening of potential patients.

The random forest (RF) machine learning algorithm (R statistical programming, package - randomForest) was applied to aggregated pathology data for ME/CFS and healthy control (HC) participants recruited by CFS Discovery, and arranged into weighted standing time (WST – severity proxy) categories. RF analyses identified the following profile of routine pathology markers - lymphocyte count, serum urea, mean corpuscular haemoglobin (MCH), alkaline phosphatase (ALP) and 24-hour urinary creatinine excretion rate, as correctly classifying ME/CFS at an accuracy of 62 – 65%. Receiver operator characteristics (ROC) confirmed the capacity of this pathology test profile to discriminate between WST categories.

Serum activin B concentration significantly separated ME/CFS from HC cases. The inclusion of activin B in RF models did not improve general classification accuracy, but analyses via RF - ROC found that activin B improved the classification of mild to moderate cases. Participants correctly predicted as ME/CFS by RF were extracted, and these data utilised to determine reference intervals for each pathology test, and activin B.

Unique reference intervals for predictive routine pathology tests were generated via RF, with severity classification enhanced by serum activin B results. Combined with clinical assessments, the identification of specific pathology test profiles and reference intervals provides effective screening tools for clinicians.

DAY 2

Session 1: Research Innovation, Big Data, Bioinformatics.

“Establishing new mechanistic and diagnostic paradigms for ME/CFS”

Speaker: Dr Ron Davis

A significant portion of the Stanford Genome Technology Center’s advanced technologies are being focused on myalgic encephalomyelitis / chronic fatigue syndrome (ME/CFS), which afflicts up to an estimated two million people in the United States. There is currently no biological test to diagnose ME/CFS and as a result, diagnosing ME/CFS patients is a lengthy and costly process, constituting a fundamental impediment to patient care. This lag in diagnosis also erects barriers to research, complicating patient recruitment for clinical trials of new treatments. To remedy this situation, the SGTC is pioneering several areas of research on ME/CFS in order to search for a cause and establish a diagnostic that can be rapidly disseminated. Potential causes of ME/CFS are being sought by searching for traces of DNA from pathogens in patients’ blood and potential environmental toxins. Progress on a number of biomarker assays that have been developed at the SGTC will be presented. These biomarker assays analyze several physical properties of ME/CFS patients’ blood cells including both red and white blood cells. Current efforts named a “bake-off,” are directed towards comparing these different assays on individual patient samples and determining how consistently they perform in distinguishing patients from healthy controls.

“The Severely Ill Patient Study of ME/CFS”

Speaker: Dr Wenzhong Xiao

ME/CFS is a serious, long-term illness affecting multiple systems of the body. We conducted at Stanford a big data study to systematically
characterize the clinical phenotypes and multiple omics of severely ill patients as well as healthy controls. While the clinical phenotypes showed that the patients suffer from severe debilitating disease, no significant differences were found in detecting viruses and bacteria between the patients and the controls. Clinical lab tests, cytokine and metabolite results showed decreased levels of circulating neuroprotective molecules in the patients, some of which are correlated with the gut microbiome of the patients. Further, genome sequencing analysis revealed likely pathogenic variants in patients suggesting an interaction between genetic and environmental factors in ME/CFS.

“Metabolic Traps in ME/CFS”

Speaker: Robert Phair

This is a talk at the intersection of genomics, biochemistry, and nonlinear systems theory. There is a growing realization among systems biologists that ME/CFS has the characteristics of a bi-stable nonlinear system. Moreover, from the history of ME/CFS epidemics/outbreaks, we argue that genetic mutations predisposing to ME/CFS must be common, not rare. Hence, we searched the Stanford/OMF WGS data collected during the Severely Ill Patient Study (SIPS) for common mutations that appear to uncover metabolic bistability. To date, we have identified two genes carrying such mutations in our ME/CFS population: IDO2 and TYR. We have shown, using computer simulation, that the IDO system, which is the first step in the kynurenine pathway of tryptophan metabolism does indeed show bistability when IDO2 is non-functional. Both the IDO system and the TYR system are prominent in the regulation of the diffuse modulatory systems of the human midbrain. These systems result in what we have called metabolic traps and have the potential to account for ME/CFS symptoms. In collaboration with Drs. Fischer, Davis, and Wilhelmy at Stanford, we are subjecting the IDO metabolic trap theory to experimental tests and will provide an update on these results.

Session 2: Metabolomics and Transcriptomics

“Longitudinal metabolomics research”

Speaker: Dr Chris Armstrong

ME/CFS is a debilitating long-term disease that is multisystem symptomatic with key symptoms of post-exertional malaise (PEM), fatigue, sleep dysfunction, cognitive impairment and pain. The symptoms of ME/CFS vary from person to person and change over the course of the illness in both symptom type and severity. The illness is dynamic and personal and to understand a complex disorder without a biomarker it is pertinent to begin phenotyping individual relationships between symptom expression and metabolism overtime. Furthermore, PEM is a key feature of the illness and is represented as the altered normal response and repair to recover from stress or exertion.

Using metabolomics and longitudinal study design we can begin to characterise the role metabolites may play in signalling the underlying disrupted biology in people with ME/CFS. The extension of this work is the development of monitoring systems to define treatments specific to correct altered biochemical signatures – a personalised medicine approach.

“Transcriptome analysis of QFS and CFS in the Netherlands Background”

Speaker: Ruud Raijmakers

Q fever fatigue syndrome (QFS) is a well-documented state of prolonged fatigue following around 20% of acute Q fever infections. It has been hypothesized that low grade inflammation plays a role in its aetiology. In this study, we aimed to identify transcriptome profiles that could aid to better understand the pathophysiology of QFS.

Materials/methods: RNA of monocytes was collected from QFS patients (n = 10), chronic fatigue syndrome patients (CFS, n = 10), Q fever seropositive controls (n = 10), and healthy controls (n = 10) who were age- (± 5 years) and sex-matched. Transcriptome analysis was performed using RNA sequencing.

Results: Mitochondrial-derived peptide (MDP)-coding genes MT-RNR2 (humanin) and MT-RNR1 (MOTS-c) were differentially expressed when comparing QFS (-4.8 log2-fold-change P = 2.19 x 10-9 and -4.9 log2-fold-change P = 4.69 x 10-8), CFS (-5.2 log2-fold-change, P = 3.49 x 10-11 -4.4 log2-fold-change, P = 2.71 x 10-9), and seropositive control (-3.7 log2-fold-change P = 1.78 x 10-6 and -3.2 log2-fold-change P = 1.12 x 10-5) groups with healthy controls, resulting in a decreased median production of humanin in QFS patients (371 pg/mL; IQR 325 – 384), CFS patients (364 pg/mL; IQR 316 – 387), and asymptomatic Q fever seropositive controls (354 pg/mL; 292 – 393).

Conclusions: Expression of MDP-coding genes MT-RNR1 (MOTS-c) and MT-RNR2 (humanin) is decreased in CFS, QFS, and, to a lesser extent, in Q fever seropositive controls, resulting in a decreased production of humanin. These novel peptides might indeed be important in the pathophysiology of both QFS and CFS.
Session 3: Genomics & Proteomics

“An Omic Analysis of ME/CFS: An assessment of potential mechanisms”


Background: The presentation evaluates the metabolomes, genome and faecal microbiomes in relationship to symptoms expression. Onset studies have found that there is no consistent triggering event, in fact many of the triggering events seem to be quite different in mechanism. The onset studies have found that each of the different triggers will only induce an MECFS-like syndrome in a small percentage of cases effected by the trigger. This suggests that the sufferers of MECFS may have some common host response to multiple different stimuli. Analysis of the available data from multiple different university MECFS research groups will be presented to evaluate the underlying mechanisms. These show a common hypermetabolic state exists in the patients which affects not only glucose metabolism, muscle and connective tissue amino acid metabolism, cardiac pulse rates, renal concentrating abilities and gastrointestinal barrier function. These changes seem to be related to a hypoacetylation state which deregulates not only nuclear histone acetylation but also cytoplasmic enzymes regulation. Factors that exacerbate the illness, such as exercise, do alter these underlying hypermetabolic issues.

“Role of Stress-Activated Circulating MicroRNAs in ME/CFS Pathophysiology”

Speaker: Dr Alain Moreau, Université de Montréal, Québec, Canada

Background: Myalgic encephalomyelitis (ME)/chronic fatigue syndrome (CFS) is a complex chronic disease and whose etiology remains poorly understood. It is a debilitating condition characterized by unrelenting fatigue, post-exertional malaise, cognitive impairment, and musculoskeletal pain, which further amplify a wide variety of other symptoms. ME is very common in all populations and more than 560,000 Canadians are affected. ME is life altering and for its more severe forms, can be life threatening. Currently, there are no specific diagnostic tests and no cure for this disease exists. It is widely accepted now that ME/CFS pathophysiology results from a combination of predisposing genetic factors and environmental exposures. Indeed, there is growing recognition that ME/CFS is a serious illness rooted in fundamental metabolic dysfunctions that are impairing energy production, immune functions and physiological response to exertion. Given the clinical heterogeneity of the disease and sex differences, the timing of progression from an acute to chronic state varies between individuals from months or years or never occurs. Thus, we search for novel biomarkers that reflect the systemic pathophysiological state associated with ME/CFS with a focus on circulating microRNAs (miRNAs) that have gained increased attention in molecular medicine. The latter is a class of small non-coding RNA molecules that play an important role in the post-transcriptional regulation of gene expression in different human diseases. Although there have been significant advances, the function of miRNAs in ME/CFS still need further research while stress-activated miRNAs have never been explored opening a new promising frontier.

Hypothesis: We propose that miRNAs could be the link between environmental factors, genetic predisposition, and phenotypic differences observed in ME/CFS. Our central hypothesis is that ME/CFS is caused by a dysregulation of circulating miRNAs that modulate immune functions, energetic metabolism and physiological response to exertion facilitates the onset of ME/CFS and its range of symptoms.

Objective of the presentation: During the presentation, experimental data will be shown to demonstrate that stress-activated circulating miRNAs could be more informative about the pathophysiology of ME/CFS than those detected at baseline and could represent unique molecular signatures to better stratify individuals affected by ME/CFS to illuminate ME/CFS pathophysiology and could be used to identify actionable therapeutic targets.

Professor Moreau’s research program on ME/CFS is funded by The Sibylla-Hesse Foundation and approved by CHU Sainte-Justine IRB (protocol #4740)

Awaiting abstract

Speaker: Dr. Jonas Bergquist, Uppsala University, Sweden
Session 4: ME/CFS Methodology, Computer Interaction & Collaboration

“Using N-of-1 methods to identify patterns and predictors of ME/CFS symptom exacerbations”

Abstract Presenter: Dr. Suzanne McDonald, The University of Queensland

Authors: Shamima Banu, Samuel X. Tan, James McGree, Geoffrey Mitchell, Jane Nikles & Suzanne McDonald

Background: There is significant inter-individual variation in the type and severity of symptoms experienced by patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS). Most existing studies in the area of ME/CFS have used group-based research designs. However, these do not capture patterns and predictors of ME/CFS symptoms and exacerbations at the individual level. Quantitative N-of-1 methods, which involve repeated measurements of symptoms and triggers over time from the same individual, may provide a crucial perspective.

Questions: Can N-of-1 methods be used to explore fluctuations in ME/CFS symptoms over time and predictors of symptom exacerbations within individuals? Are N-of-1 studies acceptable to individuals with ME/CFS?

Methods: A series of quantitative N-of-1 observational studies lasting 6-12 weeks was conducted.

Results: To date, ten patients with an existing clinical diagnosis of ME/CFS have taken part in an n-of-1 study. Participants completed questionnaires assessing symptom severity and potential triggers three times per day via an electronic diary. Questionnaires were tailored to each participant’s unique symptoms and the factors they hypothesised to trigger their symptoms (e.g. sleep, stress, dehydration, high cognitive demand, emotional distress). Physical activity levels were objectively measured by the electronic diary’s inbuilt accelerometer. Correlations between triggers and symptoms were analysed using Bayesian dynamic regression modelling. After completing the study, participants were provided with personalised feedback about the patterns and predictors of their symptoms.

Conclusions: Symptom severity fluctuated considerably over time in all participants. There was no clear common pattern of symptom triggers across participants. Participants considered the personalised feedback session to be informative for the management of their symptoms. N-of-1 studies are feasible for identifying factors associated with ME/CFS symptom exacerbation on an individual level and they have potential clinical utility in informing highly personalised interventions for ME/CFS symptom management.

“ME/CFS Research in Human-Computer Interaction”

Abstract Presenter: Dr Ryan Kelly & Melissa Rogerson, Interaction Design Lab, School of Computing and Information Systems, The University of Melbourne

Research has shown that technology has the potential to support self-management of ME/CFS. This presentation describes findings from two projects that illustrate a user-centred approach to the design of self-management technologies for people living with ME/CFS.

The first project used surveys and online discussion groups to understand users’ current approaches to condition management using digital technologies. We found that people with ME/CFS want to use technology to support management of primary symptoms—particularly those related to physical and cognitive fatigue—and currently do so by using a variety of commercial activity trackers. However, people report difficulties with these technologies because they do not provide tailored support for practices such as activity pacing. This that future technologies should be designed to reflect elements of daily living with ME/CFS.

The second project involved early-stage design of My CFS Buddy, a mobile self-management application that draws on insights from field interviews with parents of teenagers with ME-CFS. All interviewees identified communication as a key challenge – particularly between young people and their parent-carers, but also in the selection and curation of material to present to doctors at irregular appointments. The prototype app acts as a diary tool for both the patient and their carer, logging mood and medication and importing other data (e.g. sleep and movement tracking). It allows for material to be tagged at the time of recording for later sharing with doctors.

Our presentation will focus on communicating key insights from these projects and will illustrate the potential for Human-Computer Interaction research to provide a non-clinical source of expertise as a contribution to the design of technology for people living with ME/CFS. We will conclude by describing design recommendations for ME/CFS self-management applications, which we hope can be elaborated through collaboration with researchers from other disciplines.
“The European Network on Myalgic Encephalomyelitis/Chronic Fatigue Syndrome - how best to connect research and healthcare across Europe”

Abstract Submission Presenter: Dr Eliana Lacerda, London School of Hygiene and Tropical Medicine

Authors: Eliana M Lacerda on behalf of the EUROMENE network

Background: The European Network on ME/CFS (EUROMENE) was launched in 2016, funded by the European Cooperation in Science and Technology (COST), to galvanise a collaborative approach between research centres to maximise multidisciplinary research efforts.

Question: What progress has been made by EUROMENE so far, and what are the potential benefits of a collaborative, multi-centre network for people with ME/CFS?

Methods: EUROMENE's methodological strategy is structured around six working groups: Epidemiology, Biomarkers, Socio-Economics, Clinical Research, Postgraduate Training, and Dissemination and Patient Involvement. The groups have worked on deliverables, aiming to: i) describe the landscape of ME/CFS-related research and health care provision in European countries; ii) describe the burden of disease; iii) promote multidisciplinary ME/CFS research (leading to improved diagnosis), development of biomarkers, treatments, and preventative strategies for improving quality of life; and iv) disseminate best practices on research and healthcare provision for people with ME/CFS. EUROMENE's Management Committee is continuously assessing the working groups' progress against the project's timeframe.

Results: Since its outset, the number of countries participating in EUROMENE has increased to 21 (a 50% increase). The network has published peer-reviewed papers describing the current European landscape in epidemiology, biomarkers research, and socioeconomic direct and indirect costs caused by ME/CFS in Europe. A survey on practices for ME/CFS diagnosis and treatment has been conducted among the participating countries. Recommendations for clinical diagnostic criteria and research protocol standardisation are being discussed among the network members. Furthermore, the network has awarded short-term scientific fellowships for young investigators, and hosted two summer training schools on translational research in ME/CFS.

Conclusions: EUROMENE is helping to raise awareness about ME/CFS in the EU, by establishing a strong research platform with standard protocols, bioinformatics data and samples repositories; and promoting public engagement from the scientific community and health providers.

DAY 3

Management and Treatment of ME/CFS

Keynote Presentations by Dr Mark Donohoe and Dr Bruce Wauchope

Clinical Q&A Panel with Dr Mark Donohoe, Dr Bruce Wauchope, Dr Don Lewis, Dr Lisa McLindon-Smith and Dr Nicole Phillips

We want to extend our sincere thanks to everyone who has assisted in the organisation and hosting of the 2019 Emerge Australia ME / CFS International Research Symposium. We want to thank all of our keynote speakers, our guests and presenters, all of the delegates attending, Emerge Australia volunteers, the Novotel Geelong, the Emerge Australia Committee of Management and the incredible team at Emerge Australia who have all contributed to making this event a success.
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