Chronic Fatigue Syndrome

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History of CFS

- Hammurabi (Greek philosopher) 1800 BC
- Icelandic disease - 1948
- Royal Free Hospital, London epidemic - 1955
- “Psychiatric” phase – 1965 (McEvedy&Beard)
- Present day research: Florida, Stanford, Griffith, Norway, Harvard, London etc. → explanation of symptoms

3800 years of history

Early years (1977→)

- A mixture of fatigued patients
- A multitude of symptoms
- Many with psychiatric and mixed diagnoses
- Patients tended to be treated with skepticism

e.g. Often told: “All your blood tests are normal so there is nothing wrong with you!”

About 1/3 eventually had alternative diagnoses

2/3 fulfilled Fukuda criteria for CFS

Presentation (all ages)

- Incidence (3 per 1000)
- Genetic predisposition
- Viral illness
- Prolonged recovery (6 months → long-term)
- Multiple symptoms (often mimicking initial illness)
- A continuum of severity (Severe → mild)

- Diagnostic criteria – Canadian 2003. (Adopted by UK in 2011)
- Diagnosis cannot be made in presence on untreated psychiatric illness

Core Symptoms

- Fatigue and myalgia
- Post-exertional malaise
- Severe cognitive difficulties
- Chronic daily headache
- Sore throat and lymphadenopathy
- Orthostatic intolerance
- Non-restorative sleep

Co-morbidities/differential diagnoses

- Ehlers Danlos Syndrome
- Chiari Syndrome
- Fibromyalgia/Chronic pain
- Irritable Bowel Syndrome
- Mast cell activation syndrome
- POTS etc

Current research

- Griffith, Australia (Staines,Gradisnik) – immune signature
- Miami, USA (Klimas, Fletcher) – gene & immunology studies
- Stanford (Naviaux, Davis) - metabolomics
- Cornell,USA (Hansen) - microbiome
- Newcastle,UK (Newton) – cellular bioenergetics, mitochondia
- Bergen,Norway (Fluge & Mella) - metabolic profiling
- London (Kerr) – gene expression = subtypes
- California (Davenport) – 2-day exercise tests

Griffith University Research (Staines and Gradisnik)

- “Impaired calcium mobilization and dysregulation of transient receptor potential melastatin 3 (TRPM3) ion channels in Natural Killer (NK) cells from CFS/ME”.
- Their group were the first to identifyTRPM3 on NK cells. There is impaired calcium mobilization. They also have studied NK cells, and find function and expression significantly reduced. Calcium is an important intracellular cell-signalling messenger.
- Significant changes are also evident in the genetics of NK cells. SNPs may change the function. TRPM3 stood out as abnormal. TRPM3 is the predominant ion channel and its expression is down in ME/CFS. This is very important in glial cells in the brain. The calcium signals can be measured.

Naviaux et al (Stanford)

- Abnormal metabolomics = “Dauer”
- 612 metabolites
- Significant abnormalities in 20 pathways (45 subjects + controls)

= Hypometabolic Syndrome

Management

- A diagnosis of exclusion: history, symptoms, examination, investigations etc = **long consultations**
- Validation
- Patient and family education (handouts)
- Stress management
- Cautious exercise plan (Note PEM)
- Diet, ??supplements
- Sleep and pain management

Longer term

- Should be managed in General Practice
- But may need multidisciplinary team
- General medical care needed
- Ongoing surveillance vital:
    - Increased risk of cancers
    - Increased ageing of telomeres
    - ? Diagnosis correct

After 40 years of seeing 6000 CFS patients:

- All CFS patients can experience a better quality of life with firm diagnosis, compassionate care and a multidisciplinary approach.

Patient needs to feel in control of health rather than being controlled by the disease

RESOURCES

- **Physicians' Primer: IACFS/ME**:
- www.iacfsme.org

- **Paediatric Primer**:
- https://www.frontiersin.org/articles/10.3389/fped.2017.00121/full