An Evocative Autoethnography of Living Alongside Myalgic Encephalomyelitis (ME):

Reimagining a Self

An Evocative Autoethnography of Living Alongside Myalgic Encephalomyelitis (ME):

Reimagining a Self

By

Orlagh Farrell Delaney

Cambridge Scholars Publishing

An Evocative Autoethnography of Living Alongside Myalgic Encephalomyelitis (ME): Reimagining a Self

By Orlagh Farrell Delaney

This book first published 2021

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN (10): 1-5275-7165-3
ISBN (13): 978-1-5275-7165-5

This book is dedicated to the ME "millions missing"
may we all find our own way home...and to Paul
who bravely walked the long and weary road and could not stay.

Two brown hens picking
in the early morning sunshine.
A glimpse ~
perhaps no day is better
than mine.

—Orlagh Farrell Delaney

My life is my message.

—Mohandas K. Gandhi

TABLE OF CONTENTS

Poems:

ACKNOWLEDGEMENTS

I would like to thank my inspirational friend and mentor Trish (Dr Patricia Kennedy). You have paved the way and had my back for years (I have the Doc Martin print on my backside to prove it). Thank you for your guidance and the blessing of your friendship. A very sincere thank you to Maurice (Professor Maurice Devlin) who both travelled and moved mountains over the years to facilitate this work, together we made a 'radiance'.

Thank you to my parents Ann and Jack Farrell, to Fi, Ber, Mary, Mona, Seamus, Kathleen and Sue and to all my family, friends and neighbours who in so many ways loved, supported and provided.

Finally, thank you to all my beloved critters, who sat patiently looking at me sitting looking at books.

CHAPTER 1

INTRODUCTION

As a woman who has lived with Myalgic Encephalomyelitis (ME) for the last sixteen years, I am personally aware of the dearth of research on the lived experience of ME and the knowledge and information gaps that such a paucity of useful information and experience creates for sufferers, who often must struggle alone to find a way through an illness such as ME. I am moderately affected by the illness. The estimated percentage of the ME population considered to be moderately affected by the illness is 60% (MacIntyre 1998). Although this population cohort is frequently discussed within the medical research literature, they are not as visible as the severely affected category in terms of the portrayal of their lived experiences. As an incurable, invisible contested and unsupported illness (Jason 2007; Dimmock and Lazell-Farnen 2015) the lived experience of the illness is mostly portrayed in pessimistic, hopeless and option-less terms (Munson 2000; Mitchell 2003).

Myalgic Encephalomyelitis (ME) as a Chronic, Invisible and Contested Illness

Myalgic Encephalomyelitis ME is described as an acquired, complex disorder characterised by a variety of symptoms, principally extreme fatigue or malaise following exertion lasting six months or longer (Carruthers et al. 2011). ME is revealed as a chronic, invisible, contested illness (Blease et al, 2017) with a global prevalence of 17-24 million sufferers (www.meaction.net). It is estimated that 12,000 people in Ireland live with ME. ME and Chronic Fatigue Syndrome have been entwined in a 'web of confusion' (Carruthers et al 2011) for thirty years. This has delayed research into the illness and fostered stigma and disbelief within the medical profession (Dimmock and Lazell-Farnen 2015). ME is a spectrum illness with patients falling somewhere under or within the categories of mild, moderate or severe (Twisk 2014). To date there are no laboratory or diagnostic tests to identify ME, and no cures or treatments (Green et al 2015). The causes of the extreme nature of the fatigue and related

symptoms associated with ME are not yet understood, although recent biomedical hypotheses have been contributed from various medical specialities. However of greatest relevance to this book is that ME is considered a contested illness. A contested illness is a disorder that is considered medically suspect because it is not associated with any known physical abnormality (Conrad and Barker 2010). The legacy of this contested nature is that the ME patient's credibility is undermined and as a result diagnosis and adequate treatment and support are delayed. Patients are wrongly psychologised and not appropriately referred (Blease et al 2017; Jason et al 2009). Patients who feel disbelieved, mistrusted or misjudged by the healthcare system may choose to withdraw from healthcare altogether and go it alone. It is the contested and unsupported nature of the illness that is most burdensome to the ME population. ME constitutes a relatively new area of research, as the first outbreak of the illness only occurred in the late 1980s (Ramsey 1988). The recent increase in research interest is biomedical in origin (Montoya et al 2017; Naviaux et al 2017) and a dearth of research on the experience of living with ME persists (Bell 2000).

The central aim of this book is to explore and explain the experience of living alone with a chronic, invisible, contested illness such as ME. Given the pessimistic, hopeless portrayal of ME, I ask: is it possible to reimagine a self or a life living with a chronic, invisible, contested illness like ME? If it is, can such a life be considered a good life?

The purposeful design of this book is an evocative autoethnography. Autoethnography is research writing and method that connect the autobiographical and personal to the cultural and social (Ellis 2004). Evocative autoethnography is a research process that allows for expression of 'the consciousness and subjectivity of the author/researcher through a personal, vulnerable, reflective, self-reflexive narrative voice' (Bochner and Ellis 2016). I came to autoethnography as a research method as a result of an encounter in July 2009, at the 5th Annual Mixed Methods Conference at Leeds University. I was co-presenting a paper with a colleague and dear friend on our experience of making a radio documentary about both our experiences of getting divorced in Ireland and the resilience of our friendship (http://www.rte/doconone/therewaslove). Following our presentation, the conference organiser Dr Tessa Muncey (who had just written a book *Creating Autoethnographies*, 2010) named our research as autoethnography and identified it as a relevant and potential method for my research interests.

Story

I am standing with you at the conference.
You are fluently academic
I have been sick and silent for years.
All around me I hear "nomothetic" and "idiographic"
These are no longer my words
this would not be my message now.
"Who are these people? " I ask.
"That doesn't matter" you smile.
But I have changed.
Now if I do not know who you are
I cannot take to heart what you say.

Too often people with illness are the passive subjects of investigations by researchers who do not have experience of illness. An autoethnographic approach can help to redress this imbalance and generate unique insights (Ettore 2010). My primary data in this book is drawn from some field notes but principally from journal entries kept over the course of my illness. The early years of illness, from 2002 to 2008, are presented as vignettes written from field notes and from journal entries. A narrative gap between 2009 and 2013 is identified as a result of relapsed illness following a lengthy divorce. Vignettes describing memories of events relevant to this timeframe are presented. Journal entries cover the period of 2014 to late 2018. The journal entries are written in narrative form, in the first person voice and are by their nature evocative. I also include my own photographs as data in this book. I received a gift of a small, light digital camera in 2009 which lived constantly in my pocket and I began capturing moments in time. Often, over the many years of my illness, my only capacity to record a moment or a fleeting story, was to capture it in a photograph. In 2015 an unwanted iPhone replaced the camera in my pocket and captured moments in time from then to 2018. Finally, I include poetry in this book and the majority of it is my own poetry written over the course of my illness.

Structure of the Book

Chapter two outlines the nature of ME, its conflicted history, its contested nature and the thirty years of disagreement with regard to the naming and diagnosing of the illness. The medical and psychosocial natures of the illness are also discussed, along with the consequences for the ME community of the negative legacy from the years of disagreements and misunderstandings.

Chapter three outlines the methodology and conceptual frameworks employed in this book. An evocative autoethnography is adopted using personal journals, field notes, photography and poems as data. Four narratives spanning the length of the illness are presented and subsequently put under analysis in four corresponding chapters. The 'Struggle Cycle" is an original conceptual framework created by the author which is influenced by and also draws on Witkin's 'Conditions Facilitative Of Transformative Change' (2014) and the Buddhist philosophy of the Four Noble Truths. These three conceptual frameworks are used in collaboration to analyse the narratives.

Chapters four, five, six and seven present the four evocative autoethnographical narratives which express the lived experience of ME through the lenses of 'struggle', 'surrender', 'seeing' and 'sanctuary' respectively. The narratives are comprised of journal entries with the first ('struggle') also containing vignettes and poems. I present the narratives in the language they were written in in my journal entries which is creative, prosaic and at times poetic. Evocative autoethnography employs the tools of literary and aesthetic practitioners. I was influenced in my writing by the literary works of John O'Donoghue, John McGahern, May Sarton and the Zen poets. Poems can also be found in the other narratives. The evocative narratives also reveal the 'story' of a woman living alone over a sixteen year period, in a remote, rural setting, alongside rescued animals and aspiring towards a Buddhist philosophy. Photographs revealing the ordinary moments of such a life conclude this narrative section. I selected photographs from a collection of over a thousand. I chose not to narrate them individually, but instead to allow them to portray visually, and reflect, images and stories already described in the narratives themselves.

Chapters eight, nine, ten and eleven put the four narrative chapters, 'struggle', 'surrender', 'seeing' and 'sanctuary' under analysis using the conceptual frameworks already mentioned. Salient concepts and themes are identified and discussed including: voluntary simplicity and authenticity, enoughness, compassionate and harmless living, reverence for nature, gratitude and Zen consciousness. The trajectory of the transformative process or reimagining of a 'self' living with ME is illustrated using the 'struggle cycle'.

A collection of my own poetry, *SIT*, is included after this narrative-under-analysis section. The poems reflect the sentiment in both the narrative and narrative-under- analysis sections as well as reflecting the photographs.

Chapter twelve presents a discussion and conclusion. Finally there is a narrative epilogue.

The experience of living alone with an invisible, chronic, contested illness like ME is under-researched and under-reported. This is not unrelated to the nature and constraints of the illness. Using autoethnography as a method, made undertaking this research possible, while still taking its toll. The contribution to knowledge of this autoethnographical research is that it can serve to inform medical professional and other support services dealing with ME patients. It can also inform the general public and those who, although they share their lives with people who live with ME, can still struggle to understand. This book can assist others who live with ME to reflect on and understand their own experiences and increase their own options for change. This book makes a methodological contribution because an autoethnography on living with ME has not been written to date. Also, an evocative autoethnography with the additional creative strands of original poetry and photography is an emergent research approach at doctoral dissertational level and it is hoped that this book will pave the way for others interested in this field.

CHAPTER 2

UNDERSTANDING MYALGIC ENCEPHALOMYELITIS (ME)

Introduction

In researching any medical condition, it might be expected that it could be explained quite succinctly in terms of definition, symptoms, diagnosis, causes, treatments and outcomes. What becomes clear early on in researching Myalgic Encephalomyelitis (ME) is that ME is considered to be a contested illness. A contested illness is an illness that is questioned or disputed by members of the medical field (Blease et al 17). There is dispute over what the illness should be called, how to define it, and how to separate it from Chronic Fatigue Syndrome (CFS) (Hyde 2009) and the complex relationship that they have shared since the late eighties (Dimmock and Lazell-Farnen 2015). The contested nature of ME is also dominant in the dichotomous nature of the research in terms of the biopsychosocial or psychogenic theory, and the biomedical theories of the illness (Maes and Twisk 2010). The result of this dichotomy is that ME advocacy groups and the world of medicine differ greatly on how to view and manage the illness (Hossenbaccus and White 2013).

Myalgic Encephalomyelitis: ME

Myalgic Encephalomyelitis (ME) is an acquired complex disorder characterized by a variety of symptoms, primarily extreme fatigue or malaise following exertion, lasting six months or longer (Carruthers et al 2011). Many cases are preceded by a viral infection, usually a flu-like or upper respiratory illness. It can also be preceded by a non-viral illness or other trauma such as chemical exposure. Onset is usually rapid (acute) but gradual onsets are reported (Ramsey 1988). Affected individuals do not recover from the infection and instead experience a wide variety of inflammatory type symptoms, including an inability to produce sufficient energy to meet daily demands (Klimas and Koneru 2007). Marked fatigue and weakness, sickness, cognitive dysfunction and symptom flare–up

follows physical and cognitive exertion. ME represents a complex, multi-system group of afflictions adversely affecting the brain, heart, neuro-endocrine, immune and circulatory systems (Hyde 2003). Myalgic Encephalomyelitis is a chronic and disabling disorder. Moderate to severe cases leave patients housebound or bedbound respectively. (Jason et al 2015). People with ME may not look ill but maintaining employment or study may prove difficult or impossible. Family and social interactions may also be compromised (Institute of Medicine 2015). To date there are no laboratory or diagnostic tests to identify ME, and no cures or treatments (Green et al 2015). ME is a spectrum illness but it is widely accepted that patients fall under or somewhere within one of three sub-groupings (Twisk 2014; Brurbeck et al 2014). The UK National Institute for Health and Clinical Excellence (NICE) guidelines (2007) describe them as follows:

> MILD: where people are mobile, can care for themselves and can do light domestic tasks with difficulty. Most are still working or in education, but to do this they have probably stopped all leisure and social pursuits. They often take days off, or use the weekend to cope with the rest of the week.
>
> MODERATE: where people have reduced mobility and are restricted in all activities of daily living, although they may have peaks and troughs in their levels of symptoms and ability to do activities. They have usually stopped work, school or college and need rest periods, often sleeping in the afternoon for one or two hours. Their sleep at night is generally of poor quality and disturbed.
>
> SEVERE: where people are unable to do any activity for themselves, or can carry out minimal daily tasks only (such as face washing and cleaning teeth). They have severe cognitive difficulties and depend on a wheelchair for mobility. They are often unable to leave the house, or have a severe and prolonged after-effect if they do so. They may also spend most of their time in bed, and are often extremely sensitive to light and noise.

Disease Prevalence

The Institute of Medicine (IOM) in the USA reviewed the ME/CFS literature in 2015 and estimated that between 836,000 and 2.5 Americans have ME/CFS. It is most common in people between forty and sixty years old with a female to male ratio of 6:1. The estimated prevalence of ME/CFS is 0.4-1% or between 17 to 24 million sufferers worldwide. Mean illness duration ranges from 3-9 years (Capelli et al 2010). It is estimated that there are about 12,000 people with ME in Ireland. (www.imet.ie).

Myalgic Encephalomyelitis (ME) and Chronic Fatigue Syndrome (CFS)

Myalgic Encephalomyelitis (ME) is the original term for the illness. It was re-categorised by the CDC (Center for Disease Control) in the USA in 1988, who considered the illness to be more about fatigue than neurology, and renamed Chronic Fatigue Syndrome (CFS). The name CFS, despite objections from clinical, research and patient groups, persists today in the USA, and has far reaching effects globally on the reality of ME. There are experts who consider it to be a matter of 'semantics' and argue that CFS is 'American' for ME and therefore, particularly in more recent times, they are used interchangeably in both research and clinical environments, particularly in the US but increasingly in research parlance globally. I am discussing the realities of ME but when quoting research and clinical experts that speak and publish in terms of ME/CFS or CFS/ME I must refer to it as it is quoted. However, as will become clear, when the CDC renamed ME as CFS in the USA in 1988 they changed the identity and integrity of the classical ME disease entity and made it a syndrome (a collection of disorders) (Johnson 1996). They also introduced psychiatry whose legacy has been very damaging for ME patients and their families (Deale et al 2001). CFS as a 'syndrome' has become an umbrella term under which some true ME patients have had to shelter, sharing it with other related and unrelated illnesses and conditions. ME is a neuro-immune condition of severe onset whose degree of central nervous system dysfunction and post exertional malaise are its identifying features and are in a different severity league to those of CFS as defined in its criteria (Hyde 2003, 2009).

A Web of Confusion

ME and CFS have been entwined in a 'web of confusion' (Caruthers et al 2011) for thirty years now. This 'web of confusion' has particularly delayed any focused investigation into ME as it laboured under orphan medical status, stigma and disbelief. Research was unfunded until the last decade when it became funded privately in the majority (www.meresearch.uk.com). Significant other contributing factors to this web of confusion, have been the constant disagreement about, and re-categorization of, the diagnostic criteria for ME internationally. For the purpose of this chapter and to expose the sources of the contested nature of ME, which again is central to this work, a short overview of the most

relevant facts is essential here, with some additional criteria provided in appendix 1.

In the UK in the 1950s a series of outbreaks of a mysterious illness similar to poliomyelitis occurred. A Dr Ramsey, after studying such an outbreak that had occurred principally among doctors and nurses at the Royal Free Hospital in London, declared it to be 'benign encephalomyelitis'. What each outbreak had in common were symptoms of sore throat, tender lymph nodes, pain and signs of encephalomyelitis (Lancet 1955). The term 'benign' was used as it was not proving fatal (as opposed to a malignant nature), and the encephalomyelitis because of the 'evidence of parenchymal damage to the nervous system and the presumed inflammatory nature of the disorder' (Acheson 1959: 593). However, in 1970 two psychiatrists concluded that the outbreaks were 'psychosocial phenomena' caused either by 'mass hysteria on the part of the patients' or 'altered medical perception in the community' (Mc Evedy and Beard 1970: 13). They came to these conclusions because of the lack of physical signs in the patients and because of the considerably higher prevalence of the disease in women. They never actually saw or interviewed any of the patients at the time. Despite all Dr Ramsey's science and his objections (Ramsey et al, 1977) health professionals at that time subscribed to hysteria as a plausible explanation for the condition (Speight 2013). In 1984 there was an outbreak of an ME type illness in Incline Village, a popular ski town in Lake Tahoe, Nevada (Johnson 1996). In 1985, there was a similar type of outbreak in a relatively poor country town in Lyndenville, upstate New York (Bell, Jordan and Robinson 2001).

Subsequent to these two significant outbreaks, in 1986 Ramsey became the first to coin the term Myalgic Encephalomyelitis based on his description of symptoms of the 1955 Royal Free outbreak. Myalgic = muscle pain, Encephalomyelitis = brain and spinal column inflammation. His categorization and identification of symptoms has been refined several times since 1986 including his own modifications in 1988, but they have never been deviated from substantially in terms of ME. He identified:

(1) Muscle phenomena: Fatigability, pain, clumsiness.
(2) Circulatory impairment: Cold extremities, hypersensitivity to climate change, ashen grey face pallor 20-30 minutes before the patient complains of feeling ill.
(3) Cerebral dysfunction: Impairment of memory, impairment of concentration, emotional lability. Alteration in sleep rhythm, vivid or violent dreams. Autonomic nervous system symptoms including

orthostatic tachycardia (increased heart rate on standing), frequency of urination.
(4) Symptoms and physical findings may vary greatly in the course of any day.
(5) This illness has 'an alarming tendency to become chronic' (Ramsey 1988).

In 1988, the Center for Disease Control (CDC) in the USA, having visited the outbreak site in Incline Village, but *not* in Lyndonville wrote a report. They based their findings largely on one symptom; fatigue and renamed the illness chronic fatigue syndrome (CFS). They also included neuropsychological symptoms in their criteria.

In 1991, a group of British doctors unhappy with some of the American CFS criteria, met at Oxford to discuss and subsequently publish their own criteria for CFS. The Oxford Criteria for CFS (of which Sharpe and Clare, both psychiatrists, were the main protagonists) included fatigue as the main symptom (as opposed to post-exertional malaise) and included depression and anxiety disorders as criteria (Sharpe et al 1991). The Oxford criteria did not require a patient to have any of the cardinal, distinguishing features of ME that Ramsey had identified:

- abnormal muscle fatigue after trivial exertion with abnormally prolonged recovery.
- neuro-cognitive symptoms such as loss of memory and concentration.
- variability in severity from day to day or longer.
- the tendency for the illness to become chronic.

Also the Oxford Criteria included people with fatigue due to burn out as a result of stress of overwork or over training.

In 1994 the CDC updated their 1988 Criteria (Fukuda et al 1994). As Fukuda was the first research author listed, these have become known as the Fukuda criteria. The main difference between the 1988 and 1994 versions was that now the 1994 criteria also allowed minor psychiatric disorders such as anxiety, depression as causes for fatigue in CFS. Oxford and Fukuda's new criteria for CFS had moved away from Ramsey's initial descriptions of ME.

As a new millennium dawned, there were significant consequences for ME as a result of the name change to CFS, and the changing of the diagnostic criteria, particularly to include psychiatry and somatization or when

psychological concerns are converted into physical symptoms. Anne Macintyre, a British medical pioneer in ME (who also had the condition herself for many years and sadly passed away in 2018) offers this summary:

- CFS now covers a broader range of conditions causing fatigue, not only ME.
- These criteria concentrate on one symptom, 'fatigue', which is part of everyday life and present in many illnesses and has meant that the many neurological symptoms and disabling aspects are ignored and denied.
- Cases are included whose fatigue is due to depression, anxiety, stress or burn out which dilutes the potential severity of some patients' illness.
- This has posed difficulty in qualifying for social security or illness pensions because the illness is perceived by many doctors and officials as being mainly psychiatric.
- Conflicting results in research studies as different criteria are used and the characteristics of patient groups used in research may not be the same (1998, 93).

The Canadian Consensus Criteria 2003

Up to this point Canada was using Fukuda's (revised 1994 CDC) definition for ME/CFS. However as this was primarily designed to standardize *research* processes and not for use as a *clinical* case definition for ME/CFS patients, family physicians and other clinicians were in need of a clinical case definition. By the CDC singling out prolonged fatigue as the sole compulsory criterion, it minimized and de-emphasized the importance of the other cardinal and identifying signs of ME/CFS (post-exertional fatigue/malaise, cognitive dysfunction, pain and sleep disturbances etc). This made it increasingly difficult for a clinician to distinguish the pathological fatigue of ME/CFS from other fatiguing illnesses and indeed ordinary fatigue. In response to this, in 2001, Health Canada in co-operation with the National MEFM Network (FM is Fibromyalgia), established terms of reference and formed an expert medical consensus panel, comprising treating physicians, teaching faculty and other researchers (www.me/ fmaction.co). This panel had collectively either diagnosed or treated (or both) 20,000 patients with ME/CFS. Their task was to conduct a review process and establish consensus for a clinical working case definition and diagnostic and treatment protocols. In 2003

they published the Canadian Consensus Criteria (CCC) which provided a working clinical case definition (Caruthers et al 2003). This clinical case definition would include the pattern of positive signs and symptoms unique to ME/CFS. These signs and symptoms would identify ME/CFS as a distinct entity and distinguish it from other overlapping clinical entities. In fact the panel concluded that the more prominent signs which it identified (see criteria categories to follow) should be considered as compulsory for a positive ME/CFS diagnosis. In order to help focus a clinical encounter and facilitate a diagnosis, the panel grouped together symptoms which shared a 'common region of pathogenesis' versus the patient presenting with a 'laundry list of seemingly unrelated symptoms' (Carruthers et al, 2003, 10).

Diagnostic Criteria as proposed by the Canadian Consensus

A patient with ME/CFS will meet the criteria for all of 1-4 below*

1. Fatigue of recent onset, unexplained, persistent or recurrent physical and/or mental fatigue that restricts activity.
2. Post-exertional malaise (PEM) – inappropriate loss of physical and mental stamina, cognitive and muscle fatigability post exertion, tendency for clusters of symptoms to worsen on activity, pathologically slow recovery periods.
3. Sleep dysfunction: un-refreshed sleep, decreases in sleep quantity and quality and/or significant dream disturbances.
4. Pain: significant degree of myalgia, pain in muscles and/or joints, that is widespread and migratory. Headaches of new and/or severe origin.

A patient will have two or more of the following neurological/cognitive manifestations:

5. Impairment of concentration, confusion, short-term memory problems, disorientation, difficulty processing information or finding words. Muscle weaknesses, overload phenomenon: sensory (photosensitivity, hypersensitivity to noise) and/or emotional overload which can lead to 'crash periods'.

A patient will have at least ONE symptom from 2 of the following 3 categories (6) a, b, c:

6. a) Autonomic disorders: orthostatic intolerance (light headedness on standing), POTS, (fast heart rate on standing), neurally mediated hypotension (low blood pressure on standing from faulty brain signals), extreme pallor, nausea, Irritable Bowel Syndrome (IBS), urinary problems, palpitations with or without cardiac arrhythmias.
   b) Neuroendocrine manifestations: Loss of thermostatic stability, intolerance of extremes of heat or cold, subnormal body temperature, feverless sweats, anorexia or abnormal appetite, loss of adaptability and worsening of symptoms with stress.
   c) Immune manifestations: tender lymph nodes, recurrent sore throat, recurrent flu-like symptoms, new food sensitivities, new medication and chemical sensitivities.
7. The illness persists for six months or more. It is usually of distinct sudden onset although it may be gradual. To be included under this diagnosis criteria the symptoms must have begun or have been extremely altered after the onset of this illness. The physical disturbances tend to form 'symptom clusters' which fluctuate over time. It is unlikely a patient will suffer from all the symptoms in 5 and 6.
   *Condensed for convenience of reading purposes from MEpedia.

The Canadian Criteria framework was welcomed internationally and is still used widely today with many ME patient advocacy groups and ME support organizations and charities advocating its use. However it was improved upon by another international consensus criteria body (ICC) in 2011. This body in particular wanted to dissociate ME further from CFS. Due to the constraints of this chapter, I refer the reader to Appendix 1 for details of the ICC criteria for the diagnosis of ME. Principally, and of most relevance here is that because of the more recent research findings that had become available since 2003 when the Canadian Criteria was written, the ICC (Carruthers et al 2011) asserted that the research strongly pointed to widespread inflammation and multi-systemic neuropathology that indicated a specific underlying pathophysiology peculiar *only* to ME. Therefore, ME having its own identifiable pathophysiology suggested the need for a refinement of patient stratification. It also recommended that the illness once again be called ME. It suggested it was the most accurate term as it reflected the underlying multi system pathophysiology of the disease. It considered ME and CFS to be two widely diverse conditions. It rejected the interchangeable uses of the terms ME and CFS and it rejected their combined uses as ME/CFS or CFS/ME. It suggests again that a disease entity should have only *one* name consistent with the WHO classification

rule that a disease cannot be classified under more than one rubric. Finally of interest here is that the ICC (similar to Ramsey) also put as its major criterion post exertional fatigue or malaise, although it chose to rename it with a more descriptive term: 'post exertional neuroimmune exhaustion' (PENE). The ICC maintains that patients who exhibit low thresholds of physical and mental fatigability in response to exertion (which can be confirmed scientifically) along with the multi-system pathophysiology *have* ME. Others that do not belong under the more encompassing CFS classification. It subscribed to the belief that science cannot be advanced without a relatively homogenized patient group. Other researchers agreed that it was counterproductive to use inconsistent and overly inclusive criteria to glean insight into the pathophysiology of ME, if up to 90% of the patient sets researched may *not* meet the criteria (Jason et al 2009).

This lengthy web of confusion in terms of naming the illness and agreeing on the criteria for its diagnosis had not occurred without a significant impact on the ME community. In *Thirty Years of Disdain* Mary Dimmock explains the impact of bad definitions and their legacy for ME patients, firstly in terms of research and secondly in terms of clinical practice (Dimmock and Lazell-Farnen 2015). In research:

- It has resulted in flawed epidemiological studies, faulty prevalence numbers and erroneous claims of risk and prognosis (Jason and Richman 2007).
- It has virtually stalled drug development and severely impacted the ability to attract private and commercial investment into the disease (the primary clinical trials for disease modifying treatments for this disease have been Ampligen in the USA and Rituximab in Norway, neither of which have become available).
- It has impaired the development of diagnostic biomarkers leaving the diagnosis one of subjectivity and exclusion (Jason at al 2009).
- It has generated such disdain and scepticism in the research community that researchers avoid the disease like leprosy out of a fear that it could kill their careers. (One such example is Stanford's Professor Jose Montoya whose early mentor suggested that he could end up homeless if he pursued research into ME).

Secondly, in clinical care and practice the lengthy web of confusion that has surrounded ME:

- Has warped the physician's understanding of the disease leading to medical disbelief, hostility and inappropriate treatments.

- Has facilitated and allowed flawed 'evidence based' clinical guidelines that include maladaptive personalities and recommendations for Cognitive Behavioural Therapy CBT and Graded Exercise Therapy GET which continue to hurt patients today.
- Has made it very difficult for patients to get disability payments and insurance reimbursements because most tests and treatments are considered experimental.
- Has stigmatized disabled patients terribly and sentenced them to abysmal clinical care.
- Worst of all, has directly enabled and nurtured psychogenic views. This has dramatically altered the perception of ME by the public at large ensuring that neither the disease or its victims are taken seriously by anyone (Jason et al 2004).

The Psychogenic View

Undisputedly the greatest obstacle to both scientific and medical/clinical progress in ME has been the psychogenic view and its legacy. After one of the first American ME outbreaks at Incline Village, Nevada, Dr William Reeves (2005) of the Center of Disease Control (CDC) characterized the outbreak as hysteria. He would go on to lead the CDC CFS program, the biggest in the world, for ten years. Reeves's contribution was a CDC 'toolkit' of Cognitive Behavioural Therapy (CBT) and Graded Exercise Therapy (GET) and sleep hygiene for a 'condition of un-wellness' not a disease (www.Pheonixrising.me 2012). In 1988, Dr Stephen Strauss of the National Institute for Health (NIH) published that patients with CFS:

> …were educated white women, were more likely to get the disease which could either reflect their resources to access evaluations or some unique constitutional frailty of such individuals. Most had excellent health and some were competitive athletes at least with aggressively maintained physical conditioning. A less casual approach however often uncovered histories of unachievable ambition, poor coping skills and somatic complaints. It is difficult and at times unpleasant to address the demands of such patients or to test the hypotheses as to the aetiology of their woes (Straus et al 1988, 793).

In 1988, Simon Wessely, a British psychiatrist at Kings College, promoted the 'biopsychosocial' theory of ME, saying it was caused by psychological factors and physical deconditioning. Wessley has recently retired from the ME field as he has been severely trolled and threatened on the internet because of his views. By the late 1980s and early 1990s the mainstream

press began to reflect these views and the dismissive term 'Yuppie Flu' appeared (Boffey 1987). Munson (2000: 108-109) describes how ME/CFS became an 'accepted social parody', the Yuppie Flu serving as a 'catchall metaphor'. The biopsychosocial theory persists today despite all the evidence that ME is an ongoing organic disease. Its theory suggests ME patients 'maintain' or 'perpetuate' their illness by activity avoidance, which causes deconditioning, which causes the ongoing disability and symptoms that patients experience. ME patients' beliefs, behaviours and other social factors are 'keeping them sick' (Prins, van der Meer and Bleijenberg 2006). In order for the ME patient to combat these beliefs, biopsychosocial theory recommends CBT (cognitive behavioural therapy) to reverse the patients' presumed 'fear of activity' and 'false beliefs' that their disease is actually 'organic'. It also recommends GET (Graded Exercise Therapy) to reverse their presumed deconditioning (White et al 2011). The claim that patients can recover as a result of CBT and GET is not justified by the data and is considered to be highly misleading.

In 2011, the largest clinical trial conducted to date in the UK on ME was called the PACE trial (Pacing, Graded Activity and Cognitive Behaviour Therapy: a Randomized Evaluation). It was funded by the National Health Service, the Medical Research Council and the Department for Work and Pensions for a cost of £5million. The published results, which claimed a recovery rate of 20%, are still a source of controversy (White et al 2011; see also Wiltshire et al 2017; Kindlon 2017). Biopsychosocial theory explains patients' poor response to treatment and poor prognosis overall as stemming from the patient's own beliefs that they have an organic disease, or their desire to be in receipt of disability allowance (Yancey and Thomas 2012).

Even though the long awaited Institute of Medicine (IOM) (2015) report declared ME a 'serious chronic, complex, multi-system disease that can consume the lives of those whom it afflicts' and went on to highlight how 'the proposed psychological etiology created great controversy and convinced health professionals that this was a plausible explanation for the condition', the psychogenic view still has its proponents today (Wessley, Nimnunan and Sharpe, 1999; Knapson, 2015). The enduring legacy of the psychogenic bias and actual unproven biopsychosocial theories are, according to Dimmock in *Thirty Years of Disdain*, responsible for the way the Human Health Services (HHS) in the USA, the UK government and the international research community as a whole have viewed, studied and treated ME for the last 30 years. Mary Dimmock summarizes the era of the psychogenic view and misjudged interventions as follows:

> Medical mistreatment can cause great physical harm from inappropriate treatments. But the neglect, stigma and disbelief they [ME patients] experience are crushing to the psyche. When a horrible disease has ripped your life to shreds, isolated you from your family and friends, destroyed your career, left you destitute and grasping for a life you no longer have, it is deeply demoralizing and heartbreaking to have to fight off suggestions that you just want to be on disability or that you could overcome your ill health with 'positivity' and exercise (Dimmock and Lazell-Farnen 2015, 13).

The drive for a wholly physical etiology, according to feminist health psychologist Lara Stapleman, does not allow for the inclusion of other 'empirically supported and /or theoretically grounded possibilities for disease etiology...that consider the complex relationship of biological, social and psychological phenomena to medical illness' (2005: 264). However, Stapleman can concede that that is a 'luxury' afforded to illnesses that are 'visible and accepted by conventional medicine' and certainly not psychologised and contested like ME.

Another unfortunate legacy from the psychogenic view of ME is the subsequent portrayal of the illness by the media, on which the public depends for its social view. A 2017 analysis of the content of American newspaper articles (n=214) from 1987-2013 revealed the following. The etiology (cause) was portrayed as organic in 138 of the articles (64.5-%). There was no mention of case definitions or diagnostic criteria in 120 (56.1%) of the articles. The most common co-morbidity was depression in 49 (22.9%) of the articles. In 42 of the articles (19.4%) the headlines mislabeled the name of the illness. In 109 of the articles (50.9%) there was no mention of any form of treatment for the illness. In 119 of the articles, (55.6%) there was no mention of the prevalence rates (Siegel, Brown and Devendorf 2017).

Recent Research and the Physiological View

In recent years there has been an increase in biomedical research almost entirely funded from within the ME advocacy community. In the USA, the Open Medicine Foundation has raised and funded 8 million dollars of research into ME since its inception in 2012. Its CEO is Linda Tannenbaum who founded the foundation after her 16 year old daughter became ill with ME/CFS. In the UK, charities like Invest in ME (www.investinme.org) fundraise for research. The charity is run by volunteers with no paid staff. They have never received any government funding. Pia and Richard Simpson, are two such volunteers, they are

parents of two daughters with ME. Every year since 2006 they have organized an annual 'Invest in ME International Conference' which brings together world leaders in biomedical research, from 15 countries, to share research and ideas.

ME Research UK (www.meresearch.org.uk), exists to fund biomedical research into ME/CFS, to find its cause, develop treatments and find a cure. In fifteen years it has funded 38 specific research projects in the UK and beyond, which is more than any other organization outside the USA. It is estimated that there are approximately 250,000 people in the UK with ME (www.meaction.net). In Ireland it is estimated that there are approximately 12,000 people with ME (www.imet.ie). The ME community in Ireland is supported by the ME Association (www.irishmecfs.org) and the Irish ME Trust (www.imet.ie). In 2002. the Irish ME Trust sent a questionnaire to all GPs in the country. Of those that responded, 85% favoured a properly structured referral system orchestrated by the Department of Health. They are still lobbying the government for a medical consultant to oversee ME patients in Ireland. To date there is none. The Irish ME Trust offers information and support to ME patients in Ireland along with free classes and subsidized mini breaks away, in safe (chemical free) and supportive environments. However it has not generated any research of its own. It contributes its fundraised monies to ME Research UK (www.meresearch. org.uk) and is a sponsor for the Invest in ME Research Conferences (www.investinme.org/iime.shtml). Tom Kindlon, the Assistant Chairperson for the Irish ME/CFS Association, is himself a long time ME patient and an independent researcher. He has published widely, particularly in relation to the PACE trial (the use of graded exercise and cognitive therapy as ME treatments) and its ongoing controversy (Kindlon 2017; Wiltshire et al 2018).

Possible Causes of ME

The cause of ME remains unknown. Generally symptoms are triggered by some sort of 'prodromal event' such as 'infection (which is the most common) immunization, anaesthetic, physical trauma, exposure to environmental pollutants, chemicals and heavy metals and rarely blood transfusions' (Carruthers and Van der Sande 2005). It is generally accepted that a precursor, such as a virus, occurs with ME. There can be other factors such as an accident or trauma coupled with a genetic predisposition. Klimas (2019) asserts that genetics loads the gun and environment pulls the trigger. What follows the prodromal event is some