

What is M.E.? A historical, medical and political overview: Extra extended version

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Myalgic Encephalomyelitis (M.E.) is a debilitating acquired neurological disease which has been recognised by the World Health Organisation (WHO) since 1969 as a distinct organic neurological disorder. M.E. can occur in both epidemic and sporadic forms, and over 60 outbreaks of M.E. have been recorded worldwide since 1934.

M.E. is similar in a number of significant ways to multiple sclerosis, Lupus and poliomyelitis (polio). M.E. can be extremely severe and disabling and in some cases the disease is fatal.

Is M.E. a new illness? What does the name Myalgic Encephalomyelitis mean?

The disease we now know as Myalgic Encephalomyelitis is not a new disease. M.E. is thought to have existed for centuries (Hyde 1998, [Online]) (Dowsett 1999a, [Online]).

In 1956 the name Myalgic Encephalomyelitis was created. The term was invented jointly by Dr A Melvin Ramsay, who coined this name in relation to the Royal Free Hospital epidemics that occurred in London in 1955 – 1957, and by Dr John Richardson, who observed the same type of illness in his rural practice in Newcastle-upon-Tyne area during the same period. It was obvious to these physicians that they were dealing with the consequences of an epidemic and endemic infectious neurological disease (Hyde 1998, [Online]) (Hyde 2006, [Online]).

The term Myalgic Encephalomyelitis means: My = muscle, Algic = pain, Encephalo = brain, Mye = spinal cord, Itis = inflammation (Hyde 2006, [Online]).

As M.E. expert Dr Byron Hyde writes:

The reason why these physicians were so sure that they were dealing with an inflammatory illness of the brain is that they examined patients in both epidemic and endemic situations with this curious diffuse brain injury. In the epidemic situation with patients falling acutely ill and in some cases dying, autopsies were performed and the diffuse inflammatory brain changes are on record (2006, [Online]).

The Wallis description of M.E. was created in 1957, and in 1959 Sir Donald Acheson (a former UK Chief Medical Officer) conducted a major review of M.E. (Hooper et al. 2001, [Online]). In 1959 Dr. Donald Henderson (a CDC epidemiologist) and Dr. Alexis Shelakov (a NIH epidemiologist), published a comprehensive review paper in the New England Journal of Medicine describing several outbreaks. Dr. Henderson noted: ‘The pattern of the epidemic, the absence of any common exposure factors and the high incidence among medical and hospital personnel were consistent only with an infectious disease transmitted from person to person’ (McLaughlin 2004, [Online]). In 1962 the distinguished neurologist Lord Brain included M.E. in the standard textbook of neurology. In recognition of the large body of compelling research that was available, M.E. was formally classified as an organic disease of the central nervous system in the World Health Organisation’s International Classification of Diseases in 1969 (Hooper et al. 2001, [Online]). Professor Malcolm Hooper explains that:

The term myalgic encephalomyelitis has been included by the World Health Organisation (WHO) in their International Classification of Diseases (ICD), since 1969. The current version ICD-10 lists M.E. under G.93.3 - neurological conditions. It cannot be emphasised too strongly that this recognition emerged from meticulous clinical observation and examination (2006, [Online]).

In 1978 the Royal Society of Medicine held a symposium on Myalgic Encephalomyelitis at which M.E. was accepted as a distinct entity. The symposium proceedings were published in The Postgraduate Medical Journal later that same year. The Ramsay case description of M.E. was published in 1981 (Hooper et al. 2001, [Online]).

Since 1956 the term Myalgic Encephalomyelitis has been used to describe the illness in the UK, Europe Canada and Australasia. This term has stood the test of time for more than 50 years. The recorded medical history of M.E. as a debilitating organic neurological illness affecting children and adults is substantial; it spans over 80 years and has been published in prestigious peer-reviewed journals all over the world (Hyde 1998, [Online]) (Hooper 2003a, [Online]) (Dowsett 2001b, [Online]).

As award winning microbiologist and M.E. expert Dr Elizabeth Dowsett explains: ‘There is ample evidence that M.E. is primarily a neurological illness, although non-neurological complications affecting the liver, cardiac and skeletal muscle, endocrine and lymphoid tissues are also recognised’ (n.d.b, [Online]).

M.E. is not defined by mere ‘fatigue’

M.E. is not synonymous with being tired all the time. If a person is very fatigued for an extended period of time this does not mean they are having a ‘bout’ of M.E. such a suggestion is no less absurd than to say that prolonged fatigue means a person is having a ‘bout’ of multiple sclerosis, Parkinson’s disease or Lupus. If a person is constantly fatigued this should not be taken to mean that they have M.E., no matter how severe or prolonged their fatigue is. Fatigue is a symptom of many different illnesses as well as a feature of normal everyday life – but it is not a defining symptom of M.E., or even an essential symptom of M.E.

The terms ‘fatigue’ and ‘chronic fatigue’ were not associated with defining this illness until the entity of ‘Chronic Fatigue Syndrome’ was created in 1988 in the USA (Hyde 2006, [online]). But M.E. and ‘CFS’ are *not* synonymous terms.

‘Fatigue’ and ‘feeling tired all the time’ are not at all the same thing as the very specific type of *paralytic muscle weakness* or *muscle fatigue* which is characteristic of M.E. (caused by mitochondrial dysfunction) and which affects every organ and cell in the body, including the brain and the heart. This causes – or significantly contributes to – such problems in M.E. as cardiac insufficiency (a type of heart failure), orthostatic intolerance or POTS (inability to maintain an upright posture), blackouts, reduced circulating blood volume (and pooling of the blood in the extremities), seizures (and other neurological phenomena), memory loss, problems chewing/swallowing, episodes of partial or total paralysis, muscle spasms/twitching, extreme pain, problems with digestion, vision disturbances, and breathing difficulties.

These problems are exacerbated by even trivial levels of physical and cognitive activity, sensory input and orthostatic stress beyond a patient’s individual limits. People with M.E. are made very ill and disabled by this problem with their cells; it affects virtually every bodily system and has also lead to death in some cases. Many patients are housebound and bedbound and are often so ill that they feel they are about to die. People with M.E. would give *anything* to only be severely ‘fatigued’ or ‘tired all the time’ (Bassett 2010, [Online]).

M.E. expert Dr Melvin Ramsay explained that this unique characteristic: ‘is virtually a sheet-anchor in the diagnosis of Myalgic Encephalomyelitis and without it a diagnosis should not be made’ (1986, [Online]). This intolerance of certain levels of physical or cognitive activity, sensory input and orthostatic stress is one of the many things which separates M.E. so distinctly from various post-viral fatigue states or other illnesses involving ‘chronic fatigue.’

M.E. expert Dr Byron Hyde also writes that: ‘In MRI spectography it has been shown that because of an abnormal buildup of normal metabolites, the muscle cell actually shuts down to prevent cell death’ (Hyde 2003, [Online]). People with M.E. are experiencing a form of heart failure which can be exacerbated by even relatively low levels of activity. People with M.E. are made very ill and disabled by this problem with their cells (and their mitochondria, and so on), it affects virtually every bodily system, and has also lead to death in some cases. Many patients are housebound and bedbound and often are so ill that they feel they are about to die.

People with M.E would give *anything* to instead be severely ‘fatigued’ or tired all the time.

Fatigue, post-exertional fatigue or malaise may occur in many different illnesses such as various post-viral fatigue states or syndromes, Fibromyalgia, Lyme disease, and many others, but what is happening with M.E. patients is an entirely different and unique problem of a much greater magnitude. These terms are not accurate or specific enough to describe what is happening in M.E. M.E. is a neurological illness of extraordinarily incapacitating dimensions that affects virtually every bodily system – not a problem of ‘chronic fatigue’ (Hyde 2006, [Online]) (Hooper 2006, [Online]) (Hooper & Marshall 2005a, [Online]) (Hyde 2003, [Online]) (Dowsett 2001, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Dowsett 1996, p. 167) (Dowsett et al. 1990, pp. 285-291) (Dowsett n.d., [Online]).

- For more information see M.E. is not fatigue, or 'CFS'. Many of the world's leading M.E. experts have spoken out strongly against claims that 'fatigue' is the defining/essential symptom of M.E. see M.E. is not defined by 'fatigue' to read some of their comments.
- For more information on the symptoms of M.E., including the unique reaction people with M.E. have to activity, see: The Ultra-comprehensive M.E. Symptom List.

If M.E. and 'CFS' are not synonymous terms, why do some groups claim that they are? What is 'CFS'?

The disease category of 'CFS' was created in a response to an outbreak of what was unmistakably M.E., but this new name and definition did not describe the known signs, symptoms, history and pathology of M.E. It described a disease process that did not, and could not exist.

The new name and case definition for 'CFS' was created in the US in 1988 by a board of 18 members, few of which had either looked at an epidemic of M.E. or examined any patients with M.E. Indeed three of the most experienced members of the board refused to sign the final document and withdrew themselves from the (CDC) definitional committee because the proposed new name for the illness and the definition that went with it were just too different from the M.E. with which they were so familiar (Hooper et al. 2001 [Online]). With very few exceptions the majority of the remaining members never published on M.E. 'again' in their careers (Hyde 1998, [online]).

The UK definition of CFS was also arrived at without the vast majority of those involved having had the benefit of examining either individual patients *or* an outbreak of the illness. Many of them were also psychologists rather than physicians. It was a similar story worldwide. As M.E. expert Dr Byron Hyde writes, 'The inclusion of psychiatrists in the defining of an epidemic and [what is] obviously a disease of infectious origin, simply muddies the water for any serious understanding of that disease' (1998, [Online]).

Why was the renaming and redefining of the distinct neurological disease M.E. allowed to become so muddled? Indeed, why did M.E. suddenly need to be renamed or redefined at all?

The answer is **Money**. There was an enormous rise in the reported incidence of M.E. in the late 1970s and 1980s, alarming medical insurance companies in the US. So it was at this time that certain psychiatrists and others involved in the medical insurance industry (on both sides of the Atlantic) began their campaign to reclassify M.E. as a psychological or 'personality' disorder, in order to side-step the financial responsibility of so many new claims (Marshall & Williams 2005a, [Online]).

As Professor Malcolm Hooper explains:

In the 1980s in the US (where there is no NHS and most of the costs of health care are borne by insurance companies), the incidence of M.E. escalated rapidly, so a political decision was taken to rename M.E. as "chronic fatigue syndrome", the cardinal feature of which was to be chronic or ongoing "fatigue", a symptom so universal that any insurance claim based on "tiredness" could be expediently denied. The new case definition bore little relation to M.E.: objections were raised by experienced international clinicians and medical scientists, but all objections were ignored... To the serious disadvantage of patients, these psychiatrists have propagated untruths and falsehoods about the disorder to the medical, legal, insurance and media communities, as well as to government Ministers and to Members of Parliament, resulting in the withdrawal and erosion of both social and financial support [for M.E. patients]. Influenced by these psychiatrists, government bodies around the world have continued to propagate the same falsehoods with the result that patients are left without any hope of understanding or of health service provision or delivery. As a consequence, government funding into the biomedical aspects of the disorder is non-existent. (2003a, [Online]) (2001, [Online])

The psychiatrist Simon Wessely – arguably the most powerful and prolific author of papers which claim that M.E. is merely a psychological problem of 'fatigue' – began his rise to prominence in the UK at the same time the first CFS definition was being created in the USA (1988). Wessely, and his like-minded colleagues – a small group made up mostly but not exclusively of psychiatrists (colloquially known as the 'Wessely School') has gained dominance in the field of M.E. in the UK (and increasingly around the world) by producing vast numbers of papers which purport to be about M.E.

Wessely claims to specialise in M.E. but uses the term interchangeably with chronic fatigue, fatigue or tiredness, plus terms such as neurasthenia, CFS and 'CFS/ME' (a confusing and misleading term he created himself). He claims that psychiatric states of ongoing fatigue and the distinct neurological disorder M.E. are synonymous. Despite all the existing contradictory evidence, Wessely (and members of the Wessely School) assert that M.E. is a behavioural disorder, with no physical signs of illness or abnormalities on testing, that is perpetuated by

‘aberrant illness beliefs’ ‘the misattribution of normal bodily sensations,’ and that patients ‘seek and obtain secondary gain by adopting the sick role’ (Hooper & Marshall 2005a, [Online]).

The Wessely School and collaborators have assiduously attempted to obliterate recorded medical history of M.E. even though the existing evidence and studies were published in prestigious peer-reviewed journals and span over 70 years. Wessely’s claims, and those of his colleagues around the world, have flooded the worldwide literature to the extent that medical journals rarely contain any factual and unbiased information about M.E. most clinicians are effectively being deprived of the opportunity to obtain even the most basic facts about the illness.

For at least a decade, serious questions have been raised in international medical journals about possible scientific misconduct and flawed methodology in the work of Wessely and his colleagues. It is only relatively recently however that his long-term involvement as medical adviser – and board member – to a number of commercial bodies with a vested interest in how M.E. is managed have been exposed.

The government funded research produced by this group continues to be rigorously criticised on the grounds that it is methodologically flawed and biased and that it relies on a highly selective and misrepresentative choice of references, and too often cites their own studies as the sole or primary references. Despite this, and the fact that this coterie of psychiatrists has a number of outrageous conflicts of interest and proven affiliations with corporate industry they have managed to assiduously infiltrate all the major institutions – including government – directing funding for M.E. research into an exclusively psychiatric model of the illness; and which involves studying ‘fatigue’ sufferers instead of those with M.E. All under the ‘anything-goes’ banner of ‘CFS’ (Mar 2004, [Online]) (Hooper 2003, [Online]) (Hooper et al. 2001, [Online]).

This is the sole reason the myth that M.E. is a psychiatric or behavioural ‘fatiguing’ disorder or even an ‘aberrant belief system’ continues: not because there is good scientific evidence for the theory, or because the evidence proving organic causes and effects is lacking, but because such a theory is so **financially and politically convenient and profitable** on such a large scale to a number of extremely powerful corporations (Hooper et al 2001, [Online]).

Members of the ‘Wessely school’ in the UK including Wessely, Sharpe, Cleare and White, their US counterparts Reeves, Straus etc of the CDC, in Australia Lloyd, Hickie etc and the clinicians of the Nijmegen group in the Netherlands each support a bogus psychiatric or behavioural paradigm of ‘CFS’ and recommend rehabilitation-based approaches such as cognitive behavioural therapy (CBT) and graded exercise therapy (GET) as the most useful interventions for ‘CFS’ patients. It is important to be aware that none of these groups is studying patients with M.E. Each of these groups uses a definition of ‘CFS,’ or has created their own, which does not select those with M.E. but instead selects those with various types of psychiatric and non-psychiatric fatigue. (These inappropriate interventions are at best useless and at worst extremely harmful or fatal for M.E. patients. See [Smoke and mirrors](#))

‘CFS’ makes getting disability payouts almost impossible, as there are no tests whatsoever that can be used to prove the existence of ‘CFS’ and because there is also so much bogus ‘information’ available about how easily and successfully ‘CFS’ can be managed or even cured. The CDC (and all other) ‘CFS’ definitions define ‘CFS’ as a psychological illness – which many health insurance policies explicitly exclude and many limit to two years’ cover. ‘CFS’ allows insurance companies and governments to evade or at least greatly limit claims all over the world. **If the US has only had a universal healthcare system in place in the 1980s, and there hadn’t been obscene profit to be made by denying the existence of serious organic illnesses, this ‘CFS’ mess would never have happened.**

Among his 53, largely undeclared, conflicting interests Wessely is a member of the supervisory board of a company named PRISMA. This same company is being paid many millions of pounds to supply ‘rehabilitation’ programs (such as CBT and GET) to the NHS for use on ‘CFS’ patients (Mar 2004, [Online]). Wessely is also an officer of UNUM. (The facts on Wessely’s colleagues are equally disturbing. White is Chief Medical Officer of Swiss Re. LoCascio of UNUM advised the UK DWP (Welfare Office) on welfare reform while Aylward was in charge of UK DWP and then head of UNUM’s research establishment at Cardiff University. The list goes on.) Wessely was also recently reprimanded by the World Health Organisation (WHO) for attempting to subvert the ICD definition of M.E. due to the fact that he did not, as he claimed, have the authority to issue a WHO definition (Hooper 2003a, [Online]) (Hooper et al. 2001, [Online]) (Marshall & Williams 2005a, [Online]).

This group has also driven government policy on M.E. in the UK to an overwhelming extent. Wessely is adviser to the UK government and his wife (a GP and psychiatrist) is Senior Policy Adviser to the Department of Health.

This large scale deception by insurance companies has been made possible largely because of the fact that holding some of the most powerful advisory positions in government (as some of these vested interest psychiatrists do) does not seem to be mutually exclusive with also having direct ties and allegiances to industry, even if those industries are directly affected by the decisions made by the government department/adviser in question (as the

giant chemical, pharmaceutical and insurance industries are in M.E.) (Hooper 2003a, [Online]). As Professor Malcolm Hooper goes on to explain:

Increasingly, it is now "policy-makers" and Government advisers, not experienced clinicians, who determine how a disorder is classified and managed in the NHS: the determination of an illness classification and the provision of policy-driven "management" is a very profitable business. To the detriment of the sick, the deciding factor governing policies on medical research and on the management and treatment of patients is increasingly determined not by medical need but by economic considerations.

Given that what Wessely promotes is contrary to the established scientific evidence, how does he manage to maintain such power and control? Many knowledgeable people believe he maintains it by singing the desired political tune; by scientific misconduct; by manipulation of other people's published work; by flawed methodology; by deception and by the circularity of self-references. Substantial evidence clearly reveals that in pursuit of his personal ideology or, alternatively, that of his corporate masters, Wessely abuses the scientific process. The implementation of his personal philosophy is not based on medical science and has had devastating consequences, not just for sufferers of M.E. but for their families as well.

There is a gross mismatch between the severity and complexity of M.E. and the medical and public perception of the disorder, but until Simon Wessely is held to public account, and medical professionals and public alike are informed and educated about the reality of M.E., this will continue (2003a, [Online]).

These inappropriate interventions are at best useless and at worst extremely harmful or fatal for M.E. patients.

In addition to insurance companies, who else benefits from the 'CFS,' 'ME/CFS,' 'CFS/ME' and Myalgic 'Encephalopathy' and so on, fictions continuing? From M.E. and 'CFS' not being clearly separated and all patient groups involved being correctly diagnosed and treated based on science?

Other groups which benefit financially, politically or in other ways include the following:

- A. Governments
- B. The vaccine industry
- C. The chemical industry
- D. Psychiatrists
- E. 'CFS' doctors
- F. Medical doctors
- G. The media (including medical journals)
- H. CFS' or 'ME/CFS' (and other) groups that sell vitamins and other supplements to 'CFS' patients
- I. CFS' or 'ME/CFS' (etc.) so-called patient support and advocacy groups.

How have these groups each managed to avoid society's various checks and balances?

The creation of the bogus disease category 'CFS' has been used to impose a false psychiatric paradigm of M.E. by allying it with various unrelated psychiatric fatigue states and post-viral fatigue syndromes for the benefit of various (proven) financial and political interests

The resulting 'confusion' between the distinct neurological disease M.E. and the bogus disease category of 'CFS' has caused an overwhelming additional burden of suffering for those who suffer from M.E. and their families. **It's a huge mess, that is for certain - but it is not an accidental mess - that is for certain too** (Hyde 2006a, [Online]) (Hooper 2006, [Online]) (Hyde 2003, [Online]) (Hooper 2003a, [Online]) (Dowsett 2001a, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]).

It is also a certainty that the medical insurance companies could not have achieved the current state of affairs alone, with the concept of 'CFS' as their only weapon. All of the groups listed above *collaborate*.

There are different corporate and government interests involved, and they share a financial interest in suppressing M.E. and promoting 'CFS,' so they work together. For example, pharmaceutical companies fund the research, psychiatrists define the illness, assess the patient, advise the government departments in creating definitions and policy, insurance companies rely on official definitions and policy and employ psychiatrists to assess the patients, government welfare departments use the definitions and policy in assessing claimants, sell-out so-called advocacy groups support the latest government 'awareness' campaign in return for getting government funding. Most journalists act as mere stenographers when they write about 'CFS' rather than investigative journalists; they copy the government press releases almost word for word rather than doing any genuine research into the facts. This is just a brief summary of a small number of the deals we know about. There are clearly many more.

That is how these groups have been successful and how they have for the most part avoided society's checks and balances, by collaborating with each other to protect their shared financial or political gains. A group acting alone can be stopped, by making other groups aware of what is happening. But what happens when almost all of the different groups which are there to protect the interests of the victims are actually in on the scam themselves? What do the victims do then? How does one convince others of the truth when so many seemingly benign companies or supposedly patient-based organisations are producing so much completely mutually supportive and superficially convincing propaganda? This is the problem facing M.E. patients.

What makes the problem even worse is that unlike AIDS patients who in the early stages of their illness are able to march and rally and organise protests, most M.E. patients are far too ill to participate in such activism efforts. They may often not even be well enough to read the basic facts about what is happening. Thus nothing has changed for the better in the 20 years since the 'CFS' scam began. Thanks to the increasing psychological emphasis of succeeding CDC definitions of 'CFS,' 'ME/CFS' replacing M.E. in official policy in UK, Australia and Europe, and the covert infiltration of patient advocacy groups by vested interest groups, and so on, the level of abuse affecting M.E. patients is only *worsening* as time goes on

- For more information on this topic, including how each of these groups benefits from 'CFS' and 'ME/CFS' see: [Who benefits from 'CFS' and 'ME/CFS'?](#)
- See also: [A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic Fatigue Syndrome & A Brief History of Myalgic Encephalomyelitis & An Irreverent History of Chronic Fatigue Syndrome](#) and [The Nightingale Definition of M.E.](#) by Dr Byron Hyde, and [Research into ME 1988 - 1998 Too much PHILOSOPHY and too little BASIC SCIENCE!](#) and [The Late Effects Of M.E.](#) and [A Rose by Any Other Name](#) and [Redefinitions of ME - a 20th Century Phenomenon](#) by Dr Elizabeth Dowsett, plus [What is ME? What is CFS? Information for Clinicians & Lawyers](#) and [The Mental Health Movement: Persecution of Patients?](#) by Professor Malcolm Hooper and [A Public Statement to Government Health Ministers and an ALERT to citizens worldwide.](#) [Activism Articles](#), [Skewed](#) (book) and [Osler's Web](#) (book).

What does a diagnosis of 'CFS' actually mean?

There are now more than nine different definitions of 'CFS.' Each of these flawed 'CFS' definitions 'define' a heterogeneous (mixed) population of people with various misdiagnosed psychiatric and non-psychiatric states which have little in common but the symptom of fatigue.

The fact that a person qualifies for a diagnosis of 'CFS', based on any of the 'CFS' definitions: (a) does not mean that the patient has M.E., and (b) does not mean that the patient has any other distinct and specific illness named 'CFS.' A diagnosis of 'CFS' – based on any of the 'CFS' definitions – can only ever be a *misdiagnosis*. All a diagnosis of 'CFS' actually means is that the patient has a gradual onset fatigue syndrome which is usually due to a *missed major disease*. As [Dr Byron Hyde](#) explains, the patient has:

- Missed cardiac disease,
- Missed malignancy,
- Missed vascular disease,
- Missed brain lesion either of a vascular or space occupying lesion,
- Missed test positive rheumatologic disease,
- Missed test negative rheumatologic disease,
- Missed endocrine disease,
- Missed physiological disease,
- Missed genetic disease,
- Missed chronic infectious disease,
- Missed pharmacological or immunization induced disease,
- Missed social disease,
- Missed drug use disease or habituation,
- Missed dietary dysfunction diseases,
- Missed psychiatric disease (2006, [Online]).

The only groups which gain from this 'CFS' confusion are insurance companies and other organisations and corporations which have a vested financial interest in how these patients are treated, including the government. Under the cover of 'CFS' certain vested interest groups have assiduously attempted to obliterate recorded medical history of M.E., even though the existing evidence has been published in prestigious peer-reviewed journals around the world and spans over 70 years. The only way forward for M.E. patients and all of the diverse patient groups commonly misdiagnosed with 'CFS' (both of which are denied appropriate support, diagnosis and treatment, and may also be subject to serious medical abuse) is that the bogus disease category of 'CFS' must be abandoned. As M.E. expert [Dr Byron Hyde](#) explains:

Do not for one minute believe that CFS is simply another name for Myalgic Encephalomyelitis. It is not. The CDC 1988 definition of CFS describes a non-existing chimera based upon inexperienced individuals who lack any historical knowledge of this disease process. The CDC definition is not a disease process. It is (a) a partial mix of infectious mononucleosis /glandular fever, (b) a mix of some of the least important aspects of M.E. and (c) what amounts to a possibly unintended psychiatric slant to an epidemic and endemic disease process of major importance. Any disease process that has major criteria, of excluding all other disease processes, is simply not a disease at all; it doesn't exist. The CFS definitions were written in such a manner that CFS becomes like a desert mirage: The closer you approach, the faster it disappears (2006, [Online])

People with M.E. must be diagnosed with M.E., and treated for M.E. based on information gained solely from studies involving authentic M.E. patients. People with depression must be diagnosed and treated for depression. People with cancer must be diagnosed with cancer and then treated as appropriate for the type of cancer they have, and so on. Physicians who diagnose 'CFS' in any patient experiencing fatigue without looking and testing for the true cause of the symptoms (and who choose not to familiarise themselves with the scientific facts about M.E. do their patients – and themselves – a great disservice. (This misdiagnosis and mistreatment may also create legal consequences for the physician.)

Some of the conditions commonly misdiagnosed as CFS are very well defined and well-known illnesses and very treatable – but only once they have been correctly diagnosed. Some conditions can become very serious or can even be fatal if not correctly diagnosed and managed, including M.E. Every patient deserves the best possible opportunity for appropriate treatment for their illness and for recovery and this process must begin with a correct diagnosis if at all possible. *A correct diagnosis is half the battle won* (Hyde 2006a, 2006b, [Online]) (Hooper 2006, [Online]) (Hyde 2003, [Online]) (Hooper 2003a, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Dowsett n.d., [Online]).

- For more information on why the bogus disease category of 'CFS' must be abandoned see: [The misdiagnosis of CFS](#), [Why the disease category of 'CFS' must be abandoned](#) and [Smoke and Mirrors](#).
- Those patients misdiagnosed with 'CFS' (and who do not have M.E.) are advised to read the following papers: and *An additional note on 'fatigue'*: Just as some M.E. sufferers will experience other non-essential symptoms such as vomiting or night sweats some of the time, but others will not, the same is true of fatigue. The diagnosis of M.E. is determined upon the presence of certain neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms – the presence or absence of mere 'fatigue' is irrelevant. In addition to these other (far more serious) symptoms, some M.E. sufferers may also suffer with mild, moderate or severe fatigue some of the time, while others will not. Thus the symptom of fatigue is not an essential symptom of M.E. and does not define M.E., **although the symptom of fatigue is certainly essential to qualify for a (mis)diagnosis of 'CFS.'**

What do the terms CFIDS, ME/CFS, CFS/ME, Myalgic Encephalopathy and ME-CFS mean?

When the terms CFS, CFIDS, ME/CFS, CFS/ME, or Myalgic Encephalopathy are used, what is being referred to may be patients with any combination of:

1. Miscellaneous psychological and non-psychological fatigue states (including somatisation disorder).
2. A self limiting post-viral fatigue state or syndrome (e.g. following glandular fever).
3. A mixed bag of unrelated, misdiagnosed illnesses (each of which features fatigue as well as a number of other common symptoms; poor sleep, headaches, muscle pain etc.) including Lyme disease, multiple sclerosis, Fibromyalgia, athletes over-training syndrome, depression, burnout, systemic fungal infections (candida) and even various cancers.
4. M.E. patients.

The terminology is often used interchangeably, incorrectly and confusingly. However, the DEFINITIONS of M.E. and 'CFS' are very different and distinct, and it is the definitions of each of these terms which is of primary importance. *The distinction must be made between terminology and definitions.*

1. **Chronic Fatigue Syndrome** is an artificial construct created in the US in 1988 for the benefit of various political and financial vested interest groups. It is a mere diagnosis of exclusion (or wastebasket diagnosis) based on the presence of gradual or acute onset fatigue lasting at least 6 months. If tests show serious abnormalities, a person no longer qualifies for the diagnosis, as 'CFS' is 'medically unexplained.' A diagnosis of 'CFS' does not mean that a person has any distinct disease (including M.E.). The patient population diagnosed with 'CFS' is made up of people with a vast array of unrelated illnesses, or with no detectable illness. According to the latest CDC estimates, 2.54% of the population qualifies for a 'CFS' diagnosis. Every diagnosis of 'CFS' can only ever be a misdiagnosis.
2. **Myalgic Encephalomyelitis** is a systemic neurological disease initiated by a viral infection. M.E. is characterised by scientifically measurable damage to the brain, and particularly to the brain stem which results in dysfunctions and damage to almost all vital bodily systems and a loss of normal internal homeostasis. Substantial evidence indicates that M.E. is caused by an enterovirus. The onset of M.E. is always acute and M.E. can be diagnosed within just a few weeks. M.E. is an easily recognisable distinct organic neurological disease which can be verified by objective testing. If all tests are normal, then a diagnosis of M.E. cannot be correct.

M.E. can occur in both epidemic and sporadic forms and can be extremely disabling, sometimes fatal. M.E. is a chronic/lifelong disease that has existed for centuries. It shares similarities with M.S., Lupus and Polio. There are more than 60 different neurological, cognitive, cardiac, metabolic, immunological and other M.E. symptoms. Fatigue is not a defining or even essential symptom of M.E. People with M.E. would give anything to be only 'fatigued' instead of having M.E. Far fewer than 0.5% of the population has the distinct neurological disease known since 1956 as Myalgic Encephalomyelitis.

But aren't alternate concepts such as 'ME/CFS, Myalgic 'Encephalopathy' and 'CFIDS' at least an improvement on 'CFS'?

No, they are not. Most often when the term 'ME/CFS' is used, it refers to a bizarre mix of facts relating to both M.E. and 'CFS' or instead purely facts relating to any of the various bogus 'CFS' definitions. The same applies to the terms 'CFS/ME,' 'CFIDS' and 'Myalgic Encephalopathy.'

Terms such as '**ME/CFS**' and '**CFS/ME**' make about as much scientific sense as terms such as diabetes/Alzheimers, depression/HIV or headache/peanut allergy. They join together two completely opposed entities as if they were synonymous terms, only increasing the present confusion between M.E. and 'CFS' that causes patients (and the community) so much harm.

The groups that benefit from 'ME/CFS' are the same groups that benefit from 'CFS.' It is hardly a coincidence that Professor Simon Wessely – the most powerful and influential of the group of doctors who have made themselves the tools of insurance companies – is the person credited with inventing the mixed term 'CFS/ME.' The mixing of M.E. and 'CFS' into 'CFS/ME' or 'ME/CFS' serves vested interest groups well. This is why so many of the very worst government reports (and so on) in the UK, Australia and the Netherlands which talk about patients as if they were mildly ill malingerers who could easily improve if not recover from their 'fatigue' if only they could be convinced to try CBT or GET, and so on, (a) often use terms such as 'CFS/ME' or 'ME/CFS' in the titles and throughout and (b) very often mix in some of the facts about M.E. (i.e. symptoms, history, severity/disability etc.) with bogus information about 'CFS' while of course the entirety of the all-important CONCLUSIONS given (i.e. aetiology, psychological status, improvement of symptoms, response to treatments and recovery rates) is drawn exclusively from non-M.E. patient groups, and from the most mildly affected physically and the most primarily psychologically ill members of these 'CFS' diagnosed groups.

'ME/CFS' and 'CFS/ME' lets these vested interest groups have it both ways. They get to continue happily with their unscientific and unethical 'CFS' obfuscation agenda, which allows governments and insurance companies to deny medical and welfare claims, and they get to do so with far less opposition from the patients they're harming, or even with the support of some of these patients and patient groups who are taken in by the superficial appearance of progress conferred by a mere terminology mix.

This is why 'ME/CFS' articles and studies are even more dangerous in many ways than pure 'psychological CFS' ones. The issue is not that 'ME/CFS' just isn't a very good solution that will not do much good, as many have been arguing. There is so much more than that at stake here. Not only will 'ME/CFS' not help, it can and will make things so much worse. It will make the truth about M.E. even more invisible (or 'inaccessible') by hiding it in plain sight. make it harder than ever for anyone to separate M.E. out from the vague mess of 'CFS' or for those misdiagnosed as 'CFS' to be receive a correct diagnosis and appropriate treatment, as is their basic right.

There is no agreed definition for these terms (many different groups use these terms to mean very different things), nor any World Health Organization classification. Much of the very worst 'CFS' propaganda that has been produced has used these terms throughout.

The 'ME/CFS' concept is confusing, illogical, strongly reinforces the same misinformation which is the cause of the problem (i.e. that M.E. and 'CFS' are the same and that 'CFS' actually exists), and holds back the fight for justice and recognition of authentic neurological M.E. immeasurably. The mixing of M.E. and 'CFS' was invented by these vested interest groups and it is a tool they use to good effect and as much as possible. The countries in which the 'ME/CFS' concept is commonplace are those in which patients are subject to the most shocking abuse, far worse than that which occurs now in the US.

None of the justifications made by so-called advocacy groups for using the term 'ME/CFS' hold up. Putting M.E. together with 'CFS' doesn't add to the credibility of 'CFS' – it just strips M.E. of credibility and scientific legitimacy – which indeed seems to be the entire point of the exercise.

For more information on why advocates and patients must reject 'ME/CFS' see: [Problems with the so-called "Fair name" campaign: Why it is in the best interests of all patient groups involved to reject and strongly oppose this](#)

misleading and counter-productive proposal to rename 'CFS' as 'ME/CFS' and Problems with the use of 'ME/CFS' by M.E. advocates

The term **CFIDS** (Chronic Fatigue and Immune Dysfunction Syndrome) was created in the USA. When this term was first used, it was used in some ways as a synonymous term for M.E., but this is no longer the case. Although the end result is the same however, the term 'CFIDS' in contrast with the term Myalgic 'Encephalopathy' was at least well intentioned and a genuine attempt at differentiating those who merely qualify for a 'CFS' misdiagnosis and genuine epidemic and sporadic M.E. patients. Today this term is almost entirely just another term used to describe 'CFS.' Much of the very worst 'CFS' propaganda that has been produced has referred to the illness as CFIDS. The term CFIDS has no agreed definition, and no World Health Organization classification. It is a term that could be taken to mean anything and which means very different things to different people. (M.E. is not defined by 'fatigue' and many different non-M.E. illnesses – including depression – have immune system abnormalities, so this term is still very vague and inclusive and non-specific to M.E.). The term 'CFIDS' should in no way be considered as synonymous with M.E. This term is unhelpful and unscientific and only adds to and aids the obfuscation by vested interest groups.

Myalgic 'Encephalopathy' is a made-up term that was created only *after* the disastrous 'CFS' definitions. The term Myalgic 'Encephalopathy' was created in the UK, for reasons involving politics and vested interests rather than science. The claimed scientific justifications for the creation and use of this made-up name are bogus. Myalgic 'Encephalopathy' is linked to no specific definition and no specific patient group. The term was not created through a careful examination of the evidence or because of any specific research findings. There is no scientific evidence behind ME'opathy whatsoever and (as is appropriate) this term has no WHO ICD classification. ME'opathy is merely another name for 'CFS.' It is a term that could be taken to mean anything and so is just as meaningless *and as harmful* as 'CFS' is.

Do not be fooled by the merely superficial similarity of these terms – Myalgic Encephalomyelitis is not at all the same thing as Myalgic 'Encephalopathy.' Patients with authentic M.E. do have the damage to the brain referred to in the name Myalgic Encephalomyelitis; however this damage is of course not found in patients suffering various types of chronic fatigue illnesses which are commonly misdiagnosed as 'CFS.' Legitimate M.E. experts, advocates and researchers do not support the name change from Myalgic Encephalomyelitis to Myalgic 'Encephalopathy.' Patient advocates Margaret Williams and Eileen Marshall write:

Despite the relentless financial, psychosocial and political engineering that seems to underpin the current determination to remove the term "myalgic encephalomyelitis" (M.E.) from the medical lexicon (where, based on accurate published evidence of the nature of the disorder, it has resided for the last half century), the present proponents of its demise have failed to produce any evidence-base to support their clamour for its removal and its replacement by the less specific term "myalgic encephalopathy" (2004a, [Online])

The Committee for Justice and Recognition of Myalgic Encephalomyelitis also explain that:

None of the contemporaries of Ramsay, such as Dowsett and Richardson, who have been asked to comment on the appropriateness of a change from ME'itis to ME'opathy have found ME'opathy an acceptable explanation. Myalgia means muscle pain. Encephalo - means brain, myelitis has two meanings, some say it refers to inflammation of the spinal chord, others to inflammation of the myelin, the covering of the brain. Both are physical descriptions. Oopathy, on the other hand means pathology - which can mean 'the science or origin, nature, and courses of diseases', but another meaning is 'any abnormal state: social pathology' (Delbridge 1998). Hence encephalopathy can mean 'brain abnormal state' and this meaning would therefore endorse treatments such as CBT and GET - which do not work in those with neurological ME (which meets the Ramsay criteria). This change of name to 'opathy' can therefore be seen to endorse psychological therapies as treatment. Muscle pain brain myelin inflammation is not the same as muscle pain brain abnormal state. And the neurological damage which is evident in ME can be explained by myelin inflammation but it cannot be explained by 'brain abnormal state'. Evidence for brain damage has been found in the research of persons such as Casse et al. (2001), Poser (1992) and others. And there is often confusion with MS by persons in the medical profession - where there is myelin damage ([2007, [Online]).

Support for this term is red flag that lets you know a group is not to be trusted and is not involved in genuine advocacy. The use of the meaningless term Myalgic 'Encephalopathy' is a dishonest attempt to divest Myalgic Encephalomyelitis of the legitimacy and protection of its correct WHO classification. It is also an attempt to cover up the links between M.E., polio and the polio vaccine indicated by the term 'Encephalomyelitis'; to try to hide the 'smoking gun' of the term M.E. as it were. The term 'Myalgic Encephalopathy' is a political creation with no scientific validity, just as 'CFS' is. *It is a trap, a trick.* The last thing needed is yet another vague and ill-defined umbrella term that can easily be manipulated by vested interest groups. As Professor Malcolm Hooper explains:

There have been persistent and frequently covert attempts by these [vested interest] psychiatrists to subvert the international classification of this disorder, with destructive consequences for those affected. Correct classification does matter because it impacts on correct referral to an appropriate specialist, correct investigations, correct diagnosis, correct management and / or treatment, correct State benefit support [and] correct insurance policy payments (2003a, [Online]) (Hooper & Marshall 2005a, [Online]).

- For more information on the name Myalgic Encephalomyelitis (and the political motivations behind terms such as ME'opathy) see: [On the name MEitis](#) and [Problems with the so-called "Fair name" campaign: Why it is in the best interests of all patient groups involved to reject and strongly oppose this misleading and counter-productive proposal to rename 'CFS' as 'ME/CFS'](#)

It should also be noted that Wessely, Sharpe, Cleare and White (etc.) in the UK, their counterparts (and sometime collaborators) in the US; Reeves and Straus (etc. of the CDC) in Australia Lloyd and Hickie (etc.) and the clinicians of the Nijmegen group in the Netherlands often use the terms 'fatigue' 'chronic fatigue' 'CFS' CFS/ME' Myalgic 'Encephalopathy' (or Myalgic Encephalomyelitis) INCORRECTLY as if they were synonymous. Such obfuscation has greatly hindered research and caused considerable harm to many hundreds of thousands of patients (Hooper n.d., [Online]).

- For more information on the name Myalgic Encephalomyelitis (and the problems with some of these other terms including ME'opathy) see: [The definitions of M.E., Meitis? A slender string to our bow](#), [The Terminology of ME & CFS](#), [What is ME? What is CFS?](#), and [ME and CFS, The Definitions](#). See [On the name MEitis](#) for more further articles.
- For more information on why the bogus disease category of 'CFS' must be abandoned, (along with the use of other vague and misleading umbrella terms such as 'ME/CFS' 'CFS/ME' 'CFIDS' and 'Myalgic Encephalopathy' and others), see: [The misdiagnosis of CFS](#), [Why the disease category of 'CFS' must be abandoned](#) and [Smoke and Mirrors](#). M.E. and 'CFS' are not synonymous terms, and should not be used interchangeably. *The distinction must be made between terminology and DEFINITIONS*. The truth about the organic and distinct neurological illness M.E. must not be allowed to be buried under cover of 'fatigue' and 'CFS' for another 20 years!
- **A note on the current 'CFS' name change proposal:** It is *madness* to suggest that CFS should be renamed as ME-CFS or CFS/ME, as some US CFS groups are currently advocating. M.E. and CFS are not the same, only a small percentage of those (mis)diagnosed with CFS qualify for a diagnosis of authentic M.E., the vast majority do not. People with depression, Lyme disease, candida, etc. do not need to be given an additional misdiagnosis of ME/CFS, they must instead be given a correct diagnosis finally. It is unethical to use the name of another patient group to further your own interests, particularly when this causes significant additional harm and hardship to that group (and also when, generally speaking, that group is far more severely ill and vulnerable). People with various non-M.E. disorders misdiagnosed as CFS, have no more right to use the name Myalgic Encephalomyelitis (or any modified version of it) than they have to use terms such as 'Cancer' or 'HIV' just because they decide that it would suit their own purposes to do so. The suggestion that CFS should be renamed in any way as M.E. is grossly illogical, unethical and unscientific.

It may well be the case that patients with various post-viral fatigue syndromes (caused by glandular fever or mononucleosis, hepatitis, Ross River virus etc.), some of the main groups pushing for this absurd renaming, would benefit from these conditions being renamed in some way, but this new name must be one which is not TAKEN already by an entirely unrelated and already well-defined patient group. (The fact that some of these patients, and others, may fit the Canadian criteria for 'ME/CFS' does not mean that these patients can be correctly diagnosed with M.E. – as per Ramsay/Richardson/Dowsett and Hyde – nor that these illnesses are the same or 'virtually the same' as M.E. They are not. The Canadian 'ME/CFS' Guidelines and the newer version titled the International Consensus Criteria (ICC) are not accurate M.E. definitions. They are not definitions of M.E. at all. They are both redefinitions of 'CFS' which unscientifically add in a few facts about M.E. and by doing so unhelpfully worsen the confusion between these two very different entities. Read more about the benefits and the limitations of the Canadian Guidelines and the ICC at: [Canadian Guidelines Review](#) and [Testing for M.E.](#))

See also: [Where to after a 'CFS' \(mis\)diagnosis?](#), [Why the bogus disease category of 'CFS' must be abandoned](#) and [Problems with the so-called "Fair name" campaign: Why it is in the best interests of all patient groups involved to reject and strongly oppose this misleading and counter-productive proposal to rename 'CFS' as 'ME/CFS' for more information.](#)

But isn't the name 'CFS' a big part of the problem?

The reason so many patients are ridiculed, sneered at, belittled, disbelieved, accused of exaggerating or malingering or laziness by medical staff and by friends and family members etc. IS NOT BECAUSE OF THE NAME 'Chronic Fatigue Syndrome'!

If 'CFS' had instead been given a neutral name, say 'Reeves' syndrome' or 'Holmes' syndrome,' the problems would still be *exactly the same*. Vested interest groups – helped in this task IMMEASURABLY by the creation of the bogus disease category of 'CFS' – would still be flooding the medical, political and media communities with

lies and propaganda which could only have the end result of making patients seem utterly pathetic and undeserving of any respect or sympathy.

What else could anyone think of patients who supposedly have an illness that is mild and short lived, but which some patients pretend is severely disabling because they 'enjoy the sick role'? What else could anyone think about an illness that cannot in any way be proved despite vast sums being spent on tests and must be taken completely on faith. What else could you/anyone think about an illness that has been proven to be psychological or behavioural but where patients would prefer to actually stay ill rather than to admit that they are mentally ill?

Every media article and government press release about 'CFS' is filled with fictional statements which make it very clear in many different ways that the illness has no scientific validity, and that the patients do not deserve the same respect as other patient groups, but should be treated with contempt. Patients are not merely wrongly categorized as psychologically ill; it is so much more than that. It is persecution; patients are talked about (and lied about) as if they were malingerers and deviants, as if they were beneath contempt and not worthy of even basic respect or medical care, or even any level of kindness or compassion – even from their own friends and family. Whatever 'CFS' had been called, these problems would be EXACTLY THE SAME.

There is no such disease as 'CFS' and 'CFS' is merely an artificial entity created for the benefit of financial vested interest groups – that is the real problem, not the name 'CFS.'

The infectious neurological disease known since 1956 as Myalgic Encephalomyelitis already has a historically and medically correct name and definition and WHO classification. We also have clear definitions and names for Fibromyalgia, post-viral fatigue syndromes, PTSD, burnout, Lyme disease, Candida, Adrenal exhaustion, cancer, depression, athletes over-training syndrome and the other illnesses commonly misdiagnosed as 'CFS.'

Again, the only thing that makes any sense is for advocates and patients to fight together to get rid of 'CFS' in name and definition, and to have patients correctly diagnosed with and treated for whichever illnesses they actually have, including M.E., in a scientific and ethical manner – without any self-interested interference by financial vested interest groups.

- For more information see: [Problems with the so-called "Fair name" campaign](#) and [Problems with the use of 'ME/CFS' by M.E. advocates](#).

What does the term ICD-CFS mean?

The various definitions of 'CFS' *do not* define M.E. Myalgic Encephalomyelitis is an organic neurological disorder as defined at G.93.3 in the World Health Organization's International Classification of Diseases (ICD). The definitions of 'CFS' do not reflect this. The 'CFS' definitions are not 'watered down' M.E. definitions, as some claim. They are not definitions of M.E. at all.

However, ever since an outbreak of M.E. in the US was given the label 'CFS,' the name/definition 'CFS' has prevailed for political reasons. 'CFS' is widely though wrongly applied to M.E. as well as to other diseases. The overwhelming majority of 'CFS' research does not involve M.E. patients and is not relevant *in any way* to M.E. patients. However, a minuscule percentage of research published under the name 'CFS' clearly does involve a significant number of M.E. patients as it details those abnormalities which are unique to M.E. Sometimes the problematic term 'ICD-CFS' is used in those studies and articles which, while they use the term 'CFS,' do relate to some extent to authentic M.E.

In addition to its use in relation to research, some people use the term 'ICD-CFS' to refer to the disease generally. The term is usually used by people who are aware of the psychological paradigm of 'CFS,' and who want to indicate a real, biological disease rather than a psychological one. However, which exact disease or diseases are being referred to with this term varies considerably from one author to another. Sometimes the term is used to describe M.E. while others use it to describe all those patients who qualify for a 'CFS' misdiagnosis but who are not mentally ill (incorrectly calling these diverse patient groups 'subgroups of 'ICD-CFS' and so on), while others use it to describe patients with Lyme disease, various post-viral fatigue syndromes and so on. As with terms such as 'ME/CFS' the term is meaningless and only increases confusion as it has no agreed definition and many different groups use it to refer to very different, and often very mixed, patient groups.

Problems with 'CFS' or so-called 'ICD-CFS' research

The overwhelming majority of 'CFS' research does not involve M.E. patients and is not relevant *in any way* to M.E. patients. A small number of 'CFS' studies refer in part to people with M.E. but it may not always be clear

which parts refer to M.E. Unless studies are based on an exclusively M.E. patient group, results cannot be interpreted and are meaningless for M.E.

As with research which uses the term 'CFS' (or ME/CFS, or CFIDS etc.), so with research which uses the term 'ICD-CFS': virtually all of the research which does relate to M.E. (at least in part) is also significantly contaminated by 'CFS' propaganda.

While it is important to be aware of the small amount of research findings that do hold some value for M.E. patients, using the term 'ICD-CFS' to refer to this research is misleading and in many ways just damaging as using terms and concepts like 'ME/CFS' or 'CFS/ME.'

- For further details of the WHO ICD classifications of M.E. and 'CFS' worldwide (and why terms such as 'ICD-CFS,' 'ME/CFS' and Myalgic Encephalopathy' must be avoided) please see the new paper by patient advocate Lesley Ben entitled: [The World Health Organization's International Classification of Diseases \(WHO ICD\), ME, 'CFS,' 'ME/CFS' and 'ICD-CFS'](#)
- Note that virtually all of the research which does relate to M.E. (at least in part) but which uses the term 'CFS' (or ME/CFS, or CFIDS etc.) is also contaminated in some way by 'CFS' misinformation. Most often these papers contain a bizarre mix of facts relating to both M.E. and 'CFS' and imply that M.E. and 'CFS' represented one and the same patient group. Not all those involved with 'CFS' have vested financial and political interests, yet often these non-vested-interest groups still also produce significantly flawed, psychiatrically biased and 'fatigue' based information. Unfortunately these other groups have been unduly swayed and manipulated to varying extents by the enormous amount of superficially legitimate information widely disseminated by such powerful vested groups and individuals. Some researchers have seemingly been taken in entirely by such scientifically unsupportable theories, as have the large majority of the world's journalists and politicians (albeit with some notable exceptions). Even some of the best research on the illness is shrouded in heavy usage of misleading and propagandising language and false statements which often bizarrely contradict the harsh realities uncovered in the studies themselves, unfortunately.
For information on some of the most common inaccuracies and 'CFS' misinformation included in (to some extent) M.E. relevant research, see the papers: [Putting research and articles on M.E. into context](#) and [A warning on 'CFS' and 'ME/CFS' research and advocacy](#).

What does define M.E.? What is its symptomatology?

M.E. is a systemic acutely acquired illness, initiated by a virus infection, which is characterised by post encephalitic damage to the brain stem (CNS) - a nerve centre through which many spinal nerve tracts connect with higher centres in the brain in order to control all vital bodily functions. this is always damaged in M.E., Hence the name Myalgic Encephalomyelitis.

The CNS is diffusely injured at several levels; these include the cortex, the limbic system, the basal ganglia, the hypothalamus as well as areas of the spinal cord and its appendages. This persisting multilevel CNS dysfunction is undoubtedly both the chief cause of disability in M.E. and the most critical in the definition of the entire disease process.

M.E. represents an acute change in the balance of neuropeptide messengers, and consequently, a resulting loss of the ability of the CNS to adequately receive, interpret, store and recover information which enables it to control vital body functions (cognitive, hormonal, cardiovascular, autonomic and sensory nerve communication, digestive, visual auditory balance etc). It is a loss of normal internal homeostasis. The individual can no longer function systemically within normal limits.

The problem is one of maintaining systemic functioning within normal limits in the face of a chronic infectious stress. The resulting loss of normal internal homeostasis arises from the fact that a chronic viral infection provokes reactive changes in these peptides with the consequence of pathophysiological changes and autonomic dysregulation. These powerful excitatory and inhibitory mechanisms for rapid physiological adjustment work well with short-term stressors. Frequently, these mechanisms shut down the biological system allowing for compensatory adjustments of the homeostatic mechanisms. When the stressor is an infectious agent, the messenger mechanisms stimulate compensating immune reactions to rid the body of this stressor and ultimately return the individual to a normal internal homeostasis. By definition, chronic infections have managed to escape these initial compensatory immune mechanisms. However, the neurochemical homeostatic events continue to be employed uselessly and to the detriment of the organism. This modulatory biochemical complex, biologically derived over the millennium to assist the organism, destabilises the autonomic neuronal outflow and the individual

can no longer function systemically within normal limits. This dysfunction also results in the inability of the CNS to consistently programme and achieve normal smooth end organ response.

M.E. is primarily neurological, but because the brain controls all vital bodily functions virtually every bodily system can be significantly affected by M.E. Again, although M.E. is primarily neurological it is also known that the vascular and cardiac dysfunctions seen in M.E. are also the cause of many of the symptoms and much of the disability associated with M.E. – and that the well-documented mitochondrial abnormalities present in M.E. significantly contribute to both of these pathologies. There is also multi-system involvement of cardiac and skeletal muscle, liver, lymphoid and endocrine organs in M.E. Some individuals also have damage to skeletal and heart muscle. Thus M.E. symptoms are manifested by virtually all bodily systems including: cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage. (Chabursky et al. 1992 p. 20) (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 pp. x-xxi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]) (Dowsett & Ramsay n.d., pp. 81-84). M.E. expert Dr Byron Hyde explains that:

A significant number of the initial and long-term peripheral or body symptoms, as well as clinical and technological body abnormalities in the M.E. patient, are caused by variable changes in the peripheral and CNS vascular system. The vascular system is perhaps the largest of the body's organs and both its normal and patho-physiological functions are in direct relationship to CNS and peripheral vascular health or injury, to CNS control mechanisms and to the difficulty of the peripheral vascular system and organs to respond to CNS neuro-endocrine and other chemical and neurological stimuli in a predictable homeostatic fashion. Depending upon the degree and extent of the ongoing CNS and peripheral vascular injuries, these patho-physiological changes in turn may give rise to both transient and in many cases permanent systemic organ changes in the patient (2007, [Online]).

M.E. is an infectious neurological disease and represents a major attack on the CNS – and an associated injury of the immune system – by the chronic effects of a viral infection. There is also transient and/or permanent damage to many other organs and bodily systems in M.E. M.E. affects the body systemically. Even minor levels of physical and cognitive activity, sensory input and orthostatic stress beyond an M.E. patient's individual post-illness limits causes a worsening of the illness, and of symptoms, which can persist for days, weeks, months or even longer. In addition to the risk of relapse, repeated or severe overexertion can also cause permanent damage (e.g. to the heart), disease progression and/or death in M.E.

M.E. is not stable from one hour, day, week or month to the next. It is the combination of the chronicity, the dysfunctions, the instability and the lack of dependability of these functions that creates the high level of disability in M.E. It is also worth noting that of the CNS dysfunctions, cognitive dysfunction is a major disabling characteristic of M.E.

All of this is not simply theory, but is based upon an enormous body of mutually supportive clinical information which has been published in prestigious peer-reviewed journals all over the world and spans over 70 years. Modern technology has now served to confirm and to detail the meticulous clinical and scientific observations made about M.E. before 1988.

Confirmation of this hypothesis is now supported by electrical tests of muscle and of brain function (CT, MRI, SPECT and PET scans clearly indicate that metabolic dysfunction in the brain stem and the spinal nerve radiations which transverse it, are associated with viral (inflammatory) damage and are the major cause of the cardinal symptoms of M.E.) and by biochemical and hormonal assays, and microbiology (for example PCR – a microbiological technique capable of amplifying and identifying minute fragments of viral genes, hidden away in internal organs (such as brain, heart or muscle). Many aspects of the pathophysiology of the disease have been medically explained in volumes of research articles. These are well-documented, scientifically sound explanations for why patients are bedridden, profoundly intellectually impaired, unable to maintain an upright posture and so on (Chabursky et al. 1992 p. 20) (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Hyde 2009) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 pp. x-xxi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92).

- *What is Homeostasis?* Homeostasis is the property of a living organism, to regulate its internal environment to maintain a stable, constant condition, by means of multiple dynamic equilibrium adjustments, controlled by interrelated regulation mechanisms. Homeostasis is one of the fundamental characteristics of living things. It is the maintenance of the internal environment within tolerable limits.

What are some of the symptoms of M.E.?

More than 64 distinct symptoms have been authentically documented in M.E. At first glance it may seem that every symptom possible is mentioned, but although people with M.E. have a lot of different minor symptoms because of the way the central nervous system (which controls virtually every bodily system) is affected, the major symptoms of M.E. really are quite distinct and almost identical from one patient to the next. (Hooper & Montague 2001a, [Online]) (Hyde 2006, [Online])

Individual symptoms of M.E. include:

Sore throat, chills, sweats, low body temperature, low grade fever, lymphadenopathy, muscle weakness (or paralysis), muscle pain, muscle twitches or spasms, gelling of the joints, hypoglycaemia, hair loss, nausea, vomiting, vertigo, chest pain, cardiac arrhythmia, resting tachycardia, orthostatic tachycardia, orthostatic fainting or faintness, circulatory problems, ophthalmoplegia, eye pain, photophobia, blurred vision, wavy visual field, and other visual and neurological disturbances, hyperacusis, tinnitus, alcohol intolerance, gastrointestinal and digestive disturbances, allergies and sensitivities to many previously well-tolerated foods, drug sensitivities, stroke-like episodes, nystagmus, difficulty swallowing, weight changes, paresthesias, polyneuropathy, proprioception difficulties, myoclonus, temporal lobe and other types of seizures, an inability to maintain consciousness for more than short periods at a time, confusion, disorientation, spatial disorientation, disequilibrium, breathing difficulties, emotional lability, sleep disorders; sleep paralysis, fragmented sleep, difficulty initiating sleep, lack of deep-stage sleep and/or a disrupted circadian rhythm.

Neurocognitive dysfunction may include cognitive, motor and perceptual disturbances. Cognitive dysfunction may be pronounced and may include: difficulty or an inability to speak (or understand speech), difficulty or an inability to read or write or to do basic mathematics, difficulty with simultaneous processing, poor concentration, difficulty with sequencing, and problems with memory including difficulty making new memories, difficulty recalling formed memories and difficulties with visual and verbal recall (e.g. facial agnosia). There is often a marked loss in verbal and performance intelligence quotient (IQ) in M.E. (Bassett 2010, [Online]).

- For a more complete symptom list see: [The Ultra-comprehensive M.E. Symptom List](#)
- See also: [What it feels like to have M.E.: A personal M.E. symptom list and description of M.E.](#)
- See the [Research and Articles](#) section for many hundreds of different articles and medical studies into M.E.

What other features define or characterise M.E.?

What characterises M.E. every bit as much as the individual neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms is the way in which people with M.E. respond to physical and cognitive activity, sensory input and orthostatic stress;. -n other words, the pattern of symptom exacerbations, relapses and disease progression.

The way the bodies of people with M.E. react to these activities/stimuli post-illness is unique in a number of ways. Along with a specific type of damage to the CNS, this characteristic is one of the defining features of the illness and must be present for a correct diagnosis of M.E. to be made. The main characteristics of the pattern of symptom exacerbations, relapses and disease progression in M.E. include the following:

- A. People with M.E. are unable to maintain their pre-illness activity levels. This is an acute, sudden change. M.E. patients can only achieve 50% or less of their pre-illness activity levels.
- B. People with M.E. are limited in how physically active they can be but are also limited in similar ways with cognitive exertion, sensory input and orthostatic stress.
- C. When a person with M.E. is active beyond their individual physical, cognitive, sensory or orthostatic limits, there is a worsening of various neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms.
- D. The level of physical activity, cognitive exertion, sensory input or orthostatic stress that is needed to cause a significant or severe worsening of symptoms varies from patient to patient, but is often trivial compared to a patient's pre-illness tolerances and abilities.
- E. The severity of M.E. waxes and wanes throughout the hour/day/week and month.
- F. The worsening of the illness caused by overexertion often does not peak until 24 - 72 hours or more later.
- G. The effects of overexertion can accumulate over longer periods of time and lead to disease progression or death.
- H. The activity limits of M.E. are not short term: an increase in activity levels beyond a patient's individual limits, even if gradual, causes relapse, disease progression or death.

- I. The symptoms of M.E. do not resolve with rest. The symptoms and disability of M.E. are not caused only by overexertion: there is also a base level of illness which can be quite severe even at rest.
- J. Repeated overexertion can harm the patient's chances for future improvement in M.E. Patients who are able to avoid overexertion have repeatedly been shown to have the most positive long-term prognosis.
- K. Not every M.E. sufferer has 'safe' activity limits within which they will not exacerbate their illness: this is not the case for very severely affected patients.

A. People with M.E. are unable to maintain their pre-illness activity levels. This is an acute (sudden) change. M.E. patients can only achieve 50%, or less, of their pre-illness activity levels.

B. People with M.E. are limited in how physically active they can be but they are also limited in similar way with cognitive exertion, sensory input and orthostatic stress.

The bodies of people with M.E. respond inappropriately to anything that forces the body to have to react in some way or work harder in some way, in order to maintain internal homeostasis, including (but not limited to): physical activity, cognitive exertion (including emotional stress), sensory input and orthostatic stress (maintaining an upright posture).

C. When a person with M.E. is active beyond their individual (physical, cognitive, sensory or orthostatic) limits this causes a worsening of various neurological, cognitive, cardiac, cardiovascular, immunological, endocrinological, respiratory, hormonal, muscular, gastrointestinal and other symptoms.

When a person with M.E. is active beyond their individual post-illness limits, there is a worsening of all sorts of different symptoms and of the severity of the illness generally. Overexertion causes an exacerbation of symptoms which can be mild, moderate, severe, or even life threatening (eg. seizures and cardiac events).

The types of symptoms produced in response to certain levels of physical activity, cognitive activity, sensory stimuli or orthostatic stress may or may not vary depending on the type (and severity) of the activity or stimuli involved. But very often the types of symptoms worsened or produced by overexertion are fairly similar regardless of which exertion or input was involved. Overexertion can sometimes cause just one or two symptoms to worsen (eg. cardiac problems) but often a large cluster of symptoms are worsened. The cluster of symptoms made worse by excessive exertion or stimulus is often very similar from patient to patient, as generally it is a worsening of the most common symptoms of the illness. Patients commonly experience a combination of the following symptoms:

Profound cognitive dysfunctions (and various other neurological disturbances), muscle weakness (or paralysis), burning eye pain or burning skin, subnormal temperature or low-grade fever, sore throat or painful lymph nodes (and/or other signs of inappropriate immune system activation), faintness, weakness or vertigo, loss of co-ordination, dyspnoea, an explosion of sensory phenomena (low level seizure activity), cardiac and/or blood pressure disturbances, facial pallor and/or a slack facial expression, widespread severe pain, nausea or feeling as if 'poisoned,' feeling cold and shivering one minute and hot and sweating the next, anxiety or even terror (as an organic part of the attack itself rather than as a reaction to it) and hypoglycaemia. Often the patient will feel an urgent need to retreat from all homeostatic pressures. The types of symptoms triggered vary widely from patient to patient, but some combination of these is common. There may also be an accompanying exacerbation of other symptoms. These symptoms often combine to create an indescribable and overwhelming experience of terrible illness that is unique to M.E, and can be profoundly incapacitating. At its most severe, the patient feels as if they are about to die.

D. The level of physical activity, cognitive exertion, sensory input or orthostatic stress needed to cause a significant or severe worsening of symptoms varies from patient to patient, but is often trivial compared to a patient's pre-illness tolerances and abilities.

When there is talk of 'overexertion' leading to an exacerbation of symptoms in M.E. what is being referred to is not hard exercise, it is not anything resembling what healthy people would recognise as 'overexertion.' This term just refers to any activity which goes beyond a person's individual post-M.E. limits. Relapses can be very severe and prolonged (or even permanent) even if a person with M.E. has only gone past their individual limits in a seemingly minor way.

E. The severity of M.E. waxes and wanes throughout the hour/day/week and month.

One can probably observe people with some illnesses carefully for an hour or so and collect a lot of good information about what they can and can't do, how severe their illness is, and what their usual symptoms are from day to day, and so on. However M.E. is not one of those illnesses. M.E. is *not* a stable illness.

Observing the average M.E. sufferer for an hour – or even a week or more – will not give an accurate indication of their usual activity level because the severity of M.E. can wax and wane throughout the month, week, day and even hour. Also, people with M.E. can sometimes operate significantly above their actual illness level for

short periods of time thanks to surges of adrenaline – albeit at the cost of severe and prolonged worsening of the illness afterward. Relapses and worsening of symptoms are also very often also significantly delayed (there may be both an acute AND a delayed reaction).

Just observing someone with M.E. do a certain task should not be taken to mean (a) that they can necessarily repeat the task anytime soon, (b) that they would have been able to do it at any other time of day, (c) that they can do the same task every hour, day or even every week, or month, or (d) that they won't be made very ill afterwards for a considerable period because they had to really push themselves (and make themselves ill) to do the task.

Often a considerable rest period is needed before and after a task, which may be hours, days, weeks or months long. For example, someone may need 2 weeks rest before an outing, for example, and may then spend 3 weeks extremely ill afterwards recovering from it. Just observing them in the 2 hours they were 'out and about and mobile' is of course not at all representative of their usual ability levels.)

Most importantly, because the worsening of the illness caused by overexertion may not even begin until 48 or more hours afterwards (when most observers are long gone), it's impossible to tell by seeing an M.E. patient engaged in an activity, whether that activity is so far beyond the patient's limits that it will end up causing a severe or even permanent worsening of the illness (or 'relapse'). To be blunt, the activity may even end up killing the patient. This isn't common (the death rate is estimated at 3%), but deaths can and do occur. Thus, observers who see an M.E. patient engaged in an activity have no idea what the consequences of this activity may be.

- What is an adrenaline surge? Adrenaline is often referred to as the 'fight or flight' hormone as it kicks into action in situations of potential danger. However, adrenaline also kicks in when the body is in physiological difficulty, which is very often what is happening to severe M.E. sufferers. Adrenaline surges make the heart pump faster and raise the blood pressure, forcing blood around the body with greater force to supply the muscles with more oxygen, so that they can make a greater effort. Surges of adrenaline increase the metabolism. They also relax and dilate the airways so that more oxygen than usual can be taken in. Adrenaline surges can also decrease the amount of pain felt. As a result of all of these factors, adrenaline surges – while they last – have the ability to increase physical speed, strength and other physical abilities. Unfortunately, when these bursts of adrenaline wear off – as they must – people with M.E. are left far more ill as a result for many days, weeks, months or even years. People with M.E. are harmed by adrenaline surges, both by the physiological stress to the body of the changes caused by adrenaline, and by the extra activity which adrenaline enables, which may be far beyond the body's normal limits so that such activity causes damage. For every short term 'gain' there is a far greater loss overall. For more information on adrenaline surges in M.E., and the different order in which certain bodily systems may be affected by M.E. (and by overexertion), see the Dr Cheney section in The effects of CBT and GET on patients with M.E. or Treating M.E. - Avoiding Overexertion.
- There is also a waxing and waning of the *physical signs* of M.E. throughout the day, as Dr Hyde and Dr Jain explain, "A patient examined in the morning might have nystagmus, which would disappear at midday, recur later, disappear later and recur the next day."

F. The worsening of the illness caused by overexertion can be acute, but often does not reach its peak until 24 - 72 hours (or more) later.

The onset of the worsening of symptoms caused by overexertion is sometimes be acute but often will not peak until 48 hours or more afterward (this is particularly true with regard to physical, cognitive and orthostatic exertions). Symptoms will then persist for hours, weeks or many months, or longer. For many M.E. sufferers, the effects from significant overexertion will very often peak on day three.

Sometimes there is a significant worsening of symptoms evident at the time of overexertion. At other times, there may only be a minor worsening of symptoms at the time of overexertion, but the delayed effects will be severe. Sometimes the acute effects and the delayed effects will both be severe. It varies depending on the type and severity of the overexertion involved, and so on.

G. If people with M.E. push past their individual limits too deeply or too often, the effects of overexertion can also accumulate over longer periods of time and lead to disease progression, or death.

In addition to the effects of overexertion commonly being delayed by 48 hours or so, the worsening of symptoms caused by overexertion can also sometimes be delayed (and accumulate) over weeks or even many months at a time until they are realised in a 'crash.' This is a period of intense worsening of the overall condition followed by a gradual return to the patient's base level of illness over weeks, months or even years.

When the body is confronted with activity (or inputs) beyond the patient's individual limits severely and/or repeatedly over time, these effects can also become cumulative in the long term; the patient becomes unable to return to their base level of illness at all. What this means is that long-term or permanent worsening of the

overall severity of the condition is caused. Thus some patients are still dealing with the severe physical effects of inappropriate advice to be more physically or mentally active etc. five, ten, fifteen or more YEARS afterward and for some patients the damage caused is permanent. Overexertion has also resulted in death in some cases of M.E.

Strong evidence exists to show that overexertion can have extremely harmful effects on M.E. patients. Patient accounts of leaving exercise programs much more severely ill than when they began them; wheelchair-bound or bed-bound or needing intensive care or cardiac care units, are common. (Recent research has shown that postural stress and physical and mental overexertion exacerbate cardiac insufficiency in this disease; see the notes below for more information.) In addition to the risk of relapse, permanent damage, and disease progression, there have also been reports of sudden deaths in M.E. patients following exercise. As M.E. expert Dr. Elizabeth Dowsett explains, ‘20% have progressive and frequently undiagnosed degeneration of cardiac muscle which has led to sudden death following exercise. Prompt recognition and advice to avoid over-exertion is mandatory.’

- For more information on the question of “*Can M.E. patients really die just from being forced out of bed, or to leave the house etc.?*” please see the paper: Why patients with severe M.E. are housebound and bedbound
- Cardiac and vascular abnormalities have been documented from the earliest outbreaks of M.E. to the present day. Dr. Paul Cheney explains that when M.E. patients stand up, they are on the edge of organ failure as their cardiac output has dropped to the extremely low level of 3.7 litres per minute, a 50% drop from the normal output of 7 litres per minute. Without exception, says Cheney, every M.E. patient ‘is in heart failure.’

Recent research shows that mitochondrial and other dysfunction leads to diastolic dysfunction and reduced stroke volume/low cardiac output in M.E. – and that certain levels of orthostatic stress and physical and mental activity etc. exacerbate this cardiac insufficiency. Dr Cheney explained recently that because it takes more metabolic energy for the heart to relax and fill with blood than it does for it to squeeze and pump blood, the hearts of people with M.E. don’t fill with the proper amount of blood before they pump which is what causes the reduced cardiac output and many of the symptoms of M.E. (and much of the disability of M.E.). So the tachycardia – fast heart rate – often seen in M.E. in response to orthostatic stress and so on is actually compensating for low stroke volume to help increase cardiac output. The heart doesn’t fill with enough blood before each beat of the heart so it is forced to beat faster to try to make up some of the shortfall, but people with M.E. are still left with reduced cardiac output which leaves them very ill and disabled. If this problem is severe enough it can result in death.

As one M.E. advocate explains: ‘Cardiac output is sometimes too low to meet the demands of movement, and any attempt to exert oneself beyond one’s own capacity for cardiac output - that is when demand exceeds cardiac capacity - would indeed result in death. Studies on dogs have shown that when the demands of the body exceed cardiac output by even 1%, the organism dies. M.E. patients [must] reduce demand and reduce their exertion level to stay within the bounds of their low cardiac output to stay alive.’

H. The activity limits of M.E. are not short term, a gradual (or sudden) increase in activity levels beyond a patient’s individual limits can only cause relapse, disease progression or death in patients with M.E.

Increasing the activity levels of someone with M.E. beyond their individual limits, can only ever be counterproductive. It really doesn’t matter if you do this gradually or all at once. Raising the limits gradually may well delay the onset of the relapse in some patients, but the end result will still be relapse and/or disease progression, or death.

- M.E. is a chronic illness which affects the vast majority of (if not all) sufferers for many years or decades at a time, or for the rest of their lives. A person who has been correctly diagnosed with M.E. will naturally raise their activity levels when/if they have had an improvement in their illness – but it can never work the other way around. See: Smoke and mirrors for more information.

I. The symptoms of M.E. do not resolve with rest. The symptoms and disability of M.E. are not just caused by overexertion, there is also a base level of illness which can be quite severe even at rest.

There is a base level of illness that is always present in M.E., even at rest. This is because the metabolic problems of M.E. are only one part of M.E., they are not the only cause of symptoms or of the worsening of the illness.

But even those symptoms which are caused by the metabolic problems of M.E. (and the loss of homeostasis etc.) do not always resolve with rest. For severely affected patients, just keeping the body going at the lowest possible level can count as ‘overexertion’ – not only can the bodies of these people not cope with extra activity, but they also cannot even cope with keeping the bodily systems and organs going at the lowest possible level – at rest.

Virtually all bodily systems are affected in some way by both the damage to the central nervous system and the metabolic problems of M.E. (including the cardiac insufficiency this causes) etc. so it is no wonder people with

M.E. feel so terribly ill, have such a reduced level of functioning in so many different bodily systems and have so many restrictions and limits on how active they can be. Even with complete rest – and some people with M.E. can do almost nothing else – many M.E. sufferers are still very ill and disabled.

J. Repeated overexertion can harm chances for future improvement in M.E. M.E. patients who are given advice to rest in the early stages of the illness (and who avoid overexertion thereafter) have repeatedly been shown to have the most positive long-term prognosis.

Thus it is vital that M.E. patients are never encouraged to be active beyond their individual limits, as this can only ever be counterproductive. People with M.E. must be allowed to determine for themselves how much rest they need and how active they can be. Giving people with M.E. the support they need to limit their activities in this way is actually the best way to ensure that they each get to be as active as possible in the long term. The importance of getting appropriate rest and avoiding overexertion in M.E. cannot be overstated. Encouraging people with M.E. to engage in even low levels of physical and cognitive activity, sensory input and orthostatic stress beyond their individual limits can have catastrophic long-term consequences.

- For more information about the effects of overexertion on M.E. patients, including statements/research from some of the world's leading M.E. experts about why overexertion is so physically harmful, see: [Smoke and Mirrors](#). (This paper also includes links to many different patient accounts of the effects of overexertion on people with M.E.). If you have M.E. see [Treating M.E. - The Basics](#) and [Treating M.E. - Avoiding Overexertion](#) for more on the importance of avoiding overexertion.

L. Not every M.E. sufferer has 'safe' activity limits within which they will not exacerbate their illness, this is not the case for the very severely affected.

For very severely affected M.E. sufferers there is virtually no 'safe' level of physical or mental activity, orthostatic stress or sensory input; no level which does not produce a worsening of symptoms, and perhaps also contribute to disease progression. Even the most basic actions – speaking a few words, being exposed to moderate light or noise for a few minutes, turning over in bed, having hair or body washed in bed by a carer or chewing and swallowing food – cause severe and extended symptom exacerbations in such patients. It is not uncommon to hear of very severely affected sufferers who are unable to bathe themselves (or even be bathed by a carer) more often than once a week, or even once every few weeks, or even less. Some sufferers cannot chew or swallow food any longer and need to be tube fed. Many patients with severe M.E. are no longer able to toilet themselves, and so on. Either sufferers are just too ill to do these things at all, or they cannot tolerate the very long and severe relapses that come after such activities (Bassett 2009, [Online]).

- For more information on severe M.E. see [The severity of M.E.](#) and [M.E. Fatalities plus Why patients with severe M.E. are housebound and bedbound](#). For the full-length version of this text and for a full list of references for this text see: [The Ultra-comprehensive M.E. Symptom List](#). (The reference list for this text is virtually identical to the references listed after the paragraphs taken from the 'A one-page summary of the facts of M.E.' text, featured below.)

A summarised explanation of why M.E. patients are so severely and uniquely disabled, taken from the 'A one-page summary of the facts of M.E.' text, is as follows:

For a person to stay alive, the heart must pump a certain base-level amount of blood. Every time a person is active, this increases the amount of blood the heart needs to pump. Every movement made or second spent upright, every word spoken, every thought thought, every word read or noise heard requires that more blood must be pumped by the heart.

However, the hearts of M.E. patients only pump barely enough blood for them to stay alive. Their circulating blood volume is reduced by up to 50%*. Thus M.E. patients are severely limited in physical, cognitive and orthostatic (being upright) exertion and sensory input.

This problem of [reduced circulating blood volume](#), leading to cardiac insufficiency, is why every brief period spent walking or sitting, every conversation and every exposure to light or noise can affect M.E. patients so profoundly. Seemingly minor 'activities' can cause significantly increased symptom severity and/or disability (often with a 48-72 hour delay in onset), prolonged relapse lasting months, years or longer, permanent bodily damage (eg. heart damage or organ failure), disease progression or death.

If activity levels exceed cardiac output by even 1%, death occurs. Thus the activity levels of M.E. patients must remain strictly within the limits of their reduced cardiac output just in order for them to stay alive. *M.E. patients who are able to rest appropriately and avoid severe or prolonged overexertion have repeatedly been shown to have the most positive long-term prognosis* (Hyde 2007, [Online]) (Hyde 2003, [Online]) (Cheney 2006, [video recording]) (Hyde & Jain 1992a, pp. 375-383) (Ramsay 1986, [Online]) (Peckerman et al. 2003, [Online]) (Dowsett 2002b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online])

(Dowsett 1999a, [Online]) (Dowsett 1999b, [Online]) (Dowsett & Ramsay et al. 1990, pp. 285-291) (Dowsett & Ramsay n.d., pp. 81-84) (Dowsett n.d., [Online]) (Dowsett n.d.b, [Online]). (Dowsett n.d.d, [Online]) (Ryll 1994, [Online]) (Jones 1998, [Online]) (25% M.E. Group 2005, [Online]) (25% M.E. Group 2004, [Online]) (MESA 2007, [Online]) (Marshall & Williams 2005d, [Online]) (Williams 2004, [Online]).

This one page text is included in most document downloads available on the site.

- *A note about low circulating blood volume findings in M.E.* Dr Byron Hyde explains that circulating blood volume levels of 60 - 70% of expected are very common in M.E. and that readings as low as 50% are common in more severely affected patients. However, it should be noted that as the vast majority of those with very severe M.E. are of course far too ill to have this test and so they are not represented in these statistics. Thus it should not be assumed that this 50% percentage represents the 'worst case scenario' as regards reduced circulating blood volume test results in M.E. The findings from patients with very severe M.E. (if these patients could safely be tested) would likely be even more startling. For more information on this test see: [Testing for M.E.](#)

What causes M.E.?

M.E. expert [Dr Byron Hyde](#) explains that:

[The] prodromal phase is associated with a short onset or triggering illness. This onset illness usually takes the form of either, or any combination, of the following, (a) an upper respiratory illness, (b) a gastrointestinal upset, (c) vertigo and (d) a moderate to severe meningitic type headache. These are only the most common onset illnesses or symptoms of which there are several. The onset illness is associated with either a low grade or subnormal temperature, headaches, sometimes persisting and accentuated by movement with intermittent attacks of vertigo or dizziness. Evidence of a previous immune insult [such as a recent immunisation] is found regularly in both epidemic and sporadic cases. The incubation period of the triggering illness is 4-7 days. The second and third phases of the illness are usually always different in nature from the onset illness and usually become apparent within 1-4 weeks after the onset of the infectious triggering illness (1998 [Online]).

Despite popular opinion, (and the vast amount of 'CFS' government and media propaganda which purports to be relevant to M.E. but is not), there is **no** link between contracting M.E. and being a 'perfectionist' or having a 'type A' or over-achieving personality. M.E. **cannot** be caused by a period of long-term or intense stress, trauma or abuse in childhood, becoming run-down, working too hard or not eating healthily. M.E. is not a form of 'burnout' or nervous exhaustion, or the natural result of a body no longer able to cope with long-term stress.

Research also shows that it is simply not possible that M.E. could be caused by the Epstein-Barr virus, any of the herpes viruses (including HHV6), glandular fever/mononucleosis, Cytomegalovirus (CMV), Ross River virus, Q fever, hepatitis, chicken pox, influenza or any of the bacteria which can result in Lyme disease (or other tick-borne bacterial infections). These theories have been disproven with regard to M.E. M.E. is also not a form of chemical poisoning.

M.E. is undoubtedly caused by a virus, a virus with an incubation period of 4-7 days. There is also ample evidence that M.E. is caused by the same type of virus that causes polio: an enterovirus (Hyde 2006, [Online]) (Hyde 2007, [Online]) (Hooper 2006, [Online]) (Hooper & Marshall 2005a, [Online]) (Hyde 2003a, [Online]) (Dowsett 2001a, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Ryll 1994, [Online]).

- See [The outbreaks \(and infectious nature\) of M.E.](#) section for more information. [A New and Simple Definition of Myalgic Encephalomyelitis](#)
- For information on the outrageous hype surrounding the recent XMRV 'CFS' research, please see the [XMRV, 'CFS,' and M.E.](#) paper by Sarah Shenk as well as the HFME press release:

Are there outbreaks of M.E.?

One of the most fundamental facts about M.E. throughout its history is that it occurs in epidemics. There is a history of over sixty recorded outbreaks of the illness going back to 1934 when an epidemic of what seemed at first to be poliomyelitis was reported in Los Angeles. As with many of the other M.E. outbreaks, the Los Angeles outbreak occurred during a local polio epidemic.

The presenting illness resembled polio, so for some years the illness was considered to be a variant of polio and classified as 'Atypical poliomyelitis' or 'Non-paralytic polio' (TCJRME 2007, [Online]) (Hyde 1998, [Online]) (Hyde 2006, [Online]). Many early outbreaks of M.E. were also individually named for their locations so we also have

outbreaks known as Tapanui flu in New Zealand, Akureyri or Icelandic disease in Iceland, Royal Free Disease in the UK, and so on (TCJRME 2007, [Online]) (Hyde 1998, [Online]).

A review of early M.E. outbreaks found that clinical symptoms were consistent in over sixty recorded epidemics spread all over the world (Hyde 1998, [Online]). Despite the different names being used, these were repeated outbreaks of the same illness. It was also confirmed that the epidemic cases of M.E. and the sporadic cases of M.E. each represented the same illness (Hyde 2006, [Online]) (Dowsett 1999a, [Online]).

M.E. is an acutely acquired neurological illness (with systemic effects) initiated by a virus infection. This point of view is supported by **history** (M.E. epidemics have followed polio epidemics and serological studies have shown that communities affected by an outbreak of M.E. were effectively blocked (or immune) from the effects of a subsequent polio outbreak) **incidence** (correlation with a flu-like prodromic illness), **symptoms** (painful or tender lymph nodes, low-grade fever, sore throat), and **similarities** with other viral ailments (including post-polio syndrome). A large body and a large body of medical **research** also supports a viral causation for the illness (Gellman & Verillo 1997, p 19) (Dowsett 1999a, [Online]).

Transmission of M.E. to monkeys has been successfully demonstrated and has produced central nervous system and parasympathetic nervous system injury in at least two separate sets of experiments; in 1934 where 'cross sections of the spinal cord demonstrated numerous minute haemorrhages in the grey matter' and in 1949-51 in the Adelaide, Australia epidemic of M.E. where a radiculitis of the sciatic nerve was demonstrated with small punctate lesions of the myelin sheath (Hyde & Jain 1992, p. 40).

M.E. is an infectious neurological disease and represents a major attack on the CNS by the chronic effects of a viral infection. The world's leading M.E. experts, namely Ramsay, Richardson, Dowsett and Hyde, (and others) have all indicated that M.E. is caused by an enterovirus. The evidence which exists to support the concept of M.E. as an enteroviral disease is compelling (Hyde 2007, [Online]) (Hyde 2006, [Online]). An enterovirus explains the age variation, sex variation, obvious resistance of some family members to the infection and the effect of physical activity -particularly in the early stages of the illness- in creating more long-term/severe M.E. illness in the host (Hyde & Jain 1992a, p. 40).

Enterovirus infections are able to cause:

- a. a chronic host infection
- b. major or no cardiac disease depending on the virulence of the subtype
- c. cardiac injury dependent upon the sex of the patient and of the level of physical activity of the patient during the acute or infectious stage
- d. cardiac disease depending upon the immunological variability of the host (Hyde & Jain 1992a, p. 40).

An enterovirus would also explain the; age variation, sex variation, obvious resistance of some family members to the infection and the effect of physical activity (particularly in the early stages of the illness) in creating more long-term/severe M.E. illness in the host (Hyde & Jain 1992a, p. 40). There is also the evidence that:

- M.E. epidemics very often followed polio epidemics.
- M.E. resembles polio at onset.
- serological studies have shown that communities affected by an outbreak of M.E. were effectively blocked (or immune) from the effects of a subsequent polio outbreak.
- evidence of enteroviral infection has been found in the brain tissue of M.E. patients at autopsy. (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]).

The US Centres for Disease Control (CDC) placed 'CFS' on its "Priority One, New and Emerging" list of infectious diseases some years ago; a list that also includes Lyme disease, hepatitis C, and malaria' (Gellman & Verillo 1997, p. 19). Despite this, no real research into transmissibility (or more importantly on reducing infection rates) has been done by any government on patients with M.E. (or even 'CFS') despite ample evidence that this is an infectious disease. There have been many well-documented clusters or outbreaks of the illness, reports of as many as 4.5% of M.E. sufferers contracting the illness immediately after blood transfusions (or after needle-stick injuries involving the blood of M.E. patients) and evidence of the disease spreading through casual contact amongst family members (Johnson, 1996) (Carruthers et al. 2003, p.79). As Dr Elizabeth Dowsett explains: 'The problem we face is that, in spite of overwhelming epidemiological and technical evidence of an infectious case, the truth is being suppressed by the government and the 'official' M.E. charities as 'too scary' for the general public' (n.d.a, [Online]).

This pretence of ignorance on behalf of government worldwide has had enormous consequences: for example, only in the UK are people with M.E. specifically banned from donating blood. Consequently, the number of people infected with M.E. continues to rise unabated and largely unnoticed by the public (Johnson, 1996). M.E. expert [Dr Elizabeth Dowsett](#) writes about M.E. that: 'This illness is distinguished from a variety of other post-viral states by a unique clinical and epidemiological pattern characteristic of enteroviral infection. Prompt recognition and advice to avoid over-exertion is mandatory' (Dowsett et al. 1990, pp. 285-291).

- See: [The outbreaks \(and infectious nature\) of M.E.](#) section for more information.
- For more information about the effects of overexertion on M.E. patients, including statements/research from some of the world's leading M.E. experts about why overexertion is so physically harmful, see: [Smoke and Mirrors](#). (This paper also includes links to many different patient accounts of the effects of overexertion on people with M.E.). If you have M.E. see [Treating M.E. - The Basics](#) and [Treating M.E. - Avoiding Overexertion](#) for more on the importance of avoiding overexertion.

[Is the onset of M.E. gradual or acute?](#)

Again, only being able to achieve 50% or less of your pre-illness activity level immediately upon becoming ill is virtually universal in M.E. (Although a small percentage of sufferers may possibly be somewhat less severely affected at onset.) It must be emphasised that this is not a gradual change in ability levels (etc.) which occurs slowly over weeks, months or years; it is an acute (sudden) change.

The onset of M.E. is always acute and is frequently very dramatic; M.E. patients can very often tell you not just the day that they became ill, but the exact *hour* they became ill (Chabursky et al. 1992, p.22) (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hyde 2003, [Online]) (Hyde et al. 1992, pp. 25-37) (Hyde & Jain 1992, pp. 38-65).

- M.E. is always an acute onset illness, however it should be noted that: (a) some sufferers will be unsure of their onset type (they may not recall it, or may not recall it accurately, for various reasons) and (b) in some cases, acute onset M.E. is preceded by a series of unrelated minor infectious episodes (in a previously well patient) which may be misinterpreted as being a gradual onset of the M.E. (These minor infectious episodes may be due to the immune system being under temporary or chronic stress from events such as; recent immunisation, repetitive contact with a large number of infectious persons, or the effect of travel; as in exposure to a new subset of virulent infections. This pre-existing temporary or chronic immune system weakness is not seen in all patients and is *not* what causes M.E., although a compromised immune system will of course make the body more vulnerable to all types of infections, including M.E.)

[Is M.E. difficult to diagnose? What tests can be used to diagnose M.E.?](#)

M.E. is a distinct, recognisable disease entity that is not difficult to diagnose and can in fact be diagnosed relatively early in the course of the disease (within just a few weeks), providing that the physician has some experience with the illness. There is just no other illness that has all the major features of M.E.

As with a wide variety of illnesses; (Lupus, multiple sclerosis, and ovarian cancer for example) there is as yet no *single* test which can diagnose M.E. in all patients. Therefore, along with these other illnesses, M.E. must instead be diagnosed by a combination of: taking a detailed medical history (to rule out other possible causes of symptoms), noting the type and severity of symptomatology and other characteristics of the illness, the type of onset of the symptoms (a acute or sudden onset of symptoms is always seen in M.E. If present this characteristic rules out a wide variety of other illnesses associated with gradual onset) and looking for some of the physical signs of illness. A *series* of tests may also then be necessary both to rule out other illnesses, and/or to help confirm a suspected M.E. diagnosis.

Although there is (as yet) no single test which can be used to diagnose M.E. there are a series of tests which can confirm a suspected M.E. diagnosis. Virtually every M.E. patient will also have various abnormalities visible on physical exam. If all tests are normal, if specific abnormalities are not seen on certain of these tests (e.g. brain scans), then a diagnosis of M.E. cannot be correct (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hooper et al. 2001, [Online]) (Chabursky et al. 1992, p.22).

As M.E. expert [Dr Byron Hyde](#) explains:

The one essential characteristic of M.E. is acquired CNS dysfunction. A patient with M.E. is a patient whose primary disease is CNS change, and this is measurable. We have excellent tools for measuring these physiological and neuropsychological changes: SPECT, xenon SPECT, PET, and neuropsychological testing (2003, [Online]).

Tests which together can be used to confirm an M.E. diagnosis include:

- SPECT and xenon SPECT scans of the brain
- MRI and PET scans of the brain
- Neurological examination
- Neuropsychological testing (including QEEG scans)
- the Romberg or tandem Romberg test
- Various tests of the immune system (including tests of natural killer cells number and function)
- Insulin levels and glucose tolerance tests
- Sedimentation rate testing (M.E. is one of less than half a dozen diseases which can cause sedimentation rates as low as zero)
- Circulating blood volume tests (which may show a reduced circulating blood volume of up to 50%)
- 24 hour Holter monitor testing (a type of heart monitor)
- Tilt table examination and blood pressure tests
- exercise testing and chemical stress tests
- Physical exam

Physical signs of illness commonly observed in M.E. patients include:

- Nystagmus; nystagmus is jelly-like and variable (15% of M.E. patients will have nystagmus)
- Sluggish visual accommodation
- Unequal pupils and contrary pupil reaction to light
- A labile blood pressure (sometimes as low as 84/48 in an adult at rest)
- Shortness of breath (particularly on exertion)
- Sometimes marked falling pulse pressure in arterial pressures taken first when prone, then sitting, then standing
- Rapid heart rate on minor activity such as standing
- Subnormal temperature
- Patients show significant reduction in all lung function parameters tested
- Liver involvement (an enlarged liver or spleen)
- Abnormal tandem or augmented tandem stance
- Abnormal gait
- Hand tremor
- Incoordination
- Cogwheel movement of the leg on testing
- Muscular twitching or fasciculation
- Hyper-reflexia without clonus
- Facial vasculoid rash
- Vascular demarcation which can cross dermatomes with evidence of Raynaud's syndrome and / or vasculitis and spontaneous periarticular bleeds in the digits
- Mouth ulcers
- Hair loss
- Destruction of fingerprints is sometimes seen (atrophy of fingerprints is due to perilymphocytic vasculitis and vacuolisation of fibroblasts)
- Ghastly pallor of face with frequent lupus-like submaxillary mask
- Parkinsonian rigidity of facial expression
- Scanning, disjointed speech, or speech reversals
- Nasal passage obstruction and inflamed areas around tonsillar pillars
- Sicca syndrome of conjunctiva and mucous membranes
- Frequent equivocal Babinski/plantar reflex on one side
- Unusual sensitivity of cervical vertebrae area
- Nodular thyroid

The abnormalities visible on physical exam in M.E. patients are not usual in healthy patients but they are also found in people with other illnesses (so they are not specific to M.E.). In cases of severe M.E. there are always definite physical signs indicative of physical illness but virtually all patients will have some abnormality on physical exam. Not all patients will have all signs and along with a fluctuating course of the illness from hour to hour and from day to day being one of the key characteristics of M.E., signs of the illness may also change or

fluctuate during the course of a day (Hyde 2007, [Online]) (Hyde 2006, [Online]) (Hooper 2006, [Online]) (Hooper & Marshall 2005a, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Hyde et al. 1992, pp. 25-37) (Dowsett et al. 1990, pp. 285-291) (Ramsay 1986, [Online]) (Dowsett n.d., [Online]) (Dowsett & Ramsay n.d., pp. 81-84) (Richardson n.d., pp. 85-92).

While various ‘fatiguing conditions’ with a variety of different aetiology’s may be made up of vague and mild ‘everyday’ type symptoms, have no physical signs and no tests which can aid diagnosis, this is not the case with M.E. M.E. is a distinct neurological illness with a distinct list of symptoms, physical signs and diagnostic (and other) tests – it bears no relationship to such unrelated ‘fatiguing conditions.’ As authors Verillo and Gellman explain: ‘Contrary to popular belief, M.E. is a distinct, recognisable entity that can be diagnosed relatively early in the course of the disease, providing the physician has some experience with the illness’ (1997 p. 21). New TESTABLE definitions such as The Nightingale Definition of M.E. now also make diagnosis easier than ever before; even for those with no experience with the illness. Again, if all tests are normal then a diagnosis of M.E. **cannot** be correct.

Tests will only all be normal in M.E. patients – as with all illnesses – if completely the wrong tests are done, or if those tested do not in fact have M.E. in the first place.

- See: Testing for M.E. for more information on the various tests which can aid M.E. diagnosis.
- Objective scientific tests *are* available which can aid in the diagnosis of M.E. (and easily prove the severe abnormalities across many different bodily systems seen in M.E.), but unfortunately many patients are not given access to these tests. Many (probably most) patients with M.E. are only ever given access to the most basic of tests and these tests are normal in 90% of M.E. patients (as they are with many other serious illnesses). Patients are often given only basic tests and when these are pronounced normal, are told that there is ‘nothing wrong with them.’ This is an abuse of science. In short, a political decision has been made that allowing M.E. patients appropriate testing would be ‘inconvenient.’ All illnesses can only be tested for if the CORRECT tests are used. For more information on the lack of access to appropriate testing for M.E. patients see: The Montague/Hooper Paper (CONCERNS ABOUT THE FORTHCOMING UK CHIEF MEDICAL OFFICER’S REPORT NOTABLY THE INTENTION TO ADVISE CLINICIANS THAT ONLY LIMITED INVESTIGATIONS ARE NECESSARY IN ME)

How quickly can M.E. be diagnosed?

M.E. can commonly be diagnosed within just a few weeks, if the doctor has some experience with M.E. (Chabursky et al. 1992, p.22).

The definitions of ‘CFS’ state that 6 months must pass until a (mis)diagnosis of ‘CFS’ can be given, but this simply DOES NOT APPLY to the diagnosis of M.E. (None of the definitions of ‘CFS’ are M.E. definitions.) It is very important that M.E. is diagnosed promptly, and that patients are advised to rest adequately as soon as possible to prevent further bodily damage (eg. to the heart).

- See: Testing for M.E. for more information on the various tests which can aid M.E. diagnosis.
- For more information on the viral infection evident at onset in people with M.E., and the outbreaks of M.E. etc. see: The outbreaks (and infectious nature) of M.E.

How common is M.E.? Who gets M.E. and how?

Although the illness we now know as M.E. has existed for centuries, for much of that time it was a relatively uncommon disease. Following the mass polio vaccination programs of the 1960s, cases of polio were greatly reduced and outbreaks of M.E. seemed to be similarly affected. It wasn’t until the late 1970s that M.E. began its dramatic increase in incidence worldwide. Over 20 years later, M.E. is a worldwide epidemic of devastating proportions. Many people have died from M.E. and there are now many hundreds of thousands of people severely disabled by this epidemic (TCJRME 2007, [Online]) (Hyde 1992, p. xi).

The main period of infectivity of M.E. peaks at the time just before symptoms appear through to the initial acute phase of the illness (which lasts for several months or in some cases years). M.E. appears to be highly infective but also highly selective. The major mode of infectivity is by an airborne or respiratory route. Modes of transmission are thought to include: casual contact (respiratory), salivary transmission (e.g. kissing), sexual transmission and transmission through blood products (Hyde et al. 1992, pp. 25 - 37). (A recent study of 752 patients found that 4.5% of them – almost one in twenty – had had a blood transfusion days or a week before experiencing acute onset of M.E.) (Carruthers et al. 2003, [Online]) (Hyde et al. 1992, pp. 25 - 37).

There is also evidence that asymptomatic carrier of the illness may be able to pass the illness on to others for a brief period following their exposure to the illness. During the recovery and/or chronic stages of the illness however M.E. does not present a significant infective risk. There is no evidence in the literature that the infection can be waterborne or that potables (drinkable liquids) were the cause of the illness – although bodies of water, or sewage, may possibly sustain the causative infectious agent. (Hyde et al. 1992, pp. 25 - 37)

M.E. has a similar strike rate to multiple sclerosis (or possibly somewhat higher), and is estimated to affect roughly 0.2% of the population. Children and teenagers are also susceptible to the illness and children as young as five have been diagnosed with M.E. (M.E. can occur in children younger than five, but this is thought to be rare.) One hundred thousand kids are estimated to have M.E. in the US alone and a recent study in the UK found that M.E. was by far the most common reason for a child's long term absence from school. (Munson 2000, p. 198) (Dowsett 1997, [Online]) (Hyde 1992, pp. x - xxi)

There appears to be somewhat of an occupational bias towards teachers (students) and health care workers in the incidence of M.E. cases (and outbreaks). These are jobs which require higher rates of immunisation than others. This relationship with inoculation is often seen in infectious illnesses (Hyde et al. 1992, pp. 25 - 37). All ages are affected but most commonly sufferers are under 45 at onset. Women are affected around three times as often as men, a ratio common in autoimmune disorders (although in children the sexes seem to be afflicted equally

M.E. affects all ethnic and socio-economic groups and has been diagnosed all over the world. There are more than a million M.E. sufferers worldwide (Hooper et al. 2001 [Online]) (Hyde 1992, pp. x - xxi).

- See: [The outbreaks \(and infectious nature\) of M.E.](#) and [The Clinical and Scientific Basis of Myalgic Encephalomyelitis](#).
- The CDC has recently released vastly inflated estimates for figures affected by 'CFS' but it should be noted that the number of people suffering with sustained fatigue has no more relevance to patients with M.E. than to those with M.S. or AIDS or any other distinct illness. see: [More medical 'firsts' from the CDC?](#)

Are there different stages of M.E.?

Different stages of M.E. have been identified by a number of M.E. experts. [Dr Byron Hyde](#) explains that, 'M.E. is an acute onset biphasic epidemic or endemic (sporadic) infectious disease process, where there is always a measurable and persistent diffuse vascular injury of the CNS in both the acute and chronic phases' (2007, [Online]).

- For more information on the stages of M.E. see: [The Nightingale Definition of M.E.](#) by Dr Byron Hyde and [The Late Effects Of M.E.](#) by Dr Elizabeth Dowsett

Are there any treatments for M.E.?

There are no easy or quick cures for M.E., nor are any on the horizon – despite a lot of hype about various fairly unpromising 'CFS' research endeavours. intelligent nutritional, pharmaceutical and other interventions can make a significant difference to a patient's life, however.

Appropriate biomedical diagnostic testing should be done as a matter of course (and repeated regularly) to ensure that the aspects of the illness which are able to be treated *can* be diagnosed, monitored and then treated as appropriate. Testing is also important so that dangerous deficiencies and dysfunctions, which may place the patient at significant risk, are not overlooked (Hooper et al. 2001 [Online]). [Treating M.E. - The Basics](#)

For specific information on M.E. treatment, the following HFME papers are recommended reading:

- [Treating M.E. - The basics](#) and [Treating and living with M.E.: Overview](#)
- [Finding a good doctor when you have M.E.](#)
- [Symptom-based management vs. deep healing in M.E.](#)
- [A quick start guide to treating and improving M.E. with aggressive rest therapy, diet, toxic chemical avoidance, medications, supplements and vitamins](#)
- [Why research and try treatments when some groups claim an M.E. cure is coming soon?](#)
- [What if vitamin/mineral/protocol 'x' didn't work for me?](#)
- [Deep healing in M.E.: An order of attack!](#)

- Treating M.E. in the early stages

What is known about M.E. so far?

There is an abundance of research which shows that M.E. is an organic illness which can have profound effects on many bodily systems. These are well-documented, scientifically sound explanations for why patients are bedridden, profoundly intellectually impaired, unable to maintain an upright posture and so on. More than a **thousand** good articles now support the basic premises of M.E. Autopsies have also confirmed such reports of bodily damage and infection (Hooper & Williams 2005a, [Online]).

Many different organic abnormalities have been found in M.E. patients (in peer reviewed research). Patient advocates Margaret Williams and Eileen Marshall explain that:

- there is evidence of disrupted biology at cell membrane level
- there is evidence of abnormal brain metabolism
- there is evidence of widespread cerebral hypoperfusion
- there is evidence of CNS and immune dysfunction
- there is evidence of CNS inflammation and demyelination
- there is evidence of hypomyelination
- there is evidence that M.E. is a complex, serious multi-system autoimmune disorder
- there is evidence of significant neutrophil apoptosis
- there is evidence that the immune system is chronically activated (e.g. the CD4:CD8 ratio may be grossly elevated)
- there is evidence that natural killer (NK) cell activity is impaired (i.e. diminished)
- there is evidence that the vascular biology is abnormal, with disrupted endothelial function
- there is novel evidence of significantly elevated levels of isoprostanes
- there is evidence of cardiac insufficiency and that patients are in a form of cardiac failure (which is exacerbated by even trivial levels of physical activity, cognitive activity and orthostatic stress)
- there is evidence of autonomic dysfunction (especially thermoregulation; frequency of micturition with nocturia; labile blood pressure; pooling of blood in the lower limbs; reduced blood volume (with orthostatic tachycardia and orthostatic hypotension. Findings of a circulating blood volume of only 75% of expected are common, and in some patients the level is only 50% of expected.)
- there is evidence of respiratory dysfunction, with reduced lung function in all parameters tested
- there is evidence of neuroendocrine dysfunction (notably HPA axis dysfunction)
- there is evidence of recovery rates for oxygen saturation that are 60% lower than those in normal controls
- there is evidence of delayed recovery of muscles after exercise (Affecting all muscles including the heart.)
- there is evidence of a sensitive marker of muscle inflammation
- there is evidence that the size of the adrenal glands is reduced by 50%, with reduced cortisol levels
- there is evidence of at least 35 abnormal genes, (these are acquired genetic changes, not hereditary), specifically those that are important in metabolism; there are more abnormal genes in M.E. than there are in cancer
- there is evidence of serious cognitive impairment(Worse than occurs in AIDS dementia.)
- there is evidence of adverse reactions to medicinal drugs, especially those acting on the CNS.
- there is evidence that symptoms fluctuate markedly from day to day and even from hour to hour (2006, [Online])

Note that this is only a sample of some of the research available, not an exhaustive list. Dr Sheila Bastien PhD. has over 20 years experience in neuropsychological testing and more than 6 years experience in applying these tests to M.E. patients. She explains that:

Deterioration of IQ levels, as well as cognitive and motor dysfunction in these patients, suggest a pathological process in the brain. The pattern of focal and lateral impairments is consistent with patients who have this particular neurologic dysfunction. The impairment pattern is consistent across the study group [of M.E. patients] although impairment levels vary. This pattern is not seen in other diseases or injuries, such as Alzheimers, stroke, head injuries, multiple sclerosis, systemic lupus erythematosus, personality disorders, depression, psychosis, malingering, anxiety or panic disorders, somatisation or situational stress disorders. The pattern of impairment is one of focal and lateral deficits, consistent with a multi-focal organic brain syndrome. Tests suggest that the most impaired focal areas are the left temporal, right parietal, and left temporal lobes; although there are lesser bilateral impairments in the opposite lobes as well. (1992, pp. 453 - 454)

From Professor Malcolm Hooper:

In M.E. there is evidence of inflammation of the central nervous system (CNS). In some cases of ME, as in multiple sclerosis, there is evidence of oligoclonal bands in the cerebrospinal fluid. It is accepted by the most experienced M.E. clinicians that some degree of encephalitis has occurred both in patients with M.E. and in those with post-polio syndrome: the areas chiefly affected include the upper spinal motor and sensory nerve roots and the spinal nerve networks traversing the adjacent brain stem (which is always damaged).

M.E. is an autoimmune disorder, with similarities to systemic lupus erythematosus. Evidence of antilamin antibodies has been found in the blood of M.E. patients: antibodies against this protein are proof of autoimmunity and of damage to brain cells. The occurrence of autoantibodies to an intra-cellular protein like lamin B1 provides laboratory evidence for an autoimmune component in M.E.

A particularly important piece of research in these patients has demonstrated sensitivity of the vascular endothelium to acetylcholine (a major neurotransmitter and vascular dilator) and this finding may have implications for many other cholinergic pathways (which are extensive throughout the body). In M.E. there is evidence of disruption in ion channels in the cell membranes; changes in ion channel function from time to time offer a rational basis to explain the fluctuating symptoms, and such ion channel changes are known to be induced by physical activities, stress and fasting. If sodium channels are blocked in the open mode, this causes entry of sodium into neural tissues and muscles. This ingress of sodium is followed by water, which in turn leads to swelling of the neural tissues, a phenomenon observed both electron microscopically and by laser scanning microscopy. There is a continued loss of post-exertional muscle power (giving an additional loss of power), with delayed recovery for at least 24 hours, whereas sedentary controls recovered full muscle power after 200 minutes.

Autonomic nervous system testing has revealed abnormalities of the sympathetic and parasympathetic systems. There is considerable evidence from different investigators, using different technologies and studying different groups of patients, of a state of chronic immune activation (Hooper et al. 2001 [Online])

From M.E. expert Dr Elizabeth Dowsett:

The brain has often been likened to a computer. However, there are fundamental differences in its essential function of processing, comparing and storing information. This is highly developed in humans, making us uniquely creative and better adapted to our environment than any animal. Unlike a computer, which can be switched on and off and is programmed to give set answers to a single question, the chemical transmitter bridging the synapse introduces a variability into the on-going message and "Neuronal Plasticity" into the receiving/transmitting network. It has been shown that similar modifications in response may be induced by virus infection. The brain contains some 100 billion neurons connected to some 10,000 relay stations and this enormous electrical activity creates a massive need for energy, using up 20% of the entire body's demand for oxygen and glucose. Recent studies of the brain stem by SPECT scan, indicate hypoperfusion and low metabolic activity in subjects with M.E.

The brain is continuously bombarded by incoming signals each of which, after information processing and co-ordination, will initiate an appropriate muscular response (however small). However, there is no single "movement centre" and incoming signals will either be directed via the brain stem to the spinal cord, undergoing processing on the way from specialised centres such as the cerebellum (the brain's autopilot) or the Thalamus and Basal Ganglia beneath the cerebral hemispheres, all of which act as subsidiary control areas, relieving higher motor centres in the cerebral cortex for more intricate muscular action. Thus, semi-automatic movements (eg swimming) co-ordination of movement with visual and sensory input, determination of balance and the mediation of individual limb movements, will pursue a devious pathway, while direct connection is made between the higher motor cortex and muscles requiring exceptionally fine co-ordination such as those of the hand, face and mouth - an arrangement appropriate to the evolutionary toll making, and communication skills of humans. Such muscles are allotted an especially large share of the motor cortex and, when a motor impulse reaches the nerve end plate eg: in finger muscles, it is allocated to a few individual fibres rather than spread over large areas, as in the leg. Modern research indicates disturbed metabolism in many areas essential to motor control in the brain stem of patients with M.E., the majority of whom have evidence of inco-ordinated muscle twitching after slight exertion.

Hypothalamic function is often disturbed in ME. The Hypothalamus is a central relay station for collecting and integrating signals from diverse sources (including the thalamus, limbic system and reticular activating system in the

brain stem and mid brain) and for producing hormones which affect kidney function and lactation before funneling them into the dependent Pituitary Gland, as well as inhibiting or promoting the release of pituitary hormones. In this fashion it has a major influence on specific reaction to stress, thyroid function, weight, appetite and control of glucose metabolism, as well as regulation of female sex hormones and the circadian sleep/temperature rhythm.

A good memory demands normal functioning of almost all areas of the cerebral cortex, the basal nerve centres of the mid brain (eg the thalamus and hippocampus) and their interconnecting pathways through the brain stem. Fluctuations of metabolic activity in these areas (often made worse by physical and mental [overexertion]) have been reported in SPECT scans of patients with M.E., the vast majority of whom complain of difficulty with short-term memory. (n.d.c, [Online])

M.E. expert Dr Byron Hyde, explains that:

I have some ME patients with a circulating red blood cell volume less than 50% of expected and a very large number with the range of 60% to 70%. What this test means is that blood is pooling somewhere in the body and that this blood is probably not available for the brain. When blood flow to the heart decreases sufficiently, the organism has an increased risk of death. Accordingly, the human body operates in part with pressoreceptors that protect and maintain heart blood supply. When blood flow decreases, pressoreceptors decrease blood flow to noncardiac organs and shunt blood to the heart to maintain life. This, of course, robs those areas of the body that are not essential for maintaining life and means the brain, muscles, and peripheral circulation are placed in physiological difficulty. (Hyde 2003, [Online])

This physiological difficulty is exacerbated by physical and mental activity and orthostatic stress. Dr Byron Hyde goes on to say that: 'In MRI spectography of arm muscle of M.E. patients, it has been shown that because of an abnormal buildup of normal metabolites, the muscle cell actually shuts down to prevent cell death.' Dr Hyde explains that this is what is happening to the M.E. patient's cell physiology in the brain, and in muscle as a result of certain levels of physical and mental activity; there is 'cell field shutdown' to prevent the death of the cell (Hyde 2003, [Online]).

Dr Byron Hyde, also explains that the vascular and cardiac dysfunctions seen in M.E. are often the most obvious set of dysfunctions when looked for, and are the cause of a significant number of M.E. symptoms:

The subject of vascular pathology is not new. The fact of the children dying of a Parkinsonian-like vascular injury to the basal ganglia in Iceland during the Akureyri M.E. Epidemic is an obvious indication of the CNS vascular effects in M.E. Vasculitis has been well documented by Dr. E. Ryll in his description of the epidemic in the San Juan Mercy, Sacramento California Hospital in 1975. He described this M.E. epidemic as an epidemic vasculitis. He was correct. Following my 21 years of examining M.E. patients and 16 years of subjecting M.E. patients to brain imaging techniques suggested by Goldstein and Mena, it has become obvious to me that we are dealing with both a vasculitis and a change in vascular physiology. Numerous other physicians have supported this finding.

The recent interpretation of the cause of Multiple Sclerosis (MS), as an injury of the microvasculization causing the injury of the schwann cells that in turn causes the demyelination injuries of MS has been added to that of paralytic poliomyelitis as an essential vascular injury. Paralytic poliomyelitis was thought to be a primary injury to the anterior horn cells of the spinal cord but is now recognized as a vasculitis injuring the circulation to the anterior horn cells. Poliomyelitis is generally a non-progressive, specific site injury, although post-polio syndrome with demonstration of subcortical brain changes has challenged that belief. MS is a recurrent more fulminant physiological vascular injury. M.E. appears to be in this same family of diseases as paralytic polio and MS. M.E. is definitely less fulminant than MS but more generalized. M.E. is less fulminant but more generalized than poliomyelitis. This relationship of M.E.-like illness to poliomyelitis is not new and is of course the reason that Alexander Gilliam, in his analysis of the Los Angeles County General Hospital M.E. epidemic in 1934, called M.E. atypical poliomyelitis (2007, [Online]).

It is well known that enteroviruses may cause chronic cardiac disease as well as major neurological injury. Kandolf states that "enteroviruses are capable of causing dilated cardiomyopathy of sudden onset or lead to a variety of common arrhythmias." Utilizing mouse models, Wilson and again Reyes demonstrated that Coxsackie infected [enterovirus infected] mice, forced to swim to the point of exhaustion during the acute phase of infection, developed chronic heart disease whereas Coxsackie infected mice who were allowed to rest during the acute phase, did not develop chronic heart disease. M.E. represents a possibility of serious cardiac injury primarily in patients who exercise or maintain exhaustive work efforts during the onset of their illness. It is possible that some of these patients who die and others that develop major cardiac changes are never recognised as M.E. (Hyde & Jain 1992a, pp. 375-383)

Dr. Paul Cheney explains that when disabled M.E. patients stand up, they are 'on the edge of organ failure' due to extremely low cardiac output as their Q drops to 3.7 litres per minute (a 50% drop from the normal of 7 litres per minute). Without exception, according to Cheney, every disabled M.E. patient 'is in heart failure' and the disability level is exactly proportional to the severity of their Q defect, without exception and with scientific precision (Marshall & Williams 2005a, [Online]) (Cheney 2006, [video recording]). Findings which showed mitochondrial

metabolic dysfunction similar to mitochondrial encephalomyopathy also led Dr Cheney to comment, ‘The most important thing about exercise is not to have [patients with M.E.] do aerobic exercise. I believe that even progressive aerobic exercise is counter-productive. **If you have a defect in mitochondrial function and you push the mitochondria by exercise, you kill the DNA**’ (Williams 2004, [Online]).

It is known that M.E. is:

1. An acute onset (biphasic) epidemic or endemic infectious disease process
2. An autoimmune disease (with similarities to Lupus)
3. An infectious neurological disease, affecting adults and children
4. A disease which involves significant (and at times profound) cognitive impairment/dysfunction
5. A persistent viral infection (due to an enterovirus; the same type of virus which causes poliomyelitis and post-polio syndrome)
6. A diffuse and measurable injury to the vascular system of the CNS.
7. A CNS disease with similarities to M.S.
8. A variable (but always serious) diffuse, acquired brain injury
9. A systemic illness (associated with organ pathology; particularly cardiac)
10. A vascular disease
11. A cardiovascular disease
12. A type of cardiac insufficiency
13. A mitochondrial disease
14. A metabolic disorder
15. A musculo-skeletal disorder
16. A neuroendocrine disease
17. A seizure disorder
18. A sleep disorder
19. A gastrointestinal disorder
20. A respiratory disorder
21. An allergic disorder
22. A pain disorder
23. A life-altering disease
24. A chronic or lifelong disease associated with a high level of disability
25. An unstable disease: from one hour/day/week or month to the next
26. A potentially progressive or fatal disease

M.E. affects every cell in the body and almost every bodily system (Hyde 2007, [Online]) (Hooper et al. 2001, [Online]) (Cheney 2007, [video recording]) (Ramsay 1986, [Online]).

- For more information see the [General articles and research overviews](#) section. See also articles by: [Dr. Elizabeth Dowsett](#) and [Byron Hyde MD](#).

Is there a legitimate scientific debate about whether or not M.E. is a ‘real’ neurological disease?

Despite popular opinion there simply is no legitimate scientifically motivated debate about whether or not M.E. is a ‘real’ neurological illness or not, or whether it has a biological basis.

Substantial evidence exists to show that it is simply not possible that somatisation, secondary gain, malingering, aberrant illness beliefs, too much focus on normal bodily sensations, irrational fear of exercise leading to deconditioning, being rich and white, being poor and from an ethnic minority, being lazy and unwilling to work, being too highly driven and perfectionistic and working too hard, faulty thought processes, lack of motivation, long-term stress, acute stress, abuse in childhood, a genetic inability to deal with normal levels of stress, inadequate coping strategies and contagious sociological hysteria – or any or the other ridiculous and often

contradictory 'theories' put forward by these vested interest groups – play a role in causing or perpetuating authentic M.E.

The psychological or behavioural theories of M.E. and claims that M.E. is just another term for 'CFS' are no more scientifically viable than theories of a flat earth. They are pure fiction. Strong evidence of the biological basis for the illness has existed since the 1930s and 1950s and more than a thousand good articles now support the basic premises of M.E. as a debilitating organic neurological illness. Thus this is not simply theory, but is based upon an enormous body of clinical information including objective scientific testing. Newer scientific evidence is increasingly strengthening this hypothesis (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001a, [Online]). M.E. is not 'medically unexplained' or 'unexplainable.'

The reality is that anyone, whether medically qualified or not, who looks at the worldwide published medical evidence on M.E. could not fail to recognise that the psychological or psychiatric theories could not possibly explain the many different and profound physical abnormalities seen in M.E. (nor the many other characteristics of the disease which are not consistent with psychological or behavioural illness). There are only two ways that a person could reach a different conclusion:

1. Bias due to vested political or financial (or other) interests
 2. Lack of access to a truly representative selection of the evidence (ie. an individual has only availed themselves of the pseudo-science provided by financial stakeholders and not a representative selection (or indeed *any*) of the legitimate and unbiased science.)
- For more information see: [Who benefits from 'CFS' and 'ME/CFS'?](#), [Smoke and Mirrors](#) and [Putting research and articles into context](#). See also: [Are we just 'marking time?'](#)

What are some of the most common myths about M.E.?

There are many, many myths about M.E. Unfortunately, it is very common to read government reports and media articles about M.E. (or 'CFS') which do not contain even a SINGLE fact. Some of the most common myths include that:

MYTH: M.E. is consequent from an organic (viral) trigger but the illness is short lived unless there are psychological and social factors which perpetuate the illness long term

MYTH: It is only recently that researchers have finally shown that M.E. has a physical or organic basis

MYTH: The only treatments shown to be useful in treating M.E. are CBT (cognitive behavioural therapy) and GET (graded exercise therapy). CBT/GET treatments are useful in 'rehabilitating' M.E. sufferers. These treatments are also completely safe for sufferers and there is no risk of these therapies worsening the illness in the short or long term

MYTH: M.E. can be caused by the Epstein-Barr virus, candida, adrenal exhaustion, hepatitis, Q fever, Ross river virus or glandular fever/mononucleosis

MYTH: Evidence exists which suggests or shows that M.E. is caused (partially or completely) by XMRV infection, and this theory fits all the major facts of M.E. (with no big 'holes')

MYTH: The recent XMRV 'CFS' research was conducted on a distinct and 100% M.E. patient group. This research clearly separates M.E. patients from those with 'CFS.'

MYTH: The recent XMRV 'CFS' research shows promise in providing a unique test for M.E.

MYTH: Evidence exists which suggests or shows that anti-retroviral treatments (perhaps specific to XMRV) are the treatment breakthrough that M.E. patients have been waiting for, for so long. This type of treatment represents real hope (or certainty) of a cure for M.E. patients.

MYTH: XMRV is believed to be an important and absolutely vital scientific lead to follow, by all of the M.E. community.

MYTH: The name CFS was chosen in 1988 by a group of experienced M.E. clinicians who thought it was the most medically accurate name for the illness at that time (given that fatigue was always the worst symptom and that so little was known about the pathology of M.E. at the time)

MYTH: It is the name CFS itself that is the cause of all the misunderstandings about the illness. If the name Myalgic Encephalomyelitis was renewed (for example) patients would automatically start to get the recognition and respect they deserve, more money for legitimate research and everything else they so desperately need

MYTH: Once we have enough hard science behind M.E. – in particular a single diagnostic marker for the illness – things will improve and M.E. sufferers will automatically start to get the medical recognition and respect they deserve

The truth is that every one of these statements is completely untrue despite how often they have been repeated and presented as 'facts'.

- For more information, and a longer list of M.E. myths, see: [The myths about M.E.](#)

Similar Medical Conditions?

There are a number of post-viral fatigue states or fatigue syndromes which may follow common infections such as mononucleosis/glandular fever, hepatitis, Q fever, Ross river virus and so on. M.E. is an entirely different condition to these self-limiting fatigue syndromes however (and is *not* caused by the Epstein Barr virus or any of the herpes or hepatitis viruses), the science is very clear on this point. People suffering with any of these post-viral fatigue states or fatigue syndromes do not have M.E.

M.E. does have some limited similarities – to varying degrees – to illnesses such as multiple sclerosis, Lupus, post-polio syndrome, Gulf War Syndrome and chronic Lyme disease, and others. But this does not mean that they represent the same etiological or pathobiological process. They do not. M.E. is a distinct neurological illness with a distinct; onset, symptoms, aetiology, pathology, response to treatment, long and short term prognosis – and World Health Organization classification (G.93.3) (Hyde 2006, [Online]) (Hyde 2007, [Online]) (Hooper 2006, [Online]) (Hooper & Marshall 2005a, [Online]) (Hyde 2003a, [Online]) (Dowsett 2001a, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Dowsett 1996, p. 167) (Dowsett et al. 1990, pp. 285-291) (Dowsett n.d., [Online]).

What illnesses such as Gulf War Syndrome, Fibromyalgia, multiple chemical sensitivity and chronic low-dose organophosphate poisoning do have in common with M.E. however is that the same bogus psychiatric model applied to M.E. by vested interest psychiatrists and others has also been extended to these other conditions, leaving many sufferers of these illnesses also without the help and support they so urgently need (Hooper & Marshall 2005a, [Online])

- See [M.E. and other illnesses](#) for more information. See also the new paper: [M.E. vs MS: Similarities and differences](#).

How well is research into M.E. research funded by government?

Funding for legitimate M.E. research by government can only be described as a disgrace.

M.E. is a comparable illness physically to multiple sclerosis and yet M.E. research receives not even a fraction of the government research funding that MS does – this despite the fact that M.E. is at least as common as MS and can also often be far more physically disabling. A recent article explained that:

Breast cancer is diagnosed in 26 women per 100,000 and receive \$716 million dollars in government research funds; lung cancer is diagnosed in 63 women per 100,000 and receives \$300 million in government research funds; HIV is diagnosed in 125 women per 100,000 and receives \$2921 million in government research funds; and CFS is diagnosed in anywhere from 300-540 women per 100,000 and receives \$6 million in government research funds. Research funding for CFS [in the US] is about 107th out of 110 diseases funded (SDCD, [Online]).

But even this small amount of money for 'CFS' research does nothing to help people with M.E., nor patients with any other distinct disease. Studying vague and mixed groups of 'fatigue' sufferers is of no benefit to any patient group; particularly as many of those conducting this 'research' are known to have vested interests in certain outcomes – and even to have determined the outcomes of some of this 'research' before the actual studies have been conducted (Hooper 2003a, [Online]). As if that weren't bad enough, all too commonly the results of these studies on fatigued people (and people with all sorts of different illnesses both psychiatric and non-psychiatric) are then applied to people with M.E. as if they somehow represented the exact same patient group. This leaves people with M.E. not only without the new treatments which legitimate research would bring but often forced to undergo all sorts of useless and even severely harmful treatments in their stead (such as psychotherapy, antidepressants and exercise programs). Thus because of the confusion between M.E. and 'CFS' increased funding for 'CFS' research is far more likely to actually cause further harm to people with M.E. than to provide any benefit.

In reality, governments around the world are currently spending \$0 a year on M.E. research. Considering the brutal severity of the illness, the vast numbers of patients (adults and children) involved worldwide and the number of patient deaths (many of them preventable), this is a worldwide disgrace.

Often the research that offers a glimmer of genuine hope to M.E. patients is research into diseases that share significant similarities with M.E. including Alzheimer's, Polio, Parkinson's, AIDS, Lupus, [Multiple Sclerosis](#) and so on. (Alzheimer's, Parkinson's and Multiple Sclerosis are listed along with M.E. under 'Diseases of the nervous system' in the ICD Classifications.) These studies have far more relevance to M.E. patients than almost all of the 'CFS' studies produced which lack scientific merit and use exclusively or almost exclusively non-M.E. patient groups.

In future, it is essential that M.E. research again be conducted using only M.E. defined patients and using only the term M.E.

- See [Research and Articles in Context](#) for more information about research into M.E. and the challenges involved. See the [Donations](#) page to make a donation towards M.E. research and advocacy.

Abuse and M.E.

Two of the most common interventions people with M.E. are encouraged to participate in are cognitive behavioural therapy (CBT) and graded exercise therapy (GET).

However, despite the misleading claims to the contrary made by various vested interest groups, no evidence exists which demonstrates that CBT and GET are appropriate, effective or safe treatments for M.E. patients. Studies by these groups (and others) involving miscellaneous psychiatric and non-psychiatric 'fatigue' sufferers, and their positive response to these treatments, have no more relevance to M.E. sufferers than they do to patients with multiple sclerosis, diabetes or any other illness. Patients with M.E. are routinely being prescribed these treatments on what amounts to a random basis medically.

As (very bad) luck would have it, graded exercise programs are probably the single most inappropriate 'treatment' that an M.E. sufferer could be encouraged to undertake. This is because exercise or exertion intolerance is one of the many characteristics which separates M.E. so distinctly from various post-viral fatigue states or other illnesses involving 'chronic fatigue.' People with M.E. do not improve with exercise. They cannot; exercise intolerance is a large and essential part of *what M.E. is* (Ramsay 1986, [Online]) (Hyde 2003, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001a, [Online]).

Overwhelming evidence exists to show that exercise can have extremely harmful effects on M.E. patients; permanent damage may be caused, as well as disease progression: recent research has shown that postural stress (as well as excessive physical or cognitive activity) exacerbates cardiac insufficiency in this disease. Patients with M.E. are in a form of heart failure and even minor or moderate levels of physical activity (etc.) can cause severe and prolonged relapse in M.E. Patient accounts of leaving exercise programs much more severely ill than when they began them; wheelchair-bound or bed-bound or needing intensive care or cardiac care units, are common. The damage caused is often severe and either long-term or permanent: some patients are still dealing with the effects of inappropriate advice to exercise five, ten or more YEARS afterwards, and for some patients this damage is permanent. Sudden deaths have also been reported in a small percentage of M.E. patients following exercise. As [Dr. Elizabeth Dowsett](#), explains; '20% have progressive and frequently undiagnosed degeneration of cardiac muscle which has led to sudden death following exercise' (2000, [Online]) (2001a, [Online]).

In 2004 a survey of severely affected M.E. sufferers found that graded exercise was by far the single most harmful treatment they had undertaken. A shocking 82% reported that it had made their condition worse. A significant number of those surveyed indicated that they were not severely affected *before* GET (25% M.E. Group 2004, [Online]). Thus GET should not be considered safe for M.E. sufferers of any severity. Graded exercise *cannot* improve authentic M.E.; disabled patients who improve with exercise do not qualify for a diagnosis of authentic M.E. (Ramsay 1986, [Online]) (Hyde 2003, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43) (Dowsett 2001a, [Online]).

The effect of cognitive behavioural therapy (CBT) on patients with M.E. varies depending on the type of CBT used, and the severity of the M.E. At best this intervention is useless, but CBT which aims to convince a physically ill person that he/she does not have a physical disorder is disrespectful, inappropriate and cruel. It places an additional (and bogus) psychological burden on a person already suffering with severe physical illness, and can cause significant psychological harm. This abusive form of CBT can undoubtedly cause significant psychological harm, but it is the additional associated burdens; severe and prolonged physical relapse, the withholding of basic medical care, the removal of children from their parents and parents being falsely charged with making their children ill themselves (etc.) which combine to make CBT so potentially harmful. Thus the negative effects of CBT can sometimes be equally as devastating as those of GET, or in some cases, even worse (for sufferers and their families).

CBT and GET are at best useless and at worst extremely harmful for M.E. patients.

Despite this, people with M.E. are routinely being recommended these 'treatments' while also being assured that they are completely safe. These interventions are also not just being offered to M.E. patients solely on a voluntary basis; many have been treated as psychiatric patients against their will (or against the will of the parents of children with M.E.). In some cases it is a condition of receiving medical insurance entitlements that M.E. patients first undergo 'rehabilitation' such as CBT and GET programs. This is also true of government welfare entitlements, particularly in the UK, as [Professor Malcolm Hooper](#) explains:

[In the UK] many patients are simply too sick to be forced to attend psychiatric units and to participate in compulsory "management strategies" which involve exercising, but if they fail to attend, they are deemed not to want to get better and their State benefits are withdrawn because of Wessely's dogmatic advice to Government that M.E. is nothing more than an "aberrant illness belief". There are many such known cases, including those in which ME patients have been threatened with being sectioned (ie. compulsorily detained under the Mental Health Act) unless they comply with psychotherapy. (2003a, [Online])

In a scientifically enlightened age such as this, how is it acceptable that the results of studies using one patient group can be used to determine the aetiology and appropriate treatments for a completely separate and unrelated patient group? How is this scientific? How is this ethical? Does this mean that research conducted using patients with diabetes (for example) can now also be applied to all those who have cancer, nut allergies, broken legs or any number of other unrelated problems; merely because the researchers involved have decided that they would like it to, and that doing so would suit their own vested interests?

If a prescription drug had anything like the appalling track record exercise has with people with M.E. (or even a small fraction of it, even 2%) it would be a worldwide scandal. The drug would be immediately banned, there would be some form of inquiry and serious criminal charges may well be laid. Yet the rate of people with M.E. encouraged or even *forced* to exercise continues to rise, and with the full support of governments. This is despite the fact that legitimate research clearly shows that along with the huge risk involved, it has a ZERO percent chance of providing any benefit to people with authentic M.E. That this can be allowed to go on in such a supposedly enlightened day and age as ours defies belief.

It is also of great concern that so many M.E. patients are ONLY offered 'treatments' such as CBT and GET, while access to even basic appropriate medical care is withheld. Of the 25% of patients who are severely affected by the illness (and are bed-bound and housebound), the majority have no contact with the health service at all as they are seldom able to obtain house calls (Dunn 2005, [Online]). Many sufferers are also refused the basic welfare support to which they are entitled. Thus a significant percentage of very physically ill and vulnerable M.E. patients are simply left to suffer and die at home without any medical care, welfare or social support (Hooper 2003a, [Online]).

From Professor Malcolm Hooper:

Patients with myalgic encephalomyelitis, particularly children, have suffered gross and barbaric abuse and persistent denigration as a consequence of the beliefs of certain psychiatrists who are attempting to control the national agenda for this complex and severe neuro-immunological disorder. These psychiatrists are shown to be clearly in breach of the first tenet of medicine --- first do no harm--- in that by their words and deeds they have wreaked havoc in the lives of M.E. patients and their families by their arrogant pursuit of a psychiatric construct of the disorder which ignores the abundant clinical and scientific evidence (widely presented in the international medical and scientific literature) of the organic nature of M.E. (2003a, [Online])

This problem with forced participation in CBT and GET for patients with M.E. is undoubtedly at its worst in the UK and the Netherlands, and to a lesser extent Australia. These treatments are not so commonly recommended in other countries, such as the US, at present. However, the latest information produced on 'CFS' by the CDC in the US makes it clear that this seems very likely to change in the near future. US M.E. patients are increasingly concerned, and with good reason, that the US will soon follow the abusive UK model of forced CBT and GET and so on. *This abuse must be stopped, and most importantly, not allowed to spread further and claim untold numbers of new victims.*

To claim that there is "medical disagreement" over M.E. is simply not accurate. As Professor Malcolm Hooper explains:

People in positions of power are misusing that power against sick people and are using it to further their own vested interests. No-one in authority is listening, at least not until they themselves or their own family join the ranks of the psychiatrically-persecuted, when they too come up against a wall of utter indifference (2003a, [Online])

What is happening to people with M.E. is a gross violation of basic human rights. It amounts to legalised medical torture and abuse of some of our most vulnerable members of society. The damage perpetrated on those with M.E. by Wessely School adherents cannot be quantified (Hooper & Marshall 2005a, [Online]).

- These brief comments on the effects of CBT and GET are taken from the more detailed paper: [The effects of CBT and GET on patients with M.E.](#), see this paper for more information.
- For more information about the effects of overexertion on M.E. patients, including statements/research from some of the world's leading M.E. experts about why overexertion is so physically harmful, see: [Smoke and Mirrors](#). (This paper also includes links to many different patient accounts of the effects of overexertion on people with

M.E.). If you have M.E. see [Treating M.E. - The Basics](#) and [Treating M.E. - Avoiding Overexertion](#) for more on the importance of avoiding overexertion.

- For more information see also: [Comments the 25% M.E. Group to the Gibson Enquiry](#). Many more articles on this topic are also available in [Section 3](#), [Section 5](#) and [Section 6](#) of the CBT and GET Database.

Two case studies of abuse in M.E.

Case study 1: A recent example of a M.E. sufferer being taken into psychiatric care against their will is the case of Sophia Mirza in the UK. Tragically, Sophia died of her illness shortly after being wrongly sectioned under the Mental Health Act. (Sophia was not mentally ill, nor a danger to herself or others). Sophia was severely ill with M.E. and bedbound but she was refused even basic medical care, and this is believed to have contributed greatly to her death. She was only 32 when she died. Sophia's mother writes: 'In July, the professionals returned - as promised by the psychiatrist. The police smashed down the door and Sophia was taken to a locked room within a locked ward of the local mental hospital. Despite the fact that she was bed-bound, she reported that she did not receive even basic nursing care, her temperature, pulse and blood pressure (which had been 80/60), were never taken...' (Bagnall 2006, [Online]) (Marshall & Williams 2006b, [Online]).

- For more information on this tragic case, and entirely avoidable death, see: [Inquest Implications, Civilization: Another word for barbarism](#) and [The Story of Sophia and M.E.](#) Let there be no misunderstanding about deaths as a result of M.E. they occur far more commonly than many would care to acknowledge. (Marshall & Williams 2006b, [Online]).

Case study 2: An example of a child being forcibly removed from his parents, and subjected to what amounts to torture is the case of Ean Proctor, also in the UK. In 1988, a formerly healthy 12 year old boy named Ean Proctor from the Isle of Man had been suffering from M.E. (part of which involved an inability to speak) since the autumn of 1986. In June 1988 (shortly after Simon Wessely became involved in the case) without ever having spoken to his parents, [Professor Malcolm Hooper](#) explains that:

Social workers supported by psychiatrists and armed with a Court Order specially signed by a magistrate on a Sunday, removed the child under police presence from his distraught and disbelieving parents and placed him into "care" because psychiatrists believed his illness was psychological and was being maintained by an "over-protective mother". Everything possible was done to censor communication between the child and his parents, who did not even know if their son knew why they were not allowed to visit him.

In this "care", the sick child was forcibly thrown into a hospital swimming pool with no floating aids because psychiatrists wanted to prove that he could use his limbs and that he would be forced to do so to save himself from drowning. He could not save himself and sank to the bottom of the pool. The terrified child was also dragged out of the hospital ward and taken on a ghost train because psychiatrists were determined to prove that he could speak and they believed he would cry out in fear and panic and this would prove them right. Another part of this "care" included keeping the boy alone in a side-ward and leaving him intentionally unattended for over seven hours at a time with no means of communication because the call bell had been deliberately disconnected. The side-ward was next to the lavatories and the staff believed he would take himself to the lavatory when he was desperate enough. He was unable to do so and wet himself but was left for many hours at a time sitting in urine-soaked clothes in a wet chair.

Another part of the "care" involved the child being raced in his wheelchair up and down corridors by a male nurse who would stop abruptly without warning, supposedly to make the boy hold on to the chair sides to prevent himself from being tipped out; he was unable to do so and was projected out of the wheelchair onto the floor, which on one occasion resulted in injury to his back. This was regarded as a huge joke by the staff. Ean Proctor was kept in "care" and away from his parents for over five months (2003a, [Online]).

- To read more about this appalling case see: [The Mental Health Movement: Persecution of Patients?, To set the record straight about Ean Proctor from the Isle of Man, Another Meadow?](#) and [Considerations of some issues relating to the published views of Psychiatrists of the Wessely School in relation to their beliefs about the nature, cause and treatment of myalgic encephalomyelitis \(ME\)](#).
- For more information about forced exercise and other 'treatments' used on M.E. children (and adults) see: [Children with M.E.](#) and [Sara Bass's Testimony](#). For more about the new (and very concerning for M.E. patients) 'fatigue' treatment centres in the UK see [A CALL FOR HELP FROM THE M.E. COMMUNITY, THE TRAVESTY OF SO-CALLED "M.E. TREATMENT" Critical Considerations, THE UNDESERVING ILL - A WARNING, Science or Psychology](#) and many more articles in the 100+ page [CBT and GET Database](#).
- Note that this is *not* an isolated case of mistreatment of a child, or removal of a child from the home due to the diagnosis of M.E. and the involvement of vested interest groups in M.E. Children continue to be removed forcibly from their homes today, and parents have in some cases also had formal charges laid against them of child abuse. (This happens most often in the UK and the Netherlands, and to a slightly lesser extent Australia. Both of these

case studies are from the UK. But the latest information produced on 'CFS' by the CDC in the US makes it clear that similar problems for M.E. patients may well occur in the US in the near future, unfortunately. This may soon be a far more widespread problem, unless something can be done to stop it.)

It is only M.E. patients who are negatively affected by the bogus creation of 'CFS'?

The creation of the bogus disease category of 'CFS' (and the equally flawed government policies that have gone along with it) have had a devastating effect on hundreds of thousands of M.E. sufferers around the world, including young children. People with M.E. however are not the only patient group to be negatively affected by this politically-modified science. Other patient groups misdiagnosed as CFS are also denied appropriate diagnosis and treatment and they may also routinely be subjected to inappropriate psychological interventions such as CBT and GET.

Today it is common for patients with a variety of different illnesses with fatigue as major symptom to be misdiagnosed as having 'CFS.' Patients 'diagnosed' with Fukuda or CDC CFS (or any other CFS definition) may have any one of a number of different illnesses. It is vitally important that each of these patients discovers their true diagnosis so that they may finally receive appropriate treatment and support.

Every patient deserves the best possible opportunity for appropriate treatment for their illness, and for recovery and this process must begin with a correct diagnosis if at all possible; a correct diagnosis is half the battle won. Lumping these disparate patient groups together under a vague and meaningless category of 'fatiguing illnesses' only hinders each of the patient groups involved in their battle to regain their health. Treating this diverse and heterogenous patient group as if their illnesses each shared the same symptoms, aetiology, pathology and response to treatment is inappropriate and highly unlikely to benefit the health and wellbeing of any of the patient groups involved. Treating this 'CFS' group as if they each shared a specific psychological or behavioural illness is also clearly inappropriate. Aside from representing a heterogenous patient group, many (likely the vast majority) of those with the diagnosis are not mentally ill, and do not suffer from behavioural problems. (This includes of course, those patients with authentic M.E.) (Hooper 2006, [Online]) (Hyde 2006, [Online]) (Hooper et al. 2001, [Online]).

There are also a variety of negative impacts on doctors and the public (and others) caused by the 'CFS' insurance scam. As one M.E. advocate explained recently: 'So many abnormalities have now been shown to occur regularly in cases of authentic M.E. that it is not only bad science to attempt to dismiss, ignore or deny a reality that can be scientifically measured, but to continue to do so must, as others have noted, border on the criminal' (Marshall & Williams 2006c, [Online]). This is particularly relevant to those doctors which recommend CBT or GET to their patients. Whether they are aware of it or not, these doctors are leaving themselves open to being sued when (inevitably) a proportion of these patients (those with M.E.) are made sicker by these therapies, or being sued by the families of M.E. sufferers who die as a result of these inappropriate interventions.

Again, the only groups which gain from the 'CFS' confusion are insurance companies and various other organisations and corporations which have a vested financial interest in how these patients are treated, including the government.

- For more information see: [Who benefits from 'CFS' and 'ME/CFS'?](#), [Smoke and mirrors](#) and [The misdiagnosis of CFS](#)
- This misdiagnosis of 'CFS' and lack of appropriate medical treatment can have many negative effects on this heterogeneous group of patients. For example, there have been cases where cancer sufferers suffering severe fatigue (as is common in cancer) have been misdiagnosed as 'CFS' and subsequently died due to lack of treatment. This is not uncommon. Dr Byron Hyde's paper [The Complexities of Diagnosis](#) mentions several such cases (as well as many other issues and case studies of CFS misdiagnosis). Patients with a variety of different illnesses have died and continue to die because they have been misdiagnosed with 'CFS' and denied appropriate treatment. Every diagnosis of CFS is a *misdiagnosis*.

How severe is M.E.?

Although some people do have more moderate versions of the illness, symptoms are extremely severe for at least 25-30% of the people who have M.E., significant numbers of whom are housebound and bedbound.

Dr. Paul Cheney stated before a US FDA Scientific Advisory Committee:

I have evaluated over 2,500 cases. At worst, it is a nightmare of increasing disability with both physical and neurocognitive components. The worst cases have both an M.S.-like and an AIDS-like clinical appearance. We have lost five cases in the last six months. 80% of cases are unable to work or attend school. We admit regularly to hospital with an inability to care for self (Hooper et al. 2001 [Online]).

M.E. patients have been found to experience greater functional severity than the studied patients with heart disease, virtually all types of cancer, and all other chronic illnesses.’ An unrelated study compared the quality of life of people with various illnesses, including patients undergoing chemotherapy or haemodialysis, as well as those with HIV, liver transplants, coronary artery disease, and other ailments, and again found that M.E. patients scored the lowest. "In other words", said one M.E. expert in a radio interview, “**this disease is actually more debilitating than just about any other medical problem in the world**” (Munson 2000, p. 4).

For very severely affected M.E. sufferers there is virtually no ‘safe’ level of physical or mental activity, orthostatic stress or sensory input; no level which does not produce a worsening of symptoms, and perhaps also contribute to disease progression. Even the most basic actions – speaking a few words, being exposed to moderate light or noise for a few minutes, turning over in bed, having hair or body washed in bed by a carer or chewing and swallowing food – cause severe and extended symptom exacerbations in such patients. It is not uncommon to hear of very severely affected sufferers who are unable to bathe themselves (or even be bathed by a carer) more often than once a week, or even once every few weeks, or even less. Some sufferers cannot chew or swallow food any longer and need to be tube fed. Many patients with severe M.E. are no longer able to toilet themselves, and so on. Either sufferers are just too ill to do these things at all, or they cannot tolerate the very long and severe relapses that come after such activities.

For people with severe M.E. even the smallest movement, thought, touch, light, noise or period upright can make their already very severe symptoms far worse. Thus few illnesses demand such isolation and loss of quality of life as severe M.E. Very often people with very severe M.E. can barely communicate, or even tolerate the presence of another person. This is what makes M.E. such a cruel disease and such an isolating disease. The illness can cause an unrelenting level of disability, suffering and isolation that is just unimaginable to anyone not familiar with very severe M.E. (Bassett 2009, [Online]).

In the 1980s Mark Loveless, an infectious disease specialist and head of the AIDS and M.E. Clinic at Oregon Health Sciences University, found that M.E. patients whom he saw had far lower scores on the Karnofsky performance scale than his HIV patients even in the last week of their life. He testified that an M.E. patient, ‘feels effectively the same every day as an AIDS patient feels two weeks before death’ (Hooper & Marshall 2005a, [Online]). But in M.E., this extremely high level of illness and disability is not short-term. it does not always lead to death and it can instead continue uninterrupted for **decades**.

- For more information on severe M.E. see [The severity of M.E.](#) and [M.E. Fatalities](#) and [Why patients with severe M.E. are housebound and bedbound](#)
- It should also be noted that even those patients with moderate M.E. are far more severely affected than many patients with a variety of other illnesses. Of course severe M.E. is even worse, but moderate M.E. can also cause severe symptoms and a relatively high level of disability and suffering, compared to many other illnesses.

Recovery from M.E.

M.E. patients who are given advice to rest in the early stages of the illness, and who avoid overexertion thereafter, have repeatedly been shown to have the most positive long-term prognosis. As M.E. expert [Dr Melvin Ramsay](#) explains:

The degree of physical incapacity varies greatly, but the [level of severity] is directly related to the length of time the patient persists in physical effort after its onset; put in another way, **those patients who are given a period of enforced rest from the onset have the best prognosis**. Since the limitations which the disease imposes vary considerably from case to case, the responsibility for determining these rests upon the patient. Once these are ascertained the patient is advised to fashion a pattern of living that comes well within them (1986, [Online]).

M.E. can be progressive, degenerative (change of tissue to a lower or less functioning form, as in heart failure), chronic, or relapsing and remitting. Some patients experience spontaneous remissions- albeit most often at a greatly reduced level of functioning compared to pre-illness- and such patients remain susceptible to relapses for the remainder of their lives. M.E. is a chronic/life-long disability where relapse is always possible. Cycles of severe relapse are common, as are further symptoms developing over time. Around 30% of cases are progressive and degenerative and sometimes M.E. is fatal. As [Dr Elizabeth Dowsett](#) explains:

After a variable interval, a multi-system syndrome may develop, involving permanent damage to skeletal or cardiac muscle and to other "end organs" such as the liver, pancreas, endocrine glands and lymphoid tissues, signifying the further development of a lengthy chronic, mainly neurological condition with evidence of metabolic dysfunction in the brain stem. Yet, stabilisation, albeit at a low level, can still be achieved by appropriate management and support. The death rate of 10% occurs almost entirely from end-organ damage within this group (mainly from cardiac or pancreatic failure). It has to be said that suicide in younger patients and in earlier stages of the disability is related to

the current climate of disbelief and rejection of welfare support... It is an additional and potentially avoidable factor (2001a, [Online]).

When asked on CNN how many of his M.E. patients had fully recovered in fifteen years, Dr Peterson unequivocally and chillingly stated, "None" (Munson 2000, p. 5).

M.E. expert [Dr Byron Hyde](#), explains that the prognosis of M.E. also differs from patient to patient depending on the degree of damage to the brain:

If the patient's illness is not measurable using a dedicated brain SPECT scan such as a Picker 3000 or equivalent, then the patient does not have M.E. For legal purposes these changes may be confirmed by PET brain scans with appropriate software and / or QEEG. These changes can be roughly characterized as to severity and probable chronicity using the following two scales: **A:** Extent of injury and **B:** degree of injury of CNS vascular function.

Extent of Injury

Type 1: One side of the cortex is involved. Those patients labeled as 1A have the best chance of recovery.

Type 2: Both sides of the cortex are involved. These patients have the least chance of spontaneous recovery.

Type 3: Both sides of the cortex, and either one or all of the following: posterior chamber organs, (the pons and cerebellum), limbic system, the subcortical and brainstem structures are involved. Type 3B are the most severely affected patients and the most likely to be progressive or demonstrate little or no improvement with time.

Degree of injury

Type A: Anatomical integrity is largely maintained in the brain SPECT scan.

Type B: Anatomical integrity is not visible in the CNS SPECT scan. Type 3B are some of the most severely and chronically injured patients (2007, [Online]).

Clearly, many people with M.E. are significantly or severely disabled. But what is so tragic about this high level of suffering is that so much of it is needless and fairly easily avoidable. So many people with M.E. are severely affected because of inappropriate medical advice or because of a lack of support. Because of the way even minor overexertion can have such a negative effect on long term prognosis M.E. really is an illness where in terms of support and care 'a stitch in time saves nine.' The correct type of support (financial, medical and practical) can do much to prevent the physical, occupational and other deterioration in the quality of life for M.E. patients and can stabilise the illness. People with M.E. desperately need to be given the same access to basic care as those with comparable illnesses, no more and no less (Dowsett 2002b, [Online]).

It is also true that many of the deaths from M.E. could have been prevented if only those patients had been given the basic level of support and care made available to patients with illnesses with comparable care needs such as multiple sclerosis and motor neurone disease. Patients with M.E. have literally died from neglect, and continue to die from neglect (in 'first world' countries such as the UK, Australia and the USA) because of the political propaganda surrounding 'CFS' and the confusion between M.E. and CFS.

People with M.E. must – as soon as possible – be given a correct diagnosis and the appropriate advice and support to ensure that they are given a chance at achieving their best possible prognosis.

- See: [The 3 Part ME Ability and Severity Scale](#) to measure illness severity over time.
- See [Treating M.E.](#) for more information on the importance of avoiding overexertion in M.E. and how to make sure your prognosis is as positive as possible. See also [Hospital or carer notes for M.E.](#) and [Why patients with severe M.E. are housebound and bedbound](#).

Conclusion

Certain groups and individuals are benefiting enormously from this fraudulent artificial 'CFS' construct.

To say that these groups and individuals always believe what they are saying and that it is based on science or reality is ridiculous. To say that it is merely a misunderstanding or a mistake is equally ridiculous. The 'CFS' construct is a complete fiction, and exists purely because it is so financially and politically beneficial to a number of powerful groups.

The artificial 'CFS' construct is no more a scientifically accurate description of M.E. than it is a scientifically accurate description of M.S., Lupus or polio. This pretence of ignorance about M.E. and about the reality of 'CFS', particularly by governments, has had devastating consequences for people with M.E. – as well as all of those with non-M.E. illnesses who are misdiagnosed as having 'CFS' – and has also meant that the number of M.E. sufferers continues to rise unabated and largely unrecognised. The general public worldwide, including sufferers themselves, has been lied to repeatedly about the reality of M.E.

M.E. can be one of the most debilitating and devastating illness there is, yet many with M.E. are subject to repeated medical abuse and neglect and are also forced to deal with extremely severe illness without the support of friends, family or the wider community or medical or government services (and indeed often they suffer abuse at the hands of these as well) because of the way the illness has been dishonestly 'marketed' to the public as being psychological or 'behavioural' in nature or as being primarily a trivial problem of mere 'fatigue.' Professor Malcolm Hooper explains that 'Wessely school' psychiatrists, and those who follow them, have:

Built their careers and reputations on denying the physical nature of M.E., with the result that untold numbers of chronically and seriously ill patients are bullied, derided, threatened and driven to suicide by being told that they are not physically ill but are suffering from "aberrant illness beliefs". WesselySchool psychiatrists have been described in the eBMJ (N Portman, 3rd December 2003) as "*a small clique of undemocratic, unaccountable, self-serving psychiatrists who have managed to monopolise most of the research funding in this field and, thanks to their prejudices, have been its downfall ever since.*" Without doubt, the influence of Simon Wessely has resulted in a cascade of horrors which most people do not know about and when they do, they find scarcely believable (2003a, [Online]).

M.E. and CFS are *not* the same. Concepts such as 'ME/CFS,' 'CFS/ME,' Myalgic 'Encephalopathy' and 'CFIDS' are also unhelpful, unscientific and only add to the obfuscation.

'CFS' is merely a scam invented by insurance companies motivated by profit without regard for truth or ethics. These groups are acting without any regard for the extreme suffering and avoidable deaths they are causing. These groups are acting criminally. The scam is tissue thin and very easily discovered if one merely takes the time to look at the evidence.

Why is almost nobody doing this? Why is the world letting these groups get away with such a heinous scam and such appalling abuse on a massive scale? Why isn't the world caring enough or smart enough or gutsy enough to see through these slick, well-funded misinformation campaigns, and to act? How can this be, when the lies are so flimsy and scientifically laughable? Have we learned nothing from the devastating corporate cover-ups of the truth about tobacco and asbestos in our recent past? Where is the World Health Organisation? Where are our human rights groups? Where is our media? Where are our uncompromising investigative journalists?

Will it take another 20 years? How much more extreme do the suffering and abuse have to be? How many more hundreds of thousands of children and adults worldwide have to be affected? How many more patients will have to die needlessly before something is finally done? How much longer will we leave the fox in charge of the hen house? It's insupportable.

Where do we go from here?

Sub-grouping different types of 'CFS,' refining the bogus 'CFS' definitions further or renaming 'CFS' with some variation on the term M.E. would achieve nothing and create yet more confusion and mistreatment. The problem is not that 'CFS' patients are being mistreated as psychiatric patients; some of those patients misdiagnosed with 'CFS' actually *do* have psychological illnesses. with CFS actually *do* have psychological illnesses. CFS is made up of people with depression (and various other psychological illnesses), multiple sclerosis, athletes over-training syndrome, Fibromyalgia, various post-viral fatigue syndromes, candida, chronic Lyme disease, burnout, cancer and many more entirely unrelated and already well-defined conditions. To say that these conditions are all subgroups of 'CFS' is just absurd. Sub-grouping 'CFS' would only waste another 20 years or more. *There is no such distinct disease/s as 'CFS'* – that is the entire issue, and the vast majority of patients misdiagnosed with CFS (an estimated 75% *at least*) *DO NOT* have M.E. and so have no more right to that term (or a variation of it) than to 'cancer' 'diabetes' or 'multiple sclerosis.'

The distinction must be made between terminology and definitions. Changing the name of the bogus disease construct of 'CFS' to some variation on the term M.E. is not at all a step in the right direction. This move will actually be a huge step BACKWARD for everyone with M.E. *and* all those patients misdiagnosed with CFS. It will make it harder than ever for anyone to distinguish between 'CFS' and authentic M.E., and for those misdiagnosed with CFS to be able to receive a correct diagnosis and treatment finally. It must not be allowed to succeed. The groups proposing this action do NOT speak for, or represent, the M.E. community.

The 'CFS name change proposal' seems nothing more than a political stunt; designed to appease (justifiably) angry patients and make them feel like something is being done and that progress is being made finally – but not to actually effect any real change. The proposal that the name of 'CFS' should be changed to a variation on the term M.E. – despite the fact that the term is completely scientifically inaccurate for the vast majority of the patients involved and that this term has already been TAKEN by a very well-defined (and scientifically sound)

patient group for over 50 years – merely because it ‘sounds a lot more serious’ makes a mockery of legitimate advocacy, and of science, logic and ethics.

- For more information see: Why the disease category of ‘CFS’ must be abandoned, Who benefits from ‘CFS’ and ‘ME/CFS’?, Problems with the so-called "Fair name" campaign: Why it is in the best interests of all patient groups involved to reject and strongly oppose this misleading and counter-productive proposal to rename ‘CFS’ as ‘ME/CFS’ and Problems with the use of ‘ME/CFS’ by M.E. advocates, See also: Problems with ‘our’ M.E. (or ‘CFS’ ‘CFIDS’ or ‘ME/CFS’ etc.) advocacy groups (also available in an animated video format.)

So how do we stop this abuse of science? Where do we go from here?

The only way forward, for the benefit of society and every patient group involved, is that:

1. The bogus disease category of ‘CFS’ must be abandoned completely.

Patients with fatigue (and other symptoms) caused by a variety of different illnesses need to be diagnosed correctly with these illnesses if they are to have any chance of recovery; not given a meaningless Oxford or Fukuda ‘CFS’ misdiagnosis. Some of the conditions commonly misdiagnosed as ‘CFS’ are very well defined and well-known illnesses and very treatable – but ONLY once they have been correctly diagnosed. Some conditions are also very serious or can even be fatal if not correctly diagnosed and managed, including M.E. (It is not uncommon for people with cancer – which causes significant fatigue – to be misdiagnosed with CFS and to die needlessly due to a lack of appropriate treatment, for example.) Each of the patient groups involved must again be correctly diagnosed and then treated as appropriate based on legitimate and unbiased science involving the SAME patient group.

Physicians who diagnose ‘CFS’ in any patient experiencing new onset fatigue without looking and testing for the true cause of the symptoms do their patients – and themselves – a great disservice. Dr Byron Hyde explains that doctors must return to the age-old medical principals of correct diagnosis (a) careful history, (b) detailed physical examination and (c) appropriate investigation (2006, [Online]).

2. The name Myalgic Encephalomyelitis must be fully restored (to the exclusion of all others) and the World Health Organization classification of M.E. (as a distinct neurological disease) must be accepted and adhered to in all official documentations and government policy.

There were sound medical reasons for the creation of the name in 1956, and for the classification of the illness by the WHO in 1969; neither of which has changed in the interim (Hyde 2006, [Online]) (Hooper 2006, [Online]) (Hyde 2003, [Online]) (Dowsett 2001a, [Online]) (Hooper et al. 2001, [Online]) (Dowsett 2000, [Online]) (Dowsett 1999a, 1999b, [Online]) (Hyde 1992 p. xi) (Hyde & Jain 1992 pp. 38 - 43). As Professor Malcolm Hooper explains:

The term myalgic encephalomyelitis was first coined by Ramsay and Richardson and has been included by the World Health Organisation (WHO) in their International Classification of Diseases (ICD), since 1969. The current version ICD-10 lists M.E. under G.93.3 - neurological conditions. It cannot be emphasised too strongly that this recognition emerged from meticulous clinical observation and examination. (2006, [Online])

Despite misleading claims made to the contrary by vested interest groups, there is well-documented evidence of inflammation of the brain and spinal cord in M.E. spanning over 50 years, *but it is true* that there is no evidence of inflammation of the brain or spinal cord in states of ‘chronic fatigue’ or ‘tiredness’ and other non-M.E. illnesses which may be commonly misdiagnosed as ‘CFS’ (Hooper n.d., [Online]) (Hyde 2006, [Online]).

M.E. is a distinct recognisable entity, with several unique features, which can be diagnosed relatively early in the course of the disease providing the physician has some experience with the illness. M.E. can easily be distinguished from various chronic fatigue states, and other unrelated ‘fatiguing’ illnesses. People with M.E. must be diagnosed with M.E. and treated for M.E., based on research which also involves M.E. patients again, finally.

The M.E. community does not need to wait for official ‘permission’ to renew the name and scientifically and historically correct definition of M.E. These rights exist today (as they have since 1969) under the WHO ICD.

3. People with M.E. must immediately stop being treated as if they are mentally ill or suffer with a behavioural illness; as if their physical symptoms do not exist or can be improved with ‘positive thinking’ and exercise, or be mixed in with various ‘fatigue’ sufferers or patients with any other illness than authentic Myalgic Encephalomyelitis.

All forms of GET, and the abusive and unscientific form of CBT, must be banned for all M.E. patients. It is illogical and unethical (and a gross violation of basic human rights) that patients be routinely subjected to

treatments which have zero chance of providing any benefit and such a high risk of serious and long-term harm, or death. People with M.E. must also be given access to basic medical care, financial support and other appropriate services (including funding for legitimate M.E. research) on an equal level to that which is available for those with comparable illnesses (e.g. M.S. or Lupus). The facts about M.E. must be taught to medical students, and included in mainstream medical journals.

Currently many physicians and most consultants (for example, cardiologists, neurologists, chest physicians, rheumatologists, immunologists) have virtually no accurate knowledge about M.E. and therefore underestimate both its seriousness and the multi-system dysfunction it causes, so patients are simply dismissed and abandoned without support. This must change (Hooper & Marshall 2005a, [Online]). The facts about M.E. must again be taught to medical students, and included in mainstream medical journals and already practicing physicians must be brought up to speed about M.E. It must be as unacceptable for physicians to be ignorant about M.E. as it would be if doctors were ignorant of the basic facts of multiple sclerosis, diabetes or any other common and serious disease. M.E. expert Dr Elizabeth Dowsett explains that:

M.E. Research workers must be encouraged and appropriately funded to work in this field. However they should first be directed to papers published before 1988, the time at which all specialised experience about poliomyelitis and associated infections seem to have vanished mysteriously! (2001a, [Online])

M.E. is a distinct infectious neurological disease of extraordinarily incapacitating dimensions that affects virtually every bodily system – not a problem of medically unexplained ‘fatigue.’ Patients with M.E. must be treated based on the scientific facts, rather than political and financial considerations.

- M.E. is a distinct, recognisable entity that can be diagnosed relatively early in the course of the disease, providing the physician has some experience with the illness. The Nightingale Definition of M.E. now also makes diagnosis easier than ever before even for those with no experience with the illness. For an explanation of some of the issues of M.E. diagnosis in more detail see: Testing for M.E.
- See On the Name M.E. for more information on the evidence for inflammation of the brain and spinal cord in M.E. and other issues surrounding the name Myalgic Encephalomyelitis.

What can you do to help?

Unlike people with HIV/AIDS, people with M.E. do not have an initial period of their illness where they are only mildly affected. M.E. is severely disabling even in the first week of illness. People with M.E. are almost all far too ill to stage protests, rallies or marches. Many with M.E. cannot even read enough to be able to understand what is happening, and are not even aware that high quality scientific information on M.E. exists and that supporting the various ‘CFS’ and ‘ME/CFS’ faux ‘advocacy’ groups is counter-productive in the extreme.

Almost all so-called patient advocacy groups worldwide have sold patients out to the highest bidder and are now actively *collaborating* with our abusers. These groups are no longer advocates for patients with M.E. – indeed they are working directly AGAINST the interest of people with M.E. These groups also do not help all those misdiagnosed with ‘CFS’, who do not have M.E. The media too has sold-out and betrayed M.E. patients.

People with M.E. have only a tiny minority of the medical, scientific, legal and other potentially supporting professions (or the public) on their side. As Dr Elizabeth Dowsett explains:

As we approach the Millennium, it has to be acknowledged that the struggle for recognition of M.E. as a serious disabling organic disease with significant requirements for medical social, educational, and financial support has (due to media manipulation of public opinion) entered the realm of politics rather than the more desirable one of basic science (1999b, [Online]).

The Committee for Justice and Recognition of M.E. explains:

There is no immunity to M.E. The next victim of this horrible disease could be your sister, your friend, your brother, your grandchildren, your neighbour [or] your co-worker. M.E. is an infectious disease that has become a widespread epidemic that is not going away. We must join together, alert the public and demand action (2007, [Online]).

That is what is needed – people power. Educated people power. for people from all over the world to stand up for M.E. individual physicians, journalists, politicians, human rights campaigners, patients, families and friends of patients and the public, whether they are affected yet by M.E. or not, must stand up for the truth. That is the only way change will occur- through education and people simply refusing to accept what is happening any more.

Yes, there are powerful and immensely wealthy vested interest groups out there, who will fight the truth every step of the way, but we have science, reality and ethics on our side and those are also very powerful. However, for this to be of any use to us, we must first make ourselves aware of the facts *and then use them*.

So what you can do to help is to PLEASE spread the truth about M.E. and try to expose the lie of 'CFS.' You can also help by NOT supporting the bogus concepts of 'CFS,' 'ME/CFS,' 'subgroups of ME/CFS,' 'CFS/ME,' 'CFIDS' and Myalgic 'Encephalopathy.' Do not support groups which promote these concepts. Do not give public or financial help to our abusers.

The abuse and neglect of so many seriously ill people on such an industrial scale is truly *inhumane* and has already gone on for far too long.

People with M.E. desperately need your help.

More information:

- For more information about the medical and political facts of M.E. see: [Who benefits from 'CFS' and 'ME/CFS'?](#), [Problems with the so-called "Fair name" campaign: Why it is in the best interests of all patient groups involved to reject and strongly oppose this misleading and counter-productive proposal to rename 'CFS' as 'ME/CFS' and Problems with the use of 'ME/CFS' by M.E. advocates.](#) Plus also [The misdiagnosis of CFS](#), [Smoke and mirrors](#), [M.E. The Medical Facts - Extended](#), [The Ultra-comprehensive M.E. Symptom List](#), [Testing for M.E.](#) and [Putting research and articles into context](#).
- See also: [A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic Fatigue Syndrome & A Brief History of Myalgic Encephalomyelitis & An Irreverent History of Chronic Fatigue Syndrome and The Complexities of Diagnosis by Dr Byron Hyde](#) Dr Byron Hyde is the world's pre-eminent ME authority. Dr Hyde's latest paper is also a MUST-READ for those with a real interest in Myalgic Encephalomyelitis: [The Nightingale Definition of M.E.](#)
- More essential reading on M.E. for those with a real interest in M.E. includes: [What is ME? What is CFS? Information for Clinicians & Lawyers](#) by Eileen Marshall, Margaret Williams & Professor Malcolm Hooper, [The Mental Health Movement: Persecution of Patients?](#) by Professor Malcolm Hooper, [Research into ME 1988 - 1998 Too much PHILOSOPHY and too little BASIC SCIENCE!](#) and [The Late Effects Of M.E. and A Rose by Any Other Name and Redefinitions of ME - a 20th Century Phenomenon](#) by Dr Elizabeth Dowsett, [Illustrations of Clinical Observations and International Research Findings from 1955 to 2005 that demonstrate the organic aetiology of Myalgic Encephalomyelitis \(174 pages\)](#) by Eileen Marshall, Margaret Williams & Professor Malcolm Hooper, [Worldwide Epidemic:an ALERT to citizens worldwide](#) and; [ME and CFS, the Definitions from the Committee for Justice and Recognition of M.E.](#)
- To read a list of all the articles on this site suitable for different groups such as M.E. patients, carers, friends and family, the 'CFS' misdiagnosed, doctors or severe M.E. patients and so on, see the [Information Guides](#) page.

Acknowledgments

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References

All of the information concerning Myalgic Encephalomyelitis on this website is fully referenced and has been compiled using the highest quality resources available, produced by the world's leading M.E. experts.

More experienced and more knowledgeable M.E. experts than these – [Dr Byron Hyde](#) and [Dr. Elizabeth Dowsett](#) in particular – do not exist. Between Dr Byron Hyde and Dr. Elizabeth Dowsett, and their mentors the late Dr John Richardson and Dr Melvin Ramsay (respectively), these four doctors have been involved with M.E. research and M.E. patients for well over 100 years collectively, from the 1950s to the present day. Between them they have examined more than 15 000 individual (sporadic and epidemic) M.E. patients, as well as each authoring numerous studies and articles on M.E., and books (or chapters in books) about M.E. Again, more experienced, more knowledgeable and more credible M.E. experts than these simply do not exist.

This paper is intended to provide a brief summary of the most important facts of M.E. It has been created for the benefit of those people without the time, inclination or ability to read each of the far more detailed and lengthy references created by the world's leading M.E. experts. The original documents used to create this paper are essential additional reading, however, for any physician (or anyone else) with a real interest in M.E. For a full reference list please see the [References](#) page.

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Before reading any of the above links to research/advocacy information, please be aware of the following facts:

1. Myalgic Encephalomyelitis (M.E.) and 'Chronic Fatigue Syndrome' (CFS) are not synonymous terms. The overwhelming majority of research on 'CFS' or 'CFIDS' or 'ME/CFS' or 'CFS/ME' or 'ICD-CFS' does not involve M.E. patients and is not relevant in any way to M.E. patients. If the M.E. community was to reject all 'CFS' labelled research as 'only relating to 'CFS' patients' (including research which describes those abnormalities/characteristics unique to M.E. patients), however, this would seem to support the myth that 'CFS' is just a 'watered down' definition of M.E. and that M.E. and 'CFS' are virtually the same thing and share many characteristics.

A very small number of 'CFS' studies/articles and books refer in part to people with M.E., but it may not always be clear which parts refer to M.E. The [A warning on 'CFS' and 'ME/CFS' research and advocacy](#) paper is recommended reading and includes a checklist to help readers assess the relevance of individual 'CFS' studies (etc.) to M.E. (if any) and explains some of the problems with this heterogeneous and skewed research.

In future, it is essential that M.E. research again be conducted using only M.E. defined patients and using only the term M.E. The bogus, financially-motivated disease category of 'CFS' must be abandoned.

The research referred to on this website varies considerably in quality. Some is of a high scientific standard and relates wholly to M.E. and uses the correct terminology;. Other studies are included which may only have partial or minor possible relevance to M.E., use unscientific terms/concepts such as 'CFS,' 'ME/CFS,' 'CFS/ME,' 'CFIDS' or Myalgic 'Encephalopathy' and also include a significant amount of misinformation. Before reading this research it is also essential that the reader be aware of the most commonly used 'CFS' propaganda, as explained in [A warning on 'CFS' and 'ME/CFS' research and advocacy](#) and in more detail in [Putting Research and Articles on M.E. into Context](#).

Note that this list may contain some references which are not directly referenced in this paper (as this list also serves as a reference list for several other papers).

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Relevant quotes

'The problem with fatigue is that it is neither specific, definable nor scientifically measurable. Fatigue is both a normal and a pathological feature of every day life. Every normal person gets fatigued. Fatigue is a common feature of much major psychiatric disease and major medical disease. Since fatigue is such an integral part of many illnesses, by calling fatigue the primary characteristic, the authors necessitated the elimination of hundreds of other diseases. To truly follow the criteria set out by the CDC definition probably makes 'CFS' the most expensive illness to investigate of any known disease. Fatigue is not an object, it is simply a modifier in search of a noun. Also, taking fatigue as the flagship symptom of a disease not only bestows the disease with a certain Rip Van Winkle humour, but it removes the urgency of the fact that the majority of M.E. symptoms are in effect CNS symptoms. M.E. represents a major attack on the CNS by the chronic effects of a viral infection.'

BYRON HYDE MD IN 'THE CLINICAL AND SCIENTIFIC BASIS OF M.E. P 11-12

'Western newspapers and magazines are packed with trivia, television news is concealing the reality of what is happening... and investigative journalism has virtually died a death. [But] what is the point of democracy if you keep the citizens in a state of semi-ignorance?'

VETERAN ACTIVIST, PROTESTER AND AUTHOR TARIQ ALI

'Despite the claims of some psychiatrists, it is not true that there is no evidence of inflammation of the brain and spinal cord in M.E.; there is, but these psychiatrists ignore or deny that evidence. It is true that there is no evidence of inflammation of the brain or spinal cord in states of chronic fatigue or 'tiredness.'

THE TERMINOLOGY OF M.E. & CFS BY PROFESSOR MALCOLM HOOPER

'There is a principle which is a bar against all information, which is proof against all argument, and which cannot fail to keep man in everlasting ignorance. That principle is condemnation without investigation.'

WILLIAM PALEY (1743-1805)

This paper will continue to be updated regularly-at least annually. Please check back at the website periodically to make sure that you have the most up-to-date version of this paper.

The HUMMINGBIRDS' FOUNDATION for M.E. (HFME)

Fighting for the recognition of Myalgic Encephalomyelitis based on the available scientific evidence, and for patients worldwide to be treated appropriately and accorded the same basic human rights as those with similar disabling and potentially fatal neurological diseases such as Multiple Sclerosis.

A one-page summary of the facts of M.E.

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- Myalgic Encephalomyelitis (M.E.) is a disabling neurological disease that is very similar to Multiple Sclerosis (M.S.) and Poliomyelitis. Earlier names for M.E. were 'atypical Multiple Sclerosis' and 'atypical Polio.'
- M.E. is a neurological disease characterised by scientifically measurable post-encephalitic damage to the brain stem. This damage is an essential part of M.E., hence the name M.E. The term M.E. was coined in 1956 and means: my = muscle, algic = pain, encephalo = brain, mye = spinal cord, tis = inflammation. This neurological damage has been confirmed in autopsies of M.E. patients.
- Myalgic Encephalomyelitis has been recognised by the World Health Organisation's International Classification of Diseases since 1969 as a distinct organic neurological disease. M.E. is classified in the current WHO International Classification of Diseases with the neurological code G.93.3.
- M.E. is primarily neurological, but also involves cognitive, cardiac, cardiovascular, immunological, endocrinological, metabolic, respiratory, hormonal, gastrointestinal and musculo-skeletal dysfunctions and damage. M.E. affects all vital bodily systems and causes an inability to maintain bodily homeostasis. More than 64 individual symptoms of M.E. have been scientifically documented.
- M.E. is an acute (sudden) onset, infectious neurological disease caused by a virus (a virus with a 4-7 day incubation period). M.E. occurs in epidemics as well as sporadically and over 60 M.E. outbreaks have been recorded worldwide since 1934. There is ample evidence that M.E. is caused by the same type of virus that causes Polio; an enterovirus.
- M.E. can be more disabling than M.S. or Polio, and many other serious diseases. M.E. is one of the most disabling diseases that exists. More than 30% of M.E. patients are housebound, wheelchair-reliant and/or bedbound and are severely limited with even basic movement and communication.
- *Why are M.E. patients so severely and uniquely disabled?* For a person to stay alive, the heart must pump a certain base-level amount of blood. Every time a person is active, this increases the amount of blood the heart needs to pump. Every movement made or second spent upright, every word spoken, every thought thought, every word read or noise heard requires that more blood must be pumped by the heart.
However, the hearts of M.E. patients only pump barely enough blood for them to stay alive. Their circulating blood volume is reduced by up to 50%. Thus M.E. patients are severely limited in physical, cognitive and orthostatic (being upright) exertion and sensory input.
This problem of reduced circulating blood volume, leading to cardiac insufficiency, is why every brief period spent walking or sitting, every conversation and every exposure to light or noise can affect M.E. patients so profoundly. Seemingly minor 'activities' can cause significantly increased symptom severity and/or disability (often with a 48-72 hour delay in onset), prolonged relapse lasting months, years or longer, permanent bodily damage (e.g. heart damage or organ failure), disease progression or death.
If activity levels exceed cardiac output by even 1%, death occurs. Thus the activity levels of M.E. patients must remain strictly within the limits of their reduced cardiac output just in order for them to stay alive. *M.E. patients who are able to rest appropriately and avoid severe or prolonged overexertion have repeatedly been shown to have the most positive long-term prognosis.*
- M.E. is a testable and scientifically measurable disease with several unique features that is not difficult to diagnose (within just a few weeks of onset) using a series of objective tests (e.g. MRI and SPECT brain scans). Abnormalities are also visible on physical exam in M.E.
- M.E. is a long-term/lifelong neurological disease that affects more than one million adults and children worldwide. In some cases M.E. is fatal. (Causes of death in M.E. include heart failure.)

For more information, and to read a fully-referenced version of this text compiled using information from the world's leading M.E. experts, please see: What is M.E.? Extra extended version. Permission is given for this unedited document to be freely redistributed. Please redistribute this text widely.