- It also invalidates patient experience, obstructs biomedical care, and distorts epidemiological data, hindering research and policy progress.
- The conflation contravenes **ICD diagnostic codes**, **NICE guidelines (NG206, 2021)**, and **Montgomery informed consent law** if patients are not told that ME has proven biomedical abnormalities.

Ethical and Legal Distinction

- Misdiagnosing ME as FND **breaches clinical duty of candour** and may violate disability rights by denying recognition of a neurological illness. Mislabelling ME as FND denies patients appropriate care and harms them.
- Biological abnormalities are ignored.
- It also undermines **informed consent**, as patients are not informed that the "functional" label is disputed and contradicted by international biomedical evidence (IOM, 2015; NICE, 2021).
- Exercise and psychotherapy are wrongly prescribed.
- Severely affected patients risk deterioration, starvation, and death.

Why Misdiagnosis Harms Patients

Misdiagnosis is not harmless.

Rebranding a neurological illness as psychiatric leads to real suffering, neglect, and institutional abuse.

Summary:

ME is Neurological, not Psychiatric (WHO G93.3)
ME Has Proven Biological Abnormalities
FND Relies on Inconsistent Symptoms - ME Is Consistent and Measurable
Exercise Harms ME Patients - but Is Prescribed in FND
Misdiagnosis Is Dangerous and Violates Patient Rights

Know the Difference. Protect Your Rights.

Historically, medicine erroneously psychologised **serious physical diseases**, especially those that predominantly affect women, **before a biomarker was discovered**. MS was dismissed as '**hysteria'**, as **FND** was then known. Today, **ME** faces similar prejudice when it is wrongly recategorised as **FND**.

This is a **serious diagnostic error**.

- **ME is a biomedical, measurable, neurological disease** involving immune, vascular, and mitochondrial dysfunction.
- **FND is a psychiatric construct** defined by *inconsistency of symptoms* and the *absence of structural pathology*.

Labelling ME as FND erases decades of biomedical research, exposes patients to **harmful psychological or exercise-based treatments**, and denies them **appropriate medical care** for a proven multi-system disorder.

ME is not FND.
It is a distinct, physiological disease, and patients deserve to have it recognised as such.

Refs:

Myalgic encephalomyelitis: diagnosis and management: NICE guideline, Published: 29 October 2021, https://www.nice.org.uk/guidance/ng206

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2. Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: the biology of a neglected disease. Arron et al, 2024.

3. De Bellis, et al Involvement of hypothalamus autoimmunity in patients with autoimmune hypopituitarism: role of antibodies to hypothalamic cells, 2012

4.Functional (psychogenic)movement disorders: merging mind and brain, Mark J Edwards, 2012

Why ME is *NOT* FND

By Jenny Wilson

Associate Member of Doctors with ME.

A guide for patients

Many people with **Myalgic Encephalomyelitis (ME)** are being told their illness is **'Functional Neurological Disorder (FND)'** - a psychological condition ***unrelated to biomedical pathology***

This leaflet explains **why ME is not FND**, and why confusing the two is harmful.

What is ME?

ME is a **neurological and immunoendocrine disease**, recognised by the **World Health Organisation (ICD code G93.3)** since 1969.

Myalgic Encephalomyelitis (ME), also referred to as ME/CFS, is a serious, multisystem, potentially fatal disease. Over 10,000 scientific papers provide evidence that its symptoms arise from widespread dysfunction of the immune, neurological, muscular-skeletal, cardiovascular, gastrointestinal, and endocrine systems. (Naviaux 2019; Wirth & Scheibenbogen 2020; Phair 2022).

The hallmark symptom of ME is Post-Exertional Malaise (PEM), sometimes called Post-Exertional Symptom Exacerbation (PESE). This is a delayed worsening of symptoms following even minor physical, cognitive, or emotional effort that exceeds the patient's very low anaerobic threshold (Systrom 2017; VanNess 2010; Snell 2013).

Published papers show that PEM reflects a metabolic, vascular, and neuroimmune collapse triggered by exertion. Underlying mechanisms include:

- Impaired oxygen delivery to muscles and brain
- Mitochondrial energy failure
- Ionic imbalance and autonomic dysfunction
- Cerebral hypoperfusion and widespread neuroinflammation (Younger, 2024)
- Renin-angiotensin-aldosterone system (RAAS) suppression and severe hypovolemia

- Dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis, which is a potential cause of hypovolaemia (Demitrack 1991, De Bellis, 2021)
- Autoantibodies to the alpha and beta adrenergic and muscarinic receptors. (Scheibenbogen, 2020)
- Neuroinflammatory flare and cerebral/muscular hypoxia (Younger, 2024, Wirth, 2021)

Each episode of PEM can worsen the underlying disease, leaving the patient with even less capacity for activity. Recovery requires strict rest and avoidance of overexertion until vascular and mitochondrial stability return.

Prof. Carmen Scheibenbogen describes ME as an *Acquired Ischaemic Mitochondrial Myopathy* (AIMM), characterised by endothelial dysfunction, neuroinflammation, and bioenergetic collapse (Scheibenbogen, 2021). In simple terms, ME is a systemic disorder of impaired energy delivery caused by microvascular, autonomic, and mitochondrial failure.

ME inflicts profound and often lifelong suffering. It is recognised as one of the most life-destroying diseases, can be potentially fatal, and is listed by coroners as a cause of death in some cases. Autopsies, though rare, have revealed inflamed dorsal root ganglia and cardiac fibrosis even in young patients.

What is FND?

- **FND (Functional Neurological Disorder),** is diagnosed when neurological symptoms lack an identifiable structural lesion (DSM-5, 2013; Edwards et al., 2012). It replaces the term 'Conversion Disorder', which replaced 'hysteria'.
- FND is claimed to arise from **abnormal brain signalling**, not multi-system dysfunction. Treatments focus on **neurological rehabilitation and symptom retraining**, not metabolic or vascular correction.
- FND diagnosis rests on **'positive signs of inconsistency'** (e.g., Hoover's sign), not on a disease process; ME diagnosis rests on consistent, proven **post-exertional pathophysiology**.

FND's Key differences from ME:

- No post-exertional malaise
- No mitochondrial or energy collapse
- No systemic immune or vascular dysfunction

Historical and Nosological Distinction

- ME was first defined by the World Health Organisation (ICD-8, 1969) as a neurological disease (ICD code 323) - decades before 'FND' was coined.
- **FND**, by contrast, appears under **ICD-10 code F44**, in the *psychiatric* section (dissociative/conversion disorders).
- Thus, the WHO explicitly distinguishes **ME (neurological)** from **FND (psychiatric)**. → Rebranding ME as FND therefore constitutes **diagnostic misclassification**, not updated science.
- Reclassifying ME as FND **violates diagnostic standards** and can breach **Montgomery informed consent** if patients are not told the biomedical evidence.
- The **NICE guideline NG206 (2021)** confirms ME/CFS is a **chronic medical illness**, not a functional disorder.

Exercise Intolerance vs. Psychogenic Weakness

- In ME, **two-day cardiopulmonary exercise testing (CPET)** consistently shows a **reduction in oxygen uptake and workload capacity on the second day**, unique to ME/CFS (Snell et al., 2013).
- This is **objective physiological evidence of exertion-induced metabolic dysfunction**, not inconsistency or voluntary effort - key diagnostic features of FND.
- FND, by contrast, does **not** produce reproducible physiological failure on exertion testing.

FND Model Fails to Explain ME Research Findings

- FND theories centre on faulty neural prediction or

attention bias in motor/sensory processing (Edwards et al., 2012).

• ME abnormalities - such as mitochondrial fragmentation, autoantibodies to β2-adrenergic and muscarinic receptors, and impaired pyruvate dehydrogenase function - cannot be explained by misdirected attention.

• Nor can reduced total blood volume or cerebral hypoperfusion (Systrom et al., 2022; Van Campen et al., 2020) be psychogenic phenomena.

Different Pathophysiological Mechanisms

ME→ Core Mechanisms: Post-viral immune dysregulation, impaired energy metabolism, neuroinflammation, autonomic and vascular dysfunction, severe hypovolaemia, autoantibodies to adrenergic and muscarinic receptors. **Triggers:** → Viral infections (EBV, enterovirus, SARS-CoV-2), vaccination, toxin exposure. **Treatment:** Pacing, symptom management, autonomic and immune support. **Response to Exercise:** → Physiological relapse, and worsening/deterioration. **Immune Dysfunction:** → chronic immune activation, autoantibodies, IgM/fibronectin dysregulation.

FND:→ Core Mechanisms: Abnormal brain network functioning related to perception of movement and emotion regulation. **Triggers:** → Emotional stress, trauma, acute illness, non-specific. **Treatment:** →Psychotherapy, physiotherapy, retraining "maladaptive brain patterns". **Response to Exercise:** → Often encouraged as exposure therapy. **Immune Dysfunction:** No systemic immune dysfunction

Summary: ME and FND are opposites:

ME is consistent, measurable, and physiological.
FND is inconsistent, perceptual, and psychological.

→ They are diagnostically and mechanistically incompatible.

Clinical Consequences of Misdiagnosis

- Reclassifying ME as FND leads to **inappropriate behavioural treatment** (exercise, CBT) that can exacerbate disease, sometimes severely (NICE, 2021).