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Uncertainty-Driven Healthcare Epistemic Injustice: Evidence from Chronic Fatigue Syndrome

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Author's Declaration

I declare that this thesis is a presentation of original work and I am the sole author. This work has not previously been presented for an award at this, or any other, University. All sources are acknowledged as References.

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Abstract. In this thesis, I critique inappropriate and unhelpful responses to radical epistemic uncertainties in medicine, which, I argue, significantly contribute to epistemic injustice in some healthcare encounters. This uncertainty-driven explanation for healthcare epistemic injustice is additional to explanations already identified by other commentators in the literature; that is to say, the imbalance of power between patients and healthcare professionals, the effects of the differences in their phenomenological and professional perspectives, and prejudice.

In order to illustrate this critique and this argument, I use the example of the disputed, stigmatising, and inadequately-understood medical condition(s), chronic fatigue syndrome (CFS) and myalgic encephalomyopathy (ME) (which are often conflated as CFS/ME or ME/CFS), and similar medical conditions, about which there is uncertainty about causes and pathological mechanisms, where there are no curative treatments, and where there are disputes about the effectiveness or otherwise of symptomatic treatment recommendations. Many CFS patients and some of their healthcare professionals have dichotomous and oppositional responses to those uncertainties. These patients declare that their illness is biomedical (BMM), because of their phenomenological experience; therefore, psychological and behavioural treatments are inappropriate and harmful. Contrastingly, some health professionals see that the absence of objective evidence of disease in CFS patients implies that there is no disease process, and that treatments should be biopsychosocial (BPS). I argue that these conflicting responses, along with the rejection of opposing responses, lead to disputes and healthcare epistemic injustice within CFS healthcare encounters regarding conceptualisations of CFS and treatments recommendations for the symptoms.

In this thesis, I show that CFS is indeed subject to radical epistemic uncertainties, which explain those differing responses. I argue that each response is not unreasonable, bearing in mind those uncertainties; however, I also argue that the rejection of the opposing response is not epistemically virtuous.

The methodology that I use in this thesis is the logical analysis of the evidence for explanations for healthcare epistemic uncertainty, with the examination of evidence to support my argument that healthcare epistemic injustice can be significantly exacerbated by responses to epistemic uncertainty.

For amelioration, I propose two alternative conceptualisations for CFS, the network of symptoms model and the persistent physical symptoms model, each of which is additional rather than oppositional to both BMM and BPS.

In order to support my arguments, I introduce, promote, or find novel applications for, a number of concepts that are insufficiently highlighted elsewhere in the literature: *firstly*, the applicability of the economists' concept of radical uncertainty to medical knowledge; *secondly*, the existence of radical uncertainty regarding knowledge about some medical conditions; *thirdly*, a network of uncertainties hampering some medical research; *fourthly*, the distinction between uncertainty regarding a medical condition and uncertainty regarding the medical problems of a particular individual in a healthcare encounter; *fifthly* radical uncertainty as an explanation for some healthcare epistemic injustice; *sixthly*, the applicability of the concepts of epistemic virtue to responses to medical uncertainty; *seventhly*, the under-acknowledged persistence of inappropriate medical disease dualism; *eighthly*, the applicability of the network of symptoms model to non-psychiatric illness; and,

ninthly, the applicability of the persistent physical symptoms model of illness to under-explained medical conditions. These concepts have wider applicability to medical epistemology, disease ontology, healthcare ethics, and healthcare epistemic justice, thus demonstrating how the philosophical study of one medical condition (and similar conditions) can contribute to the philosophy of medicine, and some ways in which philosophy of medicine can benefit the practice of medicine.

(Word count of this Abstract – 565)

Key words and phrases: chronic fatigue syndrome; epistemic injustice; epistemic pluralism; epistemic vice; epistemic virtue; deleterious uncertainty; healthcare epistemic injustice; myalgic encephalomyelitis; myalgic encephalomyopathy; network of symptoms; network of uncertainties; pathocentric epistemic injustice; radical uncertainty; unresolvable uncertainty; uncertainty-driven healthcare epistemic injustice.

Contents

Title page	1
Author's declaration	2
Acknowledgements	3
Abstract	4
Key words and phrases	6
Contents	7
List of tables	10
List of figures	11
Abbreviations	

Chapter 1: Introduction, Preliminaries, and Outline

1.1: Introduction to This Thesis	13
1.2: The Aim of This Thesis	14
1.3: Summary of the Arguments in This Thesis	15
1.4: Scope, Time Scale, Methodology, Sources, and Theoretical Frameworks	27
1.5: Terminology	32
1.6: Caveats and Limitations	36
1.7: Outline of the Chapters in this Thesis.	43

Chapter 2: Radical Uncertainty, and Uncertainties in Healthcare

2.1: Introduction to Chapter Two	49
2.2: Concepts of Uncertainty and Radical Uncertainty	50
2.3: Uncertainties in Healthcare	63
2.4: Certainties and Uncertainties in Myxoedema and in Charles Bonnet Syndrome	74
2.5: Conclusion to Chapter Two	83

Chapter 3: Chronic Fatigue Syndrome, and its Uncertainties

3.1: Introduction to Chapter Three	85
3.2: The Symptoms of Chronic Fatigue Syndrome, and Controversies about it	86
3.3: The Areas of Uncertainty in CFS	90
3.3 (a): Introduction to Section Three	90
3.3 (b): The problem of differing definitions for chronic fatigue syndrome	90

3.3 (c): Ontological challenges to chronic fatigue syndrome	105
3.3 (d): Uncertainties about CFS/ME are reflected in the differing names for the condition	112
3.3 (e): The problem of demarcation of chronic fatigue syndrome	123
3.3 (f): The possibility of subgroups in chronic fatigue syndrome	124
3.3 (g): Uncertainty concerning the causes of chronic fatigue syndrome	126
3.3 (h): Uncertainty about pathological mechanisms of chronic fatigue syndrome	128
3.3 (i): Uncertainties regarding the role of psychological processes in chronic fatigue syndrome	130
3.3 (j): The problem of a lack of adequate progress of research into chronic fatigue syndrome	131
3.4: The Interactions Between the Differing Areas of Uncertainty in Chronic Fatigue Syndrome.	136
3.5: The Recognition of Radical Uncertainty in Chronic Fatigue Syndrome	138
3.6: Conclusion to Chapter Three, with Responses to Some Counter-Claims	145
Chapter 4 The Biomedical and Biopsychosocial Models, and their Dichotomous Applications to Chronic Fatigue Syndrome and Its Treatments	
4.1: Introduction to Chapter Four	149
4.2: The Biomedical and the Biopsychosocial Models	150
4.3: Critiques of the Biopsychosocial Model and Its Applications	157
4.4: The Dichotomous Applications of the Biomedical and the Biopsychosocial Models to Chronic Fatigue Syndrome	177
4.5: Conclusion to Chapter Four	201
Chapter 5: Stigma and Chronic Fatigue Syndrome, and ‘Medical Disease Dualism’	
5.1: Introduction to Chapter Five	203
5.2: Evidence for Stigma in Chronic Fatigue Syndrome	204
5.3: Psychiatric Attributions as Explanations for Stigma in CFS	206
5.4: The Inappropriate Physical/Psychological Disease Dichotomy	212
5.5: Conclusion to Chapter Five, and Responses to Counter-Claims	215
Chapter 6: Epistemic Injustice in Healthcare, and in Chronic Fatigue Syndrome	
6.1: Introduction to Chapter Six	218
6.2: Epistemic Injustice	219
6.3: Healthcare Epistemic Injustice	224
6.4: Healthcare Epistemic Injustice in Chronic Fatigue Syndrome and Similar Conditions	231

6.5: ‘Suzanne’s’ Explanations for Epistemic Injustice in Chronic Fatigue Healthcare Encounters	235
6.6: Healthcare Epistemic Injustice: The Perspectives of Health Care Professionals	247
6.7: the Central Argument of this Thesis	257
6.7 (a): Radical Uncertainty about Chronic Fatigue Syndrome Leads to Dichotomies of Views	257
6.7 (b): Oppositional Dichotomies Contribute to Epistemic Injustice in Chronic Fatigue Syndrome Healthcare Encounters	259
6.7 (c): Uncertainty Plus Power Differentials Leads to Uncertainty-Driven Healthcare Injustice	261
6.8 Conclusion to Chapter Six with Responses to Possible Counter-Claims	263
Chapter 7: Responses to the Problems of Chronic Fatigue Syndrome.	
7.1: Introduction to Chapter Seven	266
7.2: Generic Routes to the Amelioration of Healthcare Epistemic Injustice	267
7.3: Some Routes to the Amelioration of Healthcare Epistemic Injustice in Chronic Fatigue Syndrome	270
7.4: Standard Disease Models	277
7.5: The Network of Symptoms Model	282
7.6: Applying Network Models to Chronic Fatigue Syndrome	292
7.7: De-emphasising Disease Mechanisms	298
7.8: Conclusion to Chapter Seven	303
Chapter Eight: Summary, Conclusion, and Applications.	
8.1: Introduction to Chapter Eight	305
8.2: Summary of This Thesis	305
8.3: Summary of the Concepts in this Thesis, and The Values and Applicabilities of Those Concepts.	310
8.4: Summary of My Critiques of Others’ Contributions to the Debates Concerning Chronic Fatigue Syndrome	315
8.5: Summary of the Contributions of This Thesis, and Recommendations for Future Study	318
Appendix	
The Testimony of ‘Suzanne’	320
References and Bibliography	325

List of Tables

Table One: Five Differing Definitions for ME/CFS, CFS/ME, and ME	94-6
Table Two: Survey of the views of ME organisations and medical authorities	180-1

List of figures

Figure One:	Diagrammatic Representation of the Definitions of CFS, ME, ME/CFS, and SEID	99
Figure Two:	The Bow Tie Model of Causes and Effects	277-8
Figure Three:	A symptom network of four symptoms	284
Figure Four:	A dynamic interactive network diagram for insomnia	288-9
Figure Five:	Diagram of the CFS association model	293
Figure Six:	PowerPoint Stock Diagram	296

Abbreviations

APT:	Adaptive pacing therapy
BME:	Benign myalgic encephalomyelitis
BMM:	Biomedical model of disease or illness
BPS:	Biopsychosocial model (or conceptualisation) of disease or illness
(B)PS:	A psychosocial conceptualisation of disease or illness that appears to play down the relevance of biomedical processes
BPSM:	Biopsychosocial model (or conceptualisation) of disease or illness
CLD:	Chronic Lyme disease
CBS:	Charles Bonnet syndrome
CBT:	Cognitive behaviour therapy
CFS:	Chronic fatigue syndrome
CFS/ME:	Portmanteau term that suggests that these may be the same condition.
EBP:	Evidence-based practice
EI:	Epistemic injustice
GET:	Graded exercise therapy
HCP:	Healthcare professional
HEI:	Healthcare epistemic injustice
MDD:	‘Medical disease dualism’
ME:	Myalgic encephalomyopathy or myalgic encephalomyelitis
ME/CFS:	Portmanteau term that suggests that these may be the same condition.
MS:	Multiple sclerosis
MUS:	Medically unexplained symptoms
PEM:	Post-exertional malaise
PPS:	Persistent physical symptoms
RA:	Rheumatoid arthritis
RU:	Radical uncertainty
SEID:	Systemic exertion intolerance disease
SLE:	Systemic lupus erythematosus
UHEI:	Uncertainty-driven healthcare epistemic injustice

Uncertainty-Driven Healthcare Epistemic Injustice:

Evidence from Chronic Fatigue Syndrome

1: Introduction, Preliminaries, and Outline

1.1: Introduction to This Thesis

In this thesis, I critique inappropriate and unhelpful responses to radical epistemic uncertainties in medicine, which, I argue, significantly contribute to epistemic injustice in some healthcare encounters. This uncertainty-driven explanation for healthcare epistemic injustice is additional to explanations already identified by other commentators in the literature: that is to say, the imbalance of power between patients and healthcare professionals and the effects of the differences in their phenomenological and professional perspectives. To support my argument, I show that radical uncertainty exists in some aspects of healthcare, and that oppositional responses to this uncertainty are indeed significant explanations for epistemic injustice in some healthcare encounters. I use this analysis of these explanations to propose routes to amelioration of uncertainty-driven healthcare epistemic injustice.

In order to illustrate these critiques and lines of arguments, I use the example of the disputed and inadequately understood medical condition(s) known as chronic fatigue syndrome (CFS), myalgic encephalomyopathy (ME), or myalgic encephalomyelitis (also abbreviated to ME) (which are often conflated as CFS/ME or ME/CFS), along with similar conditions.

In this Introduction, Outline, and Preliminaries Chapter (which incorporates a literature review), I describe the aim of this thesis and a summary of my argument. I then summarise the thesis's scope, methodology, sources, and theoretical framework. I provide a definition of terms, including which terminology I prefer to use and which I prefer to avoid, along with the reasons for my preferences. I emphasise the reality of CFS/ME patients' symptoms. I accept that the inadequacy of our present knowledge about CFS/ME may be temporary. I introduce some caveats regarding the scope of this thesis, and some limitation to my conclusions. I then give an outline of the Chapters in this thesis.

1.2: The Aim of this Thesis

The **aim** of this thesis is, firstly, to give uncertainty-related explanations for epistemic injustice (EI) in some healthcare encounters, particularly explanations of how and why health care professionals (HCPs) are persistently perceived to be the agents of epistemic injustice in some healthcare encounters (Blease *et al.* 2017; Spandler & Allen 2018; Byrne 2020; Byrne 2021; Hunt *et al.* 2024), despite their training, the precepts of medical ethics (Beauchamp & Childress 2019; Della Croce 2023), the stipulations of regulating authorities such as the General Medical Council (General Medical Council, Good Medical Practice), and what I perceive as the good intentions of these medical professionals. My own background as a family doctor gives me some insight into the perspectives of other medical professionals. Secondly, I aim to show ways to ameliorate epistemic injustice in those healthcare encounters where there is significant uncertainty.

I concentrate on chronic fatigue syndrome and similar medical conditions, firstly, because there are many reports of unsatisfactory healthcare encounters concerning CFS (L. Cooper 1997; Deale & Wessely 2001; Page & Wessely 2003; Tucker 2004; Dickson *et al.* 2007; Arroll & Senior 2008; Guise *et al.* 2010; Horton *et al.* 2010; Bayliss *et al.* 2014; Marks *et al.* 2016; Blease *et al.* 2017; Lian & Robson 2017; Byrne 2020; Diserholt 2020; Franssen 2020; Fennel *et al.* 2021; Hattrick 2021; Aaron *et al.* 2024; ‘Suzanne’ 2024; Ali & Kheirabadi 2025), and, secondly, because chronic fatigue syndrome is subject to significant uncertainty, as are similar conditions (Cooper, L 1997; White *et al.* 2011; Arroll & Senior 2008; Karfakis 2018; Cortes Rivera *et al.* 2019; Harvey & Wessely 2009; Blease *et al.* 2017; Richman *et al.* 2019; Diserholt 2020; Lidbury & Fisher 2020; Fatt Scott 2021; Noor *et al.* 2021; Walitt *et al.* 2024).

1.3: Summary of the Arguments in This Thesis

I argue in this thesis for an explanatory connection between healthcare epistemic injustice and differing responses to epistemic uncertainty. In cases where there is epistemic uncertainty about some medical conditions, that uncertainty can lead to oppositional dichotomies of understanding between patients and HCPs about the nature of patients’ disease processes, and corresponding oppositional dichotomies concerning which treatments should or should not be recommended. I show how those oppositions result in unsatisfactory healthcare encounters which can be categorised as being epistemically unjust. I then argue that epistemic virtue indicates the inappropriateness of the rejection of opposing responses to that uncertainty.

Therefore, I emphasise an under-recognised explanation for epistemic injustice in healthcare that I call '*Uncertainty-Driven Healthcare Epistemic Injustice*' (UHEI). UHEI incorporates healthcare epistemic injustice (HEI), the already-recognised injustice in some healthcare encounters (Carel & Kidd 2014; Carel 2016; Kidd & Carel 2016; Carel & Kidd 2017; Kidd & Carel 2017; Buchman *et al.* 2017; Annesley 2020; G. Williams 2020; Rosen 2021; Heggen 2021; Della Croce 2023; Côté 2024), which is given the name 'pathocentric epistemic injustice' by Havi Carel and Ian Kidd and others. UHEI is an epistemic injustice problem because power imbalance in healthcare can produce both hermeneutical and testimonial injustice: *differences in response to uncertainty* can lead to testimonial injustice, and dichotomies of understanding can lead to hermeneutical injustice. I accept that this pathocentric epistemic injustice is also due to the imbalance of power between patients and healthcare professionals, and the effects of the differences in their phenomenological and professional experience (Marcum 2008, p. 280; and Reiheld 2010; Ho 2011; Freeman 2015; Carel & Kidd 2017, p. 336; G. Williams 2020; Alcalay 2024). However, in unsatisfactory CFS encounters I argue there is the *additional* explanatory power of differing responses to uncertainty concerning knowledge about the patient's medical condition and consequent treatment recommendations, along with disagreement about how that knowledge should be created and evaluated. I am not claiming that HEI in situations of uncertainty is manifested or experienced in any way significantly differently from HEI in medical situations of relative certainty; UHEI is therefore not an extension of the meaning of HEI, UHEI merely provides further explanation for some of the instantiations of HEI.

In order to illustrate UHEI, I apply my two aims of explanation and amelioration to the disputed, stigmatising, and inadequately-understood medical condition(s) chronic fatigue syndrome (also known as myalgic encephalomyelitis, myalgic encephalomyopathy, or

myalgic encephalomyopathy/chronic fatigue syndrome, and abbreviated to CFS, ME, ME/CFS, or CFS/ME), because epistemic injustice in CFS/ME healthcare encounters is not uncommon (Blease *et al.* 2017; Geraghty & Blease 2018; Geraghty *et al.* 2019; Byrne 2020; Pheby *et al.* 2020; De Boer 2021; Hng *et al.* 2021; Pot 2022), and because, I argue, HEI in CFS/ME has explanations connected to responses to epistemic uncertainty that are *additional* to explanations for EI in other better-understood medical conditions. I also, to a lesser extent, use the examples of some other conditions such as fibromyalgia, Long Covid, and chronic Lyme disease that have similar symptoms to CFS/ME, similar areas of uncertainty, and similar susceptibility to healthcare epistemic injustice.

What is ME/CFS? “Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) is a debilitating disorder characterized by prolonged periods of fatigue, chronic pain, depression, and a complex constellation of other symptoms” (Richman *et al.* 2019, p. 798) (also see descriptions in Ramsay 1986; Holmes *et al.* 1988; Lloyd *et al.* 1990; Fukuda *et al.* 1994; Sharpe *et al.* 1991; Carruthers & Vande 2003; Carruthers *et al.* 2011; National Academies of Sciences, Engineering, and Medicine 2015; Asprusten 2021). These symptoms can last for years, and usually greatly restrict the patient’s life, including long-term restrictions on work, family life, and socialisation. There are no curative treatments available, and symptomatic treatments are in many cases not particularly helpful, so many patients continue to suffer for years. I give a more comprehensive description of the symptoms and course of ME/CFS in Chapter Three, Section Two. In Chapter Four, Section Four, I describe various (oppositional) treatment recommendations for CFS.

The epistemic uncertainties about CFS are multiple: there is no agreed understanding of the pathological processes or mechanisms that cause the precipitation and/or the perpetuation of

CFS symptoms (Sotzny *et al.* 2018; Cortes Rivera *et al.* 2019; Missailidis *et al.* 2019; VanElzakker *et al.* 2019; Muller *et al.* 2020; Nacul *et al.* 2020; Noor *et al.* 2021; W. T. Lim & Torpy 2023; Thoma *et al.* 2024; Walitt *et al.* 2024; Ali & Kheirabadi 2025); there are no biomarkers for diagnosis (Brurberg *et al.* 2014; van der Meer & Lloyd 2012; Missailidis *et al.* 2019; Lidbury and Fisher 2020; Deumer *et al.* 2021; Macksoud *et al.* 2023), for the assessment of severity, for the appraisal of response to treatment (Walitt *et al.* 2024), or for the identification of subtypes; so, there are uncertainties about homogeneity and demarcation. There are no curative treatments (Missailidis 2019; Kim *et al.* 2020; Arron *et al.* 2024). Treatments intended to address possible pathological mechanisms have only limited success in some patients (Mitchell 2016; Richman *et al.* 2019, Kim *et al.* 2020; Seton *et al.* 2024), and symptomatic treatments have only partial success, and only in some patients (Arnett and Clark 2012; Lattie *et al.* 2013; Kim *et al.* 2020; Noor *et al.* 2021; Richman *et al.* 2021; W. T. Lim and Torpy 2023). Differing responses to these multiple uncertainties about CFS lead to dichotomies (or more) of conceptualisations of CFS and to consequent corresponding differences of views regarding recommendations for treatment.

The first conceptual dichotomy that I address concerns the ontological status of CFS. Many commentators refer to CFS or ME/CFS as if it is a determinate concept (Maes & Twisk 2010; Carel & Kidd 2014; O’Leary 20018; Geraghty *et al.* 2019; Sharpe & Greco 2019; Twisk 2019; Diserholt 2020; Pheby *et al.* 2020; Byrne 2021; de Boer 2021, p. 1348; Hng *et al.* 2021; Thoma *et al.* 2024; Ali & Kheirabadi 2025). Against these commentators, Roberts (2017) argues that ‘CFS’ is merely a construct and an indeterminate concept, and Scadding (1996) considers that the concept ‘chronic fatigue syndrome’ is nominalist and not essentialist, in the sense that what unifies patients presenting with this condition is merely the

labelling, because of, “... a recognition of a familiar pattern ...” (Scadding 1996, pp. 594-6), without there being adequate evidence of a common underlying disease entity.

The second conceptual dichotomy that I address is between the conception that CFS/ME is a purely physical disease needing physical treatments such as (some as-yet-undiscovered) medications, and the opposing concepts that emphasise non-physical factors in its pathological processes and management recommendations. Many commentators (Carel & Kidd 2014; Geraghty & Blease 2018; Castro-Marrero 2017; Geraghty *et al.* 2019; Lubet & Tuller 2021; Thoma *et al.* 2024; Ali & Kheirabadi 2025), CFS patients (as reported by L. Cooper 1997), and patient support and advocacy groups (as reported by de Boer 2021) contend that CFS is a physical disease with the symptoms being due to some (as-yet-undiscovered) physical pathological processes that *cannot* be improved by treatment with cognitive and behavioural treatments (MePedia, ‘Medical Hypotheses’).

By contrast to these claims of the physical nature of CFS, some healthcare professionals respond differently to uncertainty about CFS, doing so in a number of dissimilar ways. Some HCPs doubt that CFS is ‘real’ (as reported (but not endorsed) by all of: Steven *et al.* 2000; Gipps 2008, Horton *et al.* 2010; van der Meer & Lloyd 2012; Geraghty 2016; Marks *et al.* 2016; Blease, Carel & Geraghty 2017; Hand & Korsten 2020; Pheby *et al.* 2020; Hng *et al.* 2021; Hng *et al.* 2021). (I see that this doubting response is likely to lead to unsatisfactory healthcare for CFS sufferers.) Sharpe & Greco consider that CFS is not a disease, although they accept that it is an illness (Sharpe & Greco 2019). Bass (2001) suggests that CFS patients have non-organic disability. Other healthcare professionals respond to these uncertainties by conceptualising CFS as being a psychological condition, requiring treatment with, for instance, antidepressant medications (as reported (but not endorsed) by Steven *et al.*

2000; by Thomas & Smith 2005; and by Bayliss *et al.* 2014). Yet other healthcare professionals consider CFS to be a bio-psycho-social condition, needing treatments directed at patients' cognition and behaviour (Vercoulen *et al.* 1998, and as reported (but not endorsed) by Bayliss *et al.* 2014).

Medical research about CFS has not yet led to adequate knowledge of its mechanisms, nor to the discovery of any reliable diagnostic criteria or biomarker, nor to the identification of any curative treatments. The present deleterious inadequacy of scientific knowledge regarding CFS/ME pathology and treatments follows from inadequate progress in research into CFS over the past few decades. In this thesis, I argue that this research into CFS is itself hindered by uncertainties about CFS; that is to say, the present inadequacy of scientific knowledge regarding CFS/ME is *both an explanation for and the result of uncertainty*. I posit the under-recognised concept of *a network of mutually reinforcing uncertainties*, where, for instance, uncertainties about reliable diagnostic criteria hamper the choosing of people for research; and, reciprocally, the inadequacy of the results of research impedes the obtaining of reliable diagnostic criteria for choosing individuals for research. Furthermore, dichotomies of conceptualisations about CFS lead to dichotomies in the directions of research, either towards physical or towards psychological processes, rather than towards both. I therefore evaluate the differing aims, pathways, and constraints of medical research into CFS/ME.

Narratives by CFS/ME patients and others give evidence of unsatisfactory healthcare encounters in this condition (L. Cooper 1997; Deale & Wessely 2001; Page & Wessely 2003; Tucker 2004; Dickson *et al.* 2007; Guise 2010; Horton *et al.* 2010; Bayliss *et al.* 2014; Marks *et al.* 2016; Blease *et al.* 2017; Lian & Robson 2017; Diserholt 2020; Franssen 2020; Fennel *et al.* 2021; Hattrick 2021; Aaron *et al.* 2024; 'Suzanne' 2024). In Chapter Five, Section

Four, I show in which ways these unsatisfactory encounters can be categorised as being epistemically unjust, and why this unsatisfactoriness is related to epistemic uncertainty and the consequent dichotomies of view. One example of unsatisfactoriness is the situation where a healthcare professional dismisses or downplays a patient's citing of third-party-generated knowledge, such as the results of biomedical research into CFS (as I describe in Chapter Six, Section Six).

My argument concerning the origins of healthcare epistemic injustice in CFS healthcare encounters overlaps that of the philosophical contributions of Charlotte Blease and her colleagues (2017), Blease and Keith Geraghty (2018), and Kristin Heggen and Hendrik Berg (2021), the sociologist Lesley Cooper (1997), the medical doctors Hossenbaccus and White (2013), and the physiotherapist Michael J. Joyner (2012), who all see that patients' and healthcare professionals' dichotomous conceptualisations of ME/CFS (and similar conditions) lead to unsatisfactory healthcare encounters. I accept the view of Blease *et al.* (2017) that healthcare professionals who reject patients' interpretations of their condition and its causes in CFS healthcare encounters are not acting appropriately, and are thereby inflicting epistemic injustice (Blease *et al.* 2017, p. 554).

However, I differ from Vercoulen *et al.* (1998), Blease *et al.* (2017), Blease and Geraghty (2018), Heggen and Berg (2021), and from CFS patient support and advocacy organisations (MePedia, 'Medical Hypotheses'), in that I consider that both of the oppositional conceptualisations about the nature of CFS and its treatment recommendations are *not unreasonable* responses to what is known and not known objectively about CFS, *and* how CFS is experienced subjectively. Unlike many other commentators, I am even-handed when

I accept the value of these differing responses to uncertainty; I avoid supporting one side rather than the other in regard to these disputes.

However, I am also even-handed when I *critique* the positions of each side. Unlike other commentators, I argue that the inappropriate and unhelpful rejection of an opposing conceptualisation is *not warranted*, given the uncertainties about CFS, and, also, given the duties encapsulated in the concepts of epistemic virtue for knowledge acquisition and evaluation (Battaly 2017). I argue that dismissing alternative conceptualisation about CFS may be seen as a form of epistemic vice (Lahroodi 2007; Choo 2016) (with this epistemic vice being collective rather than individual). My view is that these rejections are inappropriate responses to the uncertainties about CFS, and they are unhelpful responses because they lead to conflict and to unsatisfactory healthcare encounters.

My argument is a development of that of Eleanor Byrne, who writes:

I have resisted claims that identify testimonial injustices where the epistemic problem can be just as plausibly explained by the medical professional exercising *appropriate* medical sensitivity within the context of a vast conceptual impoverishment (Byrne 2020, p. 377). [My italicisation]

Here, Byrne correctly identifies a major problem in regard to chronic fatigue syndrome, the “... vast conceptual impoverishment of CFS/ME” (Byrne 2020, p. 377; Byrne 2021, p. 154), to which I add ‘epistemic impoverishment’, in acknowledgement of the multiple profound epistemic uncertainties concerning it, which explain the conceptual impoverishment, and which amount to ‘radical uncertainty’. I argue that this radical uncertainty explains the

dichotomous responses between some CFS patients and some healthcare professionals.

Unlike Byrne, I see that oppositional responses to radical uncertainty about CFS do constitute testimonial injustice.

As Byrne acknowledges, some commentators do make the case for the interpretation of some medical professionals' responses to those epistemic problems as being epistemically unjust (Blease *et al.* 2017; Geraghty & Blease 2018; Geraghty *et al.* 2019; Byrne 2020; Pheby *et al.* 2020; De Boer 2021; Hng *et al.* 2021; Pot 2022). In the view of these commentators, these medical professionals are using the power imbalance in healthcare encounters to privilege and impose their conceptualisations over patients' conceptualisations. So, in contrast to Byrne, and in line with the views of these commentators, I question whether some medical professionals are acting 'appropriately' (as Byrne allows) in response to CFS's uncertainties.

Instead, I differ from Byrne by arguing that during unsatisfactory CFS healthcare encounters (Cooper, L. 1997; Deale & Wessely 2001; Page & Wessely 2003; Tucker 2004; Dickson *et al.* 2007; Guise 2010; Horton *et al.* 2010; Bayliss *et al.* 2014; Marks *et al.* 2016; Blease *et al.* 2017; Lian & Robson 2017; Diserholt 2020; Franssen 2020; Fennel *et al.* 2021; Hattrick 2021; Aaron *et al.* 2024; 'Suzanne' 2024), wrong is being done to people with the symptoms of chronic fatigue syndrome when those patients are subjected to inappropriate deficits of HCPs' credence. The wrong is towards these patients' capacities as knowers regarding their testimony concerning the severity and the deleterious effects of their symptoms, which is testimonially unjust according to Fricker (2007). Additionally, wrong is being done in regard to these patients' interpretations of their predicament, which is hermeneutically unjust according to Fricker (2007).

I use the concepts of stigma (Asbring & Närvänen 2002; Dickson *et al.* 2007; Fennell *et al.* 2021; Turner 202) and prejudice (Fricker 2007; ‘Suzanne’ 2024) in my analyses of healthcare injustice in CFS healthcare interactions. These are generic problems in healthcare encounters, of course, not confined to CFS healthcare interactions, nor confined to interactions where there is a great deal of uncertainty.

In this thesis I aim at mitigation of uncertainty-driven healthcare epistemic injustice—by which I mean, reduction in the deleterious effects of dichotomous responses to uncertainty that are aggravated by the power differential in healthcare encounters. Uncertainty-driven healthcare epistemic injustice is harmful, but it may be remediable if both sides are able to improve the power relationship, and their responses to uncertainty:

- *Firstly*, as a *counter to the power differential*, patients and healthcare professionals need to aim at a shared understanding of the disparate problems that contribute to epistemic injustice in CFS healthcare encounters that I itemise in the following paragraphs (Bayliss *et al.* 2014; Buchman *et al.* 2017; Pot 2022; Côté 2024).
- *Secondly*, this shared understanding requires both patients and HCPs to recognise that *there is radical uncertainty* concerning chronic fatigue syndrome and some similar conditions (*c.f.* Byrne 2020).
- *Thirdly*, there needs to be an appreciation that dichotomous and oppositional views that follow from *differing responses* to that uncertainty are unhelpful (Wojcik *et al.* 2011b), and can lead to unsatisfactory healthcare encounters in the form of epistemic injustice (Bayliss *et al.* 2014; Heggen & Berg 2021).
- *Fourthly*, there needs to be understanding that dichotomous responses to radical uncertainty can each be *explicable and reasonable*, because these differing responses

are due to the differing epistemological and hermeneutical perspectives of patients and healthcare professionals.

- *Fifthly*, epistemic virtue involves the acceptance of the legitimacy of others' differing responses to unsatisfactory states of knowledge; that is to say, radical uncertainty does not warrant rejection of an oppositional perspective, because close-mindedness is an epistemic vice (Battaly 2017, p. 224). Here I am referring to *collective* rather than *individual* epistemic virtues regarding the processes of knowledge-seeking and evidence-rejecting (as do Lahroodi 2007; Choo 2016). I am not referring to individual character traits (as described by Baehr 2006) because I see that these processes are shared among many people.

I see two potential ways to respond to dichotomies of understanding regarding CFS, by the application of two different disease models (which I describe in Chapter Seven). The first alternative model that I suggest for amelioration is the *network of symptoms model of disease* (or illness). This model envisages that some medical conditions can be considered to consist of a dynamic network of mutually reinforcing symptoms (Hevey *et al.* 2013; Fried 2015; Borsboom 2017; Blanken *et al.* 2019; Bakker 2019; Robinaugh *et al.* 2020; Byrne 2021; Byrne 2024), perhaps with a hypothecated common cause or latent variable (Borsboom *et al.* 2003; Young 2015; Epskamp *et al.* 2017). This model is applied by these commentators to medical conditions in which psychological symptoms predominate, such as depression and anxiety; but, I suggest that it also seems to have value in application to explain the symptoms and course of medical conditions in which physical symptoms predominate, such as Covid 19 after hospital discharge (Liu *et al.* 2021), and CFS (Missailidis *et al.* 2019). (A network model of disease can also be used to illustrate the relationships between pathological processes (Barabási *et al.* 2011), or to illustrate the relationships between pathological

processes and psychological symptoms (Epskamp *et al.* 2018), but I do not enlarge on that kind of model in this thesis.) A network model of symptoms or a network model of disease processes can, separately or together, be alternative to the biomedical model and the biopsychosocial model. I suggest that the acceptance of the significance of interactions between symptoms may mitigate disputes related to dichotomies of understandings between HCPs and CFS/ME patients, and between HCPs and CFS/ME patient groups of CFS/ME and its treatments; thereby, the application of a network model may help to improve CFS encounters.

My alternative suggested strategy for amelioration is the attribution of the concept of ‘persistent physical symptoms’ (Löwe *et al* 2025) to CFS, which, in my view, could reduce the stigma associated with having an ‘*unexplained*’ medical condition, because the use of the term ‘persistent physical symptoms’ involves recognising that CFS shares the characteristic of under-explanation with some of the symptoms and course of many better-understood medical conditions, such as abnormally slow physical recovery from physical trauma.

Some of these proposals for amelioration can have wider applicability, for instance, to those other medical conditions that are referred to as being ‘medically unexplained symptoms’ (MUS) or in other medical conditions where there are significant dichotomies of view due to uncertainty.

In order to support my arguments, I introduce, promote, or find novel applications for, a number of concepts some of which are insufficiently highlighted elsewhere in the literature:

- The applicability of the economists’ concept of radical uncertainty to medical knowledge,

- The existence of radical uncertainty regarding knowledge about some medical conditions,
- The existence of a network of uncertainties hampering some medical research,
- The distinction between uncertainty regarding a medical condition and uncertainty regarding the medical problems of a particular individual in a healthcare encounter,
- Radical uncertainty as an explanation for some healthcare epistemic injustice,
- The applicability of the concepts of epistemic virtue to responses to medical uncertainty,
- The under-acknowledged persistence of inappropriate ‘medical disease dualism’,
- The applicability of the network of symptoms model to non-psychiatric illness, and
- The applicability of the persistent physical symptoms model of illness to under-explained medical conditions.

I see that these concepts could have wider applicability in the philosophy of medicine and the practice of medicine, that I itemise in Chapter Eight, Section Five.

1.4: Scope, Time Scale, Methodology, Sources, and Conceptual Frameworks

The **scope** of this thesis is confined to explaining and ameliorating perceived epistemic injustice in healthcare (HEI), particularly in healthcare encounters, by showing that HEI has a close connection to epistemic uncertainty. I concentrate on chronic fatigue syndrome, and on medical conditions with similar symptoms and comparable significant lacunae in knowledge regarding causes and treatments, where these lacunae contribute to dichotomies between patients and health care professionals about understandings of the disease process and recommendations for treatments.

I confine the **time scale** of this thesis to the later twentieth century until the present day. I do not describe the well-documented history of CFS/ME and its predecessors and similar conditions in this thesis. (For a helpful historical narrative of CFS/ME, see Committee on the Diagnostic Criteria for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (2015).)

The qualitative **methodology** that I use in this thesis is the logical analysis of the evidence for healthcare epistemic uncertainty, with the presentation of evidence to support my argument that healthcare epistemic injustice can be significantly exacerbated by responses to that epistemic uncertainty. I engage with various areas of philosophy such as phenomenology, philosophical mind-body dualism, my concept of ‘medical disease dualism’, medical treatment dualism, virtue epistemology, the philosophies of psychiatry and medicine, the nature of disease, and justification for the application of various disease models including network models. I highlight scientific uncertainty in regard to CFS/ME and similar conditions as described in review articles, and the consequent difficulty of accommodating scientific uncertainty when making medical decisions in healthcare encounters. In the sections of this thesis on amelioration, I evaluate alternative conceptualisations for CFS/ME, and alternative ways of argumentation. I include analyses and interpretations of others’ rhetoric, terminology, semantics, and conceptualisations in the contributions to these debates and disputes regarding knowledge of epistemic injustice in CFS/ME and CFS/ME-related conditions. I show how some terms and phrasing used in the expressions of opposing views reduce clarity, and thereby unhelpfully augment dichotomies and disputes.

My **sources** are philosophical contributions to these debates, reviews of scientific research on CFS/ME, patient narratives, the views expressed in patient support and advocacy groups and

their websites, and the views of healthcare professionals. Most of my evidence is from English language sources. I use philosophical contributions on epistemic injustice and on healthcare epistemic injustice to connect these concepts to epistemic uncertainty. I use CFS/ME patient narratives for their perspectives on their experiences of unsatisfactory consultations, and for their explanations for those experiences; I analyse and appraise those perspectives and explanations in support of my intention to establish the existence and relevance of UHEI. I use the contributions of CFS patient support groups and websites, as these are likely to influence CFS patients' understanding of their condition and their attitudes towards treatment recommendations. I use scientific articles that describe what is known or claimed to be known about CFS/ME, its definition, its diagnosis, its pathological processes, and its treatments; for this purpose, I rely mainly on scientific *review* articles where they are available; these are articles that summarise, collate, and evaluate others' research, so they give a wider perspective than single research articles. Many of these review articles appraise the methodologies and applicability of the research that they review; I see that these appraisals give an indication of the levels of uncertainty concerning CFS. I use economists' contributions on uncertainty and radical uncertainty because much of the writing on these subjects is by economists.

I use four connected **conceptual frameworks** in this thesis—the first consisting of three paths leading to uncertainty in healthcare interactions, the second framework consisting of a network of uncertainties, the third framework consisting of three paths leading to healthcare epistemic injustice, and the fourth framework consisting of a network of symptoms. I now describe these frameworks

My first conceptual framework (that I describe in Chapter Two, Section Three) expands on uncertainty in healthcare interactions; it consists of three paths each which could give explanations for that uncertainty, but which add together to deepen the uncertainty:

- One path consists of the uncertainties in the **corpus** of medical knowledge;
- The second path is uncertainty related to the patient's **experience** of illness, which may be ineffable and difficult for the patient to understand and to communicate;
- The third path is the uncertainty inherent in the task of **matching** the patient's story to the corpus of medical knowledge.

My second conceptual framework (that I describe in Chapter Three) gives explanations for the continuing uncertainties about chronic fatigue syndrome, which I argue can be explained by a complex **network** of mutually reinforcing uncertainties.

My third conceptual framework (that I describe in Chapter Six) consists of three paths; each of these alone can explain pathocentric epistemic injustice, but which add together in some CFS healthcare encounters to, "... operate not as unitary, mutually exclusive entities, but rather as reciprocally constructing phenomena that in turn shape complex social inequalities ..." (Collins 2017, p. 115) to give explanations for healthcare epistemic injustice:

- The first of these three paths (that I describe in Chapter Six, Section Three) is the **already-recognised explanations** for epistemic injustice in healthcare (Carel 2016), which is that the differing epistemological and phenomenological perspectives of patient and healthcare professionals, and the power differential inherent between them, are explanations for the discounting by healthcare professionals of patients' testimonies about their symptoms, and for healthcare professionals devaluing patients' understandings of their medical conditions; thereby, in these circumstances,

these healthcare professionals are inflicting testimonial injustice and hermeneutical injustice.

- I add the path of the existence of significant stigma (that I describe in Chapter Five, Section Two) against some poorly-understood medical conditions and against patients with these conditions, which stigma explains the **prejudice** of some healthcare professionals against those patients, and therefore gives another explanation for healthcare professionals' discounting patients' testimonies about their symptoms, and healthcare professionals' devaluing of patients' understandings of their medical conditions. So, as I argue in Chapter Six, Section Seven, these healthcare professionals are inflicting testimonial injustice and hermeneutical injustice on patients.
- To these standard explanations for healthcare epistemic injustice, I add the path of **dichotomies of views** (that I describe in Chapter Four, Section Four) between patients and healthcare professionals about what is wrong and what treatments should be recommended. These dichotomies are explained by the deleterious uncertainty outlined in my first conceptual framework. These dichotomies help to explain the discounting by some healthcare professionals of patients' testimonies about their symptoms, and the devaluing by some healthcare professionals of patients' understandings of their medical conditions. Thereby, these healthcare professionals are inflicting testimonial injustice and hermeneutical injustice on patients.

The above three conceptual frameworks give my explanations for healthcare epistemic injustice in CFS healthcare encounters. My fourth conceptual framework is a proposal that could be a route to amelioration of epistemic injustice in CFS healthcare interactions. This framework consists of a hypothesised complex dynamic network of mutually interactive

symptoms (that I describe in Chapter Seven, Section Five) as an explanation for the disparate symptoms and disparate course of CFS between patients, and the changing symptoms over time in an individual CFS patient.

1.5: Terminology

There is dissatisfaction with the usual names for the condition(s) under discussion in this thesis. Many CFS/ME sufferers and advocates for people with CFS/ME are unhappy with the term ‘chronic fatigue syndrome’ because that phrasing seems to them to downplay the severity and intrusiveness of the symptoms and the damaging effects of these symptoms on people’s lives (L. Cooper 1997; Goudsmit 2002; Committee on the Diagnostic Criteria for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome 2015, Terminology; O’Sullivan 2015 Jason *et al.* 2020; Chivers 2021). Others claim that the alternative terms ‘myalgic encephalopathy’ or ‘myalgic encephalomyelitis’ are not adequately supported by scientific evidence. (Wojcik *et al.* 2011b, p. 501; National Academies of Sciences, Engineering, and Medicine 2015, p. 11; O’Sullivan 2015, p. 240). Further others claim that CFS and ME are two separate conditions that should not be conflated (Twisk 2019). Against Twisk, the anaesthetists Noor *et al.* (2021) find that CFS has been merged with myalgic encephalomyelitis (ME) in their literature review of the current understanding of chronic fatigue syndrome. The distinction between CFS and ME is of reduced importance according to the psychologist Jessica Beilharz and colleagues, because of their similarities: “Does it really matter whether ME and CFS are synonymous labels or not? Both labels refer to an illness with heavily overlapping subjective symptom criteria and require a 6 months’ diagnosis” (Beilharz *et al* 2019 – no page numbers). Furthermore, the distinction or not

between CFS and ME is not central to the arguments of this thesis, in which I use the examples of chronic fatigue syndrome *and similar conditions* in my search for explanations for healthcare epistemic injustice. In Chapter Three, Section Three, Subsection (d) of this thesis, I show that the alleged unsatisfactoriness of these names, and the associated disputes about them, derive from and demonstrate uncertainty. By default, I choose to use the admittedly unsatisfactory but widely-used terms, chronic fatigue syndrome, CFS, or CFS/ME, in this thesis (without specifying whether that ‘E’ refers to encephalomyelopathy or, instead, to encephalomyelitis), except when citing or referring to a particular source that uses a different specific name.

I often use the terms ‘chronic fatigue syndrome’ and ‘CFS/ME’ in the *singular* in this thesis, even though I argue in Chapter Three, Section Three, Subsection (f) that there is inadequate evidence that CFS/ME is a homogenous medical condition. That is to say, I do not see adequate support for the claims that all people with that label for their symptoms have the same pathological processes (VanElzakker *et al.* 2019), or that these people all require the same treatment recommendations. I also do not see adequate support for the view that CFS/ME can be satisfactorily demarcated from other conditions with similar symptoms. (Because of the paucity of evidence for homogeneity and demarcation in CFS and the lack of adequate evidence for disease mechanisms, I prefer the term ‘condition’ to ‘disease’ in relation to CFS.) However, for ease of reading, I do sometimes copy the practise of many of my sources and refer to CFS (or CFS/ME) in the singular, as if it were an entity. My usage of the phrase, ‘people *living with the symptoms of* chronic fatigue syndrome’, as well as ‘people *with* chronic fatigue syndrome or CFS/ME’, reflects my response to the uncertainties concerning CFS’s definitions, demarcation, and homogeneity. In this stance I am following Lesley Cooper’s guarded phrasing:

[W]hen I speak of 'sufferers' I do not mean sufferers of ME, but sufferers of such bodily experiences of pain and incapacity, which have been presently labelled as ME; I make this distinction so as not to be accused of 'ontological gerrymandering' (L. Cooper 1997, p. 188)

Like L. Cooper, I do not wish to appear to be making assumptions about the existence of CFS as a distinct entity. Nevertheless, for the sake of brevity, I sometimes use the term 'CFS patients' to refer to people living with the symptoms of chronic fatigue syndrome or CFS/ME. Again, for ease of reading, I use the term 'CFS healthcare encounters' to mean encounters between a patient and a healthcare professional where the patient's symptoms have been *or will soon be* labelled as chronic fatigue syndrome.

I use the term 'radical uncertainty' for the scientific uncertainties concerning the medical condition chronic fatigue syndrome (that I describe in Chapter Three). My view is that these uncertainties should be considered to be contingent (albeit with an uncertain timescale): the results of future scientific research may resolve some or many of the significant uncertainties concerning CFS.

I use the term 'unresolvable uncertainty' (as I describe in Chapter Two, Section Two) to describe those epistemic uncertainties about a particular patient in a specific healthcare encounter concerning chronic fatigue syndrome; these uncertainties are *not* contingent on obtaining more information, because that information *cannot be made available* by action taken during the timescale of a healthcare encounter.

I use the term ‘deleterious uncertainty’ when I wish to incorporate a negative evaluation of the effects of that uncertainty.

I sometimes choose to use the simple word ‘uncertainty’ to encapsulate the epistemic concepts of ‘severe uncertainty’, ‘epistemic uncertainty’, ‘radical uncertainty’, ‘unresolvable uncertainty’, ‘deleterious uncertainty’, and other, less profound, forms of uncertainty.

I do not distinguish between the terms ‘encounters’ and ‘consultations’ in interactions between patients and health care professionals (HCPs).

I use the term ‘health care professional’ in preference to, say, ‘doctor’, because CFS patients’ narratives give evidence that their experience of unsatisfactory consultations in healthcare encounters includes their encounters with other health care professionals, such as physiotherapists, nurses, psychologists, and psychiatrists. I note that many doctors and these other health professionals frequently share conceptualisations of medical conditions with each other, according to their training, professional roles, and experience. Some of my sources use the term ‘clinician’ in this context.

I use the terms ‘treatment recommendations’ or ‘management recommendations’ instead of simply, ‘treatments’, because much CFS management does not involve medication.

Additionally, my use of the word ‘recommendation’ respects the principle of patient autonomy, by which the patient has the right to decline treatments (Pirotte & Benson 2023/4).

When I use the term ‘physical treatments’ or ‘biomedical treatments’, I am usually referring to medications.

When using the terms ‘physical’ and ‘psychological’, I distinguish between symptoms on the one hand and medical conditions on the other hand. I use the terms ‘psychological’ and ‘physical’ in relation to symptoms and treatments, while I tend to avoid them in regard to classifying medical conditions; this is because I avoid accepting a strong distinction between physical and psychological medical conditions (see Chapter Five, Section Four). Instead, I see a non-rigid distinction between medical conditions where physical symptoms predominate (often called ‘physical’ or ‘somatic’ diseases) from medical conditions where psychological symptoms predominate (often called ‘psychological’ or ‘psychiatric’ diseases), without acknowledging them as different and distinct natural kinds. For ease of reading, I do use the terms ‘physical disease’ or ‘physical medical condition’ when distinguishing a medical condition where physical symptoms predominate from one which is considered to be mental, psychological, or psychiatric. In the place of ‘physical’ for such a medical condition, others (not unreasonably, in my view) use the terms, ‘somatic’ (as used by Carel & Kidd 2014, and as described by Vercoulen *et al.* 1998 and de Boer 2021), or ‘biomedical’ (Lubet & Tuller 2021), ‘biological’ (O’Leary 2018), or ‘medical’ (O’Leary 2018).

1.6: Caveats and Limitations

My challenges to the ontological and nosological status of the medical condition CFS/ME in Chapter Three are not a dismissal of the sufferings of people who have their illness labelled with this diagnosis. The arguments in this thesis depend on the recognition of the *reality of the symptoms* that CFS patients experience, including the severity, intrusiveness, and disabling effects of these symptoms (Joyner 2012; Barnes 2023, p. 661).

This thesis is concerned with *injustice*, so I concentrate on analysing unsatisfactory healthcare encounters, whilst acknowledging that some patient narratives also describe *satisfactory* consultations about CFS/ME. There is variability in CFS patients' satisfaction with their healthcare encounters. In regard to views about chronic fatigue syndrome, I acknowledge that different CFS patients may have perspectives that differ from each other; and, similarly, there is a variability in health care professionals' views about CFS. All of these variabilities impose some constraints to the generalisations in this thesis.

This thesis does not concentrate on the non-uncertainty-related explanations for epistemic injustice in healthcare (HEI), because there are many other commentators who have successfully undertaken these tasks:

- *Firstly*, patients' difficulty in understanding and communicating many aspects of their own experience of illness (Carel & Kidd 2014, p. 529),
- *Secondly*, power asymmetries within the healthcare encounter (Carel & Kidd 2017; G. Williams 2020),
- *Thirdly*, epistemic asymmetries—that is to say, the differences between knowledge derived from medical training and theory compared to knowledge derived from patient experience (Carel & Kidd 2014, p. 529),
- *Fourthly*, the epistemic privileging of the medical practitioner (Carel & Kidd 2014, p. 529) and,
- *Fifthly*, the downgrading of patient testimonies where that downgrading seems to be not directly related to uncertainty (Buchman *et al.* 2017).

I also do not directly address the problem of medical practitioners' poor communication skills as an explanation for patients, "... not feeling listened to ..." (Hatchwell 2023, no page numbers). It is likely that some or all of these explanations for epistemic injustice contribute

to epistemic injustice in chronic fatigue syndrome healthcare encounters, along with the problems due to oppositional responses to radical uncertainty about CFS upon which I concentrate.

In this thesis I allude to, but I do not address in much detail, the important issue of the contribution of intersectionality (as described by Collins 2017) to unsatisfactory healthcare encounters. The concept of intersectionality refers to the deleterious interaction of a number of negative factors towards increasing discrimination, prejudice, and disadvantage. In the context of the topic of this thesis, the term intersectionality could refer to the insight that, *inter alia*, (1) being ill, (2) having the disputed and stigmatising condition chronic fatigue syndrome, (3) being female (Asbring & Närvänen 2002), (4) being the less powerful person in an encounter with a healthcare professional, and (5) having an illness whose, “... experience of illness are difficult to understand and communicate ...” (Carel, 2017, p. 180) are all negative factors. These factors “... operate not as unitary, mutually exclusive entities, but rather as reciprocally constructing phenomena that in turn shape complex social inequalities ...” (Collins 2017, p. 115). These factors can interact, to the disadvantage of the CFS patient (who is more likely to be female than male). I do however address the disputes and stigma related to CFS (Asbring & Närvänen 2002), thereby indirectly responding to intersectionality in CFS healthcare encounters.

I appreciate that the lessons of standpoint theory (Wylie 2015)—by which the epistemic standpoint of the oppressed is privileged—may give further understanding of healthcare epistemic injustice. However, for the purposes of this thesis, my aim is establishment of the significance of differing responses to radical uncertainty regarding CFS, without privileging one response, or one perspective, over another.

Medical researchers have found differing and sometimes contradictory explanations for the symptoms and course of CFS, and have come to dichotomous and oppositional conclusions about the appropriateness and values of various treatment options. It is not the role of this thesis to appraise the methods or the conclusions of medical researchers, nor to adjudicate between the conclusions of those who collate and appraise the work of other researchers in review articles. Instead, I report how *review articles* that summarise, collate, and evaluate others' scientific research conclude that much of that research into CFS is not sufficiently robust to adequately eliminate uncertainty about disease mechanisms, definitions, diagnostic process, treatments, or directions for further research (Smith *et al.* 2012; Arnett and Clark 2012; Brurberg *et al.* 2014; Haney *et al.* 2015; Larun *et al.* 2017; Cores Rivera 2019; Larun *et al.* 2019; Richman *et al.* 2019; Kim *et al.* 2020; Muller *et al.* 2020; Arnet & Clarke 2021; Maksoud *et al.* 2023; Sanal-Hayes *et al.* 2023; Arron *et al.* 2024; Seton *et al.* 2024).

I do not address in much detail problems related to moral or ethical uncertainty, that is to say, the situations where it is morally uncertain how we should act.

The phrase 'medical uncertainties' has a number of related meanings, that are relevant to the arguments in this thesis. When I refer to epistemic uncertainties in medicine, I mean problems with the corpus of medical knowledge. When I refer to epistemic uncertainties in healthcare encounters, I include not only those problems in the corpus of medical knowledge, but also the other uncertainties that impact the necessary tasks for an HCP when in an encounter with a patient, such as eliciting, appraising, and analysing the patient's narrative, matching that narrative to a pattern of symptoms, matching that pattern to a particular diagnosis via the corpus of medical knowledge, and deciding between the consequent choice

of treatment recommendations, all of which tasks are subject to some degree of uncertainty.

In a healthcare encounter, the patient also has to respond to the uncertainties of the corpus of medical knowledge and then appraise the healthcare professional's formulation and treatment recommendations.

I do not analyse in detail the effects of people's differing *degrees* of uncertainty. (As I report in Chapter Two, Section Two, uncertainty is also an attribute of cognition.) These differences may well negatively impact the healthcare encounter: it may be that the CFS patient is fairly certain about the nature of her condition and what treatments are appropriate and inappropriate, while the healthcare professional may have less certainty. A mismatch of this kind may lead to an unsatisfactory healthcare encounter, which may be classifiable as epistemically unjust. In this thesis I also do not explore the phenomenology of responses to uncertainty in medicine, an area that may be able to contribute further to explaining differing responses to the uncertainties regarding CFS.

I accept that the epistemic uncertainties concerning CFS may be temporary (Lubet & Tuller 2021, p. 1). This thesis is confined to present-day knowledge regarding CFS, so it is possible that some significant aspects of the thesis may be overtaken by future events. Although, in Chapter Three, Section Six, I do argue that future hypothetical advances in scientific knowledge about CFS may still leave some patients in uncertainty.

Although much of this thesis concerns chronic fatigue syndrome (and myalgic encephalomyelopathy), I also take evidence of and look for explanations for epistemic injustice in healthcare encounters about other similarly disputed medical conditions, such as fibromyalgia, chronic Lyme disease, Long Covid, and other disputed conditions which share

many of the symptoms of chronic fatigue syndrome (Linder et al. 2002; Ulvestad 2008; Guo *et al.* 2019; Han & Korsten 2020; Joseph *et al.* 2021), where there are a lack of objective evidence, where there are uncertainties and dichotomies regarding conceptualisations and treatment recommendations (Asbring & Närvänen 2002; Eriksen 2014; Roberts 2017), and where stigma and epistemic injustice are not unusual (Heggen & Berg 2021; Della Croce 2023; Côté 2024).

In this thesis, I use the words ‘unexplained’ or ‘underexplained’ merely as a characterisation of the present knowledge relating to chronic fatigue syndrome (or the present knowledge about a particular patient’s medical condition), not as a description or a classification of CFS itself. Unlike some commentators (Ulvestad 2008; O’Sullivan 2016; Guo *et al.* 2019; Sharpe & Greco 2019), I do not conceptualise or classify CFS/ME as being in a distinct ‘unexplained’ category distinct from many other better-understood conditions, such as, say, rheumatoid arthritis (RA), multiple sclerosis (MS), or systemic lupus erythematosus (SLE). I therefore avoid the applying the terms, ‘medically unexplained symptoms’ (MUS), which could be taken to imply that CFS is in a separate epistemological, ontological, or nosological category distinct from, say, RA, MS, or SLE. Sometimes the term ‘medically unexplained’ seems to be used as a categorisation (as in the National Health Service website, *Medically Unexplained Symptoms*), rather than as a (in my view, a more appropriate) *contingent* epistemological characterisation. Additionally, MUS and similar terms can be used inappropriately to group together under one label syndromes which should be considered distinct, because many of them little in common (such as irritable bowel syndrome, fibromyalgia, myofascial pain syndrome, tension headache, multiple chemical intolerance, migraine, temporomandibular joint dysfunction, restless legs syndrome, and primary dysmenorrhoea (Guo *et al.* 2019, p. 2)). The categorisation of these conditions as being

‘MUS’ could hinder research (Tack 2019) if it were to be assumed that they have similar pathological mechanisms, and that people with these conditions are expected to have similar responses to treatments. Furthermore, research on MUS could be obstructed if the term ‘unexplained’ were to be taken as being equivalent to the more pessimistic term ‘unexplainable’. Instead, I recognise that there are degrees of explainability and uncertainty in all medical conditions, on a continuum, and that the degree of uncertainty of very many medical conditions has been shown to be contingent because uncertainty about them has reduced over time, thanks to the results of medical research. ‘Optimistic meta-induction’ leads people to believe that there could well be progress in knowledge about CFS and other underexplained medical conditions (Lubet & Tuller 2021), as has happened for many other medical conditions since the onset of scientific medicine in the nineteenth century.

Contrary to Guo and colleagues’ claim of CFS/ME being unexplained (Guo *et al.* 2019, p. 2), there are many pathological explanations for the symptoms and course of CFS/ME (Maes & Twisk 2010; Maes 2015; Geraghty 2016; Sotzny *et al.* 2018; Cortes Rivera *et al.* 2019; Missailidis *et al.* 2019; Nacul *et al.* 2020; Ali & Kheirabadi 2025). However, I see that these pathological explanations leave many major lacunae, which I describe in Chapter Three, Section Three, particularly the issues of how these disparate abnormalities are connected with each other and to the symptoms, and to the course of the condition, and which pathological abnormality is primary; so I accept that CFS/ME can be described as ‘*underexplained*’, or ‘*inadequately explained*’.

A related issue regarding the term ‘unexplained’ is that for some *individuals* the designation of their symptoms as ‘unexplained’ turns out to be a provisional description, due to the temporary inadequacy of evidence. Their (non-specific) symptoms may at first have been

given a label such as ‘unexplained’, or, perhaps, even, ‘chronic fatigue syndrome’; but, later, as their medical condition progresses and worsens, their symptoms become recognisable as being due to some better-understood medical condition, such as, say, rheumatoid arthritis, multiple sclerosis, or systemic lupus erythematosus, etc. I do not investigate epistemic injustice in these people.

Despite my previous career as a medical practitioner, I do not look to ‘solve’ the scientific problems of CFS/ME (and similar medical conditions), for instance, what the causes are and what the treatments should be. Instead, I aim at explanations regarding the epistemological and ontological problems of these conditions, understanding the deleterious effects of these problems on healthcare interactions, and I look for remediation of these effects.

This thesis is inherently interdisciplinary, so my citations derive from various sources. Many of my citations are to philosophers’ contributions to the debates about CFS and epistemic injustice. I also cite contributions from healthcare professionals. I cite many scientific contributions in order to demonstrate scientific uncertainty concerning chronic fatigue syndrome. I cite the work of economists on uncertainty. Debates about the uncertainties concerning chronic fatigue syndrome and similar conditions and debates about unsatisfactory healthcare encounters for these conditions are not only in academic articles, they are also in the public realm, so some of my citations are from people who are neither academics nor healthcare professionals. When the (principal) author of one of my references is *not* an ethicist or other philosopher, I usually specify his or her role.

1.7: Outline of the Chapters in this Thesis.

In order to support my argument that there is an explanatory connection between epistemic injustice and differing responses to epistemic uncertainty in some healthcare encounters, I show how radical uncertainties in conditions like chronic fatigue syndrome explain dichotomies of views between health care professionals and patients of what is wrong and what treatments should be offered; and, that these dichotomies, added to the power differential in medicine, can explain unsatisfactory healthcare encounters which can be classified as being epistemically unjust. Amelioration of this epistemic injustice requires reduction of the power differential between health care professionals and patients, and the mitigation of the uncertainties and of the dichotomies of views. (Here I use the term ‘amelioration’ to mean the improving of a harmful situation, and ‘mitigation’ to mean the reduction of the harmful effects of a negative situation.)

In **Chapter Two**: “Radical Uncertainty, and Uncertainties in Healthcare”, I give descriptions of the features of radical uncertainty (RU). I include different views of RU; I give examples of RU. I demonstrate how RU can be recognised. I show the drawbacks of RU, and some of its possible benefits. I show different responses to non-medical uncertainty, and then responses to medical uncertainty. I describe the epistemic status of myxoedema about which there is little uncertainty, without dichotomies of views and little evidence of contestation. I then describe the less satisfactory epistemic status of Charles Bonnet syndrome about which there is significant uncertainty, but concerning which there seems to be little evidence of dichotomy of conceptualisations, disputes about treatments, or uncertainty-driven epistemic injustice.

In **Chapter Three**, “Chronic Fatigue Syndrome, and its Uncertainties”, I describe patients’ experience of CFS/ME. I give evidence for uncertainty in CFS/ME in regard to definitions, ontology, nomenclature, demarcation, subgroups, causes, mechanisms, models, and the lack of progress in research. I then describe how many of the features of radical uncertainty are demonstrated in the scientific uncertainties about chronic fatigue syndrome, and in the consequences of that uncertainty on CFS healthcare encounters. I therefore conclude that CFS/ME is an example of radical uncertainty. I address some possible counter-claims to the arguments in this chapter.

In **Chapter Four**, “The Biomedical and Biopsychosocial Models, and their Dichotomous Applications to CFS and Its Treatments”, I explain the contrasting biomedical and biopsychosocial models of disease and illness, and I show critiques of both models. I then describe and explain the origins of the dichotomies in conceptualisations of CFS/ME, regarding its nature and treatment recommendations. I describe how HCPs’ and patients’ differing responses to uncertainties regarding CFS/ME lead to the dichotomies in understanding about what is wrong, and what should be done about the symptoms. I identify the appropriateness and inappropriateness, and values and disvalues, of these dichotomous responses.

In **Chapter Five**, “Stigma and Chronic Fatigue Syndrome, and ‘Medical Disease Dualism’”, I show evidence for stigma against chronic fatigue syndrome, and against chronic fatigue syndrome patients, associated with allegations of non-reality of the condition and accusations that the symptoms are somehow under the control of the patient. I describe how these stigmas are similar to the stigmas against mental illness and against mentally ill people. These shared stigmas are an explanation for CFS patients’ and their support and advocacy

groups’ resisting the application of psychological factors to CFS. I then address the inappropriateness of a strong distinction (that I call ‘medical disease dualism’) between psychological and physical medical conditions. I address some possible counter-claims to the arguments in this chapter.

In **Chapter Six**, “Epistemic Injustice in Healthcare, and in Chronic Fatigue Syndrome”, I use the understandings of epistemic injustice (EI) of Miranda Fricker (Fricker 2007) and others. I show the move from EI to pathocentric epistemic injustice (HEI) (Carel & Kidd 2014). I compare and contrast HEI with other forms of EI, justifying why I use these examples and why they are examples of EI. Using evidence from patient testimonies (including the Testimony of ‘Suzanne’ (2024), I distinguish HEI from bad practice, unethical practice, and/or medical professionals’ irrationality, in healthcare encounters. I then bring the example of CFS/ME into this debate and show how uncertainty and dichotomies about CFS negatively impact some healthcare encounters, again using patient testimonies. I demonstrate how these disagreements and unsatisfactory consultations in CFS/ME can be classified as EI. In Section Six, I indicate the perspectives of healthcare professionals regarding the problems of CFS, where I see some mitigations. I end Chapter Six with Section Seven, “The Central Argument of This Thesis”, which contains my main argument, that is: uncertainty about chronic fatigue syndrome and a consequent dichotomy of conceptualisations add to the well-recognised power differential in healthcare encounters and the stigma associated with CFS; together all these lead to epistemic injustice in CFS healthcare encounters, which I describe as *uncertainty-driven healthcare epistemic injustice* (UHEI). I support that argument with contrasting examples in medical conditions where there is less uncertainty and/or there is less dichotomy, leading to less uncertainty-driven healthcare epistemic injustice. I conclude by responding to some possible counter-claims to my arguments in this chapter.

Chapter Seven, “Responses to the Problems of Chronic Fatigue Syndrome”, contains my positive recommendations to ameliorate uncertainty-driven HEI, based on my analyses and on others’ contributions: we should aim to reduce the power differentials between the patient and the healthcare professional; we need to incorporate into our understanding of healthcare epistemic injustice the inherent differences between health care professionals and patients in regard to their phenomenological and epistemological perspectives; we have to live with the uncertainties; but, in our responses to uncertainties we should find ways to reduce the negative effects of the consequent dichotomies. I recommend the rejection of what I call ‘medical disease dualism’. As an alternative response to the uncertainties about chronic fatigue syndrome, I argue for either or both of two hypothesized re-conceptualisations or models of it. Firstly, CFS could be considered to consist of a dynamic network of mutually interacting symptoms, changing over time, with the possibility of one or more latent variables that are yet to be discovered. The distinctions between the dichotomous views about CFS/ME and its treatments may thereby be blunted. A second alternative is the concept of ‘persistent physical symptoms’ (PPS); PPS can be a feature of many musculo-skeletal medical conditions, where PPS refers to a significantly slower recovery. This PPS concept blunts the distinction between CFS and better-understood medical conditions, thus reducing stigma, and allowing the application of both physical, psychological, and behavioural treatment recommendations, as in many other medical conditions. I show how these two alternatives, the network of symptoms model and concept of persistent physical symptoms are compatible with each other, and add together to aid amelioration.

Chapter Eight is my “Summary, Conclusion, and Applications”. I summarise the arguments of this thesis. I describe the concepts that I promote in this thesis in order to support my

arguments, which concepts are under-emphasised elsewhere in the literature; and I show the values of those concepts and their applicabilities. I summarise my critiques of others' contributions. I summarise the contributions that the concepts in this thesis could make to medical epistemology, medical ethics, epistemic injustice, disease ontology, and the application of the concept of epistemic virtue and vice to disputes in medicine. I then suggest ways in which the lessons in this thesis could be built on and extended.

2: Radical Uncertainty, and Uncertainties in Healthcare

2.1: Introduction to Chapter Two

My argument in this thesis is that some differing responses to severe epistemic uncertainty have deleterious consequences on some healthcare encounters. In this Chapter Two, I describe this severe and deleterious uncertainty. In Chapter Three I will apply the concept of ‘radical uncertainty’ and similar terms to knowledge about chronic fatigue syndrome (CFS). I will argue in Chapter Six that these uncertainties in chronic fatigue syndrome can be explanations for epistemic injustice in CFS healthcare encounters.

In this chapter, I first show how the evaluative concept of ‘deleterious uncertainty’ shares many of its non-evaluative characteristics with what others call ‘fundamental’, ‘radical,’ ‘deep’, ‘great’, and ‘genuine’ uncertainty (Andreoletti *et al.* 2022, p. 201), or ‘radical uncertainty’ (Romeijn *et al.* 2014; Hare 2017; Kay & King 2020; Chenet *et al.* 2021; Bradley & Drechsler 2014), ‘ineliminable uncertainty’ (Knight 1933, 1921), and ‘wicked problems’ (Rittel & Webber 1973). These terms have closely-related but not necessarily identical meanings. I choose to use the term ‘radical uncertainty’ for describing the inadequate scientific knowledge about chronic fatigue syndrome, and the term ‘unresolvable uncertainty’ for the additional uncertainties in a CFS healthcare encounter (which cannot be resolved in the time-scale of that encounter).

Next in this chapter, I show how various commentators use the term radical uncertainty (and similar terms) and how they describe its characteristics. I find that radical uncertainty can be

recognised from its nature *and* its effects: radical uncertainty is a situation of inadequate information, which then negatively affects cognition, understanding of the present situation, prediction (with and without different interventions), and decision-making. By contrast, and perhaps surprisingly, I am also able to describe some potential benefits of radical uncertainty: where there are opposing and contradictory positions, the existence of radical uncertainty regarding a particular situation can allow an independent observer to consider each side's stance differing and oppositional responses to that uncertainty to be *not unreasonable*.

I continue this chapter by describing two medical conditions with contrasting certainties and uncertainties, myxoedema and Charles Bonnet syndrome (CBS). The status of myxoedema is settled, without significant uncertainties and few dichotomies of views. Charles Bonnet syndrome exemplifies a situation of a lack of objective evidence, with consequent uncertainties; however, there is little evidence of dichotomies of views between patients and health care professionals in CBS. I then summarise this chapter.

2.2: Concepts of Uncertainty and Radical Uncertainty

In this section, I describe the nature and consequences of radical epistemic uncertainty, and how it differs from non-radical or 'eliminable' uncertainty, with my aim being the recognition of instantiations of radical uncertainty. I commence with a short analysis of the concepts of uncertainty, non-radical uncertainty, and radical uncertainty (and similar concepts). I then describe the applications of radical uncertainty to economics, where the concept is prominent (although there are some economists who do not accept the category of radical uncertainty). I then summarise the characteristics and consequences of radical

uncertainty, and show its disadvantages and its possible advantages. Interspersed with these descriptions of the characteristics of radical uncertainty, I show how they illuminate some of the epistemological problems of chronic fatigue syndrome, and I show the effects of these epistemological problems on CFS healthcare encounters.

The concept, ‘epistemic uncertainty’, can refer to both the problematic nature of a situation and the problematic effects of that situation. A situation has the nature of being uncertain when we lack sufficient information to fully understand, predict, or respond to that situation. The effects of uncertainty are on cognition (the state of mind of an agent); and, there are also other consequences, such as an incomplete grasp of the current circumstances, reduced insight into underlying causes, and the hampering of prediction (including the prediction of the consequences of interventions), thereby impeding decision-making. So, uncertainty is both an obstacle to comprehension and a barrier to effective action.

The economist Frank Knight was an early identifier of the concept which is similar to what is often now called radical uncertainty, which he refers to as ‘ineliminable uncertainty’. He first distinguishes this ‘ineliminable uncertainty’ from what he calls ‘risk’, which he considers to be a lesser form of uncertainty. Knight describes risk as:

[A]n uncertainty which can by any method be reduced to an objective, quantitatively determinate probability, can be reduced to complete certainty by grouping cases (Knight 1933 (1921), pp. 231-2).

So, Knight tells us here that eliminable uncertainty refers to instances that can be grouped and then quantified, allowing the possibility of a reasonable and practicable response.

Grouping of cases can therefore be a route to reducing uncertainty.

To exemplify the benefits of grouping similar cases for the purposes of measuring and predicting, and for making informed decisions in the face of uncertainty, I give the example of actuarial science. Estimating how long a particular person of a certain age in a particular country is likely to live is uncertain in the absence of terminal illness; but, the estimate can be given a probability by obtaining pooled data such as the life expectancy at that age in that country, along with information about the person's sex and health and other relevant data, and then applying actuarial methodology, which gives a probabilistic figure for estimating the life expectancy for that person. This actuarial approach allows rational and defensible decisions to be made on probabilistic grounds: for instance, an insurance company can decide on appropriate premiums for life insurance for a particular person, even though it is inherently uncertain how long that particular individual will actually live. We can therefore recognise non-radical or eliminable uncertainty by its association with the availability of relevant information, the consequent ability to assign probabilities, and the resultant ability to make sound decisions.

In contrast to what he terms 'risk', Knight identifies a, "... higher form of uncertainty ..." (*ibid.*, p. 232) which is ineliminable because the cases are, "... to a high degree unique" (*ibid.*, p. 233); consequently, in these cases uncertainty is not susceptible to measurement by using data from previous events or to quantification by the grouping of cases. This is now referred to as 'Knightian uncertainty' (Chenet *et al.* 2021).

The economists John Kay and Mervyn King (the latter a past Director of the Bank of England) expand on Knight's account of ineliminable uncertainty. They promote the value of the concept that they call 'radical uncertainty' for situations where we have inadequate information (Kay & King 2020, p. 42), where they add that it is crucially a situation where there is, "... an inability to obtain more information" (*ibid.*, 2020, p. 10). Kay and King contrast what they call radical uncertainty with 'resolvable uncertainty', where, "... the necessary information could be available, albeit perhaps with some effort" (*ibid.*, p. 37). In contrast to situations of resolvable uncertainty, in radical uncertainty our lack of knowledge cannot be resolved by obtaining further information, because the relevant information is not available. In RU there is, "... a lack of information that in some cases but not all we may hope to rectify at some future date" (*ibid.*, p. 14). However, in these situations of RU, we do not have this information at the present time, and we do not know when or if that information will be available, let alone what it will be; so, that that possible future rectification is of no practical use at the present time.

There are economists who do not accept a strong distinction between risk and uncertainty. Frank Ramsey argues that risk *can* be quantified in situations of reduced information, by applying the concept of '*subjective* probability', whereby risk is represented numerically by an individual's subjective degree of confidence, which is shown by the extent to which an agent is willing to act (or bet) on her degree of belief (Ramsey 1926/1931). Many economists and statisticians since then have similarly elided the distinction between risk and uncertainty, emphasising the place of subjectivity in appraising risk or uncertainty (Institute for Economic Thought, accessed 1/1/26).

However, in my view, this subjectivist appraisal of ‘risk’ does not help to reduce epistemic uncertainties about chronic fatigue syndrome, because, as I describe in Chapter Four, those involved in disputes about CFS have oppositional subjective interpretations, with concomitant oppositional views on what action to take.

It is not the role of this thesis to adjudicate between the views of economists. Besides, I choose to use a qualitative rather than a quantitative understanding of uncertainty. I concentrate on the *effects* of uncertainty more than the *level* of uncertainty. In situations of inadequate knowledge and the inability to obtain more information within the necessary timescale, I attach the evaluative concept of deleteriousness to the concept of radical uncertainty. I use Knight’s and Kay and King’s understandings later this thesis. In Chapter Three, I will draw on Knight’s concept of ‘uniqueness’ to show that scientific knowledge about chronic fatigue syndrome exemplifies Knight’s concept of ineliminable uncertainty; I argue this because grouping ill people with similar symptoms in medical research may not be justified without sufficient evidence that all these people are suffering from the same underlying pathological process(es). Furthermore, the potential uniqueness of an individual CFS patient suggests that the application of grouped data from medical research to that particular patient may not be appropriate. Significant uncertainties about chronic fatigue syndrome (particularly the unclear boundaries of the condition, and the variability of symptoms between cases) hinder the obtaining of more information via laboratory or clinical research, thereby hampering the obtaining of knowledge applicable to individual CFS patients in healthcare encounters. In Chapter Three, I will also argue that Kay and King’s concept of radical uncertainty can be applied to the corpus of medical knowledge about chronic fatigue syndrome, because relevant information is not available.

The term radical uncertainty not only designates the problematic nature of a situation (how little is known, as alluded to above), it also negatively affects *cognition*. The health communication academic Mary Politi and her colleagues refer to the effect of radical uncertainty on how an agent thinks about the problem: it is, "... a cognitive state created when an event cannot be adequately defined or categorized due to lack of information" (Politi *et al.* 2007, p. 682).

In these above descriptions of radical uncertainty, it may be that agents understand where their lack of knowledge is, that is to say, what kind of knowledge is missing, which can be described as the 'known unknowns' (Djulgovic *et al.* 2011, p. 301). However, agents may be in the worse epistemic position of, "... not knowing what is unknown (unknown unknowns)" (*ibid.*, p. 301), that is to say, *where* the uncertainty is. (In Chapter Seven, Section Six, I describe how Linderman and Adams (2014), use a concept similar to 'unknown unknowns' in reference to the difficulty or impossibility to measure aspects of a network system). Even beyond unknown unknowns, Romeijn & Roy and Hansson note the possibility of higher-level uncertainty, in which an agent doesn't know *how* uncertain she is: "[R]adical uncertainty may refer to an epistemic state in which we have insufficient grasp of our uncertainty regarding a distinct set of propositions" (Romeijn & Roy 2014, p. 1222); or, agents may be in a situation of, "[m]etadoxastic uncertainty: uncertainty about the veracity of one's own beliefs" (Hansson 2022, p. 216).

Radical uncertainty negatively affects an agent's *understanding* of the present situation. Richard Bradley and Mareile Drechsler use the concept of a 'state space model' to illustrate the various levels of knowledge of or uncertainty about a situation (Bradley & Drechsler 2014). (The relevance of a state space model to this thesis is that it can be used to

illustrate what is known and what is not known about chronic fatigue syndrome, as I will allude to in Chapter Three); and, the state space model is similar to the ‘network of symptoms model’ that I will describe in Chapter Seven, Section Five.) A state space model is a *theoretical* model that illustrates what is known about the variables in a system, and the connections between those variables. These connections may be associational, causal, or mechanistic, depending on available evidence (Wilde & Williamson 2016). These connections may be one-way, or some of them may be two-way. A state space model may be linear (one-dimensional) in a simple system, or two- or three-dimensional in more complex system, or four-dimensional in a dynamic system. A successful state space model can be ‘constructed’ when supported by adequate knowledge about all the relevant variables in the system, *and* adequate knowledge about the mathematical relationships between those variables (Basdevant 2003). The model can be dynamic, changing over time. In some models it may be possible to see the results of varying interventions. A model of this kind can therefore aid an agent’s understanding concerning the elements of a system. If the model is dynamic, it can demonstrate and/or extrapolate changes in the values of the variables over time, thereby allowing predictions (with and without interventions), thus aiding decision-making.

If an agent possesses adequate knowledge, then state space modelling can be of value for making prognoses and predicting responses to interventions. For instance, a state space model could be used to first illustrate levels of knowledge about some medical condition, using the variables that are known and are considered relevant. The symptoms, the known pathological processes, the abnormal results of imaging and laboratory pathological tests in a particular person are all measurable variables; and, where the relationships between these variables are also known, a successful dynamic state space

model can be created. This model would then be able to demonstrate the course of the medical condition for that person over time, to give an estimate of prognosis (Cauchi *et al.* 2024); and may also be able to demonstrate the likely results of interventions such as treatments, and may therefore help in decision-making.

However, it may be that some of the relevant variables or their relationships in a particular system are *not* known. Under these circumstances, there are two levels of uncertainty regarding state space models: non-ideal but surmisable uncertainty, and radical uncertainty. In non-ideal but surmisable uncertainty, variables of the system may be unobservable, or even unknown; similarly, the relationships between the variables may be unobservable or unknown. Nevertheless, it may be possible to surmise the existence and characteristics of some or all of these unobserved variables or relationships, thereby giving a state space model which could well be adequate for understanding, prediction, and decision-making.

If we don't know what information is missing, that is to say, if some *significantly relevant variables* or their relationships in a state space are not only unknown but also remain un-surmisable (as are "... unknown unknowns ..." (Djulgovic *et al.* 2011, p. 301)), it is not possible to construct an effective state space model; so, a situation of radical uncertainty exists. Situations of radical uncertainty cannot be modelled, because there is a lack of understanding about the present situation: we do not know, "[w]hat is going on here" (Kay & King, p. 10); we lack adequate, "... knowledge of the state of the system, about some underlying fact of the matter, typically due to lack of useful or complete information" (Djulgovic *et al.* 2011, p. 301). Hansson describes situations of, "... uncertainty about the state of the world and about the correctness of our descriptions of it" (Hansson 2018, p. 388),

and situations where we have, “[f]actual uncertainty: uncertainty about the actual (past, present or future) states of the physical world” (Hansson 2022, p. 216). Mattia Andreoletti and colleagues note that one aspect of severe uncertainty is the absence of, “... adequate models to describe the relationship between [the] system’s variables” (Andreoletti *et al.* 2022, p. 202). Similarly, “[a]dequate models to describe the relations between system’s variables [are] unknown, unclear or undefined” (Andreoletti *et al.* 2022, p. 202). In “... model uncertainty we doubt our modelling assumptions but have insufficient means in the model to express alternative assumptions” (Romeijn & Roy 2014, p. 1222).

Bradley & Drechsler recognise that in severe uncertainty an agent is unsure even how to proceed, that is so say, how to conduct an exhaustive state space investigation (Bradley & Drechsler 2014), so it is then not possible to ‘construct’ an adequate state space model. Bradley & Drechsler explain how state-space uncertainty of this kind hinders decision-making:

State Space Uncertainty: the state space is not sufficiently detailed to capture all decision-relevant factors [and/or] ... an agent is aware of the possibility that she has entirely omitted a state of the world from the state space (Bradley & Drechsler 2014, p. 1245).

By the phrase, ‘state of the world’, here, Bradley & Drechsler are referring to the relevant variables in the system and the relevant relationships between those variables. An agent can be in a situation where she is aware that there are omissions in her state space model (the known unknowns), but she realises that she cannot eliminate them. This result of uncertainty may be compounded by ‘option uncertainty’, where the agent does not know what the consequences of an act of intervention would be at every state,

and/or the agent cannot assign precise utilities to possible consequences (Bradley & Drechsler 2014, p. 1241). Therefore the agent's decision-making is severely hampered. She then has a situation of radical uncertainty in which she has greatly reduced confidence in her decision-making.

In summary, radical uncertainty is recognisable by the presence of *all four* of these characteristics:

1. Radical uncertainty is a situation in which there is inadequate information regarding a particular state of the world; *and*,
2. Further information is not at present available to reduce this uncertainty, and is not likely to be available before there is a need to act; *and*,
3. This lack of information about the space state, its variables, and the relationships of its variables, hinders the construction of an appropriate and practicable state space model that might help understanding, prediction, and decision-making; *and*,
4. Many of the instantiations of radical uncertainty are unique; this uniqueness means that it is not appropriate to group instantiations of radical uncertainty – we are likely to get unreliable results if we choose to group instantiations of uncertainty that seem to have some features in common, if there is not enough evidence to confirm that they actually form an objective group.

Therefore, this kind of uncertainty cannot be given probability estimates, making this uncertainty *unresolvable* (or ineliminable).

The negative effects of radical uncertainty are in *all* of, cognition, understanding, causation, prediction, and decision-making; and, radical uncertainty allows a plurality of views:

1. Radical uncertainty negatively affects an agent's cognition about the situation; *and*,
2. Radical uncertainty hinders explanation for, and understanding of, the present situation; *and*,
3. Radical uncertainty hinders analysis of the mechanisms of the present situation, and the attribution of cause; *and*,
4. Radical uncertainty prevents satisfactory predictions how the present situation will change, including prediction of the outcomes of interventions; *and*,
5. Radical uncertainty gives reduced confidence regarding decision-making to ameliorate or treat the situation, or to prevent further instantiations of this situation; *and*
6. Radical uncertainty allows the existence of a plurality of views of a system; and, radical uncertainty hinders arbitration between this plurality of views.

The presence of these characteristics in any particular situation (such as a medical condition, or for a particular patient in a healthcare encounter) constitutes that situation as a case of radical uncertainty.

Nevertheless, despite the manifest problems due to radical uncertainty, I argue that there are consequences or effects of radical uncertainty which could well be seen as positive, *one or more* of which may occur in any particular situation:

1. Radical uncertainty allows people to choose what they consider to be the appropriate explanations, understandings, mechanisms, causes, predictions, treatment, and prevention regarding the present situation, if those choices are not unreasonable given the limited evidence available. These choices may bring other benefits to an individual such as permitting a desired intervention and rejecting an undesired one; *and/or*,
2. Because of radical uncertainty, people have reduced justification to criticise other peoples' reasonable explanations or choices of intervention on the grounds of irrationality; *and/or*,
3. Radical uncertainty gives some degree of freedom for people to choose to group together instantiations of the present situation that may lead to the application of probabilities to that group or to individuals in that group (although in a condition that is unsatisfactorily defined, inadequately demarcated, and imperfectly unified (such as CFS), these grouping may be inappropriate, so that the probabilities generated may be spurious or unhelpfully imprecise).

In Chapter Three of this thesis, I will critically examine cases where researchers and commentators on chronic fatigue syndrome inappropriately assume that uncertainties about it are surmisable from the available evidence; consequently, in my view, they make inapposite conjectures about what CFS is and what it is not.

In Chapter Four, Section Four of this thesis, I will show how radical uncertainty impacts debates about CFS and decisions made in CFS healthcare encounters. I will describe the two theoretical models that are usually applied to chronic fatigue syndrome, the biomedical model (BMM) and the biopsychosocial model (BPS), both of which purport to ‘show what is going on here’, that is to say they are claimed to show the mechanisms of CFS, including surmising the relationships of variables. These two models each purport to predict the effects of interventions with medical treatments and therefore to aid decision-making. However, these models have dichotomous and incompatible conceptualisations of the mechanisms of CFS, and dichotomous and incompatible treatment recommendations. *One or both* of these models is likely to be incorrect and so will give unhelpful predictions with and without interventions, and will not be able to facilitate choice between different interventions. I argue that the existence of these dichotomies and incompatibilities is explicable if these two models are recognised as being differing responses to radical uncertainty regarding CFS.

In Chapter Seven of this thesis I will describe alternative models for CFS that seems to reflect better what is known and what is experienced, and which may lead to more effective treatment recommendations. In Chapter Seven, Section Six I will show how Borsboom and colleagues’ theoretical dynamic interactive network of symptoms model (Nuijten *et al.* 2016) could be applied hypothetically to chronic fatigue syndrome. This network

model has some structural similarities to a state space model; however, in CFS there is significant paucity of information about the relevant symptomatic and pathological variables and their connections, so this network model is more limited in function than a state space model. However, a network model for CFS can illustrate what *is* known, and it can lead to suggestions for treatment options for individuals, but it does not eliminate uncertainty.

To summarise this section, radical uncertainty is recognisable by its features and its effects. It has profound disadvantages; however, it may well have some advantages for some people some of the time, in that it can enable agents to be considered to be acting not unreasonably when promoting their own interpretations of a situation.

In the next two sections of this chapter, I apply these features of uncertainty to knowledge in health care. In Chapter Three I will specify the multiple uncertainties about chronic fatigue syndrome, and I will argue that the interactions between these uncertainties concerning CFS contribute to radical uncertainty about it. I will match the features of radical uncertainty itemised above to the features and effects of uncertainties about CFS.

2.3: Uncertainties in Healthcare

In this section, I show the existence, characteristics, and consequences of epistemic uncertainty in healthcare (instantiations of which may or may not be classifiable as radical uncertainty). In the first part of this section, I describe various explanations for uncertainty in the *corpus* of medical knowledge, based on how that knowledge is created. In the second

part I describe problems in how that corpus of medical knowledge is transmitted. In the third part, I note that there may be problems with HCPs' reception of transmitted medical knowledge. In the fourth part, I describe uncertainty in the application of that knowledge *to an individual* in a healthcare encounter, which application depends on correctly identifying the patient's disease process and matching that identification to the corpus of medical knowledge.

Absolute certainty is unlikely in a biologically-based science such as medicine. Many commentators recognise this uncertainty: for instance, Bjørn Hofmann notes that,

[M]edicine uses a variety of vague concepts, making it unclear which situations, conditions, and processes that fall under them... [M]edicine deals with many indistinct phenomena resulting in borderline cases ... medicine is based on and produces uncertain knowledge (Hofmann 2022, p. 1151).

So diagnostic categories are fuzzy (Hevey *et al.* 2013). Here Hofmann is referring to knowledge about medical conditions rather than knowledge about the medical situation of a particular patient. Hofmann notes that, "... either one believes that diseases come in natural kinds or as social constructions, they appear on continuums, with numerous borderline cases, and as such are vague" (Hofmann 2022, p. 1153; and as similarly described by Scadding 1996). Here Hofmann's insight can remind us that research on a particular medical condition is hampered if its 'borders' are indistinct, as different researchers could be researching on different populations, thereby reducing the reliability and external validity of that research. ('External validity' refers to the applicability of the results of that research to patients who were not included in that research.)

Fact-value interactions come into play in the borderline cases between health and disease (Hofmann 2022, p. 1162). As an illustration of the fact-value distinction, there is uncertainty regarding the condition called ‘prolonged grief disorder’ because of the unclear distinction of it from ‘normal’ grief (Eisma 2023); this lack of clarity seems to be inherent in the concept of ‘prolonged grief’, because the actual length of grieving inevitably varies between people, and the ‘expected’ length of grieving varies between societies and cultures. Similarly, there is a fact-value borderline in regard to other symptoms: what might be considered normal (although significant) fatigue by one person could well be evaluated by another person as being pathological and part of a disease process and therefore indicative of chronic fatigue syndrome or a similar condition.

Despite the existence of borderline definitional cases such as chronic fatigue and grief, the practice of medicine depends on the existence of a degree of reliability and a degree of objectivity in the corpus of medical knowledge. However, the general practitioner and medical academic Tricia Greenhalgh reminds us that there can be,

... uncertainty *about the evidence, the ‘voice of medicine’* dimension of the visit ...
the completeness, accuracy, relevance of research-based evidence, and on the balance
between potential benefits and harms (Greenhalgh 2013, p. 41) [Italics in original].

Here, Greenhalgh reminds us that uncertainty can be a relevant issue at all stages of medical knowledge, from its creation via medical research to its application to a particular patient in a healthcare encounter. In the next few paragraphs, I describe problems with the creation, the transmission, the reception, and the application of medical knowledge

Maru Mormina expands on possible problems with the *creation of medical knowledge* in research as explanation for uncertainty in medicine. She critiques the objectivity and reliability of much of research-derived medical knowledge because of the way it is produced: she sees, "... structural inequalities in the systems of knowledge production ..." (Mormina 2022, p. 671). These inequalities are due to a, "... monolithic culture of expertise ...", from which, in her view, relevant sources of knowledge creation are excluded (*ibid.*, p. 671). By this means, medical uncertainties are not reduced, they are merely replaced by unreliable knowledge. Mormina defines a 'wicked problem' as one in which, "... facts are uncertain, values in dispute, stakes high and decisions urgent" (*ibid.*, p. 672), to emphasise the difficulty of, and the importance of, a successful and efficacious response to these uncertainties. As an example, she describes what she considers to be the unsatisfactory knowledge creation process during the Covid 19 pandemic, when decision-making certainly was urgent. For her, a wicked problem is, "... not a single problem but a web of problems intersecting in complex ways as a largely unmanageable system" (*ibid.*, p.673), or a, "... a complex interlocking of mutually reinforcing socioeconomic and environmental variables" (*ibid.*, p. 671). (Mormina's (2022) unmanageable intersecting web of problems bears some resemblance in structure (although with different parameters) to the network of uncertainties concerning knowledge about CFS that I will demonstrate in Chapter Three, Section Four, where I argue that the areas of uncertainty concerning CFS intersect to exacerbate each other.)

In response to wicked problems of this kind, Mormina, emphasises the value of *epistemic pluralism*, because that epistemic pluralism can tackle, "... the challenging of structural inequalities in the systems of knowledge production to diversify the social and epistemological foundations of science" (*ibid.*, p. 671). By 'epistemic pluralism' here, she is

referring to the many sources of knowledge that are available, and in her view should be used. Here she is alluding to (what she considers to be) the inappropriate underuse of, “... the ‘*context expertise*’ of those who experientially know about the issues at stake and feel the impact on their everyday lives” (*ibid.*, p. 681) [italics in the original], to reduce the influence of the, “... narrow ‘medical gaze ..’” (*ibid.*, p. 680) that downplays the relevance and value of patients’ contextual expertise.

In Chapter Three, I will demonstrate how Mormina’s analysis of epistemic monoliths and epistemic pluralism gives an explanation for the perpetuation of opposing conceptualisations of CFS and concomitantly oppositional research programme for it, because I see two ‘monoliths’ in chronic fatigue syndrome research and knowledge creation, one related to the biomedical model and the other to the biopsychosocial model.

Some knowledge-gathering about chronic fatigue syndrome does indeed use the context expertise of the experience of patients, as Mormina recommends. However, much biomedical CFS research goes further and *prioritises*, rather than just uses, the expertise of patients. This prioritisation enables the creation of a monolithic, rather than a pluralistic, research programme, which investigates only biomedical causes of and treatments for CFS, and plays down the contribution of possible psychological and behavioural factors (as described in the review articles by Cortes Rivera *et al.* 2019; Twisk 2019; Lidbury & Fisher 2020; Thoma *et al.* 2024).

In opposition to this biomedical research programme, there is a contrasting and oppositional monolith, where some medical researchers emphasise the psychological and behavioural

causes of CFS symptoms, while de-emphasising biological factors (for instance, White *et al.* 2011); so these researchers concentrate on cognitive and behavioural factors and treatments.

Researchers on both the sides of this divide construct scientific theories within their own theory-laden world view, which view is incommensurable with oppositional views (Veatch & Stempsey 1995, p. 253). Consequently, in CFS knowledge creation we have two monoliths, each of which is epistemically non-pluralistic. In line with Mormina's (2022) understanding, one *or both* of these interpretations is leading to unreliable knowledge, and so is not contributing to the reduction of uncertainty.

Problems can arise in the *transmission of medical knowledge*. For the sake of ease of use, medical knowledge (the medical evidence base) is distilled into 'clinical practice guidelines' and then transmitted to healthcare professionals. These guidelines rely on the consensus of expert committees or panels (sometimes with patient representatives) who analyse the evidence base and summarise it into recommendations for clinical practice. However, this method may not lead to appropriately reduced uncertainty: there may be, "... mistaken consensus because individual panel members are influenced by the recommendations of other (often more influential) members of the panels" (Djulfbegovic *et al.* 2011, p. 341). Over-influential panel members who are professionals may choose to promote the results of a specific research programme rather than fulfilling their expected panel role of collating, appraising, and distilling the results of many relevant research programmes.

Alternatively, and equally unsatisfactorily in her view, Marjolein de Boer claims that ME/CFS patient representative panel members were over-influential in their contribution towards a Dutch Health Council's recommendations regarding ME/CFS and its treatment

recommendations, where these lay panel members had (in de Boer's view) an inappropriate and unhelpful bias towards a somatic biomedical understanding of ME/CFS (de Boer 2021). There seems to have been a similarly unsatisfactory process of guideline creation when NICE (the British National Institute for Health and Care Excellence) were formulating new guidelines for ME/CFS in 2021. Four medical professional members resigned not long before the planned date of publication of those guidelines, with the suggestion that these professionals did not agree with the process of evaluating evidence, nor with the recommendations of the guideline in regard to exercise therapy and cognitive behaviour therapy (Torjesen 2021; Iacobucci 2021; White *et al.* 2023).

Healthcare professionals' *reception of medical knowledge* from expert panels (or from elsewhere) can be problematic. Healthcare professionals are the intended recipients of clinical guidelines, but some HCPs may either not read those guidelines that have been transmitted to them; or, if they do read them, they may have their own theory-laden world view which leads them to re-interpret or dismiss these recommendations (see Veatch & Stempsey 1995, pp. 261, 264).

In healthcare encounters HCPs need then to apply the relevant part of the corpus of medical knowledge to individual patients. The HCP needs to fit the patient's narrative into a coherent and recognisable pattern of symptoms, then to decide whether that set of symptoms fits various medical diagnoses, then whether there is adequate medical knowledge about that diagnosis. If the patient is not convinced about that diagnosis, the healthcare encounter is likely to be unsatisfactory. Next, in regard to treatments, there is value in some degree of certainty derived from medical research and transmitted via guidelines concerning the advantages and disadvantages of various treatment options for that diagnosis; and, lastly, the

patient and HCP need confidence in their shared process of decision-making which also takes patient preferences into account. If the patient is not convinced about the value and applicability of the HCP's treatment recommendations, the healthcare encounter is likely to be unsatisfactory.

Furthermore, as Greenhalgh reminds us, in some healthcare encounters there is, "... uncertainty about *the patient's story* – the 'voice of the lifeworld' dimension ..." (Greenhalgh 2013, p. 41) [Italics in original]. There is, "[t]he inability to determine the meaning of illness-related events resulting from ambiguity, complexity, unpredictability of illness, deficiency of information about one's illnesses and its consequences" (Politi *et al.* 2007, p. 682). So, here, Greenhalgh and Politi *et al.* are telling us that it may be that it is not certain what is wrong with the patient, so it may be equally *uncertain what aspect of medical knowledge ought to be applied*. Kay and King remind us that uncertainty of this kind in medical practice might never be solved; they quote Rittel and Webber's dramatic phrasing:

Rittel and Webber [(1973)] distinguished the tame problems which had been solved from the 'wicked' problems which might never be solved, and these terms are now often used in social policy and medicine. A broken leg is a tame problem; but many patients present with symptoms whose cause is difficult to diagnose and which require treatment whose outcomes are uncertain. Doctors must tackle wicked problems (Kay and King 2020, p. 22).

In this passage, Kay and King are referring to those medical problems that are difficult to diagnose and difficult to treat because of inadequate evidence, in contrast to leg fractures which are typically easy to diagnose and typically straightforward to treat. I understand that

Kay and King are using Rittel and Webber's (1973) quotation to mean that 'wicked' medical problems are those that, "... might never be solved" (Kay and King 2020, p. 22); they are those to which the answer to the question, "What is going on here?" (*ibid.*, p. 10) is that, "[w]e do not and cannot have ..." the necessary knowledge, and, "[w]e do not make good decisions by professing knowledge that we do not and cannot have" (*ibid.*, p. 12). I will show in Chapter Three that 'wicked' medical problems of this kind exist in the corpus of medical knowledge about CFS and also in CFS healthcare encounters.

However, Gifford reminds us that decisions have to be made even in the face of uncertainty:

Physicians find themselves in contexts where the data are incomplete, and from various sources, and where it can be said of each source that there are epistemological difficulties in inferring something about the medical case before them at the moment. Yet those physicians need to act (Gifford 2011, p. 7).

Here Gifford describes the not uncommon situation of uncertainty in a medical encounter, in which a doctor may not have enough evidence, but still has to decide what to do for her individual patient. As Greenhalgh says, there may well be, "uncertainty about *what best to do for the particular patient* ..." [so there is uncertainty] how tacit knowledge informs clinical judgement..." (Greenhalgh 2013, p. 41) [Italics in original]. (In Chapter Six, "Epistemic Injustice in Healthcare and in Chronic Fatigue Syndrome" of this thesis, I will critically analyse health care professionals' actions in the face of the uncertainties regarding CFS.) Gifford sees that physicians, "... need to act..." (Gifford 2011, p. 7), whatever the level of certainty or uncertainty. Here, Gifford is not incorporating the concept of joint decision-making between HCPs and patients. Against Gifford, Greenhalgh asserts that both patient

and HCP need to assess what is going on and what should be done about it: "... [C]are becomes a *collaborative endeavour* with its mix of human-human ... interactions (Greenhalgh 2013, p. 41) [italics in original]. Both have access to medical evidence, but there also may be incommensurability here; the patient and HCP may interpret that medical evidence differently from each other: "The patient who has a different understanding of the nature of causation, evidence, probability, and disease will have a different understanding of the clinician's report" (Veatch & Stempsey 1995, p. 264). Here, Veatch and Stempsey are comparing the understanding of the patient to the understanding of the clinician. These differing understandings may add to the divergent values of the patient and the HCP, allowing these two to come to different conclusions regarding what is going on and what should be done about it. By this means, dichotomies of views can exist even in the face of the same medical scientific evidence.

To summarise this section, Kay and King (2020), Rittel and Webber (1973), and Mormina (2022) all describe 'wicked problems', the situations where we cannot obtain the necessary knowledge, which I take to be an evaluation attribution of radical uncertainty. Djulbegovic *et al.* (2011) describe the situation where the uncertainty is irreducible, where neither classification nor the monitoring of the effects of treatment are standardised, and remind us that uncertainty is also a cognitive state. Hofmann (2022) notes the difference between the concept of diseases as natural kinds or as social constructs, and that disease constructs can be unsatisfactory because of their imprecision. Andreoletti *et al.* (2022) note uncertainty due to the absence of adequate models. Mormina (2022) conceptualises medical uncertainty as an unmanageable intersecting web of problems, where, "... facts are uncertain, values in dispute, stakes high and decisions urgent ..." (*ibid.*, p. 672). She also emphasises the differences between the HCP's scientific knowledge and the patient's experiential knowledge

(as do Pomey *et al.* 2015), and that medical knowledge may be unsatisfactory if it is created in a 'monolith'. Gifford (2014) reminds us that in medical uncertainty a decision has to be made despite the paucity of adequate data. He sees that it is for the doctor to decide, while Greenhalgh sees that deciding should be a joint process between patient and doctor. Greenhalgh (2013) reminds us that in a healthcare encounter medical evidence may be incomplete, and the patient's story may not be easy to interpret. Politi *et al.* (2007) note that there is variability in the courses of healthcare encounters, due to HCPs' differing responses to uncertainty, and possible differences between HCPs' and patients' values. Veatch and Stempsey (1995) point out that medical uncertainty allows incommensurability between patients' and HCPs' understanding, arising from their distinct scientific frameworks. There is therefore, firstly, uncertainty concerning medical evidence about some medical conditions, echoing the concerns of Politi *et al.* (2007) and Mormina (2022). Secondly, there may be uncertainty about the patient's story, that is to say, the problem of matching the information from the patient to a specific medical condition. Thirdly, there may not be agreement between patient and HCP about what is wrong, and what should be done for the patient regarding what treatments are available, and which treatments should be recommended.

I conclude this section by repeating that these contributions show us that there is potential for uncertainty due to inadequacy of medical knowledge, and due to how that medical knowledge is disseminated to and received by health care professionals. Furthermore, in a health care encounter there is potential for uncertainty regarding what is wrong with the patient, that is to say, the analysis and categorisation of the patient's problems. There is also the possibility that the patient and the healthcare professional may disagree about the diagnosis or about treatment recommendations. As a result, determining the best management of the patient's problems can sometimes be hindered.

In the next section I show how some of these uncertainties manifest in two illustrative medical conditions, myxoedema and Charles Bonnet syndrome.

2.4: Certainties and Uncertainties Regarding Myxoedema and Charles Bonnet Syndrome

Before I describe the epistemic uncertainties regarding chronic fatigue syndrome in Chapter Three, I first give the examples in this section of two medical conditions, myxoedema and Charles Bonnet syndrome (CBS), that serve as contrasts with each other *and* with CFS.

Myxoedema is a condition whose ontological status is settled, and which is therefore largely uncontroversial. There is significant uncertainty about CBS, but little controversy.

There is objective evidence that those who have the diagnosis of myxoedema (otherwise known as underactive thyroid gland or hypothyroidism) share some identifiable pathological process, that is to say, failure of the thyroid gland to secrete enough thyroid hormone. There are a number of different known causes for the failure of the thyroid gland, including surgery and auto-immunity. Myxoedema can also be identified by an unusual clustering of symptoms and signs: an insidious development of a slowing down physically and mentally, weight gain but reduced appetite, a change in facial appearance, and other symptoms. The first doctor to identify myxoedema, Sir William Gull, relied on these symptoms and signs before the invention of blood tests for myxoedema (Pearce 2006). In modern times, the identification of myxoedema in an individual has been refined to include the specific abnormalities in that patient's blood thyroid function tests (TFTs), which abnormalities can be considered to be a biomarker. The thyroid function test includes measurement of

thyroxine (T4) and thyroid stimulating hormone (TSH). Many patients who are diagnosed with myxoedema have a known cause, shared with many other patients, for instance, previous damage to the thyroid gland by medication, surgery, or radioactivity. (These patients may well have had these interventions in the past for treating an overactive thyroid gland or cancer). Finally, all patients with myxoedema are likely to respond favourably to medication that replaces the deficient thyroid hormone; and, the progress of successful treatment is monitored with the same blood tests.

There are admittedly a small number of outliers where the patient's TFT results do not give clarity, for instance where the patient has symptoms that suggest myxoedema but either the T4 level is not at the level that is considered abnormally low when first tested or the TSH is at a level that is not considered to be abnormally high. However, despite the existence of these outliers, medical knowledge about myxoedema is adequate. In general there is little uncertainty about the pathological mechanism that initiates and then perpetuates hypothyroidism; all those with myxoedema share the lack of thyroxine; there is considerable understanding of how the symptoms and course of (untreated) hypothyroidism can be explained by the lack of thyroxine; for most patients there is a match between symptoms and biomarker levels on diagnosis; there is understanding of why the biomarkers are a reliable guide in diagnosis and for monitoring treatment; and, there is understanding of how the standard treatment functions to reduce symptoms and improve biomarker levels. So, for most patients with myxoedema, the biomarker results are a good guide to the patient's diagnosis, prognosis (without treatment), and their likely response to treatment, thus aiding decision-making about choice of treatment. There is therefore little uncertainty concerning myxoedema—about what is wrong with the patient and what treatment will be effective—and certainly no radical uncertainty.

In contrast to myxoedema, in chronic fatigue syndrome the precipitating and perpetuating pathological mechanisms have not been elucidated, there is no biomarker to aid diagnosis and to aid in the monitoring of progress, and there is no standard treatment. The absence of these characteristics in CFS add to the uncertainties and controversies about it.

I now describe the features of Charles Bonnet syndrome (CBS). Even though the mechanisms of CBS are underexplained, CBS is a less contested condition than CFS/ME. My view is that contestation is reduced because CBS has a unique feature: it is characterised by episodes of visual hallucinations affecting people with poor vision, but who are not showing evidence of mental illness, dementia, or objectively-diagnosed neurological disease: “... [V]isual hallucinations are common in dementia [and] Parkinson’s disease ...” (Badcock *et al.* 2020, p. 1382). A typical case history of CBS is given by the medical doctors Murali Krishna and Peter Decalmer:

A 78-year-old gentleman ... has informed the police about ‘the men’ he has seen walking around in his house, describing them as white men, six feet tall, well built and shabbily dressed’. He sees them at least twice daily. Though annoyed by their presence, he denies feeling fearful or persecuted and has made attempts to chase them away; however, he says they leave only to return. He has partial insight and does not deny the possibility of his visions being the product of his imagination (Krishna & Decalmer 2006/2024, ‘Case Scenario’).

This condition was first recognised by the naturalist Charles Bonnet in his grandfather, and then, much later, in himself:

During his latter days Bonnet's grandfather, Charles Lullin, who had low vision from cataract[s] but was otherwise psychologically sound, had experienced complex visual hallucinations of men, women, birds and buildings. Charles Bonnet himself experienced similar phantom visions when his own vision deteriorated (Krishna & Decalmer 2006/2024, 'History of Charles Bonnet syndrome', no page numbers).

After his cataracts were removed, Lullin ceased to suffer the visual hallucinations, but they returned after his vision later deteriorated (Rohas & Gurnani 2023, p. 2). Bonnet therefore had evidence that the hallucinations were related to poor vision, and seemed to Bonnet to be not related to any deterioration in brain function.

The Brain Charity provides a helpful summary of the current view of the symptoms of CBS:

The main symptom of Charles Bonnet syndrome is visual hallucinations which are extremely vivid and entirely silent. Everyone's experience of Charles Bonnet syndrome is different. Hallucinations may appear frequently or infrequently; be benign or frightening and disturbing and can last seconds or minutes. Images seen can include:

- Simple patterns,

- Words, numbers, maps or musical notes,

- People, animals, plants, insects, rodents, reptiles, buildings, vehicles, fire or water,

- Scenes of people of different sizes, and/or

- Groups / armies often wearing costume, such as Edwardian, military or medieval.

(<https://www.thebraincharity.org.uk/condition/charles-bonnet-syndrome/>) (accessed 10/1/25).

As the Brain Charity's contribution above states, there is wide variation in people's experience of the hallucinations in CBS: for instance, what is experienced (simple patterns or people, etc.), in the timing (frequent or infrequent, lasting seconds or minutes), and in the effect on the person (experiencing them as benign, neutral, or frightening). The Charles Bonnet Syndrome Foundation record the variety of people's experience of CBS (Charles Bonnet Syndrome Foundation, 'Personal Stories', and a personal story can be found on YouTube (Living with Charles Bonnet syndrome | Nathan's story).

As in CFS/ME, the mechanisms of CBS are not yet established; CBS seems to be related to reduced stimulation of the visual cortex of the brain ('de-afferentiation' – meaning a lack of information going to the brain) and may be due to some mechanism equivalent to phantom limb syndrome (Krishna & Decalmer 2006/2024). As with CFS/ME, there are no universally effective medications for CBS. However, if the patient's visual impairment is treatable—for instance, by surgery to remove cataracts—the patient's CBS symptoms may well be eradicated.

As in CFS/ME, the symptoms of CBS are all subjective – there is no objective evidence of the hallucinations for the medical professional to elicit. In addition there is no biomarker in CBS, so there is no way for a medical professional to confirm the diagnosis objectively—the diagnosis is made only on the history provided by the patient. In contrast to CFS/ME, CBS patients may well have some objective physical signs, in that CBS patients are likely to have evidence of reasons for their visual loss; although, they have no objective physical signs that

confirm the presence of hallucinations. Furthermore, the diagnosis of CBS can be made only after the exclusion of other causes of hallucinations, such as brain tumour, mental illness, or dementia. Exclusion criteria for CBS would usually be normal vision: a patient with visual hallucinations and objectively normal vision is less likely to be diagnosed with CBS, and a patient complaining of visual loss that itself is not explainable objectively would also not be given the diagnosis of CBS (Rojas & Gurnani 2023). In a patient with a mental condition such as psychosis or brain disease such as dementia as well as visual impairment would be likely to have his or her hallucinations attributed to that psychosis or dementia rather than to CBS.

Charles Bonnet syndrome is not free of uncertainty, controversy, or unsatisfactory consultations, but I understand that these are less intense and fewer in proportion compared to those of CFS/ME. The uncertainty results from the lack of understanding of its mechanisms, the lack of a biomarker, as well as the lack of effective medication. This uncertainty is reflected in controversy about the diagnostic criteria according to Rojas & Gurnani (2023): some diagnostic criteria allow the presence of mental disease or dementia and some not, while some experts do not require the presence of visual loss (Krishna & Decalmer 2006/2024, 'Diagnostic Criteria'). Podoll *et al.*'s criteria do not mandate reduced vision to be present, although they do acknowledge that, "... reduced vision can be found in the majority of patients" (Podoll *et al.* 1989, p. 43). However, according to the later criteria of Krishna and Decalmer, the diagnosis of CBS is characterised by complex visual hallucinations *generally* against the background of visual impairment and in the absence of a neuropsychiatric disorder (Krishna & Decalmer 2006/2024).

The lack of consensus regarding the diagnostic criteria for Charles Bonnet syndrome is reflected in the disparate estimates of the prevalence of CBS in different samples. These estimates, “vary from a small percentage (around 1%) to a relatively large percentage (about 10%)” (Shiraishi *et al.* 2004, p. 207), or 1.4% to 18% of those with poor vision (Jurišić *et al.* 2018). Shiraishi *et al.* attribute this large disparity to the differing diagnostic criteria being applied; some criteria do not specify the presence of optic disease. Another possible reason for this disparity is that some patients who seem to have the symptoms of CBS may actually be in the early stages of dementia, before that dementia is obvious, or diagnosable. In some of these people, their visual hallucinations will be attributed to CBS, and in others to dementia, according to the opinion of their medical professional, thereby adding to the disparity of prevalence estimates. In addition, “CBS is often missed [in] the elderly age group due to [patients’] fear of being labelled as mentally retarded and phobia of admitting hallucinations” (Rojas & Gurnani 2023, p. 2) (also, “Mrs L.”, “Mrs B.”, Charles Bonnet Syndrome Foundation (accessed 11/1/25)). Patients’ reluctance to report symptoms may be the reason why, “... this syndrome is frequently overlooked or misdiagnosed by both ophthalmologists and psychiatrists” (Siatkowski *et al.* 1990, p. 215) (also, Jurišić *et al.* 2018; Charles Bonnet Syndrome Foundation (accessed 11/1/25)).

However, the controversy about definitions and diagnosis of CBS seems only to be between experts; this controversy seems to be peripheral to patients’ experience, and peripheral to healthcare encounters about CBS, according to a CBS patient support website <https://www.charlesbonnetsyndrome.uk/> (Charles Bonnet Syndrome, ‘Esme’s Umbrella’ (accessed 11/1/25)) and a website where patients’ narratives are recorded (<https://www.charlesbonnetsyndrome.org/index.php/cbs-ii/personal-stories>). (Charles Bonnet Syndrome Foundation, ‘Personal Stories’ (accessed 11/1/25)).

Unsatisfactory healthcare encounters about CBS do occur. In the examples, “Mrs T.”, “Mrs M.”, “Mrs A.”, “Mrs D.”, and “Mrs H.” (Charles Bonnet Syndrome Foundation, ‘Personal Stories’ (accessed 11/1/25)), the health professional does not assign the diagnosis of CBS when that diagnosis is appropriate. However, this lack of assigning seems to be due more to the health professional’s ignorance of CBS rather than due to a challenge to the patients’ testimony about reality of their experiences, or a challenge to the patient’s understanding of their experiences. It therefore may not be appropriate to classify these unsatisfactory healthcare encounters as being epistemically unjust according to Fricker’s definitions (Fricker 2007).

There is little evidence of dichotomies in the conceptualisations of Charles Bonnet syndrome: experts, patients, and CBS patient support groups all conceive of the visual hallucinations of CBS as psychological experiences that seem to be due to the brain’s response to a physical visual problem. The images are not real, but the experience of the images is real (Jurišić *et al.* 1990; Siatkowski *et al.* 1990; Krishna & Decalmer 2006/2024; Rohas & Gurnani 2023; The Macular Society November 2021; Guide Dogs, ‘Living with Charles Bonnet Syndrome – Nathan’s Story’ (accessed 12/1/25); Charles Bonnet Syndrome Foundation, ‘CBS: Personal Stories’ (accessed 11/1/25)). Moreover, the absence of effective medications for CBS explains the lack of opposing viewpoints regarding treatments (Rojas *et al.* 2023). By contrast, I will give evidence for dichotomies of conceptualisations and treatment recommendations in CFS/ME in Chapter Four, Section Four.

Charles Bonnet syndrome therefore shares some of its fundamental characteristics with chronic fatigue syndrome: the hallucination symptoms of CBS are subjective, lacking any

objective evidence; there are differing diagnostic criteria for CBS (regarding the confirmed presence of visual impairment, and the confirmed exclusion of neuropsychiatric disease), so the homogeneity and demarcation of CBS are not definite; consequently, there are disparate estimates of prevalence; research has not elucidated the mechanisms for CBS; research has not led to the discovery of a biomarker; and, research has failed to discover curative treatments or widely-effective symptomatic treatments for it. However, I argue that the uncertainties about CBS are not *radical* because it has unique features. In my view, these unique features explain why CBS does not have the disputed status that CFS has; there is an absence of dichotomous views of the nature and causes of CBS; and, CBS patient support websites align to the views of medical experts regarding its nature and management.

There is evidence of unsatisfactory CBS healthcare encounters, but those episodes are due to healthcare professionals' ignorance about CBS, not to epistemic uncertainties about CBS. I attribute the paucity of disputes about CBS to the non-ineffability of the visual hallucinations, which can be described by the person living with CBS using language that makes it easy for others to appreciate what the experience is: for instance, the patient sees men walking around his house when other people can objectively confirm that there are no men walking around the house.

By contrast, symptoms such as 'fatigue', etc. that are attributed to CFS are expressed in terms that are also used for quotidian experiences – tiredness, exhaustion, malaise, muscle pain, unrefreshing sleep, difficulty with concentration and memory, etc. The severity of CFS symptoms and their deleterious effects are not easy to convey, so a healthcare professional may fail to appreciate what a CFS patient is experiencing if she uses these mundane-sounding terms. Furthermore, some CFS symptoms and some CSF biochemical abnormalities are

similar to those of deconditioning (Jewell 2012; Joyner 2012; Kreher & Schwartz 2012; Jurdana *et al.* 2015; Cadegiani & Kater 2019; Jason *et al.* 2020; Pheby *et al.* 2020; W. T. Lim & Torpy 2023; Walitt *et al.* 2024), or overtraining (Kreher & Schwartz 2012; Cadegiani & Kater 2019), so an HCP may feel that cognitive and exercise therapies are indicated, which is in opposition to the views of many CFS patients and their support and advocacy groups.

I conclude this diversion into the well-understood condition, myxoedema, and the poorly-understood condition, Charles Bonnet syndrome, by repeating that, though there is significant uncertainty concerning the latter, there is little evidence of epistemic injustice in healthcare encounters concerning either of them, because there is a lack of *radical* uncertainty. I explain this contrast with CFS as follows: both myxoedema and Charles Bonnet syndrome are fairly easy to recognise and are reasonably well demarcated from other medical conditions; there are no significant disputes about their underlying mechanisms; and, treatments recommendations for them lack the contentious dichotomies seen in CFS. By contrast, the symptoms of CFS are not unique, and it has there are oppositional mechanism attributions, with consequent oppositional treatment recommendations. I will return to the issue of epistemic injustice in healthcare encounters later in this thesis, in Chapter Six: ‘Epistemic Injustice in Healthcare, and Chronic Fatigue Syndrome’.

2.5: Conclusion to Chapter Two

In this chapter, I described the concept of radical uncertainty that I use later in this thesis to categorise knowledge and uncertainties about chronic fatigue syndrome; I described how radical uncertainty can be recognised; I listed the disadvantages and the possible advantages

of radical uncertainty in debates concerning CFS. I then described two dissimilar medical conditions, myxoedema and Charles Bonnet syndrome, in order to contrast knowledge and uncertainties about them with knowledge and uncertainties about CFS, which latter I examine in more detail in the next chapter.

3: Chronic Fatigue Syndrome and its Uncertainties

3.1: Introduction to Chapter Three

In the previous chapter, I have described uncertainty and radical uncertainty. I have shown aspects of uncertainty regarding the creation, transmission, reception, and application of medical knowledge. I have shown the levels of certainty and uncertainty in two medical conditions.

In this chapter, I describe the features that are generally attributed to chronic fatigue syndrome, and its diagnostic process. I highlight that CFS is considered by many to be a contested category. I next itemise the many areas of epistemic uncertainty regarding CFS. These uncertainties are manifested in least three main unsatisfactory epistemological deficits concerning CFS: there is uncertainty about whether CFS/ME exists as a distinct entity; whether it is a purely physical disease or whether psychological and behavioural factors also have a causative role; and, in a healthcare encounter, uncertainty whether a particular patient has the condition or not. There is problematic lack of knowledge about the cause[s] of CFS, about how it can be diagnosed, about the pathological processes that perpetuate the symptoms, about how these pathological processes cause the particular symptom pattern and course of the illness in any particular patient, and about treatments. There is a similar lack of certainty about its classification. I argue that the multiple epistemic uncertainties concerning CFS interconnect in a network of uncertainties that results in there being a situation of radical uncertainty. I match the uncertainties concerning CFS to the characteristics of radical uncertainty that I described in Chapter Two, Section Two. I conclude that radical

uncertainty concerning knowledge about chronic fatigue syndrome leads to unresolvable uncertainty in CFS healthcare encounters.

3.2: The Symptoms of Chronic Fatigue Syndrome, and Controversies Concerning It

I now describe the symptoms that lead to a diagnosis of chronic fatigue syndrome. Before these symptoms commence, the individual has usually been healthy. The onset of CFS symptoms is fairly abrupt, often following symptoms that suggest a viral illness. There are many symptoms that are attributed to CFS, including musculo-skeletal pains, concentration and memory problems, unrefreshing sleep, with many other intrusive and disabling symptoms. The collection of symptoms and the severity of these symptoms differ from patient to patient, and fluctuate in individual patients over time. For many sufferers, the most prominent symptom is disabling post-exertional malaise (PEM), which is experienced as being much worse than ordinary fatigue, and is often associated with the exacerbation of the other symptoms. PEM can follow a short time (some hours or a day or two) after any form of exertion, whether physical, psychological, emotional, or cognitive; this malaise is disabling and can last days, weeks or longer. Other CFS symptoms are often exacerbated during episodes of PEM. Sufferers respond to the likelihood of PEM by avoiding or significantly limiting these forms of exertion, with consequent effects on many aspects of their lives. The condition usually lasts for years, often greatly interfering with patients' lives, although it is not usually fatal (W. T. Lim & Torpy 2023).

There is usually nothing for the doctor to find on examination of the patient; also, special tests such as blood tests and imaging usually do not show any abnormality, and there is no

biomarker for CFS/ME. A biomarker is a specific test (often a blood test) that can enable a confident diagnosis of a particular medical condition, enabling the inclusion of all of those with the condition and the exclusion all those without the condition. Many medical conditions have a biomarker of that kind, for instance, there is a reliable blood test called the thyroid function test for myxoedema (underactive thyroid gland) which is used for diagnosis and demarcation, and for giving inclusion and exclusion criteria for research. Furthermore, the biomarker in myxoedema gives an indication of the severity of the condition on diagnosis; and tracking biomarker levels over time enables the monitoring of progress of the condition and the assessment of response to treatment.

The various symptoms attributed to CFS/ME are shared with many other conditions, so the diagnosis of CFS/ME is made only on the recognition of the grouping of the symptoms attributed to it, their severity, their effects on the patient's lives, and the course of the illness. Diagnosis also depends on excluding other more easily identified conditions that can give similar symptoms (Centers for Disease Control, 'Symptoms of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome' (accessed 8/8/24); Mayo Clinic, Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (accessed 8/8/24); National Health Service, 'Chronic Fatigue Syndrome' (accessed 12/8/24); Komaroff 2025; MSD Manual (accessed 12/1/25), Chronic Fatigue Syndrome; Arron *et al* 2024).

Philosophical commentators and other commentators note that CFS is called 'contested', 'controversial', and/or 'unexplained'. Those who describe CFS as 'contested' include Blease *et al.* 2017; Karfakis 2018; Spandler & Allen 2018; Franssen 2020; and de Boer & Slatman 2023. Hossenbaccus and White note that CFS has, "... provoked much controversy and led to arguments between the medical profession and patient organizations" (Hossenbaccus &

White 2013, p. 1). Other commentators note that CFS is subject to ‘controversies’ (Fitzpatrick 2002; Guise & McKinlay 2010; van der Meer 2021; Blease *et al.* 2017; Jason *et al.* 2020; Fennell *et al.* 2021). Tucker (2004, p. 153) calls CFS a, “controversial illness”; Nacul *et al.* (2019, p. 1765) call ME/CFS a, “controversial diagnosis”. L. Cooper (1997) notes that Myalgic Encephalomyelitis (ME) and Chronic Fatigue Syndrome (CFS) have controversial statuses. W. T. Lim and Torpy describe chronic fatigue syndrome as, ‘enigmatic’ (W. T. Lim & Torpy 2023, p. 1). Others see that CFS and/or its symptoms are ‘unexplained’ (Fitzpatrick 2002, Nisenbaum *et al.* 2004, Harvey & Wessely 2009; Wojcik *et al.* 2011b; O’Leary 2018; Franssen 2020; Haan & Korsten 2020; Missailidis *et al.* 2019; Twisk 2019; Han & Korsten 2020; Fatt *et al.* 2021; Lubet & Tuller 2021).

These references to contestation, controversy, and lack of explanation reflect uncertainty regarding almost all aspects of CFS—its causes, its mechanisms, its course, its definition, its diagnosis, its classification, and its treatment recommendations. The cause[s] for the onset and perpetuation of the symptoms of CFS/ME is/are unknown: “[T]he aetiology of CFS is unknown. No infectious, hormonal, immunologic, or psychological cause has been established” (MSD Manual, Chronic Fatigue Syndrome (accessed 12/1/25)), and, “[S]cience has not yet identified the pathological cause or ongoing disease process for ... ME/CFS” (Lubet & Tuller 2021), and, “[In] post-infectious myalgic encephalomyelitis/chronic fatigue syndrome ... [t]he physiological mechanism responsible for the persistence of fatigue and related symptoms has yet to be determined” (Walitt *et al.* 2024). Although abnormal pathological results have been identified in some CFS/ME patients (Ali & Kheirabadi 2025), they are of limited practicality: these abnormalities are not present in all, they do not adequately explain the symptoms and course of CFS/ME, and they have not enabled the discovery of an effective cure. The non-specificity of most of the symptoms and their

subjective nature, the lack of abnormal findings on examination and on special tests, the lack of a biomarker, and the lack of adequate pathological elucidations all explain my description of CFS/ME as being subject to uncertainty. Other authoritative medical organisations acknowledge this uncertainty, for instance, “[T]he cause of ME/CFS is not known” (Centers for Disease Control, ‘Symptoms of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome’ (accessed 8/8/24)); “The cause of ME/CFS is unknown, although there are many theories” (Mayo Clinic, Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (accessed 8/8/24)); “The cause of ME/CFS is unknown” (National Health Service, ‘Chronic Fatigue Syndrome’ (accessed 12/8/24); similarly, Komaroff 2025). Spandler & Allen (2018) note that the understanding, framing and treatment of ME/CFS are subject to heated debate. Diserholt describes chronic fatigue syndrome’s diagnostic category as being, “... questionable and mysterious ...” (Diserholt 2020, p. 9). Guise *et al.* see that, “.. the aetiology, symptoms, diagnosis and treatment of ME/CFS are controversial” (Guise *et al.*, 2010, p. 426). The health researchers Simon Horton and colleagues complain that there are problems with, “... the diagnostic process [for CFS/ME], especially the degree of uncertainty which may be shared by primary care physicians and patients alike” (Horton *et al.* 2010, p. 1). Prins *et al.* (2006) note the, “... several controversies over CFS with respect to its definition, diagnosis, pathophysiology, and treatment” (p. 346). Sharpe & Greco (2019) also note controversy regarding treatments, and many others highlight uncertainty about what treatments should be recommended (as also noted by Friedberg 2016; Sharpe & Greco 2019; Wilshire & Ward 2019; Twisk 2019; L. Smith & Archer 2020; Kim *et al.* 2020; Lubet & Tuller 2021). There are disputes regarding its appropriate classification (Jason *et al.* 2010, O’Leary 2018, Nacul *et al.* 2019). There are related disputes about whether a biomedical model is appropriate (Bayliss *et al.* 2014, Karfakis 2018, Missailidis *et al.* 2019). A biomedical model is

inappropriate, Wojcik and colleagues regret, because of, "... the absence of findings [that would confirm] the presence of organic disease ..." (Wojcik *et al.* 2011b, p. 503).

In this section, I have indicated the variety, severity, and intrusive nature of the symptoms of chronic fatigue syndrome, and the impediments to diagnosing it. I have highlighted some of the controversies about it, and the widespread recognition of uncertainties about it. In the next section I describe in more detail some of the uncertainties about chronic fatigue syndrome, and I give explanations for those uncertainties.

3.3: The Areas of Uncertainty Regarding Chronic Fatigue Syndrome

3.3 (a): Introduction to Section Three

What are the epistemic uncertainties about CFS, why are there so many, and why do they persist? In the following subsections, I itemise the many uncertainties regarding CFS concerning definitions, ontology, nomenclature, demarcation, biomarkers, homogeneity, subtypes, precipitating causes, perpetuating causes, and mechanisms; and, I describe the lack of adequate progress of CFS research on mechanisms and treatments, as explicated in review articles. In Section Four, I show how the elements of uncertainty are interconnected in a network of uncertainties; I then argue that this interconnectedness explains the persistence of these uncertainties, and give one explanation for the contested status of CFS..

3.3 (b): The problem of differing definitions for chronic fatigue syndrome

A disease definition identifies the criteria for diagnosis. In this subsection, I describe how disease definitions are created and verified. I then show that there is a lack of a generally-agreed definition for CFS, and I argue that this lack impacts CFS, impedes research, hampers estimates of prevalence, and impacts CFS healthcare encounters negatively.

The purpose of a precise and generally-agreed definition for a medical condition is that it allows the identification of individual patients who have that condition. van der Meer & Lloyd (2012) explain the differing reasons for creating diagnostic criteria:

One of the first questions in this process is: what is the purpose of the criteria? It makes quite a difference whether they intended for research – in which case specificity is paramount (that is, do the criteria reliably identify only subjects with the disorder?). By contrast, if the purpose is epidemiological, for instance to understand the burden of disease in the community, sensitivity becomes paramount. Finally, if the intent is clinical – that is to facilitate the diagnosis in individual patients – a mix of these parameters is relevant (van der Meer & Lloyd 2012, p. 29).

So, according to van der Meer & Lloyd (2012), there are three distinct applications for disease definitions:

- Firstly, for identifying patient groups for research on pathological mechanisms or treatments;
- Secondly, for epidemiological estimates to give a measure of the burden of the medical condition in the community; and,
- Thirdly, to facilitate the diagnosis of individual patients in healthcare encounters.

In the above extract, van der Meer & Lloyd tell us that a definition for the purposes of research on mechanisms and treatments should be specific, that is to say, as restrictive as possible, aiming at a homogenous group. The specificity of a test is a measure of how well it excludes those people who do not have the condition; the measure of how well a test includes all those people who have the condition is called its sensitivity. A definition for the estimation of prevalence may be sensitive and less specific, in order to avoid excluding people who may well have the condition. A definition for the purposes of diagnosis in healthcare encounters is a compromise that aims to include as few as possible people with the condition, but to exclude as many as possible who do not have the condition.

The creation of a disease definition usually depends on the identification of the presence of one or more shared specified elements, which are chosen because they are shared by all those with the medical condition, and which then allows for one or more of them to be part of the definition. Shared elements can include: identified pathological process(es), and/or a unique clustering of symptoms, signs, and/or other clinical features, and/or known shared cause(s), and/or a shared successful responses to treatment, and/or a biomarker (Coggon *et al.* 2005; Livingstone-Banks 2018). A ‘biomarker’ is a test, often a chemical one, that is specific and sensitive enough to exclude all those who do not have the condition and include all those who do have it. A definition needs verification, which is often supplied by a biomarker, as argued by van der Meer & Lloyd (2012):

Designing diagnostic criteria is very difficult without such a marker, i.e. a gold standard [21]. In the absence of biomarkers, the resultant criteria are very prone to the individual biases of the group formulating them (van der Meer & Lloyd 2012, p. 29).

So, according to van der Meer and Lloyd, in the absence of a biomarker (which they also call a ‘marker’) for the verification of a definition of a medical condition, there is difficulty in confirming the validity of any particular definition. The consequence of that lack of a verification standard is that it allows a definition to reflect the biases of the formulating group (which I see is one explanation for differences between definitions).

Myxoedema and Charles Bonnet syndrome are examples of medical conditions that have generally-agreed definitions. People with myxoedema have shared identified pathological process(es), known shared cause(s), a shared unusual clustering of symptoms, signs, or other clinical features, a biomarker (the thyroid function blood test), and shared generally-successful responses to treatment. In Charles Bonnet syndrome there is an unusual clustering of symptoms. In these two medical conditions these elements contribute to a ‘gold standard’ for the validation of their definitions.

At this stage, CFS/ME lacks elements that are part of the definitions of other medical conditions: it lacks unique feature(s) so it does not have a single generally agreed definition. Instead, definitions for CFS/ME have been created by consensus of groups of experts in the field, based on collections of patients’ reported subjective symptoms, with the experts’ weighting of those symptoms. However, different experts have contributed differing definitions, that use not only differing symptom collections but also differing weightings of those symptoms.

I now give summaries of commonly-used case definitions for CFS, ME/CFS, and ME, as supplied by United States National Academies of Sciences, Engineering, and Medicine, Institute of Medicine:

Fukuda Case Definition for CFS (1994)

Terminology: CFS

Method of Development: Consensus process involving an international collaborative group of leading CFS researchers and clinicians (including input from patient group representatives).

Required Symptom(s): Prolonged or chronic fatigue that persists or relapses for $\geq$ 6 months.

Four or more of the following concurrently present for $\geq$ 6 months impaired memory or concentration, sore throat, tender cervical or axillary lymph nodes, muscle pain, multi-joint pain, new headaches, unrefreshing sleep, post-exertion malaise.

Canadian Consensus Criteria for ME/CFS (2003)

Terminology: ME/CFS

Method of Development: Expert Medical Consensus Panel that comprehensively reviewed and analyzed CFS research evidence; grouped symptoms together that share a common region of pathogenesis.

Required Symptom(s): Fatigue, Post-exertional malaise and/or fatigue, Sleep dysfunction, Pain, Two or more neurological/cognitive manifestations. At least one symptom from two of the following categories: autonomic, neuroendocrine, immune. Illness lasting $\geq$ 6 months

NICE Clinical Guidelines for CFS/ME (2007)

Terminology: CFS/ME

Method of Development: The guideline was developed by the National Collaborating Centre for Primary Care, which worked with a group of health care professionals, patients, and caregivers, and technical staff who reviewed the evidence and drafted the recommendations. The recommendations were finalized after public consultation.

Required Symptom(s): Fatigue (characterized by post-exertional malaise and/or fatigue). One or more of the following: difficulty with sleeping, muscle and/or joint pain, headaches, painful lymph nodes without pathological enlargement, sore throat, cognitive dysfunction, physical or mental exertion makes symptoms worse, general malaise or flulike symptoms, dizziness and/or nausea, palpitations in the absence of identified cardiac pathology. Persistence of symptoms ≥ 4 months for adults and ≥ 3 months in children or young people.

Revised Canadian Consensus Criteria for ME/CFS (2010)

Terminology: ME/CFS

Method of Development: Authors reviewed previous definitions and literature available; tried to limit the types of symptoms within each of the Canadian Consensus Criteria categories to allow investigators to more reliably categorize patients.

Required Symptom(s): Fatigue, Post-exertional malaise and/or post-exertional fatigue, Unrefreshing sleep or disturbance of sleep quantity or rhythm disturbance, Pain (or discomfort) that is often widespread and migratory in nature, Two or more

neurological/cognitive manifestations. At least one symptom from two of the three categories: autonomic manifestations, neuroendocrine manifestations, immune manifestations. Persistent or recurring symptoms for ≥ 6 months but not lifelong

International Consensus Criteria for ME (2011)

Terminology: ME

Method of Development: The expertise and experience of the Panel members as well as PubMed and other medical sources were utilized in a progression of suggestions/drafts/reviews/revisions. The authors achieved 100 percent consensus through a Delphi-type process. The Canadian Consensus Criteria were used as a starting point, but significant changes were made.

Required Symptom(s): Post-exertional neuroimmune exhaustion (PENE). At least one symptom from three of the following four neurological impairment categories: neurocognitive impairments, pain, sleep disturbance, neurosensory, perceptual, and motor disturbance, Immune, gastrointestinal, and genitourinary impairments. At least one symptom from three of the following five categories: flu-like symptoms, susceptibility to viral infections with prolonged recovery periods, gastrointestinal tract, genitourinary, sensitivities to food, medications, odors, or chemicals. At least one symptom from energy production/transportation impairments: cardiovascular, respiratory, loss of thermostatic stability, intolerance of extremes of temperature

Table One: Five Differing Definitions for ME/CFS, CFS/ME, and ME (Adapted from Table 3-1 – ‘Elements of Selected Case Definitions and Diagnostic Criteria for ME/CFS’. National Academies of Sciences, Engineering, and Medicine, Institute of Medicine (2015)).

Before I critique these disparate definitions in Table One, I first indicate two elements in these definitions that I have not copied here. These definitions list a number of *exclusionary medical conditions*, that is to say, any active medical condition that can explain the presence of chronic fatigue, such as untreated hypothyroidism. Evidence for the presence of such a condition would suggest that that condition is responsible for the fatigue rather than chronic fatigue syndrome. The diagnosis of a fatigued patient with one of these exclusionary diagnoses is not relevant to the arguments in this thesis. The above definitions also give lists that indicate the possibility of *comorbidities* (which comorbidities are not necessarily exclusionary), such as fibromyalgia and irritable bowel syndrome. The existence, or not, of comorbidities in CFS patients is also not relevant to the arguments in this thesis.

I note that the lists of required symptoms between the five definitions listed in Table One vary significantly, as do the weighting that they give to these various symptoms. These dissimilarities exist because CFS, ME, and SEID lack gold standards for the validation of the definitions. Instead of using any gold standard, the ‘method of development’ for each of these definitions (how they were created) was by comparison with other definitions and by a process designed to combine the views of experts. So, the validation process for these definitions is *subjective* rather than *objective*, which may explain why the Institute of Medicine report places ‘scare quotes’ around these committees’ validation processes:

Validation was by comparison with existing criteria for ME/CFS, which they considered ‘validation’ studies (‘Validation of Diagnostic Criteria for ME/CFS’, National Academies of Sciences, Engineering, and Medicine, Institute of Medicine (2015)).

Here ‘they’ refers to the groups of experts who composed these definitions. These experts compared their definition with others’ definitions. By using scare quotes around the term validation, the writers of this Institute of Medicine document seem here to be indicating the limitations, including reduced reliability, of that kind of validation processes.

These experts use different methodologies for different purposes: some refer to CFS, some to ME and some to ME/CFS. All are hampered by the lack of a ‘gold standard’ to verify the definition (Coggon 2005). Some definitions are designed for use for the diagnosis of individual patients, and some for patient selection for research (Walitt *et al.* 2024). The differences between these definitions hamper comparison between these studies; and, these differences undermine the external validity of the result of research (Nacul *et al.* 2019), that is to say, there is consequent uncertainty about the applicability of these researchers’ results to an individual patient in a healthcare encounter, if that patient has been diagnosed according to different criteria. (‘External validity’ refers to the wider application of these results to people who were not included in the trial.)

E-J. Lim and Son (2020), give a helpful diagrammatic representation of how these definitions and others of CFS, ME, CFS/ME, and SEID differ from each other in significant ways:

“ME/CFS CCC” (Canadian Consensus Criteria) refers to Carruthers & Vande (2003);

“ME ICC” (International Consensus Criteria) refers to Carruthers *et al.* (2011);

“ME Ramsay” refers to Ramsay (1986).

As E-J. Lim and Son (2020) tell us, the definitions provided by the researchers to whom E-J. Lim and Son refer are for chronic fatigue syndrome and/or myalgic encephalomyelitis, or for systemic exercise intolerance disease. E-J. Lim and Son (2020) do not assume or claim that these are distinct medical conditions. Their diagram (my Figure One) demonstrates the distinct differences between these definitions, which indicates that they will therefore choose dissimilar patient groups.

The dissimilarities in patient groups that are selected by these differing definitions are demonstrated and confirmed by differing prevalence estimates using these definitions. In their review article, Ali and Kheirabadi (2025), find that various studies give the very large variation in prevalence rates of 0.007% to 2.8% in the general adult U.S. population. A wide spread of prevalence is also found by E-J. Lim *et al.* (2020) in their systematic review of forty-six studies of prevalence estimations from 1980 to 2018, which gives a total average prevalence of $1.40 \pm 1.57\%$ (E-J. Lim *et al.* 2020, p. 1). The large confidence interval here of 1.57% reflects large variations in prevalence in these studies. E-J. Lim *et al.* (2020) then give two explanations for this wide variation in prevalence:

... [CFS] prevalence rates are widely varied particularly by case definitions and diagnostic methods. An objective diagnostic tool is urgently required for rigorous assessment of the prevalence of CFS/ME (*ibid.*, p. 1).

As E-J. Lim *et al.* (2020) claim (and as I argue), the different case definitions for CFS and the lack of a biomarker are responsible for the different prevalence estimations, because an individual patient who fulfils the criteria for one case definition may not fulfil the criteria for a different case definition. Similarly, the recently published Cochrane Review states: “There is no specific diagnostic test, therefore diagnostic criteria are used to diagnose CFS. The prevalence of CFS varies by type of diagnostic criteria used” (Larun 2024, page 1).

Cochrane Reviews are authoritative clinical practice guidelines:

A Cochrane Review is a systematic review that attempts to identify, appraise and synthesize evidence to answer a specific research question. Researchers conducting systematic reviews use explicit, systematic methods aimed at minimizing bias, to produce reliable findings to inform decision-making (Cochrane Library, ‘*Cochrane Database of Systematic Reviews*’ (accessed 3/9/25)).

However, it is not possible to reduce uncertainty about CFS by choosing which definition seems to be the most accurate. All definitions of CFS are challengeable because the accuracy and validity of the definition cannot be checked against a reference standard or gold standard (which does not exist for CFS). (For an exploration of this problem in another medical conditions (bacterial infections), see Patel *et al.*, 2023.)

These problems of differing case definitions and the consequent differing prevalence estimates would be ameliorated if there were to be certain significant features (van der Meer & Lloyd 2012) shared by CFS patients and not present in other people, but these categorising and demarcating features have not yet been identified. Alternatively, or as well, a reliable biomarker for CFS, able to demarcate those individuals who have CFS from those who do

not, would help amelioration. However, a biomarker would need to be validated against a ‘gold standard’ (van der Meer & Lloyd 2012; Patel *et al.* 2023; Paver & Morey 2023), but CFS/ME lacks that gold standard, and so lacks legitimacy for the selection of subjects for research (Noor *et al.* 2021; W. T. Lim & Torpy 2023). The lack of a biomarker challenges the concept of CFS/ME as a homogeneous condition that is adequately demarcated from other conditions with similar symptoms.

My view is that dissimilarities between definitions for CFS are problematic for research and for patient care. Dissimilarities create doubts for inclusion and exclusion criteria in medical research (Arron *et al.* 2024). When researchers use one rather than another of these differing criteria for inclusion and exclusion criteria when selecting patient for research, there is doubt that different researchers are actually using the same patient groups, and whether or not each CFS patient belongs in that group. These doubts hinder comparison between the result of different research projects; and doubts reduce *external validity*, that is to say, the appropriateness of applying the results of any particular research project to an individual CFS patient in a healthcare encounter, when this patient may have been diagnosed using a different definition. Similarly, individual patients in a healthcare encounter may or may not fit with some of these definitions, leading to uncertainty concerning their diagnosis, that is to say, whether or not their symptoms should be labelled as CFS. In a healthcare encounter, the lack of a biomarker in CFS and lack of consensus on diagnostic criteria (Deumer *et al.* 2021; Missailidis *et al.* 2019; Walitt *et al.* 2024), bring into doubt inclusion and exclusion criteria for individual patient diagnosis.

I see a further criticism of the definitions for CFS/ME, etc. in Table One and Figure One above. This criticism is recognised by Bjørn Hofmann (2022), but is not highlighted by E.-J.

Lim *et al.* (2020) or E-J. Lim and Son (2020). The problems is, that the elements that are cited in the above definitions (for instance, reported post-exertional malaise, problems with sleep, pain, infections, sensitivities, palpitations, dizziness, concentration, memory, and reported problems with autonomic neuroendocrine, immune, neurological, gastrointestinal, genitourinary, cardiovascular and respiratory systems) are all subjective, and not objective or externally detectable by another person, let alone measurable. That intrinsic vagueness or under-specification of key diagnostic symptoms of CFS leads to uncertain knowledge, resulting in over-diagnosis and under-diagnosis (Hofmann 2022). Vagueness and under-specification therefore contribute to uncertainty, and, in my view, allow a dichotomy of conceptualisations about CFS. Medical professionals have had training in scientific medicine, and are likely to react negatively to vagueness and under-specification, thereby adding to explanations of adverse CFS healthcare interactions.

These differing definitions give problems for diagnostics, that is to say, the process of making a diagnosis of CFS for an individual in a healthcare encounter, as described in the three following quotations:

[ME/CFS] is difficult to diagnose because there is no specific test for ME/CFS...

Symptoms of ME/CFS may appear similar to many other illnesses. There is no test to confirm ME/CFS (Centers for Disease Control, 'ME/CFS, About ME/CFS, ME/CFS Basics', accessed 12/8/24).

There are no diagnostic tests for ME/CFS, but medical professionals can order laboratory tests to check for conditions that have similar symptoms or to help identify

coexisting problems that can be treated. (Johns Hopkins, ‘Chronic Fatigue Syndrome’ accessed 12/8/24).

There’s no single test to detect ME/CFS. A diagnosis is made after other possible known causes for symptoms have been excluded (NHS Inform, accessed 12/8/24).

The Centers for Disease Control (CDC) quotation above regarding the symptoms of ME/CFS reminds us that the diagnosis of ME/CFS is difficult because there is no diagnostic test, so the diagnostic process depends on the elicitation and evaluation of the patient’s symptoms by the health professional. The quotations from Johns Hopkins and the Centers for Disease Control remind us that many other conditions give similar symptoms. The NHS Inform quotation above reminds us that CFS is a diagnosis of exclusion: even if a person’s symptoms may fulfil the diagnostic criteria for CFS but laboratory and other tests confirm that the patient has, say, underactive thyroid gland, heart failure, or anaemia, then the diagnosis of CFS is excluded or ruled out.

If, in the future, there were to be reliable diagnostic criteria for CFS, it may well be that the consequent demarcation process would exclude some people who have symptoms that are typical of CFS. I argue that this exclusion is a possibility, because we do not at this stage have objective evidence that all those with symptoms that fit CFS diagnostic criteria share the same pathological processes as causes of those symptoms.

I now summarise this subsection. I first described the purposes and roles of a definition for a medical condition – for clinical care, for research, and for estimates of prevalence. I then showed how such a definition is constructed, by choosing the elements that constitute a definition, and the methods for choosing those elements. I described how definitions for

medical conditions are verified. I then moved to chronic fatigue syndrome and the varying definitions that are used to identify it, and the difficulties involved in creating a single generally-agreed definition for CFS. I explained that the differing CFS definitions have been constructed by different groups of researchers for distinctly differing purposes. These differing definitions choose different populations, as indicated by the differing prevalence estimates generated by these definitions. I argued that lack of a verifying standard prevents the comparative evaluation of these definitions. I see that vagueness and under-specification of CFS symptoms, and the consequent inability to quantify them, add to uncertainties concerning CFS, hindering the creation of a single definition, thus creating problems for research and in patient care.

I now move to show ontological challenges to CFS that are to some extent explained by problems with its definitions.

3.3 (c): Ontological challenges to chronic fatigue syndrome

In this subsection, I address the challenges to claims concerning the existence of chronic fatigue syndrome that I alluded to in Chapter One, Section Three. I show that there are dichotomous interpretations regarding the existence of chronic fatigue syndrome as an objective entity, and I give the views of those who doubt its distinct and independent existence. I then move to give reasons for my acceptance of the real existence of patients' CFS symptoms.

Many commentators refer to CFS or ME/CFS as if it is a determinate concept (or concepts) (for instance, Maes & Twisk 2010; Carel & Kidd 2014; O’Leary 20018; Geraghty *et al.* 2019; Sharpe & Greco 2019; Twisk 2019; Diserholt 2020; Pheby *et al.* 2020; Byrne 2021; de Boer 2021, p. 1348; Hng *et al.* 2021; Thoma *et al.* 2024; Ali & Kheirabadi 2025). Many of these claims are likely to be realist claims. I consider that it is not unreasonable for these commentators to refer to CFS as if it were an entity, bearing in mind the problems of discussing something if it is not assumed to exist. Furthermore, it is also not unreasonable for ME/CFS patients to consider ME/CFS an entity, because certain rights accrue from the acknowledgement that they suffer from an entity that exists (Hofmann 2022, p. 1156), such as health insurance and sickness benefits. However, in this subsection, I address the arguments that challenge the assumption that ME/CFS is an entity.

ME’s existence as a distinct physical disease entity was challenged the research by the psychiatrists Colin McEvedy and William Beard (1970b), who re-assessed evidence about fifteen outbreaks from the 1930s onwards of what was referred to by some as ‘benign myalgic encephalomyelitis’ (BME). BME was characterised by symptoms similar to CFS/ME, with a similar paucity of abnormal clinical signs and paucity of abnormal results of investigations. McEvedy and Beard concluded that these epidemics could well have been episodes of mass hysteria, without any physical disease process; though, their views were opposed by Ramsay (1986) and Wookey (1987).

However, McEvedy and Beard’s views may or may not be relevant to CFS/ME. They were studying ‘outbreaks’ or ‘epidemics’, where many people who had been in contact with each other became ill with these symptoms; so, at the time of these outbreaks it was hypothesised that BME was an infectious condition. In contrast to BME, contemporary cases of CFS/ME

are sporadic or isolated and not in epidemics, reducing the likelihood that CFS/ME is infectious in the sense that it can be fairly easily transmitted from one person to another. So, it may be that CFS/ME is not the same condition as BME. It is therefore problematic to apply McEvedy and Beard's (1970a, 1970b) conclusions to the condition suffered by today's CFS/ME patients. However, the term 'benign myalgic encephalomyelitis' is similar to the term 'myalgic encephalomyelopathy' which still is in widespread use, so the attribution of hysteria to CFS/ME patients has not disappeared.

Others express doubts concerning the ontology of chronic fatigue syndrome: "... CFS is but a name for something that, medically speaking, has no substance except for the patients' complaints" (Haan & Korsten 2020). (Here, Haan and Korsten are using the word 'complaints' to mean the patients' verbal testimony of their illness experience.) Haan and Korsten classify CFS as an 'invisible illness' like migraine. I understand that Haan and Korsten's use of the term 'invisible' is not ostensibly a challenge to the reality of the symptoms of migraine or CFS; it merely highlights the lack of objective physical signs and the lack of laboratory or imaging abnormalities, and our present lack of knowledge about the underlying pathological mechanisms. I appreciate that Charles Bonnet syndrome is also an invisible illness – see Chapter Two, Section Four. However, I see that CFS is more nebulous than migraine or CBS, because CFS does not have a distinct or unifying symptomatology as do migraine and CBS.

I see that that realist's claim that a patient's bundle of symptoms is due to the disease CFS/ME is challengeable in three ways:

- *Firstly*, there is significant variability of symptoms between people with the label CFS/ME with a lack of a shared unique feature, hindering the creation of an agreed definition;
- *Secondly*, there is inadequate evidence of distinguishing objective measurable physical or laboratory signs in CFS; and,
- *Thirdly*, there is inadequate demarcation of CFS from other similar conditions.

Therefore, the contention that CFS is the cause of the symptoms (as opposed to CFS being merely the label for those symptoms) is questionable; that is to say, the concept CFS/ME is ‘nominalist’ not ‘essentialist’ (Scadding 1996).

If the diagnostic criteria that I described in the previous subsection, “... are very prone to the individual biases of the group formulating them” (van der Meer & Lloyd 2012, p. 29), as evidenced by the consequent very differing prevalence estimates, what value do these diagnostic criteria have? Alexander Nelson Roberts’s response to these differing diagnostic criteria, disparate prevalence estimates, and the many other uncertainties regarding ‘chronic fatigue syndrome’, is to critique the concept of CFS being a distinct disease at all. He contends that any particular disease should have evidence of *something unique* that all those with the disease have, and those without the disease lack. For any medical condition, diagnostic criteria, diagnostic biomarkers, and disease labels need to be checked against this unique feature (or these features) as a gold standard. Roberts notes the absence of empirical evidence for any unique feature in CFS; he therefore declares that,

The *epistemically appropriate* way to describe the lack of consistent biological findings in CFS would be to say that no characteristic, explanatory, unique, significant, etc., pathophysiological abnormalities have been observed in patients meeting the CFS diagnostic criteria (Roberts 2024, p. 189) [Italics original].

For Roberts, it is misleading to bemoan the difficulty in diagnosing CFS and the lack of laboratory or diagnostic tests (as do Geraghty and Adeniji 2019), because that bemoaning masks what Roberts understands as the actuality, that the label ‘CFS’ is merely an indeterminate construct, not a disease. So much so, that, in Roberts’s ontological interpretation (Roberts 2024, p. 189), it is inappropriate, for instance, to say that that CFS is difficult to diagnose and has no biomarker, because these problems are inherent in a condition that is merely a construct.

I see that Roberts’s argument here as a radical response to my complaints, in Chapter Three, Section Three, Subsection (b), where I bemoaned the absence of a biomarker and/or a generally agreed definition for CFS: Roberts tells us that these biomarkers and definitions fundamentally *cannot* be discovered or agreed. Roberts’s assertion, that any particular disease should have evidence of something unique that all those with the disease have, and those without the disease do not, aligns with our knowledge of myxoedema and Charles Bonnet syndrome, which both have unique features (as I described in 2.4 of this thesis), as do most other medical conditions. Roberts therefore challenges the adequacy of the CFS disease label. My development of Roberts’s view is that research on such an unvalidated and evidence-poor construct is likely to lead to unreliable results, and will fail to adequately reduce uncertainty, as I will argue in Chapter Three, Section Three, Subsection (j).

Some HCPs share Roberts's doubts about even the existence of CFS, due, according to my understanding, to the many uncertainties about CFS: the lack of distinctive symptoms or signs (in contrast to, say, Charles Bonnet syndrome), objective abnormal physical signs and laboratory test results, or a biomarker (in contrast to many other medical conditions), and the lack of adequate progress of research to adequately reduce these uncertainties over many decades.

Doubts about the existence of as CFS remain widespread among HCPs since then. The existence of ME/CFS is challenged by one third to a half of Horton *et al.*'s (2010) sample of GPs: "There's still some doubt ... amongst quite a lot of professionals as to what ... the condition is ... in fact whether it exists at all" (Horton *et al.* 2010, p. 5). Here Horton *et al.* (2010) are quoting the words of a health care professional. A more recent literature review and synthesis of thirty-three qualitative surveys found that, "... a third to a half of all GPs did not accept ME/CFS as a genuine clinical entity" (Pheby *et al.* 2020, p. 1), and the medical researchers Hng *et al.* find that there are, "... high levels of ignorance and disbelief among [UK hospital] doctors [about ME]" (Hng *et al.* 2021/2, p. 10).

Radial uncertainty challenges existence claims regarding chronic fatigue syndrome.

Nevertheless, as my counter-argument to that challenge, I see that the lack of knowledge of a precipitating cause or of perpetuating causal processes (as in CFS/ME) has not prevented other medical conditions with similar uncertainties being recognised as existing: for instance, rheumatoid arthritis and multiple sclerosis were accepting as existing in the past when little was known about their pathological processes. So, despite the doubts that I express in this subsection, I feel that it is reasonable to accept the term 'chronic fatigue syndrome' (and similar terms), along with the apparent flaws in its conceptualisation.

Roberts's challenge to the ontological status of the medical condition CFS/ME does not seem to me to be a dismissal of the sufferings of patients who have that label. I have not found academic sources that dispute or doubt the *reality of people's experience of CFS/ME symptoms*, including the severity, intrusiveness, and disabling nature of these symptoms. For instance, the psychiatrist Wojtek Wojcik and his researcher colleagues give a reason for accepting the reality of CFS symptoms; they give an example from elsewhere in medicine where there may be an unproblematic mismatch between objective signs as elicited by the healthcare professional on one side, and the patient's report of subjective symptoms on the other side: "[I]n the pain clinic, for example, the patients' experience of pain is observed to vary with more than simply the physical pathology of each case" (Wojcik *et al.* 2011b, p. 503). Here Wojcik and colleagues acknowledge a distinction between the objective signs of disease on the one hand, and the recognition of the validity of patients' experience of symptoms on the other hand, where the existence of these symptoms is not challenged. (I will show the value of an approach of this kind, where disease mechanisms (the 'physical pathology') are de-emphasised, in Chapter Seven, Section Seven.)

My view is that the realist acceptance of the presence of symptoms in an individual CFS/ME patient should be accepted on pragmatic, empirical, rational, and medical ethical grounds. The pragmatic necessity of HCPs taking patients' testimony regarding the presence of, and the negative effects of, those symptoms at face value is a practical way to conduct the healthcare interaction: if the HCP were to reject the reality of the patient's experience that rejection would inhibit further information flow, among many other harms. Additionally, the reality of CFS symptoms is the *best empirical explanation* for patients' testimony, their responses to those symptoms, and the effects of the symptoms on these patients' home lives,

social lives, and work lives. It is not rational to assert that someone else is not experiencing the symptoms they describe. Furthermore, dismissing a patient's testimony is contrary to testimonial justice as described by Fricker (2007), and contrary to medical ethical guidelines (General Medical Council, 23b).

In this subsection, I have shown that uncertainties about chronic fatigue syndrome create doubt in some people concerning the existence of CFS. I countered that doubt by citing some medical conditions that, in the past, were subject to severe uncertainty that was later resolved. Furthermore, I argued that these doubts should not permit a challenge to the reality of the sufferings of CFS patients.

3.3 (d): Uncertainties about CFS/ME are reflected in the differing names for the condition

There is a lack of a term for this condition that is all-embracing, accurate, un-stigmatising, and uncontested (on the assumption that CFS is a *single* condition). In this subsection, I note that the two commonly-used names, 'chronic fatigue syndrome' and 'myalgic encephalomyelitis (or -pathy)', have differing implications regarding attributions to pathological processes and, seemingly, regarding the recognitions of the severity of the symptoms. These two names also have evaluative differences regarding the perception or attribution of stigma. The choice of name also reflects and/or influences power relations in healthcare interactions.

It is not *universally* agreed that one single name will suffice. Twisk (2019) uses the term 'myalgic encephalomyopathy' without attaching it with a slash to 'chronic fatigue

syndrome’, because he argues that chronic fatigue syndrome and myalgic encephalomyopathy are different conditions. He sees that, “myalgic encephalomyelitis is established as neuromuscular disease with specific signs of neurological dysfunction” (Twisk 2019, p. 1), while chronic fatigue syndrome is a distinct clinical entity whose symptoms are unexplained. He supports his claim that CFS and ME are distinct clinical entities by noting the absence of evidence for ‘conversion’: “Proof that CF [chronic fatigue], CFS, and ME are part of a, "fatigue continuum" and that CF can convert to CFS at a later stage is lacking.” (*ibid.*, p. 1).

I challenge Twisk’s (2019) confidence in the understanding of the pathological processes of ME as, “... a neuromuscular disease with specific signs of neurological dysfunction” (*ibid.*, p. 1) in Chapter Three, Section Three, Subsection (h), “Uncertainty About Pathological Mechanisms of Chronic Fatigue Syndrome”. My response of Twisk’s claim of a distinction between chronic fatigue, CFS, and ME is that there does not have to be evidence of individuals ‘converting’ from one diagnosis to another. Instead, the existence of a ‘continuum’ could merely reflect the level of severity of symptoms between different patients, with some individuals’ symptoms being less severe than others’.

In contrast to Twisk (2019), most other commentators conflate the two names and often abbreviate them as ME/CFS or CFS/ME (Gipps 2008, Blease & Geraghty 2017, Blease *et al.* 2017, Blease & Geraghty 2018, Karfakis 2018, O’Leary 2018, Byrne, 2020, Byrne 2021, de Boer & Slatman 2023). This conflation is also shown in many authoritative medical sources: Johns Hopkins, ‘Chronic Fatigue Syndrome’ (accessed 12/8/24), Centers for Disease Control, ‘Chronic Fatigue Syndrome’ (accessed 12/8/24), NHS Inform (accessed 12/8/24), and the Mayo Clinic (accessed 12/8/24). Because of the wide acceptance of the conflation of

chronic fatigue syndrome with myalgic encephalomyopathy as one condition, an agreed term that encapsulates both would be of value. In the meantime, most commentators choose to use the portmanteau term ME/CFS or CFS/ME. However, I prefer to avoid including the term ‘myalgic encephalomyelitis’, because of the present lack of evidence that this term is an accurate description of the pathological processes involved (O’Sullivan 2015, p. 240).

I recognise the view that chronic fatigue syndrome and myalgic encephalomyopathy may be separate conditions (Twisk 2019), although most commentators accept that it is reasonable to consider that they are same condition, given the present state of knowledge. However, the debate about names remains unsettled in the present state of uncertainty, as I now describe.

“Surveys conducted by ME/CFS advocacy organizations have found that 85 to 92 percent of respondents want that name to be changed” (Committee on the Diagnostic Criteria for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome 2015, Terminology). There are many CFS/ME sufferers and their supporters who feel that a ‘symptomatic’ name (like CFS) for this medical condition stigmatises the patient and trivialises their suffering in a way that a name that suggests the existence of a pathological process (such as ME) does not:

“‘[C]hronic fatigue syndrome’ trivializes the seriousness of the illness ... given that patients’ experiences could be misconstrued as everyday tiredness” (Jason *et al.* 2020, p. 229). The neurologist Suzanne O’Sullivan gives evidence for the importance of terminology to some patients:

I asked [Rachael], “Do you agree with the diagnosis of chronic fatigue syndrome?” ...
“No”, she said firmly. “Have you even been listening? I don’t have chronic fatigue

syndrome, I have ME. Fatigue is something that everybody gets. The word is an insult.” (O’Sullivan 2015, p. 231).

For ‘Rachael’, the term ‘chronic fatigue syndrome’ trivialises her problems. The Board on the Health of Select Populations agrees: “Patients and advocates told the committee that the name “chronic fatigue syndrome” leads others, including clinicians, to think that patients are malingering and to ask whether the illness is “real” (Committee on the Diagnostic Criteria for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome 2015, Terminology).

The journalist and advocate for ME patients, Tom Chivers, adds to Jason *et al.*’s (2020) condemnation of the name CFS, seeing it as a trivialisation of patients’ suffering:

There is surely no disease more cruelly misnamed than “chronic fatigue syndrome”. It is mysterious, apparently triggered by viral infection, and can last for months, decades, even a lifetime. Patients can be housebound or even bedbound for years. But the name makes it sound like they are just a little bit tired (Chivers 2021, no page numbers).

The psychologist Ellen Goudsmit condemns the term CFS not only because it trivialises, but also because, in her view, it is misleadingly narrow:

To refer to chronic fatigue syndrome as 'chronic fatigue' not only trivialises the condition, but it's also grossly misleading. The essence of chronic fatigue syndrome and myalgic encephalomyelitis is not normal 'fatigue'. The diagnostic criteria may not

be terribly specific, but they require more than the presence of 'tiredness, worry, aches and pains' (Goudsmit 5th August 2002).

Lesley Cooper expands on the kinds of stigma to which the name CFS can lead:

[T]he issue of nomenclature is not just a semantic problem: it encompasses serious disagreements, which have sadly led to ill will and abusive remarks, on such questions as whether the syndrome or some form of it exists, whether it is "real" or "organic" or "merely" psychological' (L. Cooper 1997, p. 188).

'Ill will and abusive remarks' add to the sufferings of CFS/ME people. Here, L. Cooper alludes to disputes concerning the existence of the condition, an issue that I addressed in Chapter Three, Section Three, Subsection (c). The choice of name may also reflect tensions between the perspectives of patients and their healthcare professionals. The downplaying of symptoms is recognised (but not endorsed) by some medical authorities: for instance, "[T]he term 'chronic fatigue syndrome' has led to misperceptions on the part of clinicians and the public" (National Academies of Sciences, Engineering, and Medicine 2015, p. 11).

Furthermore, some names may have positive effects on power relationships in CFS healthcare encounters:

Where generally a biomedical disease name represents the symbolic legitimization of the ideology of expertise, the label ME has come to serve as a symbol for the usurpation of power from doctors by patients (L. Cooper 1997, p. 188).

Here, Lesley Cooper recognises that the power differential between patients and HCPs is reflected in the choice of a name, and she notes that some patients choose a specific nomenclature in order to change that power differential.

The choice of a name of the condition is therefore important, because sufferers and their supporters have a wish for a name like ‘myalgic encephalomyopathy’ that seems to encapsulate and reify the severity of their symptoms, and endorses and legitimises their suffering, while those who prefer the term ‘chronic fatigue syndrome’ wish to avoid unsupported claims of disease processes.

I now expand on these terminological disputes. The terms ‘syndrome’ and ‘disease’ have differing meanings, so also have differing implications. The word syndrome often refers to a collection of symptoms where there is inadequate evidence about underlying causes or mechanisms (Livingstone-Banks 2018), unlike in a disease. W.T. Lim & Torpy give a reasonable justification the use of the word ‘syndrome’ in the name CFS, because in their view CFS is a syndrome rather than a disease: “As there is no clinical test to confirm the presence of this condition, the diagnosis is syndromic based on different clinical definitions” (W.T. Lim & Torpy 2023). (As I showed in Chapter Three, Section Three, Subsection (b), CFS is certainly diagnosed on the recognition of a grouping of symptoms rather than on any unique symptom or specific test result.) Other commentators, such as Lubet and Tuller, prefer the term ‘disease’ to ‘syndrome’; but, their preference is not based on knowledge of the relevant aspects of the condition, because they acknowledge that, “... that science has not yet caught up with the disease under investigation” (Lubet & Tuller 2021, page 2 of 3). Hofmann reminds of some of the reasons for the term disease having significant practical implications for, “... whether you get the attention of health providers, whether you are freed

from social obligations (to work), and whether you will get economic support (sickness benefit)” (Hofmann 2022p. 1155).

Amanda Diserholt notes that ‘chronic fatigue syndrome’ seems to denote a psychological/psychiatric nature and aetiology, while ‘Myalgic Encephalomyelitis’ is suggestive of a neurological manifestation and cause (Diserholt 2020), which she argues is more appropriate. By contrast, a survey of the views of members of the Association of British Neurologists found that 84% of those who responded did not view chronic fatigue syndrome as a neurological condition (Wojcik *et al.* 2011a). This evidence of neurologists’ views reduces confidence in the claim that CFS is a neurological disease.

It should be noted that Wojcik *et al.*’s (2011a) survey used only the term ‘chronic fatigue syndrome’, and not ‘CFS/ME’ or ‘myalgic encephalomyelitis’. I note that these neurologists were not asked for their views on whether *myalgic encephalomyelitis* is a neurological condition, nor whether they consider ME to be distinct from CFS. I appreciate that it may be that some of those 84% considered that myalgic encephalomyelitis (or -pathy) itself *is* a neurological condition, even if their view is that CFS is not.

The objective evidence for a neurological disease process in CFS/ME is inadequate: “[E]ncephalomyelitis’, a specific and often lethal neuropathological process, does not occur” (Wojcik *et al.* 2011b, p. 501), and, “[T]he term “myalgic encephalomyelitis,” although commonly endorsed by patients and advocates, [is] inappropriate [in ME/CFS] because of the general lack of evidence of brain inflammation in ME/CFS patients” (National Academies of Sciences, Engineering, and Medicine 2015, p. 11). Similarly, the neurologist Suzanne O’Sullivan reminds us that,

[t]here is no evidence for inflammation in the brain or spinal cord in ME/CFS sufferers, and the misnomer creates a barrier between neurologists and ME/CFS sufferers (O’Sullivan 2015, p. 240).

A ‘barrier’ between ME/CFS patients and some clinicians (as O’Sullivan sees here) due to this ‘misnomer’ is clearly unsatisfactory, and may well be an explanation for some unsatisfactory ME/CFS healthcare interactions. The alternative term ‘encephalomyopathy’ is also problematic, in that it implies that an essential feature shared by all who have the condition is an identifiable pathological process in the brain—even though the current evidence for such a pathological process is insufficient, and there is less evidence that *all* people with CFS/ME have this pathological process. ‘Myalgic’ uncontroversially refers to the muscle pain that is frequently present in CFS; but for many CFS patients pain is not the most prominent or the most disabling symptom.

My view is that for CFS or ME to be considered to be a neurological condition requires two areas of evidence, both of which are lacking at this stage: firstly, evidence that a neurological process is primary, that is to say, that this neurological process is the underlying cause for the onset, perpetuation, and course of all the symptoms, including the non-neurological majority of symptoms; and, secondly and relatedly, evidence that *none* of the many other theorised underlying causes are primary, for instance, degenerative neurological processes, infection, auto-immunity, hypo-immunity, disordered gut biome, mitochondrial dysfunction, muscle disease, blood-brain barrier dysfunction, oxidative stress, dysfunction of the hypothalamic-pituitary-adrenal axis, circulating cytokines, etc.

I emphasise here that the use of the term ‘syndrome’, and my suggestion for the avoidance of using the word ‘disease’, is not an indicative that I assume that there is *no* underlying disease process in patients with a set of symptoms that are collectively called a syndrome. Instead, the use of the term ‘syndrome’ often merely acknowledges that any such underlying *unifying disease process* has not yet been adequately identified. To illustrate this point, I give the example of ‘metabolic syndrome’, a condition associated with an increased risk of cardiovascular disease and diabetes, where the unifying disease process has not yet been identified: “The precise pathogenetic mechanisms unifying all of its components are as yet unknown” (BMJ Best Practice, accessed 7/1/26). There is similar uncertainty concerning ‘complex regional pain syndrome’, which is characterised by prolonged pain and weakness in one limb often following injury, where there are some objective abnormalities but the significant underlying mechanisms are insufficiently understood (Abd-Elsayed *et al.* 2024).

So, my view is that ‘disease’ is not the appropriate term for chronic fatigue syndrome at present, because there are large lacunae in the understanding of mechanisms of CFS, including the mechanisms that unify of all its components. The hypothetical future finding of an explanatory disease mechanism for the onset, symptoms, and course of CFS could be expected to endorse a new name that includes the word disease, and that new name could also reflect some important aspect of that newly discovered disease process.

My response to expressions of dissatisfaction with the term, ‘chronic fatigue syndrome’, is that a short label for a medical condition can never encapsulate all its relevant aspects, and may not refer at all to its most important aspects: for instance, leukaemia merely means ‘white blood’, diabetes mellitus refers to excess urine with sweetness, diabetes insipidus refers to excess urine without sweetness, and intermittent claudication refers to limping

(referencing the Roman Emperor Claudius who is said to have limped all his life so did not have what we now call intermittent claudication). All those disease terms leave out any allusion at all to those diseases' most prominent symptoms, or to their serious pathological processes, or to their severe clinical consequences. Furthermore, there are many other mundane-sounding disease labels that also could be said to downplay the serious effects of those diseases, such as multiple sclerosis (MS), cystic fibrosis (CF), and motor neurone disease (MND). However, despite their potential unsatisfactoriness, all these terms are widely used and understood, and are unlikely to be changed. The medical conditions, leukaemia, diabetes mellitus, diabetes insipidus, intermittent claudication, multiple sclerosis, cystic fibrosis, and motor neurone disease all have established and non-disputed statuses, and their names are not challenged; while, contrastingly, the status of the medical condition CFS/ME remains unsettled and disputed. I apprehend that debates about the name for CFS/ME reflect this unsettled status, and reflect the continuing disputes that I describe in Chapter Four, regarding whether CFS/ME should be considered to be a biological or a psychological and behavioural condition.

In response to dissatisfactions with both usual names for this condition, the Committee of the National Academies of Sciences, Engineering, and Medicine attempt to find a more acceptable name (Committee on the Diagnostic Criteria for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome 2015). They recommend a change to the term, “systemic exertion intolerance disease” (SEID), which they feel does manage to encapsulate the severity and deleterious effects of the symptoms, and which, in their view, alludes to the different triggers for exacerbation of the symptoms, and to the variety of those symptoms, and the severity of the symptoms:

[T]he committee recommends that the disorder described in this report be named “systemic exertion intolerance disease” (SEID). “Systemic exertion intolerance” captures the fact that exertion of any sort—physical, cognitive, emotional—can adversely affect these patients in many organ systems and in many aspects of their lives. The committee intends for this name to convey the complexity and severity of this disorder (National Academies of Sciences, Engineering, and Medicine 2015, page 11).

According to the National Academies, ‘SEID’ is an improvement on ‘CFS’ because it differentiates its symptoms from the everyday fatigue felt by healthy people from time to time. Systemic exertion intolerance disease can be taken to indicate patients’ experience, that exertion is often followed by all-embracing unpleasant and disabling sensations of malaise and debilitation that persist for days or weeks, which set of symptoms is different in character from, and worse than, just ‘fatigue’.

My view is that the first three words of ‘SEID’ therefore seem helpful and appropriate. However, I can see the appropriateness of avoiding the term ‘disease’ in relation to CFS; I prefer instead the terms, ‘illness’ and ‘medical condition’ and ‘syndrome’. As Byrne says,

I take “illness” to capture the qualitative, experiential aspects of certain changes to one’s body as well as how those changes affect how one is categorised and treated within society. “Disease”, on the other hand, requires only some physiological dysfunction (Byrne 2020, p. 17).

Here Byrne concentrates on the experience of the illness because the physiological abnormalities have not yet been delineated. As it happens, despite its seeming attractiveness, the term SEID has not gained general acceptance or usage, among patients or among professionals.

In this subsection I have shown that disputes about names for CFS/ME are not just semantic; they also show disagreements about the attribution of pathological processes; and, for sufferers, they indicate recognition (or not) of the reality, the severity, and the effect of the symptoms; and CFS/ME patients find that the term, 'CFS' can negatively affect some of their healthcare encounters.

3.3 (e): The Problem of Demarcation of Chronic Fatigue Syndrome

Disease demarcation is the feature of a medical condition that enables it to be recognised as separate from others. However, demarcation is problematic in chronic fatigue syndrome because other medical conditions are characterised by similar symptoms: “The usual symptoms [of CFS] are non-specific and overlap with [the symptoms of] other diseases” (Walitt *et al.* 2024, p. 1). There is therefore a reasonable challenge to the existence of CFS as a distinct medical condition, because there are many other named medical conditions such as fibromyalgia, long covid, ‘Candida’, chronic Lyme disease, etc., which are characterised by multiple symptoms similar to those of CFS, and are also inadequately understood, along with sharing a lack of curative treatments. Distinguishing each one of this group of illnesses from each other and from CFS/ME is hampered by a lack of a unique feature or a biomarker for any of them, because they are recognised and defined by the grouping of symptoms. Patient narratives and philosophical commentaries report the existence of healthcare

epistemic injustice in these conditions. I speculate that that these injustices are also due to uncertainties. However, for the sake of simplicity, I concentrate on CFS in this thesis.

3.3 (f): The Possibility of Subgroups in Chronic Fatigue Syndrome

In this subsection I examine contributions that challenge the claims that CFS is a homogeneous condition; where, in order to classify a medical condition as homogeneous, we need evidence that patients with this diagnosis share the same underlying pathological mechanisms.

Many commentators refer to CFS as if it were a homogeneous condition (Goudsmit 2002; Tucker 2004; Hossenbaccus & White 2013; National Academies of Sciences, Engineering, and Medicine 2015; Friedberg 2017; Blease 2017; O’Leary 2018; Clark 2019; Chivers 2021; Deumer 2021; Cortes Rivera *et al.* 2021; Larun *et al.* 2024). By contrast, other commentators and researchers suggest that CFS/ME is likely to have subgroups that may differ significantly in manifestation and/or in treatment recommendations (Nisenbaum *et al.* 2004; Jason *et al.* 2005; Jason *et al.* 2010; Richman *et al.* 2019; White 2019; Asprusten *et al.* 2021; W. T. Lim & Torpy 2023). Furthermore, according to our present knowledge, it is not impossible that CFS symptoms are the final common pathway of differing pathological processes; these differing pathological processes may in the future require classification as indicators of distinct subgroups of CFS needing different management strategies, or even as indicators of distinct medical conditions, again needing different managements (Nisenbaum *et al.* 2004; Jason *et al.* 2005; Richman *et al.* 2019):

Ultimately, there may be no single underlying cause for this illness and it is not improbable that ME/CFS may serve as an umbrella term for multiple different

diseases associated with overlapping symptoms. The diversity in symptom profiles and potential etiologies associated with ME/CFS make treatment and management of this illness extremely challenging and a treatment that may be effective for one subset of individuals may not be effective for another (Richman *et al.* 2019, p. 2).

Here, Richman *et al.* (2019) consider that the evidence from the research that they reviewed challenges the claim that ME/CFS is a homogeneous condition. As Richman *et al.* (2019) remind us, it is uncertain whether chronic fatigue syndrome is a homogeneous condition or a condition with subgroups or subtypes that differ significantly from each other in symptoms, pathological processes, and/or treatments. The medical researchers Leonard Jason and his colleagues assert that subtyping of CFS is both necessary and appropriate. They challenge the unitary model of CFS, claiming that subtypes are needed because of the, "... characteristically confusingly heterogeneous symptom profiles ..." of CFS (Jason *et al.* 2005, p. 29). They feel that the heterogeneity of CFS symptoms suggest that the condition is not unitary, and that there are significantly different subgroups (Jason *et al.* (2010).

The consequence of uncertainty about the existence or not of significantly different subgroups in CFS is that this uncertainty challenges diagnosis, interferes with treatment decisions, and hampers research. Asprusten *et al.* (2021) feel that their failure to identify CFS subgroups challenges the validity of some narrow CFS diagnostic criteria (that I discussed in Chapter Three, Section Three, Subsection (b)). White (2019) sees that the successful identification of likely subgroups would help to find causes, aetiology, and/or pathophysiology. Richman *et al.* (2019) see that uncertainty regarding the possibility of subgroups hinders choosing treatments for ME/CFS patients when it is not clear which possible hypothetical subgroup their illness belongs to. Those CFS individuals in distinct

subgroups could well need significantly different treatments. W. T. Lim and Torpy see that the possible existence of (as-yet-unidentified) CFS subgroups would hinder research: they note that neuroendocrine studies on CFS patients have led to contradictory results that may well be confounded by differences between subgroups (W. T. Lim & Torpy 2023).

Subtyping is also likely to be of value in research on treatments, because of the already known heterogeneity of responses to different treatments in CFS. Differing responses to treatments may possibly help towards the identification of subgroups (Jason *et al.* 2005).

However, there has been inadequate progress on finding subgroups. Jason *et al.* (2010) claim found ‘clusters’ of symptoms, which suggests to them that meaningful subgroups exist; but, the later research of Asprusten *et al.* (2021) failed to identify subgroups, even though they also found, by analysing symptomatology, some evidence of clustering in their sample of CFS patients.

3.3 (g): Uncertainty Concerning the Causes of Chronic Fatigue Syndrome

In this subsection, I show uncertainties regarding the initiating cause(s) of chronic fatigue syndrome. In regard to these causes a recent review of scientific research on CFS by Ali and Kheirabadi (2025) highlights the areas of uncertainty regarding its initial cause[s]:

The etiology of chronic fatigue syndrome (CFS) is controversial, complicated, and incompletely understood. Controversy exists about single versus multiple causations. Many theories exist regarding the involvement of infections, the immune system, and genetics in this complex interplay (Ali and Kheirabadi 2025, accessed 31/12/25).

Similarly, in their information for patients, Johns Hopkins Hospital tell us that, "... the cause of ME/CFS is not known" (Johns Hopkins, 'Chronic Fatigue Syndrome', accessed 12/8/24). The Centers for Disease control allude to possible causes: "Scientists don't yet know what causes myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS). The disease may have more than one cause or environmental factor that triggers it" (Centers for Disease Control, 'ME/CFS, ME/CFS Causes', accessed 12/8/24). The Mayo Clinic information site goes into more detail concerning our areas of uncertainty about the cause[s] of CFS:

The cause of myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) is still unknown. A combination of factors may be involved, including: Genetics... Infections... Physical or emotional trauma... Problems with energy usage..." (Mayo Clinic, accessed 12/8/24).

Scientists are studying whether the immune system plays a role in causing symptoms, as many people with ME/CFS have allergies, reactions to foods and medications, frequent hives and, occasionally, recurrent infections (Johns Hopkins, 'Chronic Fatigue Syndrome', accessed 12/8/24).

These authorities confirm that there is a great deal of uncertainty regarding the causation of CFS, even extending to whether it has one cause or many, and perhaps different causes in different individuals.

3.3 (h): Uncertainty about the pathological mechanisms of chronic fatigue syndrome

There is not only uncertainty about the initiating causes of chronic fatigue syndrome (as I described in the previous subsection), there is also continuing profound uncertainty about the pathophysiology of CFS, that is to say, the pathological mechanisms that perpetuate its symptoms, and its course. As Richman *et al.* tell us,

The exact etiology of ME/CFS is currently unknown though multiple hypotheses exist regarding potential triggers and mechanisms of illness, including viral infection, mitochondrial dysfunction, and neurological abnormalities (Richman *et al.* 2019, p. 798).

In scientific research, many CFS patients are found to exhibit abnormal pathological results (Maes & Twisk 2010; Sotzny *et al.* 2018; Missailidis *et al.* 2019; Twisk 2019; VanElzakker *et al.* 2019; Nacul *et al.* 2020; Shepherd 2022, pp. 18-47). Ali and Kheirabadi (2025) note that researchers have found many types of abnormality in CFS patients: alterations in the immune system, increased oxidative stress, oligoadenylate synthetase/RNase L Pathway, alteration of natural killer (NK) cells, B cell impairment, immunoglobulins, autoimmunity, alterations in the central nervous system, neuroinflammation and role of glial cells, neuronal sensitization alterations in the neuroendocrine system, changes in serotonin transmission, hypocortisolism, and genetic predisposition are variously implicated by different researchers Ali and Kheirabadi (2025), but despite these findings, the mechanisms of CFS that explain the symptoms and course remain under-clarified.

A similar uncertainty in CFS concerns whether or not the continuing symptoms are the consequence of a (at the moment undiscovered) ‘common cause’, as described above in Chapter Three, Chapter Three, Subsection (h), or, alternatively, whether *interactions between symptoms* can significantly exacerbate or ameliorate the symptoms, in the form of a dynamic mutual causal network (*c.f.* Nuijten *et al.* 2016). A common cause of a medical condition is the primary one that can give an overarching explanation of how these possible pathological cause all the symptoms and the course of the condition. A yet-to-be-discovered common cause for CFS would possibly be able to explain the variability of and severity of symptoms between CFS patients, and the changing symptomatology and severity of symptoms in any particular patient over time. Arnett & Clark (2012) are confident that ‘inflammatory fatigue’ is the cause of CFS symptoms, and this knowledge would be applicable to the diagnosis and management of CFS. Yet, standard anti-inflammatory treatments have had little success in CFS (Richman *et al.* 2019), which suggests that inflammatory pathological processes may not be the primary mechanisms underlying the symptoms of CFS. Ali and Kheirabadi (2025) are not able to state which of the processes that they allude to (see above) is likely to be the ‘common cause’ that is responsible for all the other abnormal pathological findings.

There is consequent continuing uncertainty concerning effective treatments for CFS:

Although there’s no cure for ME/CFS, there are ways to help manage your symptoms. Because of the complexity of the illness different things work for different people... There is no consistently effective single treatment for ME/CFS (NHS Inform, accessed 12/8/24).

There is no cure or approved treatment for ME/CFS. But, some symptoms can be treated or managed. Treating these symptoms provides relief for some people with ME/CFS (Centers for Disease Control, 'Living with ME/CFS', accessed 12/8/24).

The effectiveness of those symptomatic treatments varies from patient to patient, which may suggest that patients with the diagnosis of CFS may not share the same disease mechanisms; consequently, there may be distinct subtypes of CFS in regard to therapy.

3.3 (i): Uncertainties regarding the role of psychological processes in chronic fatigue syndrome

A further area of uncertainty in CFS is the relationship between the psychological and physical symptoms. In the light of that uncertainty, I see six possible variations on that relationship: it may be that the psychological symptoms that are commonly associated with CFS are,

- (i) *Secondary*, that is to say, due to the effects of the physical symptoms and other illness factors on the person's life, or
- (ii) *Primary*, that is to say, one of the significant causes of the physical symptoms (O'Sullivan 2015, p. 242), or
- (iii) The relationship could be *more complex*, not classifiable as primary or secondary, where the relationship is a dynamic interaction between psychological and physical symptoms (Byrne 2024).
- (iv) A variation on this uncertainty is that the psychological symptoms of CFS could be *neither primary nor secondary*, instead being actually an integral part of the condition, with the physical and psychological symptoms all being due to the

same (as yet unknown) root cause, with manifestations that vary between patients, varying in intensity any particular patient over time, or

- (v) One could be *agnostic* as to whether the psychological symptoms are primary, secondary, or integral, which variation would be a not-unreasonable response to the paucity of definitive evidence. Alternatively,
- (vi) There may be *significant differences between people* with CFS symptoms, that the relationship (whether causative or not) between psychological and physical symptoms varies to a major and significant extent between patients, so that different patients require different management.

Variations (iv), (v), and (vi) suggest that psychosocial treatments may benefit some CFS patients, even if these treatments don't reduce the physical symptoms. Variations (ii) and (iii) imply that psychological and/or behavioural treatments are likely to be of benefit in reducing the physical symptoms. Contrarily, many CFS patients and their advocacy groups doubt that psychological and behavioural treatments will improve the physical symptoms of CFS, because of their preference for variation (i), that their psychological symptoms are secondary to the physical symptoms. In Chapter Six, Section Seven, Subsection (b) I will argue that oppositional differences between these conceptualisations explain conflicts in some CFS healthcare encounters.

3.3 (j): The problem of the lack of adequate progress of research into chronic fatigue syndrome

In this subsection, I concentrate on review articles, in which scientific researchers collate, summarise, and evaluate others' laboratory and clinical research on ME/CFS.

There has been scientific research on chronic fatigue syndrome for many decades (for instance, see Buchwald *et al.* 1987). However, there is widespread acknowledgement that uncertainties regarding chronic fatigue syndrome have not been adequately reduced by research on causation, case definition, biomarkers, mechanisms, and treatments: “Further research is needed to confirm what causes ME/CFS” (NHS Inform accessed 12/8/24), and, “Many areas are being researched to understand what causes ME/CFS including infections and genetics” (Centers for Disease Control, ‘ME/CFS, What Causes ME/CFS Causes, Key Points’, accessed 12/8/24).

My view is that the need for further research is because of the lack of progress (as described in the next few paragraphs), not because of lack of *quantity* of research. The quantity of research into CFS is demonstrated by the large amount of published research on CFS is shown in the results of my PubMed search for "chronic fatigue syndrome" OR "myalgic enceph*" "research" on 14/8/2024, which returned the large number of 8536 results of paper reporting research on ME/CFS, from 1955 until the present time. In order to gauge how much research is still being conducted, my next search was for "chronic fatigue syndrome" OR "myalgic enceph*" "research" AND "2024" on 14/8/2024 which returned 157 results. These large numbers are evidence that a great deal of research has taken place into chronic fatigue syndrome over the years, and that research is continuing in large numbers.

Brurberg *et al.* (2014), Haney *et al.* (2015), and Arron *et al.* (2024) tell us that their reviews show that there has been no effective progress in research on the diagnosis of ME/CFS. Macksoud *et al.* tell us that there is currently no consensus on a biomarker for ME/CFS. Arnett and Clark (2012), Costa Rivera *et al.* (2019), and Muller *et al.* (2020) tell us that research on the pathological process of ME/CFS has been inadequate to give practical

applications to patient care; Smith *et al.* (2012), Larun *et al.* (2019), Richman (2019), Kim *et al.* (2020), Sanal-Hayes *et al.* (2023), Arron (2024), and Seton *et al.* (2024) all see inadequate progress on treatments for CFS and ME/CFS. Todhunter-Brown and colleagues include as many as 639 studies in their review of myalgic encephalomyelitis/chronic fatigue syndrome research from 2018 to 2023 on causes, diagnosis, and treatments. They conclude that,

Evidence relating to ME/CFS is seriously hindered by challenges and inconsistencies relating to the use of and reporting of diagnostic criteria... many studies are limited by the poor reporting of basic demographic information... we found large variations in the volume, nature and quality of research addressing different topics (Todhunter-Brown *et al.* p. 17).

However, these review articles do not show evidence of research effectively solving the uncertainties of CFS. The unsatisfactoriness of the results of CFS research is demonstrated by its inadequate progress: after some decades of research we still don't know enough about what is going wrong in CFS patients, and we don't know enough to decide what to do about how to help these patients. It may be that the lack of progress of research in reducing uncertainty about CFS is due to the quality of research rather than its quantity as Todhunter-Brown *et al.* (2025) conclude. However, for the purposes of this thesis, it is not my role to judge that quality; I merely use the words of the researchers or the reviewers of research themselves to demonstrate the continuing uncertainties regarding CFS.

In order for me to assess levels of certainty or uncertainty about CFS, my next search was, "chronic fatigue syndrome" OR "myalgic enceph*" "review" "2024" on 14/8/24. This search

returned sixty-nine results, many of which have “chronic fatigue syndrome”, “myalgic encephalomyelitis” or “myalgic encephalomyopathy” in their titles.

Uncertainty regarding CFS remains despite a great deal of research into the causes of and treatments for it: this research has been largely unsuccessful. Inclusion and exclusion criteria for selection of patients for research vary considerably between CFS research studies, which puts into question internal and external validity, and makes comparison between studies problematic. No biomarker has yet been found (Scheibenbogen *et al.*, 2017), so “ME/CFS research and patient treatment have been challenged by the lack of diagnostic biomarkers ...” (Missailidis *et al.* 2019). I further show this paucity of success by summarising the comprehensive survey of recent CFS research collated, analysed, and evaluated by Dr Charles Shepherd (Shepherd 2022). Dr Shepherd is a medical doctor with personal experience of ME/CFS and is Honorary Medical Advisor to the ME Association, a support group for patients with this condition. He lists multiple recent scientific research papers describing abnormal laboratory findings in CFS/ME (*ibid.*). He itemises abnormal pathological results in CFS/ME research that have been found in so many areas of physiology: infective, immunological, muscular, mitochondrial, central nervous system, autonomic nervous system, endocrine, cardiac, blood vessel, genetic, metabolic, gastrointestinal and psychological (*ibid.*, pp. 18-47). Despite this work, these researchers have not been able to compose a unitary understanding of CFS/ME, for instance, how these pathological findings causally connect to each other, nor how these processes could cause all the disparate symptoms and the variable course of the condition. MePedia gives links to other lines of CFS research: Abnormal neurovascular coupling Amygdala hypothesis Autoimmune hypothesis, Bacterial disease onset, Breakdown of homeostasis hypothesis, Co-conditioning theory, CYP21A2 mutation associated psychiatric spectrum, Immune

exhaustion hypothesis, Intracellular immune deregulations, Itaconate shunt hypothesis, Kindling hypothesis, Multiple virus hypothesis, Multisystemic dyshomeostasis, Neuroimmune model, Nitrogen hypothesis, Persistent infection hypothesis, Personality traits and patient attitudes in ME/CFS, Serotonin hypothesis, The 3Ps model, The Danger Model (MePedia, Medical Hypotheses, accessed 10/7/25). Furthermore, laboratory research in CFS is hampered by lack of integration of the various disparate abnormal pathological findings of, for instance, "... disturbed muscle function, metabolism, mitochondria, immunity, [calcium] signaling, neurological, adrenal, and gut health ..." that have been shown in CFS research (Ulvestad 2008) (Missailidis *et al.* 2019). It seems that very few areas of human physiology have not been implicated as causative of CFS. However, none of these abnormal findings have led to satisfactory explanations for all the symptoms and the course of CFS, let alone to successful treatments. Furthermore, in much research into CFS there may be inadequate demarcation of these pathological abnormalities from pathological findings in other medical conditions.

It may be that a future unitary concept for the pathology of CFS will be able to incorporate some or all of the abnormal physiological processes suggested by the areas of research itemised by Shepherd (2022, pp. 18-47). If so, these pathologies may be connected by a common cause (as yet not identified), or, perhaps, in a network that is similar in structure to Bringmann and Eronen's (2018) network model, that I will describe in Chapter Seven, Section Six.

These subsections (a) to (i) have demonstrated many elements of uncertainty about CFS: its definitions, its existence, its nomenclature, its demarcation, its homogeneity, its initiating cause(s), its continuing pathological processes, hypothesized common cause, treatments, the

role of psychological factors, and in laboratory and clinical research into all these uncertainties. The lack of success so far in CFS research contributes to radical uncertainty about it, and adds to the epistemological, nosological, and ontological challenges to CFS. My view is that in a healthcare encounter, these challenges may well have more import to a healthcare professional than to a patient, leading to mismatches of perspective and dichotomies of views.

In the next section, I argue that the many uncertainties concerning CFS itemised in this Section Three are interconnected.

3.4: The Interactions Between the Differing Areas of Uncertainty in Chronic Fatigue Syndrome.

In the previous section, I showed the lack of success in CFS research. In this section, I argue that the present inadequacy of scientific knowledge regarding CFS/ME is both the cause and the result of uncertainty due to a *complex network of mutually reinforcing uncertainties* regarding CFS. In order to support my argument, I show how the uncertainties in scientific knowledge about chronic fatigue syndrome (CFS) interact, so that uncertainty about one aspect of CFS is a partial explanation for uncertainties in other aspects. In the next section, I argue that this network of uncertainty can be classified as radical, problematic, and deleterious uncertainty.

There are no biomarkers for the diagnosis of CFS in an individual patient, no biomarkers for providing inclusion and exclusion criteria for selecting patients for research, no agreed

understanding of the pathological processes of that cause the precipitation and perpetuation of the condition, no curative treatments, and the absence of widely-agreed symptomatic treatments. I argue that research into CFS is hindered by uncertainty which allows the dichotomies of conceptualisations about it; these dichotomies lead to dissimilarities in the directions of research. Additionally, there are differences in the interpretation of evidence derived from research, showing that inadequate knowledge concerning CFS/ME has many of the features of radical uncertainty.

This complex network of mutually reinforcing uncertainties reciprocally impairs the reduction of uncertainty about CFS, because the existence of this network impedes research. The uncertainties consist of,

- *firstly*, the lack of biomarker able to confirm or deny the diagnosis of CFS for selecting patients for research.
- *Secondly*, the diagnosis of CFS in an individual is made only on the identification of groupings of symptoms; but, there are differing diagnostic criteria which vary from each other in their selection and weighting of those symptoms, so reducing certainty that the researchers who use different diagnostic criteria are choosing the same groups of patients.
- *Thirdly*, there is a lack of a ‘gold standard’, that is to say, a measure giving objective confirmation of the accuracy of a biomarker or of diagnostic criteria.
- *Fourthly*, the lack of confirmed biomarkers or diagnostic criteria for CFS causes reciprocal uncertainty regarding research into biomarkers and reliable diagnostic criteria based on symptoms; there is uncertainty about the homogeneity and demarcation of CFS, causing uncertainty regarding exclusion and inclusion criteria for case selection for research.

- *Fifthly*, consequently, research on of mechanisms of CFS is hindered.
- *Sixthly*, there is a lack of quantifiable biomarkers that could provide objective measurable indicators of disease progress or improvement in patients, which lack hinders the evaluation of, and comparison between, treatments, because treatment evaluation requires quantifiable outcome measures.

I see that these different elements of epistemic uncertainty regarding chronic fatigue syndrome do not exist in isolation: the uncertainty of each element is explained by or exacerbated by some or all of the others; each element contributes to or exacerbates some or all of the others, in a *network of mutual, two-way explanation*. These uncertainties therefore explain the continuing network of uncertainties about CFS.

3.5: The Recognition of Radical Uncertainty in Chronic Fatigue Syndrome

In the above subsections, I have argued that a lack of adequate progress of research into mechanisms of and treatments for CFS both explains and is explained by radical uncertainties about CFS. The paucity of knowledge reciprocally hinders the obtaining of relevant, reliable, and applicable knowledge. In this section, I argue that chronic fatigue syndrome is characterised by radical uncertainty, by matching the uncertainties concerning CFS onto those that I attributed to radical uncertainty in Chapter Two, Section Two. I then show the disadvantages, and some possible advantages, of radical uncertainty in CFS.

CFS is subject to Knightian (1933, 1921) uncertainty because of, *inter alia*, its disparate definitions and lack of biomarker; this lack of clear boundaries creates significant challenges

for selecting the appropriate people for research, and for diagnosing individual patients. In Knight's terms, the problems with 'grouping of cases' means that its uncertainty is at this stage ineliminable (Knight 1933, 1921, pp. 231-2). So, there remains a major problem for research: although that there are many people who have severe, intrusive, and disabling symptoms, there is insufficient evidence to group them together for eliminating Knightian uncertainty.

Uncertainties about CFS make researchers unsure how to create a dynamic 'state space model' (Bradley & Drechsler 2014) (that I described in 2.2) for CFS, because they don't know all the variables—for instance, all the relevant pathological processes that are perpetuating the symptoms, nor the existence or not of a (hypothesized) common cause or latent variable of CFS; they don't know the mathematical relationship between those pathological processes; they don't know the relationships between CFS patients' manifest symptoms and those pathological processes; and, they don't know whether some decision-relevant variables and some decision-relevant relationships between variables remain unknown, and unsurmised. As well as hindering research, the lack of a dynamic state space model for CFS hinders prediction of the consequences of acts of intervention, thereby hampering decision-making in healthcare encounters.

Cognitive epistemic states shaped by radical uncertainty play a significant role also in CFS healthcare encounters. In Chapter Six, Section Seven, Subsection (a), I will argue that healthcare epistemic injustice be explained if both the healthcare professional and the patient are in the cognitive states described by Romeijn & Roy (2014) (that I described in 2.2), that is to say, they are insufficiently aware of the extent of what is unknown (Djulbegovic *et al.* 2011). In these circumstances, an HCP may show unwarranted doxastic confidence, and then

use her authority to impose a prediction of outcomes of the patient's CFS, and to determine treatments plans. Meanwhile, in opposition, a CFS patient may also be doxastically confident enough to have her own (conflicting) predictions of the illness course with interventions, and her own preferred treatments. However, RU prevents arbitration between these two opposing positions.

I identify three different (although connected) areas where there is impactful radical uncertainty concerning CFS:

- *Firstly*, radical uncertainty in CFS is demonstrated in our lack of knowledge about it;
- *Secondly*, for an individual patient with the label CFS there is radical uncertainty in our knowledge concerning what pathological processes are making that patient ill, and what treatments will help; and,
- *Thirdly*, radical uncertainty about CFS reciprocally impedes CFS research intended to reduce that uncertainty.

I determine that we can recognise radical uncertainty regarding our knowledge about the *medical condition* chronic fatigue syndrome, and in the *individual patient* with symptoms that suggest CFS, by means of the five elements that I itemised above in Chapter Two, Section Two, “Concepts of Radical Uncertainty”. There, I described the features and consequences of radical uncertainty, and I argued that *all four* elements need to be present for a situation be considered one of radical uncertainty. I now match those same four elements onto knowledge about CFS:

1. Radical uncertainty is a situation in which there is inadequate information. We don't have an adequate overarching understanding of the mechanisms of CFS, and we don't know enough about what is going on for a particular patient; *and*,
2. For the medical condition CFS, decades of research have failed to provide the missing information, and further information also seems unlikely to be available in the near future. In a healthcare encounter, further information is not available in the timescale available to reduce uncertainty for an individual patient who has symptoms that suggest CFS, because the results of standard laboratory and imaging tests are unhelpful; *and*,
3. It has not been possible to construct a reliable, practical, and generally agreed model for CFS, thereby hindering understanding, prediction, and decision-making; *and*,
4. The instantiations of CFS are in many ways unique, because CFS patients' sets of symptoms differ from each other, as do the courses of their illnesses. Because this uniqueness, grouping patients for research without sufficient evidence that they are members of a homogeneous group may give unreliable results (and reduced applicability of that research to other CFS patients), even if the patients in the research study appear to share some features. Therefore, this kind of uncertainty hinders probability estimates on mechanisms and treatments for individual CFS patients.

Consequently, the uncertainties about CFS as a medical condition are at the present time 'ineliminable' (in the Knightian (1933, 1921) sense), or 'radical' (in Kay and King's (2020)

sense). I now emphasise a distinction between two situations of uncertainty regarding chronic fatigue syndrome: scientific uncertainties concerning the *medical condition* chronic fatigue syndrome, compared to the extra uncertainties in a CFS *healthcare encounter*:

- Firstly, I consider that scientific evidence concerning the medical condition CFS is *contingent* (albeit with an uncertain timescale and uncertain outcome): the results of future scientific research may resolve some or many of the significant uncertainties concerning CFS. I choose to use the term ‘radical uncertainty’ for this situation, because of the plurality of those uncertainties and their detrimental interactions.
- Secondly, and more profoundly, in a specific healthcare encounter concerning CFS, the uncertainties cannot be reduced by obtaining more information, because that information *cannot be made available during the timescale of a healthcare encounter*. I choose to use the term ‘unresolvable uncertainty’ for this particular healthcare uncertainty.

Unlike the term ‘radical uncertainty’, the term ‘unresolvable uncertainty’ is associated with an action verb, ‘to resolve’. My use of the term ‘unresolvable uncertainty’ therefore emphasises that there is no action that can be taken by the patient or the healthcare professional in the time-scale of a CFS healthcare encounter to reduce that uncertainty.

(Despite the similarity of the meanings of the words ‘unresolvable’ and ‘ineliminable’, I am obliged to avoid the term ‘ineliminable’ for this scenario because Knight (1933/1921) uses it to emphasise uniqueness and the inability to quantify risk, and I wish to use a term that also incorporates the inability to obtain further relevant information.)

The negative effects of these uncertainties concerning CFS are that it reduces or hinders *all of*, understanding, causation, prediction, and decision-making, as I described in six elements in regard to radical uncertainty in Chapter Two, Section Two. I now match all six of those same elements onto knowledge about the medical condition CFS, and in CFS healthcare encounters:

1. Radical uncertainty concerning CFS negatively affects patients' and HCPs' cognition about the situation; *and*,
2. Radical uncertainty concerning CFS limits explanation for and understanding of the present situation regarding the medical condition CFS, and of the medical situation of an individual CFS patient; *and*,
3. Radical uncertainty concerning CFS reciprocally hinders research on the mechanisms of CFS and the attribution of causes to CFS; *and*,
4. Radical uncertainty concerning CFS prevents satisfactory predictions of how the present situation will change, that is to say, there is uncertainty regarding the prognosis of an individual with CFS; *and*,
5. Radical uncertainty concerning CFS diminishes confidence when making decisions about how best to ameliorate, treat, or prevent CFS, whether in the group of CFS patients, or for an individual patient; *and*,
6. Radical uncertainty allows the existence of a plurality of views of CFS, and radical uncertainty hinders arbitration between this plurality of views.

Thereby radical uncertainty regarding CFS allows a dichotomy (or more) of views and facilitates disputes regarding explanation, understating, mechanisms, causes, predictions,

treatment recommendations, and prevention. Moreover, radical uncertainty hampers arbitration between these disputes and dichotomies of views.

However, by contrast, there are consequences or effects of radical uncertainty regarding CFS which could well be considered as being positive; these consequences match onto the three elements that I itemised in Chapter Two, Section Two in regard to radical uncertainty:

1. Radical uncertainty about many aspects of CFS allows patients and their support and advocacy groups to choose to use conceptualisations derived from what *is* known—that is, from patients’ phenomenological experience of CFS, and from analogy with medical conditions with known biomedical processes, thereby allowing people to choose what they consider to be the appropriate explanations, understanding, mechanisms, causes, predictions, treatment, and prevention regarding CFS. These choices may bring other benefits to an individual such as choosing a desired intervention and rejecting an undesired one; *and/or*,
2. Because of radical uncertainty, other people have reduced opportunity of manoeuvre to criticise those choices on the grounds of irrationality or inadequate evidence; *and/or*,
3. Radical uncertainty gives freedom for researchers to group together people with the diagnosis of CFS, in order to assess probabilities regarding treatments. (Though, I appreciate there is a disadvantage here, in that these grouping may be inappropriate, and the probabilities so obtained may therefore be spurious.)

A shared acceptance of the existence of radical uncertainty in CFS could be a route to amelioration (which I will describe further in Chapter Seven). Where there are opposing and

contradictory positions regarding the nature of a medical condition and its treatments, the existence of radical uncertainty *could* allow each of the opposing sides to consider their rivals' different interpretations to be *not unreasonable* responses to that uncertainty. The mutual acceptance of reasonableness of opponents' interpretations could be a route towards amelioration between the understandings of CFS patients and the analyses of healthcare professionals—if patients were to recognise the significance of the inadequacy of objective evidence about individual patients; and, conversely, if HCPs were to recognise and value their patients' subjective evidence.

I conclude this section by confirming that radical uncertainty in CFS is recognisable by its features and by its effects. Radical uncertainty about CFS has profound disadvantages similar to those I described in Chapter Two, Section Two, “Concepts of Radical Uncertainty”; and, in this section I have shown that radical uncertainty has disadvantages in regard to CFS. By contrast, as I described in this section, there are some potential benefits of radical uncertainty in CFS healthcare and in CFS healthcare encounters, because they can lead to a possible route to amelioration.

3.6: Conclusion to Chapter Three, with Responses to Some Counter-Claims

In this Chapter Three, I have shown that there are many uncertainties in basic knowledge about chronic fatigue syndrome. I argued that many of these areas of uncertainty mutually explain each other and explain other areas of uncertainty about CFS, so that I envisage that these areas of uncertainty are linked in a *complex network of uncertainties*. My view is that this network prevents the creation of a single generally-approved and generally-used definition, and continues to hinder research into CFS. The many uncertainties about CFS

have the negative consequence of unresolvable uncertainty in CFS healthcare encounters.

However, there are also a few positive consequences, which may include a route to compromises between CFS patients' perspectives and those of health care professionals and the possible amelioration of epistemic injustice in CFS healthcare interactions. I now move to respond to possible counter-claims to my arguments in this chapter.

One claim, which would be difficult to sustain, is that there are not severe uncertainties about chronic fatigue syndrome. It may perhaps instead be argued that the uncertainties about CFS are not 'ineliminable' in the 'Knightian' (1933/1921) sense, or 'radical' in Kay and King's (2020) sense, because some other economists assert that it is possible to estimate any risk subjectively. I respond to this counter-claim by showing that the 'subjectivist' appraisal of risk that has favour with some economists would not help to ameliorate the oppositional interpretations about CFS, of which one interpretation—based on CFS patients' understanding of their symptoms—is inherently subjective, while the oppositional interpretation—as favoured by many healthcare professionals—is based on objective evidence; and, they lead to oppositional positions. I argue that my use of the evaluative term 'deleterious uncertainty' remains appropriate in regard to CFS and supports my argument, because oppositional responses to that uncertainty have deleterious consequences in some CFS healthcare encounters.

My conclusion in this chapter—that the uncertainties about CFS are entrenched because research on CFS is itself hampered reciprocally by the many uncertainties (the 'network of uncertainties')—would be severely challenged by some possible future breakthrough in knowledge about CFS, whether that breakthrough were to be in diagnosis, in definition, in the discovery of a biomarker for diagnosis and/or for monitoring of progress, in demarcation, in

homogeneity (or the identification of significant subgroups), in the elicitation of perpetuating pathological processes, or in the discovery of successful symptomatic or curative treatments.

It may be claimed that the uncertainty problems concerning CFS will very soon be solved by one of the many current on-going research projects into biomedical mechanisms for CFS, for a biomedically-informed biomarker, or for a biomedically-based widely effective treatment.

It is true that, uncertainties in very many other medical conditions have been reduced by the application of scientific medical research during the past century and a half. However, at this stage, we have no indication of a hoped-for major breakthrough regarding CFS. My response to that challenge is that this thesis concerns the present—not the future—state of knowledge.

A hypothetical solution to as described in the preceding paragraph could well fail to reduce uncertainty and disputes in all those whose symptoms fit the present diagnostic criteria for CFS. If in the future there were to be a reliable diagnostic test or a successful treatment for CFS, these advances may not help all those whose symptoms fit with the present definitions of CFS, because of the nebulousness of the concept CFS that I have described earlier in this thesis. Some people with CFS-like symptoms may well have a negative result to that hypothetical future test or may not respond to that hypothetical future treatment. (In the following, I designate our present understanding of CFS as CFS₀, any new understanding that follows from success in research as CFS₁, and I use the term CFS₂ to designate the medical condition of those who have CFS₀ symptoms but don't fulfil the new criterion for CFS₁.) If there were to be a breakthrough in knowledge, and, say, if a biomarker for CFS₀ were to be found, then uncertainty problems regarding its definition would be reduced, creating a new, similar but not identical, condition that we could call CFS₁. However, because of the lack of homogeneity in CFS₀, it is likely that some people who are at present diagnosed as having

CFS₀ would not fulfil criteria for the new diagnosis of CFS₁. We would then designate these people as having the ill-understood and unsolved condition CFS₂. Uncertainty would remain for this (smaller) group of CFS₂ people; these people would then continue to suffer the deleterious effects of uncertainty that are now suffered by many CFS₀ patients. The lessons of this thesis would still hold for these CFS₂ patients.

Additionally, even if the uncertainties regarding CFS become reduced, there are other medical conditions that are subject at present to severe uncertainty and with evidence of epistemic injustice in healthcare encounters (such as chronic Lyme disease, fibromyalgia, long Covid, ‘Candida’, and multiple allergy syndrome), for which the lessons in this thesis would continue to be relevant.

I now move in the next chapter to expand on the deleterious consequences of radical uncertainty concerning CFS, where I describe dichotomies in CFS conceptualisations and treatment recommendations.

4: The Biomedical and Biopsychosocial Models, and their Dichotomous Applications to Chronic Fatigue Syndrome and Its Treatments

4.1: Introduction to Chapter Four

I argued in Chapter Three that knowledge regarding chronic fatigue syndrome exemplifies radical uncertainty because of multiple uncertainties, and that these uncertainties are maintained by a complex network of uncertainties. As a result of these ongoing uncertainties, our understanding of the causes of CFS symptoms—and of the applicability of treatments for those people affected by CFS—remains inadequate. In this Chapter Four I describe the two oppositional models that are applied to CFS and its treatments, the biomedical model (BMM) and the biopsychosocial model (BPS), that are differing, and oppositional, responses to that radical uncertainty. In Chapter Five I will describe other relevant continuing controversies about CFS. In Chapter Six, I will show how differing responses to each of these dichotomies and controversies can explain epistemic injustice in some CFS healthcare encounters.

The outline of this chapter is that, firstly, I describe the form of, and the claimed limitations of, the biomedical model (BMM), and then the form of, the justifications for, critiques of, and oppositions to, the biopsychosocial model (BPS). I appraise these critiques of those models. I show how these models are used to justify recommendations for medical treatments. I then show the applications of these models to CFS and its treatments, and I show criticisms of those applications.

4.2: The Biomedical and the Biopsychosocial Models

The biomedical model (BMM) and the biopsychosocial model (BPS) are models that are used in medicine to indicate disease processes, to justify treatment recommendations, and to guide the directions of research. In this section, I describe and evaluate both models, and I appraise the claim of others that the biopsychosocial model is an enhancement of and an improvement on the biomedical model.

With the establishment of scientific medicine in the nineteenth century, BMM became the prevailing approach for understanding, treating, preventing, and researching disease. Its successes and effectiveness led to strong and widespread support. The biomedical model depends on the assumption that onset, the symptoms, and the progress of a medical condition, are due mostly or wholly to its pathophysiology, with treatment recommendations being responses to those pathological processes. The patient's mind and cognition are not considered to be major considerations for understanding of the disease process, or for treatment choice, or for researching disease mechanisms and treatments. So, according to BMM, medical research on diseases should concentrate on discovering these pathophysiological processes and how treatments can reverse or at least control them.

The contrasting biopsychosocial model depends on the claim that all three elements, biological, psychological (including cognition), and social (including behavioural) contribute to the disease processes and the illness experience. Therefore, according to BPS, all these factors need to be addressed in the care of the patient, in treatment recommendations, and in medical research on mechanisms of disease and on treatments.

An early promotor of biopsychosocial model (BPS) was the physician and psychiatrist George Engel (1977). This promotion was in response to what he perceived as the limitations of the then ubiquitous biomedical model. Engel felt that BMM was inadequate, and so sought to enhance it through the incorporation of the biopsychosocial perspective. Engel introduced his concept of a biopsychosocial model in his 1977 paper, 'The Need for a New Medical Model: A Challenge for Biomedicine'. He challenged 'biomedicine' (where he was referring to what is also referred to the biomedical model), because biomedicine inappropriately depends on the assumption that disease is 'fully accounted for by deviations from the norm of measurable biological (somatic) variables' (Engel 1977, p. 130).

Engel appreciates the benefits of the epistemological and methodological aspects of the biomedical model, with its, "... record of astonishing achievements in elucidating mechanisms of disease and devising new treatments" (*ibid.*, p. 129). As the name suggests, BMM depends on understanding the abnormal biological processes underlying disease (wherever possible). However, according to Engel, BMM needs to be challenged because, when used alone, it allows the human body to be considered to be a machine, with, "... disease as the consequence of breakdown in that machine, and of the doctor's task as repair of the machine." (*ibid.*, p. 131) (also, Adler 2009, p. 609), which, "... leaves no room for psychological and behavioral dimensions of illness" (Engel 1977, p. 130). Here, Engel is using the standard distinction between 'illness' and 'disease' (as later endorsed by Sharpe & Greco 2019; Byrne 2020; Roberts 2023), in that 'illness' is the patient's experience (which includes all the physical, psychological, and behavioural deviations from being healthy), and whereas 'disease' also includes the scientific

elucidation of the pathological processes that underly the patient's illness symptoms and course.

So, in Engel's view, BMM greatly narrows the health professional's conceptualisation of what is going wrong for the patient, consequently focusing the medical professional's gaze onto the patient's body merely as a machine rather than an aspect of a complete human being. Engel sees that this narrow focusing distorts and harms doctor-patient interactions because, "...it encourages by-passing the patient's verbal account by placing greater reliance on ... laboratory measurements" (Engel 1977, p. 132), thereby reducing the medical professional's opportunity to understand the patient's predicament concerning, for instance, the severity and effects of the illness; and also allows the HCP to by-pass the patient's own interpretation and understanding of the illness. (In Chapter Six, Section Two, I will describe Fricker's (2007) concept of testimonial injustice, which, *inter alia*, criticises the by-passing of someone's verbal account; Engel claims this by-passing is more likely if the healthcare professional uses BMM.)

Engel indicates another disadvantage of BMM. He tells us the application of BMM can lead the HCP to assume that absence of abnormal examination and laboratory results in a patient with physical symptoms that there is no disease present:

The traditional biomedical view, that biological indices are the ultimate criteria defining disease, leads to ... some people with ... [negative] laboratory findings ... [who are] feeling sick are assured that they are well, that is, they have no "disease" (Engel 1977, p. 133).

Here, Engel is describing a situation which the patient has arranged a healthcare encounter, presumably because the patient is feeling ill, but where the doctor finds no objective abnormalities. According to Engel, the application of BMM could allow the HCP to tell the patient that she is well, which would be contrary to the patient's experience, and which dismissal would create an unsatisfactory healthcare encounter. (In Section Four of this Chapter, I give an example of a contribution from the medical professionals Sharpe & Greco (2019) that has superficial similarity to this situation, where they doubt that the patient has a disease, but they do accept that the patient is ill. In Chapter Six, Section Six, I report how some CFS patients may misinterpret a health professional's saying, "I cannot find a disease", as meaning, "You are not ill".)

In order to avoid these disadvantages of the biomedical model, Engel introduces his 'biopsychosocial' model (BPS) as an enhancement of the BMM. In his view, BPS encourages a wider understanding of the determinants of health and treatments, whilst still incorporating the scientific knowledge derived from the biomedical model of disease:

To provide a basis for understanding the determinants of disease and arriving at rational treatments and patterns of health care, a medical model must also take into account the patient, the social context in which he lives, and the complementary system devised by society to deal with the disruptive effects of illness, that is, the physician role and the health care system. This requires a biopsychosocial model (Engel, 1977, p. 132).

In this passage, Engel's reference to a 'medical model' aligns with what he calls the biopsychosocial model elsewhere in his 1977 article. So, according to Engel, a

biopsychosocial understanding of the causes of a disease helps the physician's choice of rational treatments. Engel does not reject what he calls biomedicine; he feels that BPS is needed to supplement it, in order to provide better healthcare interactions and better patient care.

Engel's philosophical justifications for BPS includes his view that BPS is non-dualistic, avoiding what he considers to be the harmful separation of mind from body in medical thinking and in medical care. In his view, that separation is intrinsic to the biomedical model. Unlike in BMM, in Engel's BPS the problems in a patient's body are not considered to be separate from problems in the patient's mind, thereby avoiding inappropriate mind-body dual dualism (*ibid.*, p. 130). Additionally, in Engel's view, BPS is non-reductionist in two ways: firstly, in that the patient's illness experiences are not reduced to pathological processes in the body; and, secondly, neither can the biological, psychological and social be reduced to each other. In BPS, the body and its biological abnormalities are considered to be coupled to the psychological and social factors of illness, because the illness can be understood and treated only as an integrated whole by taking all these factors into account. Engel tells us that BPS is therefore holistic and non-reductionist (*ibid.*, p. 134).

For its theoretical justification, Engel claims that BPS's holism aligns with the 'general systems theory' (GST) of the biologist Ludwig von Bertalanffy (1972). von Bertalanffy sees that systems (including organisms and their environment) can be considered to be organised into dynamically interacting levels of parts and processes (Engel 1977, p. 134; and see N. Williams *et al.* 2008). According to Engel, GST "... holds that all levels of organization are linked to each other in a hierarchical relationship so that change in one

affects change in the others ...' (Engel 1977, p. 134). Engel tells us that this GST conceptual approach is suitable for, "... studying disease and medical care as interrelated processes" (*ibid.*, p. 134), where all the levels, biological, psychological and social, are contributing to the illness.

I now give a hypothetical example that demonstrates the value of understanding disease bio-psycho-socially. This example was suggested to me as personal communication by Dr David Corfield. The closure of a coal mine and "Mr Brown's" ensuing unemployment led to depression; then his wife left him; he did not look after himself properly, and now he has coronary heart disease; the heart disease makes him unfit for many jobs, leading to poverty. Here, "Mr Brown's" case seems to demonstrate causal connections between the different elements—biological, social, and psychological—in the formulation of a patient's problems: the social problems led to psychological problems, which led to other social problems, which together led to biomedical problems, which led to more social problems. In this example, the causal connections are identifiable, and could be used as guides for suggesting interventions.

Since Engel's time, the biopsychosocial approach to medical care has gained wide support among commentators (for instance, Duncan 2000; Gatchel *et al.* 2007; Adler 2009; Farre & Rapley 2017; Bolton and Gillett 2019; Aftab & Nielson 2021; Amoretti & Lalumera 2021; Bolton 2021; Maung 2021), and amongst healthcare professionals (for instance, Horton-Salway 2002; N. Williams *et al.* 2008; Feder in Malpass *et al.* 2019, p. 300; Huda 2021; Tabb 2021). BPS is incorporated into medical training and practice: Roberts acknowledges that BPS is, "increasingly influential in medical research and practice" (Roberts 2023, p. 367). Bolton and Gillett portray BPSM as having, "...

become the orthodox overarching model for health, disease and healthcare” (Bolton & Gillett 2019, p. 5). Malpass and his philosophical colleagues (one of whom is the general medical practitioner Gene Feder) write that, “[t]he general practice gaze, developed in postgraduate training, is explicitly biopsychosocial” (Malpass *et al.* 2019, p. 300). The psychiatrist Kenneth Kendler quotes his wife (a family doctor) telling him, “[BPSM] is used widely in family medicine and is a great teaching tool, reminding the residents to consider the psychological and social influences on their cases and not just focusing on the pathophysiology” (Kendler 2010, P. 999). Similarly, “... psychosocial factors are also important in disease of known pathology” (N. Williams *et al.*, 2008). So, not only is BPS ubiquitous in medical practice, its application is appropriate and useful, according to N. Williams *et al.* (2008) and Kendler’s family doctor wife (Kendler 2010).

In summary, the biomedical model explains disease onset, symptoms, progression, and treatments solely through biomedical processes, whereas the biopsychosocial model considers not only biomedical factors, but also the influence of psychological and social aspects.

I was taught the practice of medicine according to the biomedical model when I was in medical school in the nineteen-sixties and -seventies. My experience since then has shown me that that, in general, BMM is an inadequate model for doctor-patient encounters, because, *inter alia*, people should not be treated as machines (Engel 1977); and because the present current appreciation of two-way interactions between the mind and body in illness and disease make BMM an inadequate model for understanding the processes of illnesses and for their treatment recommendations. I see that the contrasting biopsychosocial model has a much wider remit, so seems to have advantages over BMM.

However, there are major criticisms of BPS concerning both its mechanisms and the suitability for its application to medical care, as I detail in the following section.

4.3: Critiques of the Biopsychosocial Model and Its Applications

In this section, I show how despite its current ubiquity in much medical practice that I described in Chapter Four, Section Two, the biopsychosocial model is critiqued by its supporters, and criticised by its detractors, in a number of differing ways—for lacking details, that it is not a model, that it is lacking adequate metaphysical justification, that it is too narrow in its understanding of disease and treatments, that it is not as easy to use as the biomedical model, that it is inappropriate for many medical conditions, that it may not be needed, and that it can lead to adverse effects in healthcare encounters.

I order this section by, first, showing the arguments of commentators who support BPS, but who see that it has significant lacunae: Adler (2009) and Bolton & Gillett (2019). These commentators highlight what they consider to be the missing causal metaphysics of Engel's 1977 article. By contrast, I then show the arguments of commentators who play down the need for a metaphysical causal structure (Maung 2021; Aftab and Nielsen 2021). Tabb (2021) and Mescouto *et al.* (2022) suggest that BPS needs more elements. I then describe the arguments of commentators who oppose PBS: firstly, because of what is perceived as its philosophical flaws (O'Leary 2021a; O'Leary 2021b); secondly, because BPS can be misused by inappropriately adding to the medical professional's power in a healthcare encounter (Ghaemi 2009); thirdly, the application of BPS can interfere with good medical care (O'Leary 2021b); and, fourthly, because medical advances seem to

have made BPS redundant (Ghaemi 2009). I then give some responses to these critiques and criticisms.

Rolf Adler (2009) is generally supportive of BPS, but he critiques what he sees as a lack of causative mechanisms in BPS. Although Engel cites the general systems theory (GTS) of von Bertalanffy (1972) as a rationalisation for BPS, Adler complains that, “Engel has not explicitly mentioned either the question of how lower and higher systems [of GTS] are coupled and how coupled systems are separated” (Adler 2009, p. 608).

My understanding of Adler’s complaint here is that, in the analysis of a patient’s medical problems we need to understand the dynamic causative connections between the three elements of BPS; and, furthermore, for the purposes of choosing therapeutic interventions, we need to understand how to loosen or break those connections, for instance, in those cases where psychological or social problems seem to be causing physical symptoms. For example, how does BPS give clear guidance to a healthcare professional in choosing which interventions are likely to help “Mr Brown” in the example given in Chapter Four, Section Two above?

In response to Adler’s criticism of a lack of a specification of mechanisms in BPS, I note that Engel sees that in the general systems theory of Ludwig von Bertalanffy (1972), which underlies Engel’s BPS, “... all levels of organization are linked to each other in a hierarchical relationship so that change in one affects change in the others ...’ (Engel 1977, p. 134). Therefore, by using the term ‘link’ here, Engel seems to be claiming that there are causative relationships between the levels in BPS, even if he does not specify those causative mechanisms.

Derek Bolton and Grant Gillett (2019) (who are also supportive of BPS) do manage to specify what they see as those causative mechanisms. Similar to Adler's position, they stress the need for a causal theory that connects the biological, psychological, and social, in order to show how BPS functions in a way that allows these three elements to causally interact. For instance, how is it possible for there to be top-down causation from the psychological and social to the biological? That is to say, how can social or psychological problems cause biomedical disease, and how can treatments aimed at the patient's cognition and behaviour improve the biological processes of illness and disease? Bolton and Gillett respond to these questions by claiming that the various elements of BPS are connected by the flow of causative information, because these elements, "... are all in the same ontological space" (Bolton and Gillett 2019, p. 98). For Bolton and Gillett, biomedical causation differs from physico-chemical causation (and is irreducible to it) because biological causation is normative and teleological, as opposed to causation in physics and chemistry, where causation is inevitable and controlled by laws and equations that cannot be violated.

My understanding is that Bolton and Gillett's (2019) justification for allocating social and psychological elements to the same ontological space as the biological is because, in their view, all three of these elements share the property of normative and teleological causation. According to Bolton and Gillett (2019), these three elements can causally interact and be mutually causative because they are in the same ontological space; and, so, it is possible for them to affect each other, and for interventions on one of these elements to affect one or both of the others.

Hane Htut Maung (2021) challenges the need for Bolton and Gillett's (2019) causative connections in BPS. Maung notes that both he and Bolton & Gillett accept James Woodward's (2003 and 2021) interventionist (and non-metaphysicalist) account of causation. Woodward is concerned with the psychology of causal reasoning, where people choose which causes to concentrate on for the purposes of manipulation, prediction, and control. Woodward's account concentrates on recognising the *effects* of interventions rather than on the metaphysics of causation; so, for Maung, Woodward's causation is, "... metaphysically neutral and can accommodate the roles of diverse factors" (Maung 2021, p. 19). According to Maung, Woodward's restricted account makes Bolton and Gillett's (2019) causative-information-flow account superfluous, because the causal explanations that we use merely need to be relevant to our needs and aims, and these explanations can be pluralistic (Maung 2021); so, in the case of BPS, we can choose and prioritise any of the three elements of BPS, according to the seeming needs of any particular patient. (However, later in this section, I will show Ghaemi's (2009) criticism of a health professional's choosing and prioritising these elements in a healthcare encounter.)

Awais Aftab and Kristopher Nielson (2021), who also appreciate the value of BPS, go further; considering that metaphysical justification for BPS is not necessary at all, because Engel himself does not see that justification as being essential for, or integral to, his recommendations:

Engel in his vision of the biopsychosocial model was less concerned with the ontological possibility and nature of psychosocial causes, and more concerned with psychosocial influences in the form of illness interpretation and presentation,

sick role, seeking or rejection of care, the doctor-patient therapeutic relationship, and role of personality factors and family relationships in recovery from illness, etc. On the basis of this assessment, we then question Bolton and Gillett's restricted focus on accounting for biopsychosocial causal interactions (Aftab & Nielson 2021, p. 5).

Here, Aftab and Nielson (2021) see that Engel gives priority to improving medical care, and less priority to an account of medical causation.

Like Aftab and Nielson, Kathryn Tabb (2021) sees the value of BPS, but similarly attaches less importance to the metaphysics of its causal connections; instead, she feels that, in order to improve clinical care and the directions of research, BPS needs to be updated by the incorporation of ethical principles:

An updated biopsychosocial model should include explicit normative commitments about the aims of medicine that can give guidance about the sorts of causal connections to be prioritized as research and clinical targets (Tabb 2021, p. 5).

Here, Tabb sees that causal relationships in disease states can be analysed and understood, but those analyses and understandings are not enough in themselves; clinicians and researchers should also select which causal relationships are ethically more important for research and for individual patients, and concentrate on those causal relationships.

Some commentators claim that BPS is incomplete, in that it should have more than the three elements, bio-, psycho-, and social. Karim Mescouto and colleagues (2022) (who are physiotherapists and a sociologist) appreciate the value of Engel's BPS model, but their critical review of sixty-six research articles concerning how healthcare professionals apply BPS to low back pain found that, in general, the professionals' focus was narrowly on the biological and cognitive behavioural aspects of the BPS model, while, "... broader aspects such as interpersonal or institutional power relations, cultural considerations, ethical, and social aspects of health may not be incorporated into physiotherapy research and practice" (Mescouto *et al.* 2022, p. 3270).

Like Tabb (2021) and Mescouto *et al.* (2022), the academic general practitioners Nefyn Williams *et al.* see the value of managing the biological, psychological, social (and spiritual, in their view) components of illness as an integrated whole; but, they see evidence that, in practice, BPS is frequently used not used as intended, because clinicians seem to find the biomedical model easier to use, which could be the reason that some researchers find that BMM is still in use (N. Williams *et al.* 2008, p. 1).

I now move to show oppositional arguments to the biopsychosocial model.

Notwithstanding the claims of its popularity and claimed usefulness, there are commentators who see significant philosophical problems with BPS, or limitations to its applicability; and, some commentators oppose its use at all. I address all these types of criticisms in the next part of this section; I include my appraisals of and responses to these criticisms.

The psychiatrist Niall McLaren (1988) objects to BPS on both philosophical and practical grounds. McLaren notes that, even though Engel summarises the advantages of BPS in his 1977 article, he does not actually give a comprehensive elucidation of what form BPS takes, nor how it functions (McLaren 1988, p. 89). He argues that BPS should not even be considered to be a model, because it is not based on an underlying and supporting theory (McLaren 1998, p. 89), and because one cannot make long-term predictions using BPS (*ibid.*, p. 91), both of which should be possible in a model, according to McLaren. Engel's lack of specification of the details of BPS and how it is meant to function and used allows medical professionals to use it in ways that cause unsatisfactory healthcare encounters (McLaren 1998). Unlike, for instance, Aftab & Neilson (2021), McLaren (1998) does not accept that a model can still be of value even if it 'only' gives explanations rather than predictions.

However, I understand that BPS may well enable short-term prediction-making, such as estimating the prognosis of the illness in a particular patient, or predicting the consequences of therapeutic interventions; prognosis and rational treatment-choice of this kind are both necessary in medical practice. I agree with McLaren's (1998) criticism that Engel's BPS does not specify how it should be used; this lack of specification can be problematic concerning its application to CFS, as I show in the next section.

Alexander Roberts's (2023) critique of the biopsychosocial model (which he abbreviates to BPSM) goes further than that of McLaren (1998), because Roberts sees that BPSM does not aid explanation. Roberts tells that, "...although Engel later writes of 'the proposed biopsychosocial model' (Engel 1977, 134-5), he does not, in fact, propose a model" (Roberts 2023, p. 373). Roberts challenges the claim that BPSM is a model

because, in his view, it fails to fulfil the role of a scientific model, which should have explanatory power, should be able to define diseases and distinguish disease from non-disease, and should have the ability to identify genuine cause-and-effect relationships. According to Roberts, BPSM can fulfil none of these roles of a scientific model; therefore, BPSM, "... offers no tools for producing valid (or probabilistically true) knowledge claims" (*ibid.*, p. 368). Roberts claims that, "[B]iopsychosocial' can appropriately be called a ... 'conceptual framework' ..., and not an explanatory model of disease" (Roberts 2023, p. 370) that should not be used, "... for the purposes of defining disease(s) or establishing causal relationships" (*ibid.*, p. 371). Roberts (2023) evidently sees limitations to the practical value of BPS.

I agree with Roberts (2023) here that BPS as described in Engel (1977) is underspecified. I appreciate that Engel's concept of BPS may well be limited in the ways that Roberts identifies; Engel did not seem to be aiming at the establishment of cause-and-effect relationships, distinguishing disease from non-disease, or defining 'disease', as Roberts (2023) expects a model to do. Indeed, Engel's intention was more limited: to bring the psychosocial determinants of health into the healthcare encounter, for diagnostic and therapeutic purposes. In defence of Engel's use of the term 'model' (as criticised by Roberts (2023)), I see that Roberts's seemingly dismissive phrase 'conceptual framework' is similar to Engel's term, 'conceptual approach' (Engel 1977, p. 134).

The philosopher and ethicist Diane O'Leary objects to BPS because she perceives that it fails on metaphysical grounds. She sees that the BPS model has a, "... deeply inconsistent position on dualism" (O'Leary, 2021b, p. 6): it is philosophically incoherent (*ibid.*, p. 11), because she does not accept Engel's metaphysical arguments. According to

Engel, BPS's ontology is characterized by opposition to the dualism of BMM, that is to say, by the idea that mind can, "... be reduced to purely material and deterministic neurological mechanisms" (*ibid.*, p. 8). In his advocacy for BPS, Engel opposed what he saw as the reductionism and dualism of the biomedical model:

[T]he biomedical model embraces both reductionism, the philosophic view that complex phenomena are ultimately derived from a single primary principle, and mind-body dualism, the doctrine that separates the mental from the somatic (Engel 1977, p. 130).

O'Leary derides Engel's claim here, declaring that,

[R]eductive physicalism and Cartesian dualism are mutually exclusive, so it's not possible for Engel to be correct in framing the BMM as reductive dualism, or dualistic reductionism... [Therefore] BPSM is aiming for an incoherent goal, setting out to reverse a position that was impossible in the first place (O'Leary 2021b, p. 9).

Here, O'Leary sees that 'reductive dualism' and 'dualistic reductionism' are internally contradictory). O'Leary's preferred metaphysical position is non-reductive physicalism,

Nonreductive physicalism (where we accept that mental properties are distinct from physical properties, but reject dualism) [differs from] naturalistic dualism (where we accept that mental properties are distinct from physical properties and accept dualism) (O'Leary 2021b, p. 9).

In non-reductive physicalism mental events do supervene on physical events, but are not reducible to them. From this interpretation, BMM is non-dualistic, in that the physical and psychological effects of medical conditions both supervene on physical events.

O'Leary is of the view that the bases of Engel's support for BPS and his opposition to the biomedical model are flawed, because Engel has mischaracterised the ontology of BMM. Engel's opposition to dualism in the biomedical model—the separation of mind and body—seems to O'Leary to be referring merely to an epistemological practice (*ibid.*, p. 10), implying that she understands that when using Engel's BPS instead of BMM, the medical professional can choose to consider the mind and body being at times together or being at other times separate, as seems appropriate to the healthcare professional for the occasion. However, for O'Leary,

... dualism is actually not an epistemological position. Dualism does not come and go depending on the ideas we prefer or the words we choose. If the world is dualistic, then two kinds of things exist in the world, no matter what we say or think or do in medical practice (*ibid.*, p. 10).

Here O'Leary claims that ontological non-dualism disallows epistemological dualism, so Engel's metaphysics is incoherent.

I accept O'Leary's disparagement of Engel's metaphysical conceptualisation of the biomedical model, because I also see a tension between Engel's criticism of BMM being dependent on the reduction to a *single principle*, and his further criticism of BMM as a

depending on the inappropriate separation of *two entities*, the mind and the body.

However, *pace* O’Leary, it seems to me that Engel’s stance is that BMM is inappropriately *epistemologically* mind-body dualist; but, his condemnation of the *epistemological* dualism in BMM does not (in my understanding of his view) need to be supported *metaphysically*, neither by ontological mind-body dualism, nor by ontological mind-body non-dualism.

Disputes about BPS do not derive just from its theoretical justifications and metaphysics; the implementation of BPS can be problematic: “[M]any criticisms of BPSM are directed at the manner in which BPSM has been operationalized and implemented” (Aftab & Neilson, p. 11).

The psychiatrist Nassir Ghaemi (2009) has two objection to the application of BPS: that is does not give adequate guidance, and, anyway, it is often not necessary. (I address Gheami’s claim of lack of necessity later in this section.) Ghaemi sees that BPS does not give guidance to medical professionals on how to apply the biological, the psychological, and the social; he therefore challenges BPS on the grounds that the application of BPS can add inappropriately to the medical professional’s power in a healthcare encounter:

[B]iopsychosocial advocates really seek eclectic freedom, the ability to ‘individualise treatment to the patient’, which has come to mean, in practice, being allowed to do whatever one wants to do. This eclectic freedom borders on anarchy: one can emphasise the ‘bio’ if one wishes, or the ‘psycho’ (which is usually psychoanalytical among many biopsychosocial advocates), or the ‘social’.

But there is no rationale why one heads in one direction or the other (Ghaemi 2009, p. 3).

Ghaemi sees that BPS gives medical professionals freedom to enact their own dogmas, *i.e.*, “... eclecticism produces dogmatism: anarchy leads to tyranny” (*ibid.*, p. 3).

Anarchy and tyranny are strong words, so Ghaemi is clearly claiming that there are major problems with the use of BPS. Ghaemi complains that clinicians apply the elements of BPS eclectically, and, in his view, this eclecticism allows, “... scant attention [to be] given to biological or social features” (*ibid.*, p. 3) in a healthcare encounter. I extend Ghaemi’s argument here to suggest that in a healthcare encounter with a not-unusual (and unsatisfactory) power imbalance between healthcare professional and patient, BPS allows the health professional eclectic freedom to impose a bio-, a psycho-, or a socio-interpretation (and their concomitant treatment recommendations) according the professional’s own judgements (as accepted by Maung (2021), and as criticised by Maes & Twisk (2010)), without necessarily incorporating the patient’s judgements. Here Ghaemi is criticising how BPS is implemented, as do Aftab & Neilson (2021, p. 11).

The family doctor Robert Bartz gives empirical support to Ghaemi’s objection to BPS; in Bartz’s view, BPS gives healthcare practitioners freedom to impose their treatment recommendations. Bartz shows from studies of healthcare interactions that BPS seems to give too much power to the health professional (Bartz 1999, p. 601), in those cases where the health professional attempts to *change the patient’s perspective* to adapt to the HCP’s own BPS understanding. These attempts conflict with Bartz’s ideal consultation style, where, in his view, the patient and the doctor should be working together to co-construct the approach to illness care (Bartz 1999, p. 605).

In the next section (4.4), I will show how some medical commentators do interpret BPS ‘eclectically’, by playing down, or even disregarding, the biological aspects of CFS. Their interpretation seems to be, if there is inadequate evidence for biomedical processes, then the mechanisms of the patient’s symptoms are likely to be psychological (or cognitive) and/or social (or behavioural). I will show in Chapter Six of this thesis how medical professionals can use their biopsychosocial judgements in this way, along with the power imbalance, to conduct CFS healthcare encounters epistemically unjustly.

Like Ghaemi and Bartz, O’Leary objects to the application of BPS in healthcare encounters. Moreover, her objection is even more momentous: the inappropriate use of BPS results in dangerous errors. O’Leary writes:

When the [BPS] model opens the door to psychosocial diagnosis for bodily symptoms in everyday practice, clearly it opens the door to a new and threatening (*sic*) form of diagnostic error (O’Leary 2021b, p. 14). [The original seems to have a misspelling of the word ‘threatening’.]

So, according to O’Leary, a healthcare professional using BPS could fail to make an important physical diagnosis by dismissing the patient’s bodily symptoms as merely psychosocial. She gives the example of a university lecturer, Dr Smirl, whose chest symptoms were incorrectly dismissed by her doctors as being psychosocial (Smirl 2021; Philipson 2013; O’Leary 2021b). O’Leary sees this major error as being the consequence of a, “... BPS clinician managing unexplained symptoms with deliberate diagnostic vagueness, making sure never to force these disorders into either a ‘mental’ or ‘physical’

classification” (*ibid.*, p. 15). Here, O’Leary claims that a healthcare professional is being inappropriately influenced by BPS. Her view seems to be that, if a healthcare professional were to consider that all disease is biopsychosocial, then that HCP would be giving him- or herself the choice to assign the patient’s symptoms to the psychological and/or social elements of BPS, and to downplay the possibility of biomedical element. In O’Leary’s view, this dangerous ‘vagueness’ results from BPS’s metaphysical confusion on dualism; so that, in a healthcare encounter, her view is that it is a mistake, “... to avoid diagnostic practice that ‘separates mind and body’ at all costs” (*ibid.*, p. 16):

[T]hough we accept that bodily health is inextricably interwoven with psychosocial well-being, we insist that at the level of medical practice psychiatric conditions must be clearly distinguished from biomedical conditions (O’Leary 2021a, p. 1989).

Here, O’Leary insists that, in clinical practice, there should be a clear distinction between mental and physical conditions in disease classification and in disease management.

However, I add a contrasting example to O’Leary’s concern. I see that an HCP having a purely biomedical agenda may fail to elicit important psychological or social contributions to the patient’s distress and illness (as in the case of “Mr Brown” that I described in Chapter Four, Section Two). This, in my understanding, is one of Engel’s (1977) chief concerns, and motivation. My interpretation is that the possibility of each of these consequences (the inappropriate downplaying of the biomedical, or the inappropriate downplaying of the psychological or social) give support to the need for flexibility during health care encounters, with the need for a soft (or ‘vague’) demarcation

between physical and psychological interpretations of a patient's problems. This is not O'Leary's view.

Ghaemi's second objection to BPS is that it is often not necessary because, in many medical conditions, scientific medical advances have made the reductionism of the biomedical approach more appropriate than the holism of BPS:

Reductionism is not always wrong; peptic ulcer disease, long considered a classic psychosomatic illness, proves to be caused by *Helicobacter pylori*. In a hallmark of science, the apparently complex proved to be simple (Ghaemi 2009, p. p. 4).

Here Ghaemi references the discovery in the early 1980s of the role of the bacterium *Helicobacter pylori* in some people's peptic ulcers. The peptic ulcers of most of these people were found to respond to a short course of antibiotics and anti-acid medication. So, according to Ghaemi, reductionism replaces 'psychosomaticism' in regard to peptic ulcer, which in his view justifies BMM replacing BPS in this condition and, by implication, in many others.

By contrast, the neuroendocrinologist George Fink notes with disapproval that the present consensus gives support to Ghaemi's argument here: "[T]he aetiological pendulum of gastric and duodenal ulceration has swung from 'all stress' to 'all bacteria'" (Fink 2011, p. 107). (Gastric and duodenal ulcers are the commonest examples of peptic ulcer.) Here, Fink feels that the pendulum has swung too far. Fink (*ibid.*, p. 113) challenges Ghaemi in his characterisations of the *present* understanding of the causes of peptic ulcers, because

in Fink's view, the Helicobacter-peptic-ulcer theory is itself challenged by equivocal evidence:

Most people living with H. pylori never develop ulcers, about 30% of patients with ulcer do not harbour H. pylori infection and some patients in whom H. pylori colonisation has been eliminated by antibiotics subsequently develop new ulcers (*ibid.*, p. 113).

Similarly:

[T]he etiological reality clearly favours the multifactorial treatment. H. pylori is neither necessary nor sufficient for duodenal ulcer, and nor is its elimination from a patient either necessary or sufficient for the curing of an ulcer (Broadbent 2009, p. 309).

Here, Fink and Broadbent claim that, empirically, the link between Helicobacter (H. pylori) and peptic ulcer is therefore weak. Fink claims the weakness of that link is explained by 'stress' remaining an important causative risk factor (*ibid.*, p. 113); thereby, in contrast to Ghaemi, Fink sees that peptic ulcer remains a psychosomatic disease that does not reduce to causation by a bacterial infection.

Furthermore, I challenge both Ghaemi and Fink in their characterisation of *past* opinions on the causes of peptic ulcers. From the 1970s onward (before the discovery of the role of *Helicobacter pylori*), the predominant explanation for peptic ulcers was biomedical,

that peptic ulcers were due to excess acid, and that, consequently, patients were treated with operations or medications that were known to reduce that excess acid:

[In] 1972 ... Black's H₂ receptor blockers [e.g., cimetidine]... changed the ulcer world for ever, and confirmed the threshold model – that a powerful enough acid inhibitor would heal any ulcer (Dr Hugh Baron, in Christie & Tansey 2002, p. 6).

So, from the 1970s to the 1980s, the most successful interventions for peptic ulcer were in fact biological, not psychosocial. These biological interventions were antacids, anti-acid-secretion drugs such as cimetidine, and operations such as vagotomy and pyloroplasty or partial gastrectomy. Furthermore, there was evidence from the 1950s onwards that peptic ulcer prevalence and exacerbations of peptic ulcer were *not* correlated with stress (Sir Richard Doll and Professor Roy Pounder in Christie & Tansey 2002, p. 9; Dr Raymond Kirk in Christie & Tansey 2002, p. 12). Psychosocial interpretations of peptic ulcer disease, with management of the ulcer patient's stress, anxiety, or depression were usually not enough, so it is incorrect to claim that peptic ulcer was considered psychosomatic in the modern meaning of the term:

[C]onditions once thought to be 'psychosomatic' have later been determined to have physiological causes (Lubet & Tuller 2021, p. 1).

Here, Lubet and Tuller (2021), like Ghaemi (2009), note that there has been a change in the classification of some medical conditions over time, away from the 'psychosomatic' label as more knowledge has been gained about them.

I argue that this change is not only due to improvement in knowledge about the pathological processes involved in these conditions, but is also due to changes in the usage of the term ‘psychosomatic’ over the past seventy years, from a description of medical practice to an attribute or characterisation of some medical conditions. From the nineteen fifties onwards, ‘psychosomatic medicine’ used to refer to a medical practice for managing illnesses with clear physical features where psychological factors were thought also be contributory (such as asthma, peptic ulcer, ulcerative colitis, hypertension, thyrotoxicosis, rheumatoid arthritis, and neurodermatitis) (Martin 1978, p. 699). This former usage of ‘psychosomatic’ depends on the acceptance of the existence of *two-way causation* regarding physical symptoms and psychological processes—they can influence each other positively or negatively, so therapeutic interventions can be towards either, or both. By contrast, the usage of the term ‘psychosomatic’ from the nineteen eighties onwards has been as an attribute or characterisation of medical conditions that lack adequate evidence of physical processes (such as chronic fatigue syndrome). This present usage depends on the assumption of only *one-way causation*, from the mind the body; that the bodily symptoms derive from problems of the mind or cognition. The term, ‘psychosomatic’ can be derogatory if there is also an implication that the patient has the power to cause the bodily symptoms, and has the power to ameliorate them. This change in usage means that it is not appropriate to equate (as do Ghaemi (2009) and Lubet & Tuller (2021)) medical conditions that used to be managed using ‘psychosomatic medicine’ with medical conditions (such as CFS) that nowadays are sometimes dismissed as being merely ‘psychosomatic’.

I now respond further to some of the criticisms of BPS. I accept Adler’s (2009) criticism of BPS, that Engel does not specify its causal structure. I also agree with Ghaemi (2009)

and Bartz (1999); the lack of a clearly-established and generally-agreed causal structure for BPS does allow freedom to a healthcare professional to interpret BPS eclectically, whether when interpreting the patient's story, or when deciding on treatment recommendations. An HCP could also choose to ignore other factors that the patient may feel are relevant (such as past or present stressors, as itemised by Mescouto *et al.* (2022)). Each of these impositions can lead to patient dissatisfaction. Dissatisfaction of this kind contrasts with Engel's (1977) original intention, which was *not* to by-pass the patient's verbal account (Engel 1977, p. 132). The accusation of a lack of an underlying theory for BPS could be answered in a number of ways: firstly, that Engel did specify a theory; or, secondly, that there is a theory that can be applied to it (even if Engel did not specify it); or, thirdly, that it does not need an underlying theory at all. McLaren's (1998) criticism of the lack of theory for BPS can be countered by Engel's claim that there is an underlying theory that supports BPS, that is to say, von Bertalanffy's 'general systems theory' (Bertalanffy 1972; Engel 1977, p. 134). The accusation that BPS is not actually a model (despite Engel's use of the word in his title – "The Need for a New Medical Model: A Challenge for Biomedicine" (Engel 1977)), can be also answered in two ways: it is indeed a model, according to others' later interpretations with, for instance, Bolton and Gillett's theory of information transfer (Bolton & Gillett 2019); or that, even if it is not a scientific model as normally understood (with a structure that shows its functions and how it can be manipulated), it is still of practical value in healthcare interactions as an aide-memoire and a teaching tool for the practice of medicine. In response to Roberts's (2023) criticism of Engel's use of the term 'model', I accept BPS as a 'conceptualisation' (as I indicated in the Abbreviations section at the beginning of this thesis), which term aligns with Roberts's seemingly dismissive phrase, 'conceptual framework' (Roberts

2023, p. 370), and Engel's term, 'conceptual approach' (Engel 1977, p. 134); though, I follow many of my sources by referring to it as a model.

To summarise this section: Engel (1977) promotes BPS as having wide applicability towards improving healthcare encounters in physical medicine and in psychiatry. The biopsychosocial model of disease has become widely used in medical practice. Feder (in Malpass *et al.* 2009) tells us that he uses BPS as a guide in medical practice, seemingly in the way that Engel intended. However, BPS is criticised by some commentators for lacking detail, lacking a firm metaphysical foundation, and having too narrow a focus. Furthermore, in a healthcare interaction, BPS can give freedom for the healthcare professional to choose which elements of BPS to use or not use. Roberts accepts that BPSM is, "a useful tool for organizing and communicating information about the psychosocial determinants of health" (Roberts 2023, p. 367). Aftab & Neilson 2021 see that BPS can improve medical care when it is used as an aide-memoire for the clinical encounter and for education (Aftab & Neilson 2021, p. 13): "The biopsychosocial formulation [is] a useful educational and clinical tool (*ibid.*, p. 10) ... [as] a reminder not to ignore psychosocial variables" (*ibid.*, p. 12). Mescouto *et al.* (2022) and N. Williams *et al.* (2008) appreciate the value of BPS but suggest that its elements need to be widened because many clinicians misuse it too narrowly. N. Williams *et al.* (2008) remind us that it is possible for BPS be applied incorrectly, when a medical practitioner fails to apply the necessary integration of its elements into assessment and management. McLaren sees that BPS does not have the characteristics of a model because it is of little use for making predictions. Ghaemi (2009) accepts that BPS might have value as a heuristic, but he sees that BPS gives too much power to healthcare professionals so that some of them are able to misuse it; and, anyway, medical advances make BPS unnecessary. For O'Leary BPS is

too flawed philosophically to be of any value in healthcare encounters (O’Leary 2021a), and it instigates a “threat[en]ing form of diagnostic error” (O’Leary 2021b, p. 14).

4.4: The Dichotomous Applications of the Biomedical and Biopsychosocial Models to Chronic Fatigue Syndrome.

In this section I argue that uncertainty about chronic fatigue syndrome, along with the many dichotomies concerning its nature, results in the adoption of contrasting approaches to treatment for it, derived from the biomedical and from the biopsychosocial models. The biomedical and the biopsychosocial models in CFS, and their treatment or management recommendations, are in opposition; so, there may be conflict in CFS healthcare encounter where, say, the patient favours the BMM interpretation and management recommendations and the healthcare professional favours the BPS interpretation and treatment recommendations.

I order this section by first showing support for BMM by CFS patients, their support groups, and some philosophical commentators. I then show support for BPS by some medical authorities. I describe how four often-recommended management strategies follow from BMM and BPS. Pacing follows from the biomedical model, while adaptive pacing therapy, graded exercise therapy and cognitive behaviour therapy follow from the biopsychosocial model. I describe and explain support and opposition to these dissimilar treatments. I then report reviews of clinical trials that assess the effectiveness of these therapies. I report the results of surveys of CFS patients and of healthcare professionals

that show oppositional perspectives to these therapies. In Chapter Six I will show how these conflicts can explain epistemic injustice in some CFS healthcare encounters

The major dichotomy regarding CFS is the choice of the appropriate model or conceptualisation for understanding the condition, for deciding on treatment recommendations, and for directing research (Wojcik *et al.* 2011b; Geraghty & Blease 2018; Geraghty *et al.* 2019; Thoma *et al.* 2024). This dichotomy is based on differing conceptualisations of CFS, that is to say, whether the condition is just physical in causation, or whether psychological and behavioural factors are significant (with or without physical processes underlying the symptoms). Dichotomies about treatment recommendations follow from uncertainties concerning their applicability, and from uncertainties regarding the significance and reliability of the results of research into mechanisms and treatments.

The conceptualisation preferred by many people living with chronic fatigue syndrome and their patient support and advocacy groups is a biomedical model (BMM) of it, the view that the causes of CFS are only physical (although these physical processes are not adequately understood). (I apprehend that this model is preferred by CFS patients because, in their experience, physical *symptoms* predominate and seem to have the most disabling effects.) According to this biomedical model, CFS is a physical disease, with only (a) physical cause(s), and consequently requiring treatments that are physical (such as medications) (Geraghty & Blease 2018; Geraghty *et al.* 2019; Thoma *et al.* 2024) (even though effective physical treatments have not yet been discovered). In the present absence of adequate knowledge of these physical causes and physical treatments, CFS patients and their support and advocacy groups find that deliberate rest is an effective

management for controlling the symptoms. Patients' support for BMM in CFS is derived from personal experiential evidence (Pomey *et al.* 2015), analogy with other better-understood diseases (for instance, myxoedema), evidence of abnormalities in laboratory research on CFS, social (group) epistemology from their support and advocacy groups and websites, their experience of symptoms worsening following exercise and following graded exercise therapy (GET) (which targets behaviour), and the frequent failure of cognitive behaviour therapy (CBT) (which targets patients' cognition regarding their symptoms).

Many philosophical and other commentators support those CFS patients and their advocacy groups who claim that chronic fatigue can be best understood and managed according to the biomedical model (Maes & Twisk 2010; Geraghty & Blease 2018; Cortes Rivera *et al.* 2019; Geraghty *et al.* 2019; Missailidis *et al.* 2019; Twisk 2019; Lidbury & Fisher 2020; National Health Service (NHS Inform, accessed 20/5/25)). This claim is that the symptoms and course of the condition are caused by pathological physiological processes in the human body. Associated with the BMM attribution of CFS is opposition to the biopsychosocial model and its consequent treatment recommendations, on the grounds that BPS depends on an incorrect understanding of CFS (Horton-Salway 2002; Geraghty & Blease 2018; Geraghty *et al.* 2019; Thoma 2024). Furthermore, according to this BMM view, all *research* on CFS should have the aim of looking for only physical causes and treatments; so, researchers on CFS should *not* look for psychological or behavioural causes or treatments (Action for ME, 'Research Campaigns'; Invest in ME Research; Quadram Institute).

The opposing conceptualisation, as held by many healthcare professionals, is that, in the absence of objective evidence of physical disease, CFS symptoms are considered to be due to processes that are mostly psychological and behavioural; this view is usually called the biopsychosocial model (BPS) of CFS. The appropriateness of applying BPS to CFS is given some support by the continuing absence of biomedical understanding of CFS, the continuing absence of effective biomedically-based treatments, and the partial success of CBT and GET in some patients and in clinical trials (e.g., the PACE trial—White *et al.* 2011). According to the way that BPS is interpreted by some medical professionals and then applied to CFS, the continuing symptoms of CFS are due to a number of emotional or psychological factors, including patients' inappropriate cognition regarding the symptoms (the 'mistaken' idea that the symptoms are part of a major physical disease that requires management with deliberate rest); and that CFS patients' inappropriate responses to those cognitions perpetuate the symptoms because CFS patients are avoiding exercise and are resting too much (Vercoulen *et al.* 1998). Thus, according to this psychological and behavioural interpretation, CFS patients need to change their interpretation of their symptoms (helped by CBT) and to exercise more (according to an exercise schedule where the patient is encouraged to increase exercise activity).

The disparities of support for BMM and BPS in CFS/ME are shown in the analysis of one-hundred-and-six source files gathered by the medical researchers Zahra Hossenbaccus and Peter White (2013). They performed a general search on 'myalgic encephalomyelitis', 'myalgic encephalopathy', 'ME', 'chronic fatigue syndrome', 'CFS', 'post-viral fatigue syndrome', 'PVFS', and 'post-infectious fatigue syndrome'. They then analysed the attitudes of thirty-six patients' organizations, seventy-two media articles and

thirty-eight medical authorities towards the physical and psychological interpretations of CFS/ME. Here are the results of these analyses, in numbers, not percentages:

	Physical	Both	Psychological	Totals
ME organisations	32	4	0	36
Media	42	23	7	72
Medics	9	24	5	38

Table Two: The results of surveys of the views of media, patient groups and the medical establishment (from Hossenbaccus & White 2013, p. 4)

Here, ‘ME organisations’ refers to ME advocacy organisations. ‘Media’ refers to the British daily newspapers, The Daily Mail, The Guardian, and The Daily Telegraph.

‘Medics’ refers to medical authorities, that is so say, medical organisations and other authorities (NICE, NHS Plus, NHS 24, NHS Choices, Department of Health and Royal Colleges of General Practitioners, Paediatrics and Child Health, Physicians and Psychiatrists), and not individual healthcare professionals.

In this review, Hossenbaccus and White show that those ME organisations whose content they analysed all reject a ‘psychological only’ categorisation of CFS/ME, and, in general support an ‘only physical’ categorisation. By contrast, a large majority of the medical authorities examined see that psychological factors are significant; while, only a small minority of medical authorities support a ‘physical only’ categorisation. The views of three national UK newspapers were assessed by Hossenbaccus and White as being intermediate between ME patient organisations and medical authorities (*ibid.*, p. 4).

Hossenbaccus and White see the problematic consequences of these discrepancies:

The considerable disagreement, particularly between ME patient organizations and medical authorities, may help to explain the gulf in understanding between doctors and patients and the consequent reluctance of some patients to engage in behavioural treatments (*ibid.*, p. 1).

Here, Hossenbaccus and White see that these differences explain ‘gulfs of understanding’ about ME and CFS between individual patients and healthcare professionals in healthcare encounters; these gulfs of understanding explain why many patients reject behavioural therapies.

Hossenbaccus and White’s (2013) conclusions align with some of the arguments in this thesis, that disagreements about the categorisation of CFS/ME explain some of the disagreements within CFS/ME healthcare encounters, including disagreements concerning treatment recommendations.

The psychologist Megan Marks and colleagues add information concerning differences *between* healthcare professionals concerning CFS. Marks *et al.* conducted interviews with ten health professionals who had a great deal of experience working with children and adolescents with chronic fatigue syndrome, in order to explore the origin of these differences, and how these differences impact healthcare encounters. They report:

[A]ll [interviewees] recognised the contribution of physiological and psychological factors [in CFS]; however, differences appeared in the emphasis

given to these. Some HCPs believed in an undiscovered physiological cause, while others believed symptoms served particular [psychological] functions (e.g. avoiding anxiety) (Marks *et al.* 2016, p. 2262).

Here, Marks *et al.* see differences between healthcare professionals concerning physiological and psychological factors in CFS. Marks *et al.*'s 'physiological cause' refers to what other commentators call 'physical' or 'biomedical' cause. The view of Marks *et al.* is that these differences are explained by uncertainty about CFS due to, "... lack of solid empirical understanding" (*ibid.*, p. 2665). In this situation of uncertainty, Marks *et al.* see that, "... health professionals rely on previous experiences to make sense of the condition and inform their clinical practice" (*ibid.*, p. 2658). (My interpretation is that here, by the use of the term, 'previous experience', Marks *et al.* are referring to these professionals' experience in other medical conditions.) According to Marks *et al.*, the lack of agreement among HCPs allows, "... variability in how young [CFS] people's difficulties may be conceptualised, labelled and treated [by their healthcare professional]" (*ibid.*, p. 2665).

My view is that this variability between HCPs could cause difficulty for patients if they receive conflicting advice from differing health professionals. Marks *et al.*'s understanding that empirical uncertainty explains differences of perspective about CFS and differences in management recommendations aligns with a part of the main argument of this thesis.

In regard to treatment options, commentators on each side of the debate about the applicability of BMM or BPS to CFS see a correlation between the model used for

understanding CFS and consequent treatment recommendations. I first give the perspective of those who have a BMM conceptualisation of CFS; they tend to recommend ‘pacing’ as an energy management strategy, which practice is supported by the ‘energy envelope theory’ of CFS (Jason *et al.* 2008; Sanal-Hayes *et al.* 2023).

The energy envelope is given as an explanation for post-exertional malaise (Jason *et al.* 2008). PEM is one of the most intrusive and disabling symptoms of CFS; PEM has significantly deleterious effects on the patient’s ability to live a normal life:

Post-exertional malaise (PEM) is a delayed worsening of symptoms that occurs after minimal physical or mental activity. The key feature of PEM is that the malaise (extreme fatigue and flu-like symptoms) and other symptoms experienced are not in proportion to the amount of activity that has been done. PEM is often delayed and may be experienced hours or days after the activity took place but is most likely to occur 1-2 days after the exertion event (Johns Hopkins, ‘Chronic Fatigue Syndrome – Post-Exertional Malaise’).

PEM is felt as much more than simple ‘malaise’; it is significantly incapacitating, and can last for a long time; additionally many of the other symptoms of CFS are also exacerbated during episodes of PEM. The above extract, which is from a hospital patient information leaflet for people with CFS, tells us that the severity of PEM symptoms after exertion is often disproportionate to the activity that triggered those symptoms; a seemingly minor physical, social, emotional, or mental exertion may well give symptoms so severe that the CFS patient is compelled to be confined to bed for days or even weeks. PEM is so characteristic of CFS that it is part of most definitions of ME, CFS, or ME/CFS

(‘Elements of Selected Case Definitions and Diagnostic Criteria for ME/CFS’, National Academies of Sciences, Engineering, and Medicine, Institute of Medicine (2015)). The prevalence of PEM and its negative effect on people’s lives is attested to by clinical researchers (Jason *et al.* 2008) and many CFS patients. The explanation for PEM is claimed to be the existence of a hypothetical ‘energy envelope’ in CFS, meaning that CFS patients have only a set limited amount of energy to expend on any one day, and that exceeding that limit is likely to precipitate PEM with exacerbations of many CFS symptoms, within a day or so. The energy envelope theory appears to be a biomedical model for CFS; however, I see that it is a description of symptoms rather than a summation of detected pathological processes. The specific biomedical or pathophysiological processes underlying this energy envelope remain unclear, thus preventing the pinpointing or measuring of the envelope biochemically (a biomarker) in any particular patient, nor for the objective measurement of any biochemical parameters of the severity of the envelope, nor to provide targeted medications for the envelope (see Sanal-Hayes *et al.* 2023; see the references of *MePedia*, ‘Energy Envelope Theory’, accessed 1/2/26). In the absence of objective measures of, or targeted therapies for, PEM, CFS patients respond to their perception of having an energy envelope by limiting their activity, whether that activity is physical, emotional, intellectual, or social:

The Energy Envelope Theory ... posits that maintaining expended energy levels consistent with available energy levels may reduce the frequency and severity of symptoms (Jason *et al.* 2008, p. 189).

Here, Jason *et al.* tell us that CFS patients respond to their energy envelopes by limiting the expenditure of their energy up to the limits of their perceived available energy, and no

further. This coping strategy, the deliberate limiting of activity in CFS, is referred to as ‘pacing’, which can be described as, “... regulating activity to avoid post exertional malaise (PEM), the worsening of symptoms after an activity” (Sanal-Hayes *et al.* 2023, p. 1). ‘Pacing’ is an energy management strategy for CFS intended to reduce the frequency and severity of episodes of ‘post exertional malaise’ (PEM) (but not specifically to alter the course of CFS).

Reviewers of medical research regarding pacing come to differing conclusions. The medical researchers Jesus Castro-Marrero and his colleagues collate, review, and analyse the results of trials of pacing in CFS. They find that, “... pacing was consistently shown to be the most helpful treatment ...” [compared to cognitive behavioural therapy and graded exercise therapy] (Castro-Marrero *et al.*, 2017, p. 364). However, by contrast, a more recent review conducted by the psychologist Nilihan Sanal-Hayes and her colleagues of trials of pacing in CFS shows that these trials gave varying results, with some trials showing improvement, some showing no effects on fatigue, physical functioning, or pain scores in CFS, and some trials showing pacing having detrimental effects, with worsening of symptoms (Sanal-Hayes *et al.* 2023, p. 16). In addition, this team judged that many of these studies were of poor to fair methodological quality, with inconsistency and a lack of detail of the pacing strategies used (*ibid.*, p. 15), some being clinical trials, some merely reporting patients’ own experiences. These inconsistent results therefore do not give objective support to the energy envelope theory.

In response to these inconsistent results, I speculate that these results are compatible with the possibility of disparate subtypes of CFS (as promoted by Jason *et al.* 2005), with some patients having a symptomatic ‘energy envelope’, and others having a different kind

of energy problem. (As it happens, subtypes of CFS of this kind (or of any other kind of practical value) have not yet been identified.) Those patients who obtain some symptomatic benefit from pacing may actually have an ‘envelope’ of this kind. By contrast, it is possible that those others without an energy envelope (or with a less restrictive envelope) *may* benefit from allowing themselves to exercise more, thereby increasing their exercise tolerance leading to a reduction in the intrusiveness of the CFS symptoms. However, there is no objective measure to show that someone has or does not have an energy envelope, so there is no easy way to confirm the existence of these hypothetical subtypes, nor is there an easy way to identify which patient fits into which subtype, if in fact these subtypes do exist. Without objective verification in any particular CFS patient, the individualisation of treatment recommendations can be only in the light of the patient’s perception of exercise limits, or possibly his or her responses to the differing treatment regimes. Additionally, the possibility of variability between CFS patients will inevitably make the results of trials of pacing (or, alternatively, exercise therapies in CFS) more difficult to interpret, thereby giving an explanation for uncertainty about which treatments should be recommended for CFS.

In opposition to the biomedical model, its associated energy envelope theory, and the recommendation for pacing in CFS, there are commentators who claim that, instead, the biopsychosocial model can be appropriately applied to CFS. According to BPS model of CFS, cognitive and behavioural factors are claimed to have significance in the causation and/or perpetuation of the symptoms (Finnis 2000; Fatt *et al.* 2019); additionally, some supporters of BPS in CFS see that there is not enough evidence to support a biomedical understanding of CFS (Finnis 2000). According to Finnis,

The biomedical model is known for a number of assumptions, including the Cartesian dualism which places the mind apart from the body. By persisting in understanding CFS in terms of such a model, [and] ignoring the social factors, such as the many lifestyle similarities of many patients of CFS, crucial considerations are overlooked. This may then prevent adequate understandings of the syndrome (*ibid.*, p. 31).

So, here, Finnis not only supports BPS in CFS, she tells us that uncertainties concerning the understanding of CFS are in part *due to* the erroneous application of BMM to CFS.

Other researchers reject the energy envelope theory and the claimed need for ‘pacing’ in response to it (this rejection is reported by, but not endorsed by, Jason *et al.* (2008, p. 189)). Contrary to the view that the symptoms of CFS are due to the existence an energy envelope that needs to be managed by limiting energy expenditure, these other researchers hypothesise that CFS symptoms are actually *worsened by activity avoidance*, such as is likely to happen with pacing. Exercising only within limits according to the principles of pacing means that some people living with chronic fatigue syndrome will doing very little exercise each day. Jason *et al.* quote the views of the psychiatrists S. Butler and colleagues (with whom they do not agree): “As individuals continue to avoid activity, their symptoms persist and may increase in severity due to decreasing activity tolerance and increasing sensitivity to any kind of stimulation” (S. Butler *et al.* 1991, p. 153). S. Butler *et al.*’s understanding of the pathological processes involved in CFS is in complete contrast to the energy envelope theory. In order to support their case, S. Butler and colleagues conducted a trial on,

[f]ifty patients fulfilling operational criteria for the chronic fatigue syndrome (CFS), and who had been ill for a mean of five years, were offered cognitive behaviour therapy in an open trial... The rationale [of this trial] was that a distinction [should] be drawn between factors that precipitate the illness and those that perpetuate it. Among the latter are cognitive factors such as the belief that physical symptoms always imply tissue damage, and behavioural factors such as persistent avoidance of activities associated with an increase in symptoms (*ibid.*, p. 153).

Here, S. Butler and his colleagues are claiming that patients' cognition and behavioural factors seem to be unnecessarily perpetuating the symptoms of CFS. S. Butler and colleagues' assumption is that the initial trigger (such as a virus) is no longer causing harm, but the symptoms associated with that trigger persist because of the patient's response to those symptoms. S. Butler and colleagues' interpretation is that CFS patients have inappropriate cognition and inappropriate behaviour, in that they perceive erroneously that their experience of post-exertional malaise and pain indicates that tissue damage persists, and they consequently avoid exercise. This avoiding contributes to their symptoms, as similarly claimed by the medical researchers Brian Walitt and his colleagues:

With time, the reduction in physical activity leads to muscular and cardiovascular deconditioning, and functional disability (Walitt *et al.* 2024).

Here, Walitt and colleagues argue (as do Vercoulen *et al.* 1998), that limiting of activity by CFS patients causes them to become physically deconditioned, with the result that

the unpleasant physical symptoms during and following exertion worsen over time, as their exercise tolerance decreases, encouraging them to exercise less, thereby forming a vicious circle: the experience of post-exertional symptoms leading to exercising less leading to worsening post exertional symptoms, etc. CFS patients' limiting of their activity by pacing makes them more immobile, but immobility brings its own problems. The medical internist Kay E. Jewell describes these problems: deconditioning is the inevitable effect of immobility, giving the symptoms of poor exercise tolerance, fast heart rate, low blood pressure, and reduced muscle strength and stamina (Jewell 2012). The specialist in exercise medicine Michael Joyner notes the similarity between the physical symptoms of being physically deconditioned to the symptoms of CFS according to patient narratives and physician responses (Joyner 2012). Here, Jewell (2012) and Joyner (2012) give rational and evidence-based support to the concept of a vicious cycle mechanism for the perpetuation of CFS symptoms. According to the understanding of S. Butler *et al* (1991), Jewell (2012), Joyner (2012), and Walitt *et al.* (2024), management by pacing alone is the route for CFS symptoms *not* to improve, but instead is the route for the symptoms to persist for months or years.

The psychiatrists Scott Fatt and colleagues (2019) report that, in their view, their study that shows the value of BPS-derived therapeutic interventions much earlier in the illness. These are interventions in those months between the onset of unexplained debilitating fatigue and the date of diagnosis of CFS. The date of diagnosis of CFS for these people was (by the definition that these researchers used) after at least *six months* of symptoms; so those people who had had these significant symptoms were not actually eligible for the diagnosis of CFS at that stage. Fatt and colleagues are concerned that some people who have a few months of debilitating fatigue (in the absence of evidence of a physical disease

that could explain the fatigue) may not recover; their symptoms persist, and they then, as time passes, they fulfil the diagnostic criteria for CFS/ME. Fatt and colleagues' suggestion is that these people may well benefit from a management approach based on BPS early on in their illness (before the diagnosis of CFS) to change their cognition and behaviour and perhaps avoid persisting CFS symptoms.

To summarise the contributions of Walitt *et al.* (2024), Jewell (2012), Joyner (2012), and Fatt *et al.* (2019), these researchers seem to be using all three elements of a bio-psycho-social understanding for the mechanisms of CFS, where the biological aspect refers to the physical changes to CFS patient's heart, blood pressure, and muscles, etc. (all persisting, in their view, due to physical deconditioning); the psychological aspect is the CFS patient's (what these researchers consider to be, unhelpful) cognition regarding the symptoms (leading to, "... an alteration of effort preference, rather than physical or central fatigue ..." (Walitt *et al.* 2024, p. 1)); and the 'social' aspect is the CFS patient's unhelpful behavioural adaptations to the symptoms.

The academic psychiatrist Michael Sharpe and the academic social scientist Monica Greco (2019) go further, by playing down the biological component of BPS in CFS; they refer to CFS as a paradoxical, "... illness without disease ...", because, "... no good objective evidence of bodily disease to account for it" (Sharpe & Greco 2019, p. 185). Sharpe and Greco (2019) claim that CFS patients may well have no disease; however they accept that these patients do have an illness. Sharpe and Greco acknowledge that a situation of illness without disease can challenge others' belief in the reality of the illness itself:

[T]here will be those who [cast] doubt on whether the person who reports an illness-without-disease is actually ill at all... [H]ow can an illness be patently real, sometimes severely so, when doctors can find no good objective evidence of bodily disease to account for it? (Sharpe & Greco 2019, p. 185).

Here, Sharpe and Greco show us how some CFS patients have the reality of their illness experience doubted by some medical professionals. Sharpe and Greco's interpretation here has some similarity to Engel's condemnation of those of his colleagues who tell patients that they are well because the test results are negative (Engel 1977, p. 133), as I described in Section Two of this Chapter; although, doubting a patient's illness is distinct from telling a suffering patient that she is well.

Sharpe and Greco see the value of the concept of 'illness without disease', in that it encourages the application of illness-related treatments (such as exercise therapies) for CFS patients, which, in their view, will be more effective than 'disease-focused treatments' that I describe later in this section.

The BPS (or (B)PS) interpretation of the mechanisms of CFS indicate to some medical professionals that CFS patients need illness-focused treatments. In this context, illness-focused treatments are graded exercise therapy (GET) and cognitive behavioural therapy (CBT). These interventions are recommended by those who have a BPS understanding of the symptoms and course of CFS. GET and CBT concentrate on changing the psychological and social (or behavioural) components of CFS, and (like Sharpe & Greco 2019) downplay the need to address the possible biological component.

Graded exercise therapy involves, "... an exercise plan with structured incremental increases in exercise over time ..." (M.E.B. Smith *et al.*, 2012, p. 844). An exercise plan of this kind is under the instructions of, and is monitored by, a healthcare professional such as a physiotherapist. The intention here is to change the behaviour of the patient: "... [C]ognitive behavioral therapy (CBT) [is] intended to change behavioral and belief factors that may trigger and maintain symptoms ..." (*ibid.*, p. 844). Therapy of this kind is organised and led by a healthcare professional such as a counsellor or a psychotherapist. There is therefore an overlap in the conceptualisation and aims of CBT and GET, so these therapies are often applied in parallel for the same patient. As S. Smith *et al.* (2012) imply, the therapist's role in both CBT and GET is to change how the patient believes and behaves: the patient is instructed to play down or ignore the symptoms of PEM, to reject the concept of an energy envelope (despite their symptoms), and not to limit their activities in response to it.

If the BPS interpretation of CFS were to be correct, then one would expect that confirmation of the effectiveness of CBT and GET (that are justified by BPS) to be confirmed by clinical trials. However, the evidence from clinical trials is not definitive. There is evidence that CBT and GET are of value in CFS; however, that evidence is disputed. The large (and, in my view, deceptively-named) PACE trial studied 641 'eligible' CFS patients, who were randomly allocated to one of four groups with differing treatment options. One group was offered GET, one group was offered CBT, one group was offered adaptive pacing (APT), and one group were offered only standard medical care (SMC) without GET, CBT, or APT (White *et al.* 2011). These researchers found that GET and CBT were of benefit in CFS and so they concluded that,

Findings from the PACE trial suggest that individually delivered CBT and GET, when added to standard medical care (SMC), are more effective and as safe as APT added to SMC or SMC alone. Patients attending secondary care with chronic fatigue syndrome should be offered individual CBT or GET, alongside SMC (*ibid.*, p. 835).

The methods used by White *et al.* (2011), and their conclusions, have since then been disputed by those who claim that the PACE researchers seemed to have changed the parameters that they were measuring, they did not use objective evidence ('wearables' such as step-counters), and they did not demonstrate the vitally important point of *prolonged benefit* in those who, according to the PACE researchers, responded positively to CBT and GET (Friedberg 2016). (Furthermore, I see that it may be that there are (as-yet-unidentified) subtypes of CFS patients who can benefit from CBT and GET, and other subtypes that cannot benefit from those therapies.) The results of the PACE trial therefore did not settle the argument in favour of CBT and GET in the management of CFS.

Some physiotherapists offer what could be considered to be the compromise management regime of 'adaptive pacing therapy' (APT) as a therapeutic intervention. (Adaptive pacing seems to me to be an appropriate response to the 'deconditioning' interpretation of S. Butler *et al.* (1991), Vercoulen *et al.* (1998), Jewell (2012), Joyner (2012), Fatt *et al.* (2019), and Walitt *et al.* (2024)). APT gives the patient more freedom of action than GET, because APT does not necessarily sanction the physiotherapists' imposition of *planned and structured incremental increases* in activity that are a feature of GET.

However, APT does aim primarily at increasing the patient's activity (unlike patient-monitored pacing), and is still monitored and supervised by the physiotherapist:

Adaptive Pacing Therapy (APT) is ... designed to alter patients' behaviour. In contrast to CBT [cognitive behavioural therapy], however, pacing makes allowances for the characteristic fluctuations in symptom severity and delayed recovery from exercise... Patients receiving APT are instructed to set themselves reasonable targets for their daily activity and exercise and to avoid possible over-exertion (and aggravation of symptoms) by striking a balance between activity and rest. APT patients function ... within their individual limits, then gradually raise their activity and exercise levels (Castro-Marrero *et al.* 2017, p. 358).

In adaptive pacing therapy, CFS patients are encouraged to function 'within their limits'; and therefore this therapy is compatible with the possibility of the existence of an 'energy envelope'. This compatibility may allow APT to be more acceptable to patients who experience what they feel is an energy envelope. However, APT is a more formalised therapy for CFS than 'pacing'. The intention and hope are that patients following a course of APT will follow their therapist's instructions and *increase* their activity over time. (By contrast to adaptive pacing's aim of increasing activity, pacing alone is designed to keep CFS symptoms in check, without aiming to achieve an increase in activity over time). Adaptive pacing is structured and monitored by a professional such as a physiotherapist, and as Castro-Marrero *et al.* (2017), tell us; it is designed for *therapists to alter patients' behaviour*. In adaptive pacing therapy, the therapist leads the process, and expects the patient to modify her actions accordingly. Thus, this approach seems to rely on the recognition that there may be a dichotomy of understanding between

the therapist and the patient, while also depending on the acceptance that the therapist holds greater authority in the relationship.

Review articles of reports on clinical research on treatments for CFS show that there is some limited empirical support for the effectiveness of CBT and GET and other exercise therapies. Arnett and Clark's review finds that CBT and GET give variable benefit (Arnett and Clark 2012). In their review of current research on CFS, Castro-Marrero and colleagues conclude that the articles they studied show that behavioural and rehabilitation therapy programs may be of benefit for CFS/ME (Castro-Marrero *et al.* 2017, p. 345). However, Castro-Marrero *et al.* (2017) complain that the evidence of effectiveness of these interventions is still limited (*ibid.*, p. 345). In their review article for the authoritative Cochrane Library, the medical researchers and reviewers Lillibeth Larun and her colleagues found that the research studies that they studied and collated showed them that, "[t]he effectiveness of exercise therapy seems greater than that of pacing but similar to that of CBT" (Larun *et al.* 2017, p. 25). However, the conclusion of Larun and her fellow authors' later review of the then current evidence was less definite: "Exercise therapy *probably* has a positive effect on fatigue in adults with CFS compared to usual care or passive therapies" (Larun *et al.* 2019, p. 37) [my italics]. Larun and colleagues complain about limited evidence: they note that the evidence available gives only low to moderate certainty (*ibid.*, p. 37).

In contrast to the claimed limited evidence of support for treatments for CFS based on BPS, Sharpe and Greco (who are BPS advocates) see that CBT and GET have value. They claim that:

[T]reatments such as CBT and GET relieve the symptom of fatigue [in conditions such as cancer, and therefore] they offer a useful improvement of a condition [CFS] for which there was previously no treatment at all (Sharpe & Greco 2019, p. 186).

Here, Sharpe and Greco see the value of CBT and CFS in many conditions, including CFS. They note that there are no biomedical treatments (curative or symptomatic) for CFS, so they are also implicitly claiming that pacing, the alternative to CBT and GET is actually ‘no treatment at all’. They extol the benefits of GET and CBT; however, their main evidence for the effectiveness of these interventions is the disputed PACE trial; the results of the PACE trial have not been duplicated by other large trials.

I now move to describe opposition to these BPS-related management strategies. Pheby and colleagues see harm in an HCP’s reliance on a biopsychosocial (BPS) understanding of, and management for, chronic fatigue syndrome:

[The] insistence on the biopsychosocial hypothesis[,] that ME/CFS can be totally explained away as being due to faulty illness beliefs combined with deconditioning[,] ... have led to tension between patients and healthcare professionals (Pheby *et al.* 2020, p. 13).

So, Pheby *et al.* see that some HCPs are using BPS in ways that Pheby *et al.* consider to be incorrect; they claim that some HCPs are using a very restricted biopsychosocial conceptualisation for ME/CFS, one that ‘totally’ excludes non-psychological and non-behavioural explanations.

The academic general medical practitioner Nefyn Williams and his colleagues disapprove about the way some of their colleagues seem to think:

[C]linicians often have difficulty relinquishing a disease-centred approach to diagnosis, and either dismiss illness when pathology has been excluded, or explain all undifferentiated illness in terms of psychosocial factors (N. Williams *et al.* 2008, p. 1).

A ‘disease-centred approach’ is one that depends on evidence of pathology. So we see that, for N. Williams *et al.*, the application of BPS to illnesses where disease and pathology are unidentified encourages some health professionals to attribute the symptoms to social or psychological causes, or even to dismiss the symptoms (and the patient’s experiences).

Similarly, Maes and Twisk (2010) condemn this playing down or discounting of the biological aspects of CFS patients’ problems, by promoting the term ‘(bio)psychosocial’ ((B)PS). My understanding of Maes and Twisk here is that the use of (B)PS can be problematic in CFS healthcare encounters in at least three ways, by:

- *Firstly*, encumbering the medical professional’s acceptance of the reality of the patient’s symptoms; and,
- *Secondly*, encumbering the creation of a shared understanding of the patient’s problems; and,
- *Thirdly*, encumbering the selection of treatment recommendations.

I appreciate that the imposition by an HCP of such an interpretation would be likely to lead to an unsatisfactory ME/CFS healthcare encounter. However, it may be that that situation is rare; a survey of medical practitioners found that only a small minority of them considered CFS to be only psychological, without any biological component (Hossenbaccus & White 2013).

The attribution of a disease-centred conceptualisation by health professionals in the way described by Sharpe & Greco (2019), Pheby *et al.* (2020), N. Williams *et al.* (2008), and Maes and Twisk (2010), could well be the origin of the complaint by many ME/CFS patients that their condition has been dismissed by some medical professionals, and others in CFS patients' social circles, with the phrase, "... all in your mind ..." (as reported by Geraghty 2016, no page numbers). This 'in your head', or the 'in your mind' concept is bitterly opposed to as 'a myth' by the research psychologist Keith Geraghty, as highlighted in the title of his article: "The 'all in the mind' myth of myalgic encephalomyelitis/chronic fatigue syndrome" (Geraghty 2016). By contrast, the use of this kind of phrasing in reference to CFS is actually endorsed by the neurologist Suzanne O'Sullivan, in that a version of it is explicitly part of the title of her book, *All in Your Head: True Stories of Imaginary Illness* (2016).

The psychiatrist and advocate for ME patients, Fred Friedberg, has even less confidence in CBT. He sees that trials of CBT in CFS show limited effectiveness in his review of others' research; he attributes that lack of success to its misguided, "... disease-denying rhetoric and exaggerated claims regarding recovery" (Friedberg 2016, p. 129). (Friedberg's accusation of 'disease-denying rhetoric' aligns with the 'illness without disease' conceptualisation that is promoted by Sharpe and Greco (2019).) So, for

Friedberg, CBT is the wrong treatment for CFS because it does not take a biomedical approach; furthermore, he sees that the advocates for CBT in CFS overstate its success rate, another factor that causes CBT to be criticised in the CFS community.

I speculate that an alternative (or additional) explanation for the reduced effectiveness and unpopularity (among some CFS patients) of CBT in CFS may be that this CBT is too narrowly focused; CBT for CFS concentrates on changing patients' cognition concerning their physical symptoms and changing their behavioural reaction to those cognitions (Starbuck *et al.* 2022). This restricted version of CBT is not intended to elicit and address patients' emotions and cognitions about many other aspects of their present lives, such as, "... interpersonal or institutional power relations ..." (Mescouto *et al.* 2022, p. 3270), or the possible continuing negative effects of past adverse life experiences. It may be that, for some patients, their past or present negative situations are contributing to their present unhappiness; a narrowness of focus that does not acknowledge these negative life experiences may well hinder the success of CBT in CFS, and may contribute to CFS patients' negative reactions to CBT. My speculation here aligns with the complaint of commentators that the biopsychosocial model (which underpins CBT) can be interpreted too narrowly (N. Williams *et al.* 2008; Mescouto *et al.* 2022). Engel's original formulation of BPS may well not have been narrow in that way.

In this section, I have shown that the biomedical model and biopsychosocial models of chronic fatigue syndrome lead to oppositional treatment recommendations. The unsatisfactoriness of CFS healthcare encounters could well be exacerbated by these differing and contradictory treatment recommendations. The biopsychosocial model of CFS attributes the perpetuation of the symptoms to patients' cognition and behaviour,

which interpretation conflicts with the interpretation of many CFS patients and their support and advocacy groups who attribute CFS and its symptoms to underlying (although at present unknown) biomedical processes. Consequently, there is support for BPS by some healthcare professionals, in opposition to some of their CFS patients' support for BMM. However, empirical evidence for the effectiveness of any of these differing treatments is equivocal. The advocacy for oppositional conceptualisations and treatment recommendations makes significant contributions to the controversies and dichotomies regarding CFS, and to the unsatisfactory healthcare interactions that I will describe and analyse in Chapter Six of this thesis.

4.5: Conclusion to Chapter Four

I have shown in this chapter that the biopsychosocial model is now widely used in medicine, as was Engel's intention. There are commentators who see the value of BPS to understanding medical conditions and to medical practice, even though some of these see it merely as an aide-memoire for the clinical encounter and for teaching. I have shown the views of other commentators who oppose BPS on philosophical grounds, seeing problems with its metaphysical justification. Additionally, I presented the views of commentators who oppose BPS because they see that clinicians can misuse it to enhance their power in the healthcare encounter. Furthermore, the use of BPS may lead to diagnostic error. The relevance to this thesis is that BPS is applied by some HCPs to chronic fatigue syndrome as the justification for cognitive behaviour therapy and exercise therapies in CFS; however, the value of these applications is disputed by some commentators and by many CFS patients, who instead see the value of the biomedical

model for understanding CFS and for endorsing treatments. In this Chapter, I showed that differing responses to the uncertainties about chronic fatigue syndrome lead to oppositional conceptualisations, attributions of disease mechanisms, treatment recommendations, and recommendations for the direction of research programmes. In Chapter Six I will show how these oppositional stances lead to epistemic injustice in CFS healthcare encounters.

5: Stigma, Chronic Fatigue Syndrome, and ‘Medical Disease Dualism’

5.1: Introduction to Chapter Five

In this short chapter, I argue that stigma concerning chronic fatigue syndrome and towards CFS patients follows from the uncertainties about CFS that I described in Chapter Three, and from the disputed applications of the biomedical model or the biopsychosocial model to CFS that I described in Chapter Four. Stigmas about CFS seem to follow an attribution of psychological causation for CFS symptoms. Here, ‘psychological’ is misinterpreted by some as a ‘psychiatric’ attribution. There is stigma against psychiatric disease and psychiatric patients. Stigmas of this kind negatively affect CFS healthcare encounters. I argue that the mistaken attribution of ‘psychiatric’ to CFS depends on a questionable assumption, that there is a clear distinction between physical illness and psychiatric illness, with a similar clear distinction between physical and psychiatric treatments. Stigma is often associated with prejudice, which is an integral part of epistemic injustice, in the view of Fricker (2007). In Chapter Six I will show how stigmas contribute to unsatisfactory healthcare encounters that can be categorised as being epistemically unjust.

Here, as elsewhere of this thesis, and as mentioned in my Preliminaries Chapter One, I use the term ‘CFS patients’ or ‘CFS/ME patients’ to refer to those people living with symptoms that are labelled as CFS, CFS/ME, or ME, whilst I maintain a neutral stance regarding the ontology of this condition (or these conditions).

The outline of this chapter is as follows: In Section Two, I give evidence for some healthcare professionals’ stigmatising doubts or lack of belief about the ‘reality’ or ‘existence’ of CFS

as a legitimate disease, leading to negative and stigmatising attitudes not only towards the medical condition CFS but also to towards CFS patients. In Section Three, I digress slightly to give a short account of stigma against mentally ill patients. That stigma is felt by some CFS patients if they are told that their CFS symptoms are psychological or psychiatric. However, I emphasise the distinction between the meanings of the terms ‘psychological’ and ‘psychiatric’. In Section Four, I argue against the unstated assumptions, or specific claims, of a clear distinction between physical and psychological medical conditions, because my view is that these assumptions and claims are inappropriate, and contribute towards stigmas against CFS and mental illness and against patients with these conditions. In Section Five, I summarise this chapter, and respond to possible counter-claims.

5.2: Evidence for Stigma in CFS

In this section, I show the variety of stigma-related attitudes of some healthcare professionals towards CFS and CFS patients, including challenges to the existence of CFS and the attribution of CFS symptoms to psychological processes.

There is evidence that there are healthcare professionals who stigmatise the medical condition CFS by saying that it does not exist (as reported by Hng *et al.* 2021, p. 2), or by expressing doubt that CFS exists or doubt that is ‘real’ (as reported by: Cooper 1997; Geraghty 2016; and Pheby *et al.* 2020). Some HCPs seem to dismiss CFS as being, perhaps, ‘all in your head’ (O’Sullivan 2016), or ‘all in the mind’ (as reported by Tesio & Buzzoni 2021 and Geraghty 2016).

Both patients and primary care physicians share uncertainty about CFS/ME (Horton *et al.*, 2010, p.1). However, their responses to that uncertainty are often oppositional; and, some patients feel that their doctors' responses show that these doctors are blaming patients for patients' predicaments: "... [patients] felt severely ill, yet [felt] blamed and dismissed. Patients' beliefs and causal attributions oppose the doctor's understanding of the condition" (Larun & Malterud 2007, p. 20). Some health professionals allow their doubts about the condition CFS to contaminate their CFS healthcare encounters, where those HCPs stigmatise CFS patients (not just the medical condition CFS), denigrating CFS patients and their illness experiences. CFS patients can be stigmatised and negatively stereotyped by HCPs (as reported by Raine *et al.* 2004; and Geraghty & Blease 2018), for instance as being "“heartsinky”" (Raine *et al.* 2004, p. 1355), or stereotyped, "... as having certain undesirable traits" (*ibid.*, p. 1354). ('Heartsinky' patients are those make the HCP's heart sink.) During the healthcare encounter, CFS patients can experience, "... high levels of ignorance and disbelief among doctors" (Hng *et al.* 2021, p. 10). There is delegitimisation of illness experience (Hng *et al.* 2021, p. 2...), with the, "... marginalization and exclusion of patient voices" (Blease & Geraghty 2018, p. 400), with HCPs' disbelief in the severity and effects of CFS symptoms (Whitehead 2006; Pheby *et al.* 2020, p. 14). CFS patients are made to feel guilt and blame: "... patients feel guilt for seeking treatment for illnesses that only exist 'all in the mind' (Tesio & Buzzoni 2021, p. 1); and, "... defining an illness as psychosomatic [is] effectively 'shifting the blame' for what might otherwise be treated as medical failure or uncertainty (Horton-Salway 2002, p. 401). Here, Horton-Salway reminds us that an explanation for stigma in CFS is uncertainty (as I described in Chapter Three of this thesis); Horton-Salway sees that doctors are responding inappropriately to uncertainty about ME by unfairly making the patients feel blamed. (Horton-Salway is using the present-day meaning of the term

‘psychosomatic’, to refer to physical symptoms that are caused by psychological problems, as I described in Chapter Four, Section Three.)

5.3: Psychiatric Attributions as Explanations for Stigma in Chronic Fatigue Syndrome

The ‘all in your head’ attribution of chronic fatigue syndrome or myalgic encephalomyelitis (O’Sullivan 2016), Kendell 2001; Gipps 2008; Geraghty 2016; Franssen 2020; Tesio & Buzzoni 2021; Turner 2021) can imply that CFS is related to, or is, mental illnesses.

I now describe how this implication is present in commentaries from all three standpoints: those commentaries that *acknowledge the value* of that attribution, in commentaries that *oppose* the appropriateness of that attribution, and in commentaries that are *neutral* on the existence or relevance of that attribution.

The medicolegal consultant Robert Lloyd sees a ‘psychiatric genesis’ of CFS and/or ME that, in his view, should not be denied. In his 7th August 2002 response to Goudsmit (Goudsmit 2002), Lloyd claims that, “[t]here is no rational reason to fear a psychiatric label” (Goudsmit 2002). (The debate between Goudsmit and Lloyd was conducted in in the BMJ in the “Responses to Mayou and Farmer” (2002).) O’Sullivan accepts that ME/CFS is, “... a disability that has its roots in behavioural and psychological factors” (O’Sullivan 2015, p. 242), and, “... ME/CFS patients ... have a high rate of depression and anxiety” (*ibid.*, p. 244).

On the other side of the debate concerning the physical versus psychological attribution for CFS, there are commentators who oppose the claim for a ‘psychiatric’ or psychological interpretation of CFS, for instance, the contribution of the senior psychologist Ellen Goudsmit: “Somatisation and somatoform disorders [as ascribed by some commentators to CFS] are psychiatric” (Goudsmit 2002, in her response of 8th August). Similarly, O’Leary notes with disapproval that, “[p]rofessional consensus has long suggested that the condition should be classified as psychiatric” (O’Leary 2018, p. 716). The sociology researchers Helen Spandler and Meg Allen use the disparaging term ‘psychiatrisation’ for this interpretation, which they oppose (Spandler & Allen 2018, p. 127). Geraghty notes with disapproval that O’Sullivan, “argues [that] ME/CFS is strongly associated with psychological complaints and illness beliefs” (Geraghty 2016, §1). Here, Geraghty is likely to be alluding to O’Sullivan’s assertion that, “... ME/CFS patients ... have a very high rate of depression and anxiety” (O’Sullivan 2015, p. 244). (I understand that the term ‘psychological complaint’ as used here by Geraghty means a psychiatric condition.) Ramsey and Wookey reject the accusation that the 1955 outbreak in the Royal Free hospital (later named ‘benign myalgic encephalomyelitis’) was not a physical condition:

Dr Ramsay has confidently marshalled the evidence and put together the facts which will convince even the most sceptical of doctors that the cause of myalgic encephalomyelitis, popularly known as Royal Free disease, was not conversion history (*sic*) (Wookey 1987, p. 565, reviewing Ramsay 1986). [My understanding is that ‘conversion history’ here is a misprint for ‘conversion hysteria’.]

‘Conversion hysteria’ is a psychiatric condition where patients’ dramatic or florid symptoms are declared by their healthcare professional as being non-physical, instead being attributed by the

HCP to patients' psychological problems. McEvedy and Beard (1970a) gave that implication as an explanation for the Royal Free outbreak.

Additionally, the claim for the existence of a 'psychiatric' interpretation of CFS is acknowledged in other, more neutral, commentaries (Thomas & Smith 2005; Wojcik *et al.* 2011; van der Meer & Lloyd 2012; Carel & Kidd 2014; Karfakis 2018; Diserholt 2020).

The attribution of the psychiatric interpretation is not popular with many CFS patients: "Many of these [CFS] patients had received a psychiatric diagnosis for their symptoms *which they rejected*" (Thomas & Smith 2006, p. 2) (my italicisation) (also see 'Suzanne', §1). This rejection is likely to be associated with an appreciation that there is stigma against mental illness (Spandler & Allen), and against mentally ill people. This stigma against people with mental illness is identified by Anastasia Scrutton: "... [P]eople with mental illness are stereotyped in a way as to deflate their credibility" (Scrutton 2017, p. 349). She tells us that there is, "... prioritization of objective third-person accounts to the detriment and even exclusion of subjective first person accounts" (*ibid.*, p. 353) to patients' disadvantage. She rails against what she sees as, "... a voluntaristic or moralising model of depression, according to which people could stop being depressed by pulling themselves together, looking on the bright side and adopting a different lifestyle [which] is epistemically problematic ..." (*ibid.*, p. 348). Similarly, the physician and writer on medical error Pat Croskerry criticises this blaming:

Fundamental attribution error: the tendency to be judgmental and blame patients for their illnesses (dispositional causes) rather than examine the circumstances (situational factors) that might have been responsible. In particular, psychiatric

patients, minorities and other marginalized groups tend to suffer from this bias (Croskerry 2019, p. 179).

Here Croskerry emphasises that the attribution of blame to psychiatric patients is faulty reasoning; this faulty reasoning leads to medical error in the form of poor interpretation about the causes for a patient's problems.

Scrutton's (2017) and Croskerry's (2019) concerns regarding how people with mental illness are treated by healthcare professionals are similar to the concerns of many CFS patients and their supporters.

- Firstly, many CFS patients find that their first-person accounts of the severity and deleterious effects of their symptoms are discounted compared to healthcare professionals' third person reliance on the results of clinical examination and investigations.
- Secondly, many CFS patients experience exhortations to adopt a different lifestyle (that is to say, exercise more) in order to get better, and to change to a 'brighter' interpretation of their condition, which exhortations are how some CFS patients construe cognitive behaviour therapy and exercise therapies.
- Thirdly, many CFS patients see that some medical and non-medical people have, "... a voluntaristic or moralising model" (Scrutton 2017, p. 348) of CFS, where patients could get better if they wished, so these CFS patients are morally suspect; the 'voluntaristic' attribution is an implication that patients have some control over their symptoms and/or over their response to those symptoms.

- Fourthly, Goudsmit sees that CFS patients are demeaned *even more* than psychiatric patients; she supports that claim in the following passage, in which she first quotes those views of Robert Lloyd, with which she disagrees:

Lloyd goes on: "There is no rational reason to fear a psychiatric label." Yes, there is! Not all psychiatric labels are the same. In my experience, people with depression, anxiety and schizophrenia are treated much more sympathetically than patients with CFS. This is not only because of the trivialisation of the illness—well, we all get tired—but because colleagues have implied that the essential problem here is stupidity. These patients are not portrayed as sensible people, coping with their symptoms in a sensible way. They are portrayed as misguided and silly. Afraid of fatigue (Goudsmit 2002, 8th August).

Here, Goudsmit sees that CFS patients are treated unsympathetically. It is therefore unsurprising that CFS patients are likely to be reluctant to receive the stigma that a psychiatric label brings, with the implication of moral failings regarding helping themselves to get better, undesired treatment recommendations that follow that implication, and being considered ‘stupid’, ‘misguided’, and ‘silly’.

However, I see a semantic problem here, with an inappropriate conflation by some commentators of the terms ‘psychiatric’ and ‘psychological’, which usually refer to distinct concepts. The former term refers to conditions such as depression and schizophrenia, while the latter term refers merely to any of the processes in people’s minds, without those processes necessarily being manifestations of mental illness.

O’Sullivan (2015) sees that this unhelpful conflation has negative effects. She appreciates the value of treatments that address behavioural and psychological factors (ME/CFS is, “... a disability that has its roots in behavioural and psychological factors” (O’Sullivan 2015, p. 242)), but she understands that some ME/CF patients mistakenly (in her view) see these treatments as psychiatric.

Other commentators who do *not* see value in treatments that address behavioural and psychological factors seem to be inappropriately applying the term ‘psychiatric’ to categorise or attribute treatments such as cognitive behaviour therapy and/or exercise therapies in the context of the management of CFS: “[A]dvice on management [of CFS] emphasized psychiatric interventions, like cognitive behaviour therapy” (Goudsmit 2002, in her response of 8th August). Similarly, Friedberg (2016) claims, “Cognitive-behavior therapy is a well-established psychosocial intervention for psychiatric disorders ...” (Friedberg 2016, p. 127), and, “CBT and GET are based on psychiatric views ...” (Kim *et al.* 2020, p. 8).

My view is that these conflations, categorisations, or inappropriate attributions, can negatively affect CFS healthcare interactions in those situations where the healthcare professional suggests that there are contributions from *psychological* factors for the perpetuation of the patient’s CFS symptoms, with a consequent need for therapies that could counter those negative psychological factors. ‘Psychological’ is indeed a central component of the bio-psycho-social model, while ‘psychiatric’ is not; so, an HCP’s psychological explanation should not be seen as an implication that the patient has a psychiatric disease. However, a CFS patient who conflates ‘psychological’ with ‘psychiatric’ could re-interpret the HCP’s views and treatment recommendations as implying a psychiatric diagnosis, with its attendant stigma. On the other hand, and contrastingly, a CFS patient may experience stigma

when the term ‘psychological’ is used, as she might conceive a psychiatric condition as being by its nature a disease process that is outside the patient’s control, while ‘psychological’ processes could be considered to be under the control of the individual, and therefore blameworthy and stigmatising.

In this section, I have shown that, in some of their healthcare encounters, CFS patients’ experiences of blame and stigma are augmented by the perception of inappropriate psychiatric attributions of their illness.

5.4: The Inappropriate Physical and Psychological Disease Dichotomy

In this section I describe what I consider to be an unsatisfactory assumption of a physical-psychological classificatory dichotomy for medical conditions. This assumption is associated with a claim, or an assertion, or an implicit assumption, of a clear distinction between physical diseases and psychological diseases, which distinction I term ‘medical disease dualism’ (MDD). The concept of medical disease dualism seems (for most people who assume it) to be only peripherally related to the many uses of the term dualism and its concepts in other areas of philosophy, so I don’t explore in detail possible metaphysical connotations of this distinction. In this section I give examples that show or seem to show assumptions of medical disease dualism; I then show what I consider to be the unsatisfactoriness of the concept of medical disease dualism.

The relevance of this physical-psychological argument to this thesis is that many contributors to debates regarding chronic fatigue syndrome assert that it fits into one of these

physical/psychological categories and not into the other (Geraghty 2016; Spandler & Allen 2018; Ali and Kheirabadi 2025; Arron *et al.* 2024). These assertions implicitly imply the assumption that these categories are mutually exclusive, at least in the case of CFS.

O’Leary *explicitly* endorses what I call medical disease dualism: [P]sychiatric conditions must be clearly distinguished from biomedical conditions (O’Leary 2021a, p. 1989).

O’Leary’s endorsement of the distinction between psychiatric and biomedical conditions is associated with her rejection of metaphysical dualism, “... where we accept that mental properties are distinct from physical properties, but reject dualism” (O’Leary 2021b, p. 5). Her empirical endorsement for the distinction is that a vague distinction can lead to diagnostic error, in those cases where a healthcare professional can move the classification of a patient with physical symptoms (and an undiagnosed dangerous physical disease) to merely (and incorrectly) needing only psychological management (O’Leary 2021b, p. 14).

The physical-psychological classificatory dichotomy is evident in opposing views on the nature of medical condition such as chronic fatigue syndrome: either it is a physical disease where the symptoms and course are due to pathological bodily processes, or CFS’s physical symptoms and course derive from psychological processes. This dichotomy can be seen clearly in the results of the survey reported by Hossenbaccus and White (2013, p. 4) that I showed as Table Two in Chapter Four, Section Four. These medical researchers report that thirty-two of the thirty-six ME organisations consider that CFS/ME is a physical condition, while none of them consider that it is a psychological condition. By contrast, most of the medical authorities that they surveyed consider that ME is a mixed psychological and physical condition. However, fourteen of the thirty-eight medical authorities do classify ME as either physical or as a psychological condition.

I claim that both these contrasting views depend on an *implicit* assumption by those thirty-two ME organisations and those fourteen medical authorities that there are two distinct disease categories, physical and psychological. There seems to be an implicit assumption of physical-psychological dichotomies between the causes of medical conditions, and concomitant dichotomies regarding treatments that are likely to be effective for those medical conditions.

Against both sides in the physical/psychological disease dichotomy, I argue that most medical conditions are ‘mixed’ (as do twenty-four of the thirty-eight surveyed medical authorities in regard to ME, etc. (Hossenbaccus and White 2013, p. 4)), having both physical and psychological symptoms, and many having both physical and psychological causes for their onset and course, therefore requiring the medical professional to address both the physical and psychological needs—the body and the mind— of the patient. There is also a rhetorical issue: debates regarding chronic fatigue syndrome demonstrate the distortion of views of the opposing side, in that a ‘mixed’ claim by one side is sometimes re-interpreted by the opposing side as being a wholly-physical or a wholly-psychological claim. For instance, Song and Jason oppose other researchers’, “... purely psychogenic explanation for CFS” (Song & Jason 2002, p. 285), and Arron and colleagues see that some clinicians label ME/CFS patients’ symptoms as being solely psychosomatic: “... ME/CFS ... patients are labelled as hypochondriacs and their condition is attributed solely to psychosomatic origins” (Arron *et al.* 2024, p. 06). In these two quotations, the words ‘purely’ and ‘solely’ do not accurately convey the nuanced views of those people that these commentators oppose. Here Arron *et al.* are using the contemporary meaning of

‘psychosomatic’ to refer to medical symptoms that have one-way causation, from the mind to the body, as I described in Chapter Four, Section Three.

As I imply in the previous paragraph, my opposition to medical disease dualism is not an argument against the understanding that there are *symptoms* that we choose to call physical and symptoms that we call psychological. I argue that it is common for them co-exist in any particular person with a medical malady. Most conditions that require an encounter with a healthcare professional have symptoms that can be described as physical, and symptoms that can be described as psychological, and treatment for these conditions requires responding to both these kinds of symptoms. For instance, if someone is experiencing the physical symptoms of a medical problem such as myocardial infarct she is likely also to experience the psychological symptoms of apprehension and anxiety. Contrarily, someone who has anxiety related to agoraphobia will also experience physical symptoms such as a fast heart rate, sweating, and tremor. I do, of course, accept that there is institutional separation in professions, in that there are specialists such as psychiatrists, psychologists, and counsellors whose role is different from, say, surgeons and cardiologists. However, there are many specialities such as physiotherapists and my own former speciality—family practitioner—whose role is to elicit and attend to both physical and psychological aspects of their patients’ medical conditions, and to recommend treatment regimens that embrace both; they do not see a sharp distinction between the psychological and the physical.

5.5: Conclusion to Chapter Five, and Responses to Counter-Claims

The inadequacy of objective evidence of specific pathological processes in chronic fatigue syndrome leads some people to ascribe psychological factors as significant underlying mechanisms for the symptoms and course of CFS. Unfortunately, many commentators conflate the terms ‘psychological’ and ‘psychiatric’, both those people who accept the relevance of psychological factors in CFS, and those who oppose that relevance.

Consequently, some people see that the medical condition CFS and people whose symptoms are labelled as CFS then share in the stigma that accompanies psychiatric conditions and psychiatric patients. This view depends to some extent on the assumption that there is a clear distinction between physical and psychiatric medical conditions, against which I have argued. The stigma against psychiatric patients seems to derive from a mistaken view that psychiatric patients have some control over their symptoms and the course of their illness, and so are to be blamed if they do not apply that control. Blame leads to stigma. Conversely (but equally unsatisfactorily, in my view), one could see attributions of responsibility—with ensuing blame—in reference to psychological (non-psychiatric) ascriptions. The relevance of these allegations of control and attributions of responsibilities to this thesis is that CFS patients could seem to be blamed for their predicament for not exercising the alleged ability to control their symptoms. For instance, cognitive behaviour therapy suggests to some patients and commentators that patients are thinking incorrectly about their symptoms and the effects of their symptoms. Similarly, recommendations for exercise therapies seems to imply that CFS patients are responsible for the prolongation of their symptoms by their lack of activity. Therefore, according to these interpretations, CFS patients seem blameworthy to others, and consequently feel stigmatised. My view is that these attributions of blameworthiness and stigma derive from differential responses to radical uncertainty about CFS.

I now respond to possible counter-claims to my opposition to what I call ‘dualistic’ conceptualisations for the understanding and treatment of CFS. The first medical dualistic claim is that CFS is a purely *physical medical condition* without any need for research into supplementary behavioural and psychological treatments. My response is that the conceptualisation of a purely physical medical condition needing only physical treatments is counter to the understanding the management of most other medical conditions, and counter to the practice of professional care in most other medical conditions. The oppositional claim is that, according to some commentators, *CFS is merely a behavioural or psychological condition*. This claim is also medically dualistic. However, this conceptualisation requires the inappropriate ignoring of the known physiological and/or biochemical abnormalities found by researchers in many CFS patients.

In the next chapter I show how the dichotomies of views about CFS, that I showed in Chapter Four, and the stigma and blame, that I showed in this chapter, combine with other aspects of healthcare to give negative effects on some CFS healthcare encounters.

6: Epistemic Injustice in Healthcare, and in Chronic Fatigue Syndrome

6.1: Introduction to Chapter Six

In Chapter Three, I demonstrated that there is radical uncertainty concerning chronic fatigue syndrome. In Chapter Four I showed that this radical uncertainty allows dichotomous conceptualisations about CFS; this dichotomy leads to oppositional interpretations concerning what is going wrong in CFS and what should be done about it. In Chapter Five, I showed that those differing responses to uncertainty lead to the blameworthiness and stigma that are attached to the medical condition CFS, and projected onto CFS patients by some health professionals and others. In this chapter, I argue that these differing responses to uncertainty and the attributions of blameworthiness and stigma add to other better-recognised explanations for epistemic injustice in chronic fatigue syndrome healthcare encounters. However, the adoption of alternatives to these oppositional dichotomous responses may help towards amelioration, which I will advocate in Chapter Seven.

The outline of this chapter: in Section Two I describe the features of epistemic injustice. In Section Three I describe the features of healthcare epistemic injustice. In Section Four, I show my explanatory connection between uncertainty and injustice in chronic fatigue syndrome: differing responses to uncertainty about CFS give some explanations for healthcare epistemic injustice, according the concept that I call *uncertainty-driven healthcare epistemic injustice*. In Section Five, I summarise and analyse the testimony of ‘Suzanne’, who has had long experience of healthcare interactions concerning chronic fatigue syndrome and similar conditions. She appreciates that there is uncertainty about CFS, and has experienced what I argue is epistemic injustice. I show the ways in which ‘Suzanne’s’

testimony aligns with and supports the arguments in this thesis, and the ways in which her views differ from mine. In Section Six, I give some explanations for HEI from the perspective of healthcare professionals. In Section Seven I give the main arguments of this thesis, that epistemic injustice in some CFS healthcare encounters can be explained by a combination of the factors demonstrated earlier in this chapter and in chapter five, along with uncertainty, differing responses to that uncertainty, and power differentials between patient and healthcare professional.

6.2: Epistemic Injustice

I commence this section with an outline of epistemic justice (EI) as identified by Fricker (2007), including her understandings of testimonial and hermeneutical injustice. I describe how power differentials can be considered integral to understanding and ascribing EI, along with prejudice and culpability. I indicate the roles of empathy and trust. I show that the recognition of EI is dependent on identifying situations where the standpoint of the speaker's knowledge and perspective are under-recognised. I also demonstrate the different harms of EI, some of which are non-epistemic, for instance, the hindering of flourishing, and the harming of social welfare.

Fricker's interest is in, "... injustice specifically in the sphere of epistemic activity ..."

(Fricker 2007, p. viii). Here, Fricker concentrates on the social acquisition (or hinderances to the acquisition) of knowledge. In situations where knowledge is to be gained socially, human practices are, "... played out by subjects that are socially situated ... [where] social identity and power [are] centre-stage ..." (*ibid.*, p. vii). Fricker describes 'epistemic

injustice' as, "... a wrong done to someone specifically in their capacity as a knower" (*ibid.*, p. 1). Fricker tells us that she accepts that this wrong may be perpetrated unintentionally rather than deliberately; however, the wrong is caused by the hearer being, "... clouded by prejudice ..." (*ibid.*, p. 54), and is therefore culpable because, "... prejudiced thinking is almost always culpable" (*ibid.*, p. 55).

Fricker distinguishes two forms of epistemic injustice, testimonial and hermeneutical. Testimonial injustice as described by Fricker is an epistemic wrong occurring between an individual speaker and an individual hearer:

Testimonial injustice occurs when prejudice causes a hearer to give a deflated level of credibility to a speaker's word (Fricker 2007, p. 1).

So, according to Fricker, the prejudice in testimonial injustice is that, in an encounter, the speaker is misjudged and perceived as epistemically lesser than the hearer (Fricker 2007, p. 1). Testimonial injustice is associated with 'negative identity prejudice' which is, "... a negative valence held against people *qua* social type" (*ibid.*, p. 35), that is to say, people marginalised by social identity.

Fricker then describes hermeneutical injustice:

[H]ermeneutical injustice occurs at a prior stage, when a gap in collective interpretive resources puts someone at an unfair disadvantage when it comes to making sense of their social experiences (*ibid.*, p. 1).

Hermeneutical injustice ... wherein someone has a significant area of their social experience obscured from understanding owing to prejudicial flaws in shared resources for social interpretation (*ibid.*, p. 147).

In these two passages, Fricker tells us the experients of hermeneutical injustice are prevented from understanding their situation. Hermeneutical injustice does not necessarily commence with an interaction between two individuals. One of both of these individuals may already have a deficit of understanding, often due the information that they have received from elsewhere, before their interaction.

Goldwasser and Springle (2024) emphasise the significance of power differentials; they critically investigate interactions where a speaker's trust and their trustworthiness are both undermined by the more powerful listener (Goldwasser & Springle 2024). Olivia Bailey sees the significance of the relationship between the individuals that are involved; she emphasises the value of a relationship of empathy and testimonial trust (Bailey 2018). As Leah Rosen reminds us, "Epistemic injustice sits at the intersection of ethics, epistemology, and social justice" (Rosen 2021, p. 1). According to these contributions, instantiations of epistemic injustice can have a number of intersecting elements: testimonial injustice, hermeneutical injustice, prejudice, power differentials, and reduced empathy and reduced trust. In this way, EI can therefore be construed using the lens of intersectionality (Barned *et al.* 2019).

Morten Byskov argues that some circumstantial details are relevant to judgements regarding epistemic injustice. He is of the view that epistemic practices should be considered unjust in those cases where the speaker suffers a disadvantage because,

- (i) a decision is to be made,

- (ii) the speaker has relevant knowledge, and
 - (iii) the speaker is a stakeholder in a decision which affects her personally
- (Byskov 2021, p. 118).

However, Byskov sees that discounting or failing to give appropriate weight to a speaker's knowledge *may not* be unjust if the speaker lacks relevant knowledge, or if the speaker will not herself be affected by the decision.

Epistemic injustice has harmful consequences; it is a wrong because of, *inter alia*, these consequences. I now expand on those harms, some of which are integral, and some of which are consequent. Many commentators take epistemic injustice (EI) to be harmful *in its own right*:

The primary harm of testimonial injustice should be defined as [the speaker] being relegated to the role of epistemic other, being treated as though the range of one's subject capacities is merely derivative of another's (Pohlhaus, Jr. 2014, p. 107).

Here, Pohlhaus indicates that epistemic injustice is harmful of itself, because it hinders the necessary exchange of knowledge. Other commentators *add or incorporate* EI's non-epistemic consequences in their judgement of the harmfulness of EI:

The harm ... is that the speaker isn't allowed to epistemologically transact with the world in ways that enable her, as well as others', flourishing (Sullivan 2019, p. 210).

Here, Shannon Sullivan alludes to the non-epistemic harmful consequences of epistemic injustice, interference with the flourishing of the harmed individual and others. Another

commentator who negatively appraises epistemic injustice suggests that its deleterious consequences could well be integrated into the concept:

A consequentialist, harm-based critique of epistemic injustice could ... be developed from an assessment of the impact of these deleterious effects on general social welfare (Congdon 2019, p. 245).

Here, Matthew Congdon sees that it could be appropriate to appraise epistemic injustice by its consequences. His interest is the effect of epistemic injustice on *social* welfare. A consequentialist condemnation of epistemic injustice could, using similar reasoning, also reasonably incorporate an assessment of its harmful effects on *individual* welfare, as in the case of an individual patient in a healthcare encounter.

So, either, the harms of epistemic injustice are *added to* by its non-epistemic consequences (Pohlhaus 2014), or consequences are incorporated in an assessment of the harms of epistemic injustice (Sullivan 2019); or, possibly, the consequences alone are enough to criticise epistemic injustice (Congdon 2019). In any of these cases, it is appropriate to explore the *harms* of epistemic injustice in healthcare encounters, along with evidence for the existence of that EI, as I do in the next section in reference to healthcare encounters.

To summarise this section: epistemic injustice is a wrong perpetrated on the speaker by the hearer, where the speaker's credibility is discredited because of social stereotyping and where there is power differential; this injustice may be unintentional but nevertheless culpable because of prejudice. EI can be individual or systemic, and there are intersectional aspects.

The harms of EI include the failure of adequate transmission of relevant knowledge, and the hindering of flourishing.

I now move on to epistemic injustice in healthcare encounters.

6.3: Healthcare Epistemic Injustice.

Healthcare epistemic injustice (HEI), also described as ‘pathocentric epistemic injustice’, is the situation where that HEI targets ill persons (Kidd and Carel 2018). Healthcare epistemic injustice is associated with prejudice and power asymmetries; in instantiations of HEI, patients perceive that HCPs are prejudiced, and HCPs are actively attempting to retain power in the power struggle.

In this section, I give evidence for the existence of healthcare epistemic injustice, and show how it can be recognised. I show in which ways this injustice in healthcare encounters accords with Fricker’s definition of EI. I describe the negative consequences of that HEI, including the impeding of good medical care.

Patients are the experiencers of epistemic injustice in healthcare encounters, while health care practitioners act as agents of that injustice. Epistemic justice is relevant to the analysis of healthcare encounters because decisions there affect the patient personally, and unjust epistemic practices will prevent the patient imparting the relevant knowledge that she has in her possession (*c.f.*, Byskov 2021); by these failures the patient could well be harmed. For this reason, healthcare epistemic injustice conflicts with the medical duty of nonmaleficence

as defined by Beauchamp and Childress (2019), and as Della Croce (2023, *passim*) reminds us. Sigrid Wallaert and Seppa Segers's (2023) analyses of HEI provide, "... positive requirements of action, like respecting patient autonomy" (Wallaert & Segers 2023, p. 225); during experiences of HEI, patient autonomy is not being adequately respected. Here, Sigrid Wallaert and Seppa Segers (2023) see patient autonomy (another of Beauchamp and Childress's (2019) four principles) is being infringed in HEI.

Philosophical contributions to the debate regarding healthcare-related epistemic injustice note that, "... ill persons are particularly vulnerable to epistemic injustice ..." in healthcare (Carel & Kidd 2014) and as identified by Carel & Kidd 2017; Kidd & Carel 2018; and Della Croce 2023. These contributions concentrate on the analysis and evaluation of patients' experience, and on the analysis and evaluation of health care professionals' contribution to that epistemic injustice (Kidd & Carel 2017; Kidd & Carel 2018; Reynolds 2020). (To these analyses and evaluations, I add further *explanations* for the perceived unsatisfactory behaviour of health care professionals in Section Six of this chapter.)

In this section I do not widen my analysis of epistemic injustice in healthcare encounters to include other significant perspectives, for instance, the contributions that appraise agency (Mitova 2020; Radoilska 2020; L. Smith & Archer 2020; Radoilska 2022), trust (Buchman *et al.* 2017), patient empowerment (Bogaert 2021), and epistemic solidarity, which is the epistemic support that people with a shared epistemic interest can give each other (Pot 2022).

Epistemic injustice in healthcare can be recognised where there is downplaying of the patient's contribution of knowledge because of what seems to be the healthcare professional's prejudice. Some HCPs put negative valuation on some patient's testimonies:

[S]ome patient testimonies are valued above others (Rosen 2021, p. 1) ... [O]ne's identity as an "ill person" — especially if there is stigmatization or stereotyping surrounding the condition — can lead to a patient being viewed as a less credible source of knowledge (Rosen 2021, p. 3).

Here, Rosen is also claiming that the testimonies of some patients are valued *below* others' testimony, because of stigmatisation and stereotyping of these patients, due to their specific medical condition. Rosen includes CFS/ME among those conditions where medical professionals stigmatise and stereotype. (In Chapter Five, I showed the existence of and the relevance of stigma in relation to CFS.) Prejudging and stereotyping are individually culpable, according to Fricker (2007), even if unintended.

I now move to other consequences of healthcare epistemic injustice. Carel and Kidd indicate that HEI has negative consequences on the transfer of knowledge. Carel and Kidd note that, "... patients seem to occupy the position of 'other' in Pohlhaus's sense ..." (Carel & Kidd 2017, p. 343). (In this passage, Carel and Kidd are referencing Pohlhaus's (2014) contribution where she sees that in instantiations of epistemic injustice a speaker is relegated to being an 'epistemic other', merely a derivative of another's (the HCP's) epistemic task (Pohlhaus, Jr. 2014, p. 107). Pohlhaus's 'another' (2014) refers to the healthcare professional, who chooses to attend to, "... only to what stems from the perpetrator's perspective" (Carel & Kidd 2017, p. 343). The 'perpetrator' of epistemic injustice here being the healthcare professional. Carel and Kidd then indicate that patients may not be allowed opportunities to contribute their relevant experiences:

[I]ll persons ... are afforded limited capacities to epistemically contribute, usually by proving factual *information*, but not by offering their distinctive *experiences* (Carel & Kidd 2017, p. 343) [italics in original].

Here, a consequence of HEI is that both the patient *and* the professional are likely to be harmed by a deficient transfer of relevant knowledge of this kind: the professional's epistemic impoverishment in this way will lead to a reduction in that professional's ability to adequately assess the patient's symptoms for the purposes of diagnosis and for the choice of treatment options.

Ian Kidd and Havi Carel see that many patients complain when they perceive that they have been wronged epistemically; Kidd and Carel see,

... the persistent complaints patients make about their testimonial frustration and hermeneutical marginalization, and the negative impact this has on their care... (Kidd & Carel 2017, p. 172).

Here, Kidd and Carel acknowledge the relevance of the non-epistemic harmful consequences of HEI, in that there is a resultant negative impact on patients' care. Kidd and Carel give some explanations:

We identify the negative stereotypes and structural features of modern healthcare practices that generate [these injustices] (Kidd & Carel 2017, p. 172).

Here, Kidd and Carel attribute healthcare epistemic injustices to, *inter alia*, healthcare professionals' prejudice against stereotypes. Kidd & Carel's (2017) structural factors include inherent differences in social identity, the power differential between patients and HCPs, doctors' poor communication skills, and, "... doctors' perceived indifference to or ignorance of the concerns of their patients" (Kidd & Carel 2017, p. 173). Kidd & Carel's phrase, 'structural features' suggest that these injustices are systemic, and therefore likely to be widespread.

Carel & Kidd expand on explanations for healthcare epistemic injustice (HEI):

Ill persons are vulnerable to testimonial injustice through the presumptive attribution of characteristics like cognitive unreliability and emotional instability that downgrade the credibility of their testimonies (Carel & Kidd 2014, p. 529).

In this passage, Carel and Kidd see that healthcare professionals may have a low opinion of some of their patients' abilities to process and communicate relevant knowledge, because of HCPs' negative evaluation of those patients' emotions and/or cognitive ability. This low opinion may be stigma, or may be due to the HCP's appraisal of the effects of the patient's illness on their cognition, as I describe in Section Six of this chapter.

Patients suffer hermeneutical injustice when their understanding of their illness is discounted, even without testimonial injustice. In these cases patients may well be able to communicate their illness experiences adequately, and may be actively listened to, but their testimony may be ignored. As Garrath Williams says, "[P]rofessionals and organisations often ignore patients' own knowledge about their condition and treatment" (G. Williams 2020, p. 323).

This ignoring may be because of the power differential, which the HCP herself perhaps does not actually notice and accommodate for.

Kristin Heggen and Henrik Berg expand on explanations for hermeneutical injustice in healthcare, by highlighting the significance of dichotomous epistemic positions. Their contribution concerns fibromyalgia—a medical condition that shares many of the features of chronic fatigue syndrome, and whose status is similarly subject to dispute, and where patients are correspondingly subject to epistemic injustice (Della Croce 2023; Côté 2024):

First, we will argue that patients' epistemic marginalization can be reframed as an epistemological problem. There is a gap between patients' lived experience of illness [in fibromyalgia] and professionals' conceptualization of illness as disease. This gap leads to two distinct conceptualizations, possibly causing harm to patients' capacity as first-hand knowers... EBP prioritizes knowledge obtained through clinical research over other forms of clinical knowledge and has the potential for harmful epistemic devaluation of patients' stories (Heggen & Berg 2021, p. 1).

'EBP' is 'evidence-based practice', by which medical-research-derived knowledge is intended to diminish the gap between scientific knowledge and clinical practice. So, according to Heggen and Berg, there are major epistemic differences between patients and HCPs: firstly, in the means by which knowledge is acquired, and, secondly, in the content of that knowledge. Patients acquire much of their knowledge about their condition through their own experience, and the experiences of fellow sufferers in support groups; while, by contrast, HCPs' knowledge derives from the propagation of the results of clinical research in EBP. Consequently, there are differing conceptualisations concerning fibromyalgia (as with

chronic fatigue syndrome). Heggen and Berg tell us that the presence of a dichotomy of this kind between patients' and HCPs' conceptualisations of a medical condition leads to harm to patients. This harm results because HCPs prioritise their scientific knowledge over patients' experiential knowledge (*c.f.* Pomey *et al.* 2015).

This explanation aligns with a theme of this thesis, that dichotomous epistemic positions contribute to healthcare epistemic injustice.

Kidd and Carel (2018) see that dichotomous views about illness and disease exist not only within healthcare encounters but also extend beyond them, in theories and conceptualisations of disease and illness:

What's epistemically unjust might not be only agents, communities and institutions, but the theoretical conceptions of health that structure our responses to illness (Kidd & Carel 2018, p. 211).

The theoretical conceptions that Kidd and Carel (2018) oppose here are those that are naturalistic, particularly Boorse's value-free conception of 'disease' as deviations from statistical norms compared to a relevant reference class; these deviations are of internal states that depress biological functioning to below species-typical levels (Boorse 1977). In their 2018 article, Kidd and Carel (2018, p. 225) see that these naturalistic conceptualisations are ubiquitous within healthcare, in assumptions and in acts. Here, Kidd and Carel (2018) argue against the *exclusive privileging* of Boorse's naturalistic conception of health and disease, claiming that this conceptualisation is harmful if used without the testimonies, narratives, interpretations, and self-understanding of ill persons.

To summarise this section, epistemic injustice can occur in social situations where there are unequal power and prejudice; that difference in power can be inherent in the relationship, such as healthcare encounters. This prejudice in healthcare encounters may be unintentional, but it is still culpable. Testimonial injustice refers to the situation where the hearer prejudicially reduces the credibility of a speaker who has lesser power. In a healthcare encounter, a patient has less power and may have reduced ability to communicate her predicament; however, the patient has relevant knowledge and is closely affected by the decisions to be made in the encounter, so the patient suffers epistemic injustice if given reduced credibility. Hermeneutical injustice refers to situations where those with lesser power are prevented from having the resources they need in order to understand their position. In a healthcare encounter, a patient's relevant knowledge that is discounted by the health professional may include the patient's interpretation of the severity of, and the causes of, her illness. There is evidence from commentators and patients that there are episodes of reduced credibility, and therefore epistemic injustice, in CFS encounters. I now turn to an account of those episodes.

6.4: Healthcare Epistemic Injustice in Chronic Fatigue Syndrome and Similar Conditions

In this section I apply the conceptualisations of healthcare epistemic injustice that I described in Section Three of this chapter to confirm my (and others') conclusion that there is healthcare epistemic injustice in healthcare encounters concerning chronic fatigue syndrome

and similar medical conditions. I will use that confirmation in Section Seven of this chapter, where I encapsulate the main argument of this Thesis.

This section is laid out as follows. I first re-iterate some of my caveats. I then describe the significance of stigma against CFS and CFS patients as explanations for HEI in CFS healthcare interactions, as well as the ineffability of some prominent and disabling CFS symptoms. CFS patients regard the often-recommended psychological treatments as epistemically unjust.

As I expressed in Chapter One, the Introduction to this thesis, I don't claim that EI in situations of uncertainty (UHEI) is manifested in any way that is different from EI in situations without significant uncertainty; UHEI is not an extension of the meaning of EI or HEI, it is merely a further explanation for some of its instantiations. Also, as I wrote in Chapter One of this thesis, I am concerned with *injustice*, so I concentrate on analysing unsatisfactory consultations. However, I also compare these unsatisfactory consultations to healthcare encounters that are successful as reported in patient narratives, including successful health care encounters regarding CFS. The contrast between the content and outcomes of those consultations can give a guide to recognising injustice and justice. I have not identified the prevalence, or the proportion, of cases of healthcare epistemic injustice in chronic fatigue syndrome healthcare encounters. However, my argument in this thesis is not concerned directly with numbers; I merely understand that there is evidence that HEI in CFS health encounters is a significant problem:

[M]any sufferers of this disabling condition [ME/CFS] report distress and dissatisfaction following medical encounters (Geraghty & Blease 2018, p. 3093).

One reason for this dissatisfaction is shown in Hng *et al.*'s finding, that some of the doctors they interviewed failed to adequately appreciate, “ ... the impact of the disease [ME] on daily living or quality of life” (Hng *et al.* 2021, p. 1). This suggests that these doctors unjustly fail to apprehend ME patients' testimony, which may be examples of testimonial injustice.

Heggan and Berg describe the distresses that fibromyalgia patients experience in healthcare encounters: “[H]ealthcare professionals do not take their [fibromyalgia] patients' testimonies and interpretations seriously” (Heggen & Berg 2021, p. 1). Similarly, ME/CFS patients' testimonies are downgraded because of stigma:

We find that widespread negative stereotyping of patients and the marginalization and exclusion of patient voices by medical authorities provides a better explanation for expressions of frustration among patients with ME/CFS (Blease & Geraghty 2018, p. 400).

The negative consequences of this exclusion of patients' voices, as described here by Blease & Geraghty (2018), include a failure to value patients' appraisal of HCPs' treatment plans:

[P]atients with CFS/ME often experience suspicion by healthcare professionals, and many patients vocally oppose the effectiveness, and the conceptualisation, of their illness as psychologically treatable (Blease *et al.* 2017, p. 549).

Carel & Kidd expand on explanations for healthcare epistemic injustice (HEI):

Ill persons are vulnerable to testimonial injustice through the presumptive attribution of characteristics like cognitive unreliability and emotional instability that downgrade the credibility of their testimonies. Ill persons are also vulnerable to hermeneutical injustice because many aspects of the experience of illness are difficult to understand and communicate and this often owes to gaps in collective hermeneutical resources (Carel & Kidd 2014, p. 529).

In this passage, Carel and Kidd see that healthcare professionals may have a low opinion of some of their patients' abilities to process and communicate relevant knowledge, because of HCPs' negative evaluation of those patients' emotions and/or cognitive ability.

I see this low opinion as inappropriate stigmatising, and therefore blameworthy.

Psychological treatment recommendations for CFS are claimed by some patients and commentators to be epistemically unjust:

[P]eople's experiential knowledge ... [is] discredited. This stems, in part, from a psychiatric-specific form of 'epistemic injustice' which can result in unhelpful, unwanted and forced treatments. This understanding helps explain why the psychiatrisation of ME/CFS has become the focus of such bitter debate (Spandler & Allen 2018, p. 127).

Here, Spandler and Allen complain that CFS patients experience having their experiential knowledge discredited, leading to their receiving (what they see as) inappropriate treatments. They also experience moralising (being treated as though they have some control of their

symptoms (Blease *et al.* (2017)). Both these aspects of epistemic injustice are similar to those experienced by people with mental illness, as described by Anastasia Scrutton (2017 p. 348) and Drożdżowicz (2021).

We see from the above contributions (Carel & Kidd 2014; Blease *et al.* 2017; Blease & Geraghty 2018; Geraghty & Blease 2018; Spandler & Allen 2018; Heggen & Berg 2021; Hng *et al.* 2021) that many commentators find evidence for epistemic injustice in CFS and fibromyalgia healthcare encounters, often associated with stigma. In the next section, I show further evidence for that HEI, along with a CFS patient's explanations for EI in CFS.

6.5: 'Suzanne's' Explanations for Epistemic Injustice in Chronic Fatigue Healthcare Encounters.

I next analyse the anonymous Testimony of a former chronic fatigue syndrome patient whom I call 'Suzanne' (not her real name). Suzanne kindly offered to give me information about her experience of and ideas about CFS and Long Covid in order to help with this thesis, and she agreed that I could quote anonymously from her Testimony. Her Testimony (the Appendix to this thesis) is her response to my email question, "Do you have views on why there is so much epistemic injustice in regard to CFS or Long Covid?". I have numbered her paragraphs (§1, etc.). Her account illustrates many of the arguments in this thesis. Suzanne relates that she has experienced unsatisfactory CFS healthcare encounters; she also describes the experiences of other patients with whom she is in contact who have experienced similarly negative healthcare interactions. She gives some explanations for epistemic injustice in CFS,

many of which align with the conclusions in this thesis. Having established that ‘Suzanne’ is a pseudonym, from now on I usually leave off the inverted commas.

In this section, I first summarise and then analyse Suzanne’s account. I then give my interpretations of her CFS healthcare encounters. I then describe in what way some of these healthcare encounters can be described as unsatisfactory, and why they can be classified as being epistemically unjust. I highlight the explanations for epistemic injustice that I derive from her testimony, her seeming acceptance of the ‘medical disease dualism’ concept, and her suggestions for amelioration. Lastly, I show in which ways her testimony does and does not align with the arguments in this thesis.

I now summarise Suzanne’s account. Suzanne describes two episodes of long-lasting disabling and exhausting chronic fatigue. Her first episode of chronic fatigue was when she was in her twenties, and lasted for some years. She later recovered and then remained physically and psychologically well for sixteen years. The second episode was more recent when she was in her forties, when an infection with Covid 19 was followed by a recurrence of the same symptoms. (Suzanne uses the phrase ‘chronic fatigue conditions’ or ‘chronic fatigue’ when she is referring to either chronic fatigue syndrome or Long Covid.) In Suzanne’s view, both these episodes were, “... extreme physical reactions ...” (‘Suzanne’ 2024, §2), “... *following* a harsh viral illness and a period of stress ...” (‘Suzanne’ 2024, §2) [my italics]. Here, Suzanne distinguishes between the *triggers* of her episodes of chronic fatigue and the *later symptoms* of chronic fatigue. In both episodes the physical symptoms started first; later on, her mental health was negatively affected by her inability to work and the collapse of social and family structures around her. She is “now 85% better, 2 years after

getting covid, clearly back at work and coping physically and mentally with a great social and family life ...” (*ibid.*, 2024, §4).

In her Testimony, Suzanne reports that some of her CFS healthcare interactions in regard to CFS have been unsatisfactory. She tells us that the first doctor (whom I call Doctor One) whom she saw during the episode in her twenties did not believe her at all; presumably the doctor doubted Suzanne’s account of the extent, severity, and/or effects of her symptoms, or, perhaps, did not accept that her symptoms added up to any specific diagnosis. The second doctor (Doctor Two) that she saw during her first episode of illness diagnosed her symptoms as being due to depression, not due to a bodily physical condition, and that doctor offered antidepressants. She tells us that Doctor Two advised her not to join a support group because, “... it will make you worse ...” (*ibid.*, §2), and from then on Doctor Two seemed unkeen to provide help or support; Suzanne felt dismissed by Doctor Two’s manner: his, “... and off you go attitude ...” (*ibid.*, §2). When Suzanne felt that she, “... had news to bring of what appeared to help ...” (*ibid.*, §2), she was discouraged to return by the doctor’s attitude of, “... ‘oh, well she’s here again’ ...” (*ibid.*, §2). Therefore, in these accounts, Suzanne gives evidence that the unsatisfactoriness of these CFS healthcare encounters was due to the doctors’ unacceptable attitude and behaviour.

Suzanne has also found that it is fairly common for people with chronic fatigue to experiences negative healthcare encounters:

I’m in a Facebook group for Long Covid sufferers (who may suffer from chronic fatigue or other symptoms which I never had, such as heart issues and breathlessness),

and the messages there, from people all over the world are heartbreaking. Because they are not being believed by their medical professionals (*ibid.*, §5).

Here, Suzanne tells us that many of those with Long Covid have unsatisfactory healthcare encounters where they are not believed, which is similar to some the experiences of people living with chronic fatigue syndrome. Long Covid is the term used for the prolonged symptoms that sometimes follow an infection with the virus SARS-CoV-2; the symptoms and course of Long Covid are similar to those of CFS, including chronic fatigue and the disabling symptom of post-exertional malaise (PEM) (Stussman *et al.* 2025). As in CFS, many of these people have normal, "... clinical evaluations and results of routine blood tests, chest X-rays, and electrocardiograms" (Centers for Disease Control, 'Long COVID Basics' (accessed 10/6/25)). Uncertainty about Long Covid is similar to that about CFS, so it is likely that unsatisfactory Long Covid healthcare encounters have similar explanations to those of CFS.

Suzanne's accounts of the negative experiences of others and her suggestions for amelioration reveal her own and others' adverse experiences of CFS healthcare encounters. Suzanne's narrative supports a characterisation of some of her own healthcare interactions as being unsatisfactory, when she feels that her doctors disbelieved, dismissed, or stigmatised her. In her healthcare interactions with both Doctors One and Two, she sees that these doctors conducted these interactions unfairly, by disbelieving her: "...[B]eing believed is half the battle" ('Suzanne' 2024, §5); "... badged by some medical professionals ..." (*ibid.*, §5); "Sufferers really need to be heard and understood that it's more than symptoms of feeling depressed/anxious" (*ibid.*, §2); "Long Covid sufferers (who may suffer from chronic fatigue ...) ... are not being believed by their medical professionals" (*ibid.*, §5). She and her doctors

had opposing interpretations of her illness: she disagreed with Doctor Two's diagnosis of depression because she was sure that there was something, "... very physically ..." wrong with her (*ibid.*, §1). Suzanne's perception of being stigmatised relates to her view that there is stigma associated with the diagnosis of mental illness:

[S]ome medical practitioners dismiss sufferers as being depressed and so only offer psychological interventions. Because they can't prove it with a physical test. And the stigma associated with having a mental health condition is very real, which is even worse when you, as a sufferer, really don't believe that it is only a mental health condition, but that you have something very physically wrong with you (*ibid.*, §1).

Here Suzanne feels that CFS sufferers are being dismissed and stigmatised by their medical practitioners' attribution of the diagnosis of depression and their offer of psychological interventions for CFS. She feels that that stigma should *not* apply to her, because she was confident that she had a physical illness, which was being misdiagnosed.

Suzanne has evidence that sufferers from CFS and Long Covid have unsatisfactory healthcare interactions; I now make the case for classifying these unsatisfactory healthcare encounters as being epistemically unjust. Attributions of epistemic injustice require evidence of testimonial and/or hermeneutic injustice, usually associated with power differentials and culpable prejudice (Fricker 2007). In epistemic injustice the speaker has relevant knowledge and is a stakeholder in the decisions to be made (Byskov 2021), and frequently epistemic injustice is associated with negative consequences where the speaker comes to harm (Congdon 2019).

My view is that these characteristics of epistemic injustice do seem to be present when Suzanne and her correspondents in her Facebook group suffer unsatisfactory healthcare encounters ('Suzanne' 2024; *c.f.* Geraghty & Blease 2018, p. 3093. Suzanne and her correspondents have experienced doctors' disbelief, the downplaying by doctors of patients' testimonies regarding patients' experience of disabling symptoms (*c.f.* Fricker 2007, p. 1), thus giving evidence of testimonial injustice. Suzanne's difficulty in explaining her symptoms: "... sufferers can't fully explain themselves ..." ('Suzanne' 2024, §1) meshes with Carel and Kidd's recognition of the ineffability of symptoms in some medical conditions: "[i]ll persons are also vulnerable to hermeneutical injustice because many aspects of the experience of illness are difficult to understand and communicate" (Carel & Kidd 2014, p. 529). There is evidence that some CFS patients have a reduced ability to explain and communicate (Byrne 2020, p. 376-7), which will reduce their ability to influence their doctors to obtain a shared understanding of the patients' situations. Suzanne sees that some doctors reject CFS patients' interpretation, which is that their symptoms should be attributed to physical rather than psychological causes; thereby patients are hindered from being able to understand what is wrong and prevented from understanding what they can do to improve (*c.f.* Fricker 2007, p. 1). This is hermeneutical injustice. "[Some]... medical practitioners don't necessarily believe it exists at all" ('Suzanne' 2024, §1), thereby showing, "... indifference to or ignorance of the concerns of their patients" (Kidd & Carel 2017, p. 173), which similarly creates a barrier that prevent patients' understanding and interpretation. Suzanne disagrees with Doctor Two's diagnosis of depression (*c.f.* Blease *et al.* 2017, p. 549; Spandler & Allen 2018, p. 127).

Suzanne's Doctor Two then advises her not to join a support group even though the members of that group would have been able to share with her their understandings and their

interpretations of symptoms. An explanation for Doctor Two's advice could be that he was assuming some sort of 'looping effect' (Hacking 1996), whereby *CFS people* change unhelpfully in their interpretation of their symptoms and their responses to those symptoms after obtaining an *ME classification*; so that becoming a member of an ME support group could (in my understanding of Doctor Two's view) negatively affect Suzanne's interpretation and behaviour. However, I see that it is testimonially unjust when HCPs disbelieve patients, and hermeneutically unjust when HCPs prevent or reject patients' understandings and interpretations. The power differential between doctors and patients is shown in Suzanne's testimony when she describes Doctor Two's dismissal of her with him having an, "... off you go attitude ..." (Suzanne 2024, §2), which also suggests prejudice against her. The stigma attached to CFS, where patients are labelled as, "... a worrier, a hypochondriac, [and] a time waster ..." (*ibid.*, §1), also suggests prejudice (according to Fricker's 2007 definition) (*c.f.* Kidd & Carel 2017, p. 172; Rosen 2021, p. 3). Even though patients feel that they have relevant knowledge about the severity of their symptoms, and are stakeholders in the decisions about illness management, they experience downplaying of their testimonies in these encounters, which downplaying is compatible with Byskov's attributions of testimonial injustice (*c.f.* Byskov 2021 p. 118). Prejudice is integral to the recognition of and explanation for epistemic injustice (Fricker 2007, p. 54). Suzanne felt the prejudice of stigma in some of her CFS healthcare interactions. She also reminds us of the contribution of intersectionality to prejudice against CFS patients: "... most sufferers are women" ('Suzanne' 2024, §1). The epistemic injustice experienced by Suzanne can be categorised as culpable (Fricker 2007), because Doctor Two was dismissive of the results of Suzanne's self-directed research, that is to say, her, "... news to bring of what appeared to help ..." ('Suzanne' 2024, §2). Under these circumstances, Doctor Two could have become better informed by learning from and then using that information, but he chose not to; so when he was being epistemically unjust

here he was also culpable. Patients who are prevented from understanding the ways that they could help themselves and who are unfairly labelled come to non-epistemic, as well as epistemic, harm (*c.f.* Kidd & Carel 2017, p. 172; Congdon 2019). Suzanne could well have come to non-epistemic harm in that Doctor Two's actions and advice was aimed at preventing Suzanne from benefitting from the advice of those who had news for her, "... of what appeared to help ..." ('Suzanne' 2024, §2).

Suzanne therefore gives evidence from her own and others' experience that some chronic fatigue healthcare encounters do indeed exemplify epistemic injustice: these encounters show testimonial and hermeneutic injustice, in that some healthcare professionals show a lack of belief in patients' testimonies, and some HCPs do not believe patients' interpretations of their symptoms or patients' understanding of their medical condition. That epistemic injustice is related to prejudice and is culpable, and patients are coming to harm. Therefore, Suzanne's descriptions fulfil the criteria of Fricker (2007) and others for the attribution of epistemic injustice.

In Chapter Seven, I will give some suggestions for the amelioration of this epistemic injustice; my view is that this amelioration first requires *explanations* for that epistemic injustice. In order to advance my contention in this thesis that it is the differing responses by patients and HCPs to uncertainties that can explain healthcare epistemic injustice in CFS, I next analyse Suzanne's own explanations for unsatisfactory CFS healthcare encounters, her recognition of the relevance of uncertainties and dichotomies, her responses to those uncertainties and dichotomies, and the negative effects of *differing* responses to those uncertainties and dichotomies.

Suzanne gives some explanations for her unsatisfactory CFS healthcare interactions. Firstly, she notes the relevance of the ineffability of the symptoms and difficulties in articulating them: “... sufferers can’t fully explain themselves ...” (‘Suzanne’ 2024, §1). Suzanne alludes to a second explanation (one that is in accord with the theme of this thesis), that is to say, the effects of uncertainties concerning evidence about the condition: she notes the lack of objective evidence of disease in CFS cases: “... you can’t point to a test result ...” (*ibid.*, §5), and, “... they can’t prove it with a physical test” (*ibid.*, §1). Here, Suzanne highlights the dichotomy between doctors’ scientific knowledge and patients’ experiential knowledge (*c.f.* Pomey *et al.* 2015; Kidd & Carel 2018, p. 225; Heggen & Berg 2021, p. 1). Suzanne sees that there are uncertainties concerning the nature of CFS: “... it’s more than symptoms of feeling depressed/anxious” (*ibid.*, §2), and, “... there is no one proven link that the illness is physical, psychological or both, most sufferers don’t look particularly ill ...” (*ibid.*, §1). Here Suzanne seems to appreciate what Byrne calls the, “... vast conceptual impoverishment ...” (Byrne 2020, p. 377), concerning the evidence for and the nature of CFS, and that this impoverishment negatively affects some of her CFS healthcare encounters and those of other patients living with chronic fatigue.

Suzanne gives evidence supporting one of the explanations for UHEI that I give in this thesis, that some *responses* to uncertainties about CFS lead to unsatisfactory healthcare encounters. She moves away from Doctor One—the first doctor that she saw—after he, “... did not believe me at all ...” (‘Suzanne’ 2024, §2), which is likely to be that doctor’s response to the uncertainties regarding the cause, severity, and effects of her symptoms. After her encounter with Doctor Two, her next response is to attempt to reduce her own uncertainty—to search for answers to find out, “... what is wrong and do whatever you can do to improve” (*ibid.*, §1)—which implies that she responds to uncertainty by ignoring Doctor Two’s advice of, “...

never join a support group ...” (*ibid.*, §2). Thirdly, she attempts to educate Doctor Two by bringing him that information about what might help CFS patients (*ibid.*, §2), because she sees that he is under-informed about CFS. Fourthly, she continues to affirm the physical nature of CFS, because it is experienced as having, “... something very physically wrong with you ...” (*ibid.*, §1) (albeit with stress sometimes being a trigger factor (*ibid.*, §2)). Her view is that psychological symptoms do not cause the continuing physical symptoms of CFS; instead, if psychological symptoms occur, they are secondary. (I described this explanation in Chapter Three, Section Three, Subsection (i) above.) She supports this contention of psychological symptoms being secondary from her own experience: “[t]he physical symptoms happened first ...” (*ibid.*, §2), and anxiety and depression followed, “... in the months following the initial symptoms” (*ibid.*, §2). The implication here is that Suzanne has the view that her later *psychological symptoms* were caused by the symptoms of the continuing *physical condition CFS* and the effects of CFS on her social and family structures and her ability to work. As far as treatments are concerned, Suzanne’s response to uncertainty is to affirm that, “... anti-depressants don’t tend to work” for CFS. (*ibid.*, §1). She sees that the attribution of CFS as being psychological is a ‘dismissal’ of patients (*ibid.*, §1).

Suzanne’s view regarding the nature of CFS—which she sees as being physical and not psychological—depends, in my understanding, on her acceptance of a local physical-psychological dualistic conceptualisation of some medical conditions, with chronic fatigue conditions on one side and depression and anxiety on the other. We do not have information about whether, for Suzanne, this dualistic conceptualisation regarding medical conditions is global or applied just to CFS and similar conditions, and to psychological conditions. I show later, in Section Six of this chapter, that some healthcare professionals are wary of that

physical-psychological dualistic conceptualisation of medical conditions, which wariness gives an explanation for HCPs responding differently to uncertainties regarding CFS.

For amelioration, Suzanne again emphasises what she sees as the physical nature of CFS.

She recommends that doctors change how they treat CFS patients, and how they understand the nature of CFS:

To be believed and to be treated empathetically with pointers for good information on how to help yourself, and to be told that it is very much a physical illness which can and does affect your mental health, can only be positive for sufferers (*ibid.*, §5).

Suzanne's Testimony therefore gives support to many of the arguments of this thesis. She appreciates that the similarity between the symptoms of chronic syndrome and symptoms of Long Covid enables both these groups of people to be categorised them having 'chronic fatigue', and to suffer similarly unsatisfactory healthcare encounters. She sees that there are substantial uncertainties regarding chronic fatigue conditions due to, *inter alia*, the paucity of objective evidence from tests. She sees that, because of these uncertainties, there are dichotomies of views between people living with chronic fatigue on the one side and some healthcare professionals on the other side, regarding the nature of CFS and Long Covid and their consequent treatment recommendations. She sees that CFS patients suffer unsatisfactory healthcare care interactions due, in part, to these dichotomous responses to these uncertainties; and, she shows how CFS patients come to harm due to these unsatisfactory healthcare care interactions. She sees that chronic fatigue patients suffer stigma and prejudice in some healthcare encounters. Suzanne's assessment is that CFS and Long Covid patients are acting reasonably when they postulate the physical nature of CFS,

and when they tell of the reduced benefit of psychological treatments, because patients' views are based on their experience of their illnesses, where their physical symptoms precede and therefore seem to be causative of their psychological symptoms—although she sees that the physical symptoms may have originally been triggered by stress. Her suggestions for amelioration align with my own, in that she advocates for patients to be treated empathetically; she also wishes patients to be believed in their testimony that their chronic fatigue is severe—severe enough to be intrusive and affect many aspects of their lives.

By contrast, in her Testimony, Suzanne does not support two of the arguments in this thesis: firstly, my opposition to medical disease dualism, that is to say, my contention that it is inappropriate and unhelpful to claim that there is and should be a strong distinction between psychological medical conditions and physical medical conditions; and, secondly, and concomitantly, her wish for patients to be assured by their healthcare professionals that CFS is very much a physical illness clashes with my view that it is not unreasonable for healthcare professionals to conceptualise CFS as having physical, psychological, and behavioural aspects in its likely mechanisms and for its treatment recommendations. (Furthermore, my view is that it would be deceptive for HCPs to mis-state or hide their own views.)

Suzanne does not mention medical research, but it may be that, like CFS/ME patient support and advocacy groups, and unlike me, she does not see the benefit of research also on psychological and behavioural mechanisms of and treatments for CFS.

I now move to summarising and analysing the perspectives of healthcare professionals in their responses to uncertainty about CFS. These responses differ from those of many CSF patients and their support and advocacy groups.

6.6: Healthcare Epistemic Injustice: The Perspectives of Health Care Professionals.

In the previous sections of this chapter, I have shown chronic fatigue syndrome patients' first-person experiences of healthcare epistemic injustice in healthcare encounters, and I have given the (third-person) elucidations by philosophers and other commentators regarding this HEI. To these accounts, I now add explanations from the (second-person) *perspectives of health care professionals*, who, despite their professional duties, and what I conceive of as their good intents, can be the perpetrators of what patients experience as unsatisfactory healthcare encounters in CFS and other medical conditions. This line of reasoning may offer some justifications for the actions of HCPs in these healthcare encounters.

In this section, I first show arguments for the value of analysing HCPs' perspectives. I then move to show some justifications for HCPs to play down their CFS patients' testimonies, their patients' interpretations of their medical conditions, and their patients' attributions of their symptoms. I appraise these justifications in the light of Fricker's (2007) description of the characteristics of testimonial and hermeneutic injustice, to assess the possibility of mitigation for HCPs' behaviour.

As is the theme of this section, Kasper Nielsen and his colleagues stress that any judgements concerning what seem to be unsatisfactory healthcare encounters can benefit from taking into account the perspectives of healthcare professionals, as well as those of patients (Nielsen *et al.* 2025):

How do we determine that a patient has been subject to epistemic injustice? ... Many of these cases [in the academic literature] are described from a third-person perspective, leaving out the patients' first-person perspectives on situations in which they are said to be epistemically harmed... Other cases in the literature ... consist only of patients' first-person complaints, often centering on feelings of not being adequately listened to ... From such cases, we are ... forced to assume that a first-person perspective on a given situation is sufficient for determining that epistemic injustice has occurred (Nielsen *et al.* 2025, p. 5).

Nielsen *et al.* (2025) claim here that it *may not be* appropriate to consider unsatisfactory healthcare encounters to be epistemically unjust if the interactions are judged only on the perspectives of the patient or of a third party observer; for Nielsen *et al.* (2025), the missing factor is the second-person perspective of the healthcare professional:

Deciding what is medically relevant and what is not requires medical expertise. Intentionally disregarding some parts of patient testimonies, because they are medically irrelevant for the examination at hand, does not amount to testimonial injustice (Nielsen *et al.* 2025, p. 7).

So, according to Nielsen *et al.* (2025), what appears to the patient or to a third-person observer to be testimonially unjust may merely be a situation where an HCP is concentrating on what is relevant information, according to the HCP's own professional perspective. The HCP's perspective derives from her or his professional knowledge, so medical relevance may perhaps best be judged from that perspective. Elizabeth Barnes expands on the notion of

HCPs' decisions concerning relevance by giving examples that categorise what she feels an HCP would find relevant in a healthcare encounter:

There are three types of claims in [chronic Lyme disease] illness narratives:

- Claims about the experience of suffering,
- Claims about the seriousness or significance of suffering, and
- Claims about the aetiology of suffering.

Physicians should always trust (i) – the reality of suffering and functional limitation (accepting that the patient is not deceitful or malingering), almost never trust (iii) and trust (ii) only within limits. With type (iii) claims, group or collective testimony re causation (or the effectiveness of treatments) does not lead to competence (furthermore, there may well be very divergent individualised views on causation or treatments) (Barnes 2023, p. 661).

Here, Barnes is discussing healthcare encounters concerning 'chronic Lyme disease' (CLD), a condition with symptoms that are similar to CFS and fibromyalgia, and whose nature, diagnostic processes, and treatment options are similarly disputed. Many people who claim to have CLD comprehend that their continuing symptoms are due to continuing infection, requiring long-term antibiotic therapy. In opposition to this view, HCPs see no objective evidence for continuing infection, so many HCPs are reluctant to prescribe long-term antibiotic therapy for these people.

Barnes's view is that patients' testimonies should not necessarily be trusted regarding the seriousness of their medical condition, and that patients' views on the cause of their medical condition (as being due to infection, in the case of CLD – or being neurological in the case of

CFS) are probably not trustworthy at all. Barnes's understandings here align with those of Nielsen *et al.* (2025), who see that HCPs' intentionally disregarding some parts of patient testimonies may not amount to testimonial injustice if these testimonies seem to the HCP to be medically irrelevant for the examination at hand. In the case of CLD, many HCPs see the attribution of chronic infection needing long-term antibiotic therapy as incorrect, not just irrelevant.

Barnes's (2023) and Nielsen *et al.*'s (2025) stance here contrasts with that of Blease *et al.* (2017), who see that it is hermeneutically unjust for patients' interpretations to, "... play no role in the diagnostic and clinical process" (Blease *et al.* 2017, p. 555).

My view is that Barnes has made an appropriate distinction: I agree with her that patients do have unchallengeable knowledge regarding their symptoms and their deleterious effects (as I argued in Chapter Three, Section Three, Subsection (c)). However, I argue here, partially aligning with Barnes (2023) and Nielsen *et al.* (2025), that, for people whose symptoms fit with CFS, their testimony regarding the aetiology of their symptoms and the seriousness of their medical condition may not be correct *in their own individual case*, for three reasons:

- (1) The aetiology of the symptoms of CFS has not been adequately established, as I described for CFS in Chapter Three, Section Three, Subsections (g), (h), and (i);
- (2) Patients' interpretations may well be influenced by those researchers who are confident that the medical condition CFS is biomedical and not psychological, which depends on a dichotomy that I challenged in Chapter Five, Section Four;
- and,

(3) Without adequately verified and agreed definition for CFS (as I reported in Chapter Three, Section Three, Subsection (b)), or a diagnostic biomarker, or the identification of possible significantly different subgroups, it is not possible to confirm that any particular patient with CFS symptoms shares the same aetiology with others' CFS symptoms; so, the attribution of the cause of their symptoms may not be correct, or may not be relevant.

However, *pace* Barnes (2023) and Nielsen *et al.* (2025), my view is that the judgement of relevance is not straightforward in *situations of uncertainty*. Radical uncertainties regarding CFS mean that it is possible that the patient's conceptualisation may well be more correct than that of the HCP. The patient's standpoint is of value in this situation (although, in my view, it might not necessarily be privileged). So, the HCP's decision concerning, "... what is medically relevant and what is not ..." (Nielsen *et al.* 2025, p. 7) may be less relevant, despite the HCP's medical expertise. My suggestion is, therefore, that Barnes's categories (ii) and (iii) (Barnes 2023, p. 661) could be better phrased as, "It is not unreasonable for physicians to be agnostic about patients' claims of seriousness and aetiology, bearing in mind the many uncertainties about their medical condition". The word 'agnostic' here allows (and I suggest should encourage) a dialogue between an HCP and a CFS patient in a healthcare encounter, where they share their views about what is known and not known about the seriousness and aetiology of that particular patient's symptoms, which dialogue aligns more with the views of Blease *et al.* (2017, p. 555) for avoiding hermeneutical injustice.

CFS patients may claim a neurological aetiology for their condition, because the ICD-11 classification of CFS is as a neurological condition (World Health Organisation, ICD-11,

Chronic Fatigue Syndrome 8E49). However, many HCPs see this classification of CFS as inappropriate because there is inadequate evidence for abnormal neurological physiological processes in CFS patients. The British neurologists who responded to a survey arranged by the researchers Wojcik and colleagues (2011a): 84% did not view CFS as a neurological condition (as I noted in Chapter Three, Section Three, Subsection (d)). One would expect neurologists themselves to be able to distinguish neurological conditions from non-neurological conditions. Many other doctors doubt the classification of ME as a physical illness at all: “[o]nly 9.1% responded physical ... 82% of participants believed ME was at least in part psychological” (Hng *et al* 2021, p. 1).

Some HCPs not only contest the classification of CFS as a neurological condition; they have doubts about even the existence of CFS, as I described in Chapter Three, Section Three, Subsection (c). Doubt by some medical professionals about the existence of CFS can explain some unsatisfactory healthcare encounters. Some CFS patients interpret challenges to the reality of their medical condition as dismissal or downplaying of their suffering. However, as I also described in Chapter Three, Section Three, Subsection (c), there is an important distinction between doubting the existence of a medical condition and doubting the reality of patient’s symptoms. Hofmann (2022) analyses this problem:

The distinctions of the concepts of disease, illness, and sickness can explain many misunderstandings between health professionals and patients. For example, the physician can say that ‘I cannot find disease’ (meaning ‘I cannot identify any known diseases to the condition that you have’) while the patient hears “you are not ill” (meaning that ‘I do not believe that you experience illness’). Clarifying such misunderstanding can thus be of great value (Hofmann 2022, p. 1156).

Here Hofmann sees an unnecessary misunderstanding, where the health professional's words can be misinterpreted as disbelief in the patient's suffering. The HCP needs to be clear.

Another unnecessary misunderstanding concerns the mechanisms of CFS. Without definitive evidence of pathological mechanisms in CFS, either physical or psychological, then it is my view that it is not unreasonable for healthcare professionals to postulate that there may be a combination of these processes in CFS. My view here is a counter to the opinion of some CFS patients, their support groups, and some commentators, that the attribution of psychological factors is incorrect and stigmatising.

HCPs and CFS patients may understand that, because of their illness, some CFS patients have cognition and emotional problems that may interfere with understanding and articulating information (*c.f.* Byrne 2021, p. 52). Therefore, attributions of reduced cognitive ability are not necessarily due to prejudice and stigmatisation.

Diagnosing CFS/ME is challenging; according to Pheby and colleagues (2020) and Hng and colleagues (2021), diagnosis is hampered by doctors' ignorance and uncertainty about it, leading to delays in diagnosis. These commentators see that there is similar lack of HCPs' confidence in managing CFS/ME (Pheby *et al.* 2020; Hng *et al.* 2021). However, one health care professional whom Horton *et al.* (2010) interviewed does seem to have confidence in managing CFS/ME; he or she claims to have seen benefits from exercise and cognitive behaviour therapy for these patients: "... [I]f you push yourself and you get tired ... those are the people who actually do the best from CBT because it actually helps to break that cycle'" (Horton *et al.* 2010, p. 5, quoting HCP6). Here, this healthcare professional is using

his or her professional experience to evaluate treatments, which may be an explanation for that HCP's approval of CBT.

I argue here that one explanation for unsatisfactory CFS healthcare encounters can follow the good intentions of health care professionals, where an HCP may see that the CFS patient's response to uncertainty differs from the response of the HCP; but, the HCP attempts to help the patient get better by challenging the CFS patient's biomedical conceptualisation of CFS. The HCP acting in this way is likely to be because the HCP believes that the patient's preferred biomedical understanding of CFS leads to inappropriately reduced activity and therefore to exacerbation and perpetuation of CFS symptoms. The HCP may be motivated by the need to respond her patient's symptoms (even if that response lacks adequate evidence of effectiveness for that particular patient).

A further explanation for the HCPs' rejection of the biomedical model (BMM) is because the HCP may see BMM as being too narrow an explanation for the symptoms and course of CFS. In many other medical conditions psychological and behavioural factors are relevant to recovery.

I therefore see a number of explanations for the behaviour of HCPs in healthcare encounters regarding CFS and similar conditions:

- (1) HCPs may have different (and defensible) concepts concerning what knowledge is relevant to the encounter; and,
- (2) HCPs may have a greater appreciation of the deleterious uncertainties regarding some medical conditions; and

(3) Because of those uncertainties, HCPs may have (defensible) conceptualisations of the pathological processes that are making the patient ill and which treatments may help, which conceptualisations may be different from the patients' conceptualisation. I see that these responses by HCPs do not necessarily derive from blame, stigma, or prejudice.

So far in this section, I have given some explanations for healthcare professionals' actions in (what some patients and some commentators consider to be) unsatisfactory CFS healthcare encounters. I now describe the negative aspects of some CFS healthcare encounters in order to appraise whether these encounters should be considered to be epistemically unjust, according to the tenets of epistemic injustice that I described in Section Two of this chapter.

An HCP's rejection of the relevance of parts of a patient's testimony, as allowed by Barnes (2023) and Nielsen *et al.* (2025), is likely to be experienced negatively by a patient.

Similarly, a patient is likely to perceive negatively an HCP's rejection of a patient's claims regarding the seriousness or significance of her suffering, and a rejection of the patient's attribution of the aetiology of that suffering, as allowed by Barnes (2023). Wojcik and colleagues' (2011a) survey shows that many HCPs reject a neurological aetiology for CFS; others reject a physical aetiology at all. The down-playing of a CFS patient's testimony because of the attribution of psychiatric disease, or the ascription of reduced cognitive ability may be empirically based. Horton *et al.*'s (2010) 'HCP6' sees the value of exercise therapy, which many CFS patients feel ignores their testimony concerning the suffering that follows exertion. There may be rational rather than prejudicial explanations for HCPs to play down or reject CFS patients' testimonies and interpretations. So, the HCP may not be culpable, as

Fricker (2007) alleges. However, each of the behaviours of HCPs itemised in this paragraph would be *experienced by CFS patients* as being epistemically unjust.

The possible absence of ostensive blaming of, stigmatising of, or prejudice towards patients by HCPs, as I suggested above, may seem to be a counter-claim to my contention that CFS patients are subjected to epistemic injustice. My response is that I see the essence of healthcare epistemic injustice is the patient's *experience* of being epistemically marginalised, with her testimony being dismissed: "... a wrong done to someone specifically in their capacity as a knower" (Fricker 2007, p. 1), and as described by Carel & Kidd (2014); Blease *et al.* (2017); Blease & Geraghty (2018); Geraghty & Blease (2018); Spandler & Allen (2018); Heggen & Berg (2021); Hng *et al.* (2021), and 'Suzanne' (2024), and her hermeneutical experience being devalued. So, my view is that these dismissals by HCPs are epistemically unjust.

In this section, I have argued that actions that seem to be epistemically unjust may have explanations that mitigate that accusation, once the actions are inspected from the perspective of the person who appears to be the perpetrator. In healthcare encounters, health professionals can use their specialist knowledge to appraise what testimony is relevant; they can dismiss patients' under-supported attributions of aetiology (Barnes 2023). Health professionals see that uncertainty concerning CFS puts into doubt many of its claimed attributes, such as being fundamentally a neurological condition, or that behavioural and psychological factors are of little significance in the perpetuation of the symptoms. It may be that these healthcare professionals' not-unreasonable responses are *not* associated with blaming or stigmatising CFS patients, nor with prejudice against them, so these HCPs may not be blameworthy. However, as I said in Chapter Six, Section Three, patients are the

experiences of epistemic injustice, due to the actions of health care practitioners. In my view, it would be inappropriate to fail to privilege the perceived experiences of patients in these circumstances. I see mitigation for these acts by HCPs, but I still see epistemic injustice.

6.7: The Central Argument of This Thesis

In this section, I give justifications for the central argument of this thesis concerning explanations for epistemic injustice in chronic fatigue syndrome healthcare encounters, that epistemic uncertainty about CFS and a consequent dichotomy of conceptualisations add to the well-recognised power differential in healthcare encounters, and the stigma associated with CFS, together all explaining uncertainty-driven healthcare epistemic injustice (UHEI) in CFS.

In the first subsection, I argue that dichotomies of conceptualisations about CFS derive from radical uncertainty concerning CFS; uncertainty about many other medical conditions is not radical in this way. In the second subsection I show how these oppositional dichotomous conceptualisations can lead to epistemic injustice in healthcare encounters. In the third subsection, I argue that the disparities of power between patients and healthcare professionals can lead to the HCP imposing her conceptualisation on the patient, which imposition is epistemically unjust.

6.7 (a): Radical uncertainty about chronic fatigue syndrome explains dichotomies of views

In this subsection I argue that oppositional dichotomies of conceptualisations about CFS can be explained by different responses to radical uncertainty about it. In order to support this argument, I also give counter-examples in healthcare where there is uncertainty but no dichotomy.

In Chapter Three, Section Five, I established that there is radical uncertainty regarding chronic fatigue syndrome. In a healthcare encounter both parties could well be in the cognitive states described by Romeijn & Roy (2014), where they are uncertain about states of the physical world (Hansson 2022), but inadequately appreciate what is unknown (Djulgovic *et al.* 2011).

Uncertainties about CFS negatively impact clinical encounters: for example, an individual patient with symptoms that could be ascribed to CFS seeking the help of a medical professional, it is usual for there to be no objective findings when the HCP examines the patient, with standard laboratory and imaging investigations also usually showing no abnormalities. There is no biomarker to confirm or to refute the diagnosis. Consequently, there are oppositional dichotomies of views between patient and healthcare professional: the patient has experiential knowledge that he or she has a significant illness with significant disruption of his or her ordinary tasks of life, while the HCP is quite likely to have found nothing objectively wrong with the patient (*c.f.* Pomey *et al.* 2015).

Uncertainty does not always lead to dichotomies of view. There are counter-examples of other medical conditions where there is a significant level of uncertainty but no significant dichotomies. For instance, the initial and underlying causative mechanisms of multiple sclerosis and of many forms of dementia are unknown, even if many of their other

pathological processes are understood. Despite uncertainties there is little evidence of dichotomous and oppositional conceptualisations and oppositional treatment recommendations in these conditions. My view is that CFS differs from multiple sclerosis and dementia by being not just subject to *some* uncertainty but by being subject to *radical uncertainty*, in definition, demarcation, and homogeneity, as I argued in Chapter Three.

6.7 (b): Oppositional dichotomies contribute to epistemic injustice in chronic fatigue syndrome healthcare encounters

In CFS healthcare encounters, HCPs are responding not just to the epistemic uncertainties about CFS but also to dichotomies: many CFS patients oppose management strategies that are common and are accepted in other areas of medicine. I now illustrate those other management strategies.

Dichotomous views do not always lead to epistemic injustice. In other medical conditions, there can be differences of view between patients and HCPs that do need to be interpreted as being epistemically unjust. For instance, in rehabilitation after surgery it is nowadays generally agreed that patients need to exercise. Until the middle of the twentieth century the dictum was the need for rest after surgery in order to aid healing; but, more recent empirical evidence has shown that early mobilisation following most forms of surgery reduces the likelihood of major post-surgical complications such as deep vein thrombosis, pulmonary embolus, and pneumonia. Furthermore, deliberate exercise after heart surgery aids rehabilitation, enabling patients to increase their exercise tolerance and improve cardiac function and patients' general health and prognosis. There is no uncertainty concerning this recommended cardiac rehabilitation regime. Moreover, there is no dichotomy external to the

healthcare encounter, in that there are no cardiac surgery patient support and advocacy groups that condemn this exercise regime; contrarily, the advice of cardiac patient support and advocacy groups coheres with that of the medical professionals (British Heart Foundation, *‘What happens after heart bypass surgery?’* (accessed 13/7/25)).

However, on an individual basis, there may not be immediate concordance between the patient who has had cardiac surgery and her physiotherapist. The patient may well suffer chest pain during exercise and may interpret that pain as being a sign of harm or damage. If the patient has had cardiac surgery through the sternum, she will have an unhealed fracture of that bone, which will give significant pain on most movements, including exercise.

Unfortunately, that pain may feel similar to the pain she felt on exertion due to heart disease before the surgery. She may reasonably feel that her symptoms imply danger, and therefore be reluctant to continue exercising. In response, the physiotherapist then has the task of convincing the patient that the pain is not a symptom of harm, and that exercise is beneficial.

There is therefore a mismatch of understanding because of an inevitable difference between the patient’s phenomenological experience on the one hand, and the physiotherapist’s expertise (which may well be phenomenologically informed) on the other hand. By the phrase ‘phenomenologically informed’, I mean that the physiotherapist understands the patient’s interpretation of her body’s reaction to exertion, and the physiotherapist’s response is to encourage the patient in the light of that understanding. At the time, the patient may well temporarily consider that this healthcare encounter is unsatisfactory. My view is that this is not an example of radical uncertainty as in CFS, because the physiotherapist’s stance is evidence-based; I understand that the power of the healthcare professional is being used appropriately to defuse the apparent dichotomy of interpretation of symptoms. I conclude that the relative certainty concerning cardiac rehabilitation means that differences of view

between cardiac patients and healthcare professionals do not seem to be example of epistemic injustice.

The biopsychosocial conceptualisation of CFS symptoms (in opposition to CFS patients' biomedical conceptualisation) can lead to testimonial injustice, because it can allow the HCP to downplay the CFS patient's testimony concerning the severity and adverse effects of her symptoms; and, BPS can lead to hermeneutical HEI, because it is possible for the healthcare professional to apply BPS to the patients' symptoms while at the same time discounting the patient's hermeneutical understanding of her symptoms. HCPs' challenges of this kind do involve the downgrading of evidence derived from patients' lived experience, and the opposing patients' interpretation of the causes of their symptoms. This challenging and downgrading can be interpreted as being epistemically unjust; therefore, HCPs' behaviour can lead to epistemic injustice, even if motivated by good intentions.

6.7 (c): Uncertainty plus power leads to uncertainty-driven healthcare epistemic injustice.

In this subsection, I show explanations for uncertainty-driven healthcare injustice that include the power differential within the healthcare encounter.

HCPs are trained and motivated to act rather than do nothing. Professionals' response to the uncertainties and dichotomies concerning CFS are the result of their training and roles, whereby they try to fit the symptoms of CFS into an explanatory framework. Some HCPs apply a framework that consists of low mood with overtraining syndrome (Kreher & Schwartz, 2012; Cadegiani & Kater 2019), or physical deconditioning (Vercoulen *et*

*al.*1998). At least those frameworks give treatment options. The power invested in the roles of HCPs seems to be implicitly endorsed by the action of the patient in seeking professional help. It is therefore not difficult for an HCP to follow her usual path of ‘diagnosis’ leading to treatment recommendation. By contrast, many people living with chronic fatigue syndrome respond to scientific uncertainty regarding CFS by falling back on the certainties of what they know from experience about their bodies and its present failings; they experience the disease process as physical, and their experience is that exertion beyond certain constrained limits causes them harm. However, when CFS patients attempt to explain their perspective, their testimony is downgraded and their experience and understanding of their bodies is disdained by some HCPs. This is a situation crying out for amelioration.

There is differential power in healthcare encounters: the HCP has personal (interactional) power, but also uses medical authority (the power of her or his professional status), as supported by medical research and by official guidelines. The patient can choose not to co-operate with the decision-making process, or with the actual decision regarding management of the CFS; or the patient has the option of not returning to this HCP and instead arranging to change to a different HCP (as did Suzanne by changing from Doctor One to Doctor Two, as I described in Chapter Six, Section Five).

Conflicts between patients and HCPs regarding their dichotomous views of pathological processes of CFS and its treatment recommendations are compounded by what I consider to be inappropriate and unhelpful resistance to opposing interpretations. Dichotomous views are not-unreasonable responses to uncertainty regarding CFS, but the rejection of an opposing view in this situation of radical uncertainty is epistemically vicious (as I will describe in Chapter Seven, Section Three).

CFS patient narratives show that their evaluation of the results of CFS research and their application of that research to their own symptoms are often belittled by HCPs; so, uncertainty plus power leads to testimonial injustice. The biopsychosocial model (BPS) leads to HCPs' rejecting patients' understanding and interpretation of their symptoms; so, dichotomy plus power leads to hermeneutical injustice.

In this section I have shown justifications for the main argument of this thesis, that *epistemic uncertainty, differing responses to that uncertainty, and the power differential within healthcare encounters, can add together with stigma and prejudice to explain epistemic injustice in some chronic fatigue syndrome healthcare encounters.*

6.8: Conclusion to Chapter Six with Responses to Possible Counter-Claims

In this chapter (and aligning with the interpretations and arguments earlier in this thesis), I have argued that chronic fatigue syndrome is subject to a great depth of uncertainty. I have further argued that there are great and more profound *dichotomies of response* to that uncertainty in CFS. I have shown that there are opposition and resistance to others' differing responses in CFS. I have used others' descriptions of epistemic injustice, along with narratives of some CFS healthcare encounters and the interpretations of those encounters by commentators, to show that unsatisfactory CFS healthcare encounter do indeed exemplify epistemic injustice. I have agreed that there is epistemic injustice in CFS healthcare encounters. In these encounters, patients' and HCPs' different conceptualisations of what is wrong are associated with consequential mismatches regarding which treatments should be recommended. Some HCPs then seem discount their patients' knowledge. Mismatches of

these kinds, if unresolved, may explain the patient experiencing epistemic injustice, if the HCP seems unreasonable in her rejection of the value of the patient's claims of knowledge and of the patient's methods of creating knowledge. When these mismatches are associated with stigma and prejudice, they are culpable. 'Suzanne's' view is that epistemic injustice in CFS is associated with prejudice and stigma, as Fricker (2007) reminds us. I have shown stigma and prejudice against CFS and CFS patients. I concluded by claiming that all these negative factors are joined in an explanatory group that leads to uncertainty-driven healthcare epistemic injustice in many chronic fatigue syndrome healthcare encounters.

I then summarised the main argument of this thesis, that uncertainty, differing responses to that uncertainty, and the power differential within healthcare encounters can add together with stigma and prejudice to explain epistemic injustice in some chronic fatigue syndrome healthcare encounters.

A counter-claim to my conclusions in this chapter could be that there are not dichotomous responses to uncertainty in CFS. However, I have shown that there is ample evidence for these responses. It might be argued that this pathocentric epistemic injustice in CFS is not due to oppositional responses to uncertainty but is due merely to the other explanations for epistemic injustice in healthcare encounters, the already-recognised imbalance of power between patients and healthcare professionals, the effects of the differences in their phenomenological and professional experience, and HCPs' inappropriate stigmatisation and prejudice. My response to that counter-claim is that patient narratives, CFS patient support and advocacy groups, and many philosophical commentators, all confirm that disputes between healthcare professionals and CFS patients do seem to concern their differing views about the nature of CFS and what treatments should be offered to patients, and these differing

views are explicable by uncertainty about CFS. It could be argued that the many unsatisfactory CFS healthcare interactions are not instantiations of epistemic injustice. In response to that charge, I have shown examples of the dismissal by healthcare professionals of CFS patients' testimony concerning the severity and effects of their symptoms (which dismissal is testimonially unjust) and dismissal by healthcare professionals of CFS patients' interpretation and understanding of the causes of their medical condition (which is hermeneutically unjust), thereby exemplifying the two components of epistemic injustice.

7: Responses to Some of the Problems of Chronic Fatigue Syndrome

7.1: Introduction to Chapter Seven

In the previous chapters, I have identified explanations for healthcare epistemic injustice in healthcare encounters concerning chronic fatigue syndrome and similar medical conditions. I have shown the many epistemic uncertainties about CFS, the failure of research in reducing those uncertainties, and the consequent epistemically unjust CFS healthcare encounters. In this chapter, I give proposals for the amelioration of this epistemic injustice and for mitigation of its harms by addressing what I take to be some of the explanations for that epistemic injustice. I also look for amelioration for the disputes concerning the mechanisms of CFS, and for choosing directions for research into CFS. (As I stated in Chapter One, I take amelioration to mean a practice that improves an unsatisfactory situation, and mitigation to mean reducing the negative *effects* of an unsatisfactory situation.)

This chapter is ordered in eight parts. In Section Two, I describe the generic skills and strategies—as proposed by others—for reducing healthcare epistemic injustice that are also likely to improve CFS healthcare interactions. These skills and strategies aid mitigation by reducing the negative effects on healthcare encounters of the imbalance of power between patients and healthcare professionals, and the effects of the differences in their phenomenological and professional experience. In Section Three, I address CFS healthcare encounters specifically; I show that the negative effects of uncertainties and opposing dichotomies can be reduced. I propose that where there are dichotomies, there is value in the application of a process where both sides come to an understanding of their opponents' reasoning and perspective, and where both sides then find ways to move their positions to

ones that incorporate some aspects of their opponents' reasoning and perspective. By this means, CFS patients and HCPs can both move towards a shared understanding of what is wrong and what can be done about it. In Section Four, I describe standard disease models; I show the difficulties of applying these models to chronic fatigue syndrome. In Section Five, I describe the (hypothetical) network of symptoms disease model, and its hybrid variant that incorporates the possibility of a latent variable. In Section Six, I suggest a re-conceptualisations of CFS using this network model. In this way it may be possible to incorporate both oppositional interpretations regarding CFS into this new hypothesised disease model. I suggest that if this model were to be applied to CFS it may not only aid amelioration in patient/HCP encounters, it could also help direct research into the mechanisms of CFS. In Section Seven, I offer a different alternative reconceptualisation for CFS that concentrates on addressing the persistence of the symptoms of CFS; this conceptualisation eschews the necessity of incorporating claims of understanding its mechanisms. In Section Eight, I summarise the lessons of this chapter.

7.2: Generic Routes to Amelioration of Healthcare Epistemic Injustice

The mitigation of epistemic injustice in healthcare encounters requires, *inter alia*, the reduction of the effect of the power differential between patients and healthcare professionals, and a constructive response to the effects of the differences in their phenomenological and professional experience. This issues are addressed in others' contributions. (Carel & Kidd 2014; Carel 2016; Kidd & Carel 2016; Carel & Kidd 2017; Kidd & Carel 2017; Buchman *et al.* 2017; Annesley 2020; G. Williams 2020; Rosen 2021; Heggen 2021; Della Croce 2023; Côté 2024). Additionally, the presence of empathy and

testimonial trust mitigate epistemic injustice (Bailey 2018) in healthcare. My proposals add to those of other commentators in two ways. Firstly, my proposals respond to the additional explanations for HEI in CFS as outlined in this thesis, that uncertainty about CFS leads to dichotomous responses which lead to disputes which themselves lead to HEI; and, secondly, that mitigation would be helped by healthcare professionals *and* patients and their supporters recognising and acknowledging these uncertainty-driven explanations for HEI, and consequently modifying their responses.

As I wrote in my Summary of Argument in the Introduction and Outline of this thesis, and as I described in more detail in Chapter Six, wrong is being done to people living with chronic fatigue syndrome in their capacities as knowers, in that their testimony regarding the severity and effects of their symptoms and their interpretations of their predicament. As I implied there, healthcare professionals need to change, in order to eliminate their inappropriate deficits of credence. Many commentators recommend changes to reduce healthcare epistemic injustice that should be applied to any healthcare encounter, not just those concerning chronic fatigue syndrome. The general theme is that professionals should move away from the traditional paternalistic doctor-centred consultation style. As Geraghty and Blease (2018) tell us, “Clinicians should consider adopting alternative patient-centred approaches”. Others expand on what the term ‘patient-centred’ means. Côté (2024) highlights the need to improve the epistemological function of a healthcare encounter: she recommends that in information exchange in a healthcare encounter, clinicians should exercise epistemic humility, because patients’ testimony is equal and competent. This humility aligns with the appreciation of the value of epistemic pluralism that I described in Chapter Two, Section Three. Rosen also sees patients as knowledge-sources; she emphasises that healthcare professionals need to change their views of patients’ contributions in order to

see patients as a valuable source of unique knowledge (Rosen 2021). Kidd and Carel (2017) see that the patient's phenomenological experience should be central in a healthcare encounter; they recommend that HCPs listen to patient narratives, in order to grasp what the illness means to the patient. Kidd and Carel (2017) recommend a 'phenomenological toolkit' in order to overcome patients' inevitable inarticulacy and ineffability. This phenomenological toolkit prioritises patients' illness experiences and their reaction to those experiences over a belief in the reality of an objective disease as a biological process (Kidd & Carel 2017, p. 358). Carel and Kidd's (2017) phenomenological toolkit could well integrate the formulations of healthcare professionals and patients in a condition like CFS, where the patients have illness symptoms, but where the role of biological processes has not been adequately elucidated. Under these circumstances, the patient's local knowledge and epistemic privilege need to be central to the healthcare encounter. Bayliss and colleagues (2014) recommend that medical professionals should collaborate with patients in order to reach agreement. Catala (2020) similarly recommends collaboration, with the emphasis on that collaboration being two-way, giving the reason that patients have, "... local knowledge ..." (Catala 2020, p. 755). Scrutton (2017) similarly understands that the patient is epistemically privileged compared to the health professional.

The medically-qualified academic researcher into health policy Marie-Pascalie Pomey and her colleagues see patients as central to decision-making: they encourage HCPs to change towards making patients essential partners in all decisions, that is to say, treating them as experts of their own care (Pomey *et al.* 2015). Similarly, Drożdżowicz (2021) puts the onus on healthcare professionals to reduce HEI. Kidd and Carel (2017) put the onus on the structures and institutions of contemporary healthcare health to reform. However, I suggest

that there is an advantage if CFS patients and their support and advocacy groups can change too, in their stances on the biomedical model and the biopsychosocial model for CFS.

I conclude this section by repeating that the generic skills that help towards reducing epistemic injustice in healthcare interactions include the recognition where HEI is existing, the reduction of prejudice and of medical professionals' epistemological presuppositions, and the incorporation of patients' phenomenological experience as an integral part of the collaboration that leads to a shared formulation of the problem and shared responses to that problem.

7.3: Some Routes Towards the Amelioration of Healthcare Epistemic Injustice in Chronic Fatigue Syndrome.

In the previous section, I designated generic routes towards reducing epistemic injustice in healthcare encounters. In this section, I concentrate on the problems of CFS healthcare encounters. Routes to amelioration of pathocentric epistemic injustice in these encounters include identifying, examining, analysing, challenging, and (where possible) reducing, the reasons for that HEI. I suggest routes that critique unhelpful responses to unresolvable uncertainty and promote helpful responses, where I use the concepts of explicitness, non-eristic debate, epistemic pluralism, the appreciation of the reasonableness of an oppositional stance, the appreciation of the weaknesses and lacunae of one's own stance, the tenets of epistemic virtue, and tolerance for epistemic situationism.

My first recommendation for the amelioration of CFS UHEI is the need for explicitness.

Both HCP and patient need to openly acknowledge that the many uncertainties about CFS lead to dichotomies of view.

My second recommendation is that debates within CSF healthcare encounters need to be undertaken co-operatively and non-eristically, with the shared aim of finding mutual productive grounds. (Non-eristic argumentation is non-adversarial, aiming at problem-solving by finding mutual grounds, rather than ‘winning’ by refutation of one’s opponent’s position (Hundleby 2021).) I can see the value of using these methods not only in individual healthcare encounters, but they also need to be applied to the general biopolitical debate about CFS because the contents of that debate are widely disseminated and therefore impact individual healthcare encounters.

My third recommendation concentrates on accepting the reasonableness of each perspective, according to the precepts of epistemic pluralism, as I described in Chapter Two, Section Three. In my view, neither a claim for the biomedical model nor a claim for the biopsychosocial model for CFS is unreasonable; each is believable. (This believability is in contrast those cases where some people insist on an *unbelievable claim*, for instance, the claim that 5G causes Covid-19, which claim conflicts with our basic understanding of physics and physiology.) I view the BMM interpretation (with its treatment recommendations)—as favoured by many people living with chronic fatigue syndrome, CFS patient support and advocacy groups, and their philosophical supporters—as being not unreasonable, bearing in mind that this perspective is a response to the predominance of physical symptoms in CFS. Contrariwise, the BPS interpretation of many medical professionals is similarly a not-unreasonable response to *their* perspective, which is

dominated by the vast conceptual and epistemological impoverishments regarding CFS, with their rationale, that the mechanisms of the roles of unhelpful cognition and behaviour are coherent with their other knowledge. Similarly, the opposing consequent treatment recommendations that follow from those differing conceptualisations are also not unreasonable.

My fourth recommendation is the necessity of acknowledging the weaknesses and lacunae in one's own stance. I now summarise the weaknesses and lacunae in each side's understanding of CFS.

Supporters of the biomedical model for CFS need to accept there is also a major challenge to this biomedical model in that the consequent CFS management recommendation of 'pacing' is merely for symptom control; pacing is not a cure, and is not even aimed at improvement in functioning over time (Jason *et al.* 2008; Jason *et al.* 2010; Brown *et al.* 2013; O'Connor *et al.* 2019; Jason *et al.* 2020). Many CFS patients remain affected negatively by their illness for many years, and some are bed-bound. Furthermore, it may be that some CFS patients should not avoid exertion; uncertainties about CFS mean that we lack evidence that CFS is a unitary disease, that is to say, a disease where all patients share the same pathological processes and require the same treatments. So, it may be that, for some CFS patients, exercise therapy and CBT may be of benefit. After all, the PACE trial did show some benefit from exercise in some patients (White *et al.* 2011). Additionally, supporters of the biomedical model for CFS cannot fail to appreciate that biomedical research has not at this stage led to any effective biomedically-based curative treatments, nor even to biomedically-based treatments that give improvement in symptoms or functioning over time. In this unsatisfactory situation, non-biologically-based supportive psychotherapy in the form of

cognitive behaviour therapy is not an unreasonable recommendation. This recommendation of the offer of CBT is endorsed in the most recent NICE guidelines in order to, “... support people who live with ME/CFS to manage their symptoms, improve their functioning and reduce the distress associated with having a chronic illness” (NICE Guidance 2021, ‘Managing ME/CFS’). This form of CBT is limited in intent compared to the previous NICE endorsement of CBT, which was intended to, “... sustain or gradually extend, if possible, the person's physical, emotional and cognitive capacity” (NICE 2007, 41.6.2.1, p. 5). The NICE 2007 aim to extend ME/CFS patients’ physical capacity was a source of conflict. By contrast, a shared acceptance of the value of the NICE 2021 recommendation of supportive CBT could lead to a reduction in conflicts between patients and HCPs about the management of CFS. The dispute between the biomedical and the biopsychosocial conceptualisations for CFS is, for the moment, unresolvable, in that there is not enough evidence to support BMM (although the application by medical researchers and other commentators of ‘optimistic meta-induction’ indicates that the necessary evidence will appear in the future (O’Leary 2018)), and that it is ethically and epistemologically wrong to reject the evidence of the phenomenological experience of CFS—the patients’ symptoms and the effect that the symptoms have on the patients’ lives.

However, supporters of the opposing biopsychosocial model and its consequent treatment recommendations of exercise therapy and cognitive behaviour therapy need to acknowledge the paucity of empirical evidence for the effectiveness of its treatments. Many people living with chronic fatigue syndrome find that exercise therapy makes them suffer more, while the aim of cognitive behaviour therapy is to persuade CFS patients that the evidence from their experience of their own bodily sensations is incorrect. Increased suffering following BPS therapeutic recommendation suggests that the treatment and its rationale could both be

incorrect. Furthermore, denying patients the validation of their own experiences and their interpretation is hermeneutically unjust, particularly so when there is insufficient evidence to show that their experiences and interpretations are incorrect.

My fifth recommendation for amelioration uses the lens of *epistemic virtue* and its corollary *epistemic vice* to critique what I consider to be problematic rejections of oppositional models—the biomedical model and the biopsychosocial model (and their associated treatment recommendations). Epistemic virtue is a theory of knowledge that is based on the evaluation of, “... the cognitive dispositions of agents, especially their good cognitive dispositions” (Battaly 2017, p. 223). Epistemic virtues include, “... open-mindedness, intellectual courage, intellectual integrity, and epistemic responsibility ...” (Choo 2016, p. 91), “... open-mindedness ...” (Battaly, 2017, p. 223), “... fair-mindedness, open-mindedness, intellectual carefulness, intellectual courage ...” (Baehr 2006, p. 193). Intellectual epistemic “... vices include gullibility, dogmatism, prejudice, closed-mindedness, and negligence ... intellectual character vices are intellectual character traits that impede effective and responsible inquiry” (Cassam 2016, p. 159), closed-mindedness (Battaly 2017 p. 224). In contrast to Baehr’s (2006), Battaly’s (2017), Rubarth’s (2005), and Choo’s (2016) approaches, when I attribute epistemic virtue or vice I choose to appraise epistemic agents’ assumptions, their use of evidence, and their arguments. I make this choice because people’s cognitive dispositions can be best perceived through their epistemic actions. Reza Lahroodi and Choo articulate the *social dimensions* of epistemic virtues; they allow epistemic virtues to be possessed by collectives, where the collective can be an organisation (Choo 2016), or a less formalised group (Lahroodi 2007):

Character epistemology teaches that the cognitive character traits of individuals are proper subjects of epistemological inquiry. Collective epistemology, on the other hand, teaches that collectives can be genuine cognizers like individuals. Combined together, they suggest the possibility that some collectives too may have cognitive character traits and be worthy of epistemic assessment as virtuous or vicious (Lahroodi 2007, p. 282).

I follow Lahroodi (2007), in the sense that it is possible to assess the epistemological traits of collectives such as those who support one or other of the biomedical model or the biopsychosocial model for chronic fatigue syndrome.

The amelioration of disputes concerning CFS would benefit from critiquing some people's rejection of others' disease models for chronic fatigue syndrome and its treatments. My view is that the rejection of an opponent's (reasonable) view of CFS is not being open-minded (Cassam 2016; Choo 2016; Battaly, 2017; Baehr 2006), so can be classified as being epistemically vicious. Additionally, the rejection of the value of research into biomedical *or* non-biomedical causes of CFS symptoms is contrary to the recommendations of, for instance, Charles Sanders Peirce, who exhorts that we, "[d]o not block the way of inquiry!" (as reported by Haak 2014, p. 319 and Burch 2024, p. 4.) [exclamation mark original to Peirce].

My sixth recommendation is for tolerance of '*epistemic situationism*', the understanding that people can be epistemically inconsistent. Empirical evidence shows that many people have differing epistemic enquiry habits in different situations: "Philosophical situationism's fundamental empirical claim is that most people do not have any cross-situationally consistent behavioral dispositions" (Sreenivasan 2013, p. 292). Hence people can appear to

be epistemically virtuous in some situations, while in other situations they may seem to be exhibiting intellectual character traits that impede effective and responsible inquiry (Cassam 2016, p. 159). Because of the prevalence of epistemic situationism, people on each side of the dichotomies relating to CFS would benefit by being tolerant of those others who have used different epistemic methods to come to a conclusion concerning the nature of CFS. Many CFS patients use evidence from their own experience for understanding their illness, while for other medical conditions they may well prefer to use objective scientific evidence. Conversely, in some situations a healthcare professional may, perfectly reasonably, prioritise her own phenomenological experience over objective evidence, for instance if the HCP has migraine, a condition often without objective evidence.

I conclude this section by reiterating that compromises in regard to the support for postulated mechanisms can lead to shared conceptualisations (which I will describe in Sections Six and Seven of this chapter). Shared conceptualisations may lead to agreed compromised treatment recommendations.

I can identify four counters to my recommendation of coming to mutual understandings. Firstly, it may be that either side in the dispute about CFS may not agree with my suggestion that their opponent's stance is reasonable: those on each side of the dispute have already been exposed to their opponents' perspective and have not changed their own perspectives. Secondly, the appreciation that one's opponents are taking a probably reasonable stance does not force one to change one's own stance: each side can remain just as rigidly in opposition to each other; so, shared understanding may well not result in amelioration. Thirdly, it may be counter-claimed (against my arguments) that some people's opposition to either the biomedical model or the biopsychosocial model of CFS is not epistemically vicious, because

either BMM or BPS is incorrect, making rejection appropriate. My response to that counter-claim is that those people who assert that the opposing conceptualisation is incorrect are not taking into account the many uncertainties regarding CFS. Failing to appraise all the available evidence (including evidence for uncertainty) is epistemically vicious. The fourth counter to my recommendation of mutual understanding is that this is not a dispute of equals: healthcare professionals have more power.

7.4: Standard Disease Models

A possible route to amelioration of epistemic injustice in chronic fatigue syndrome healthcare encounters is for patients and their healthcare professional to see the value of re-conceptualisations of the mechanisms of CFS. I describe some re-conceptualisations in Sections Five, Six, and Seven of this chapter. Prior to that, in this section, I describe the common cause model that is applied to many other medical conditions, and its hidden variable variant. I show how much biomedical research into CFS looks for a common cause, or assumes a hidden variable.

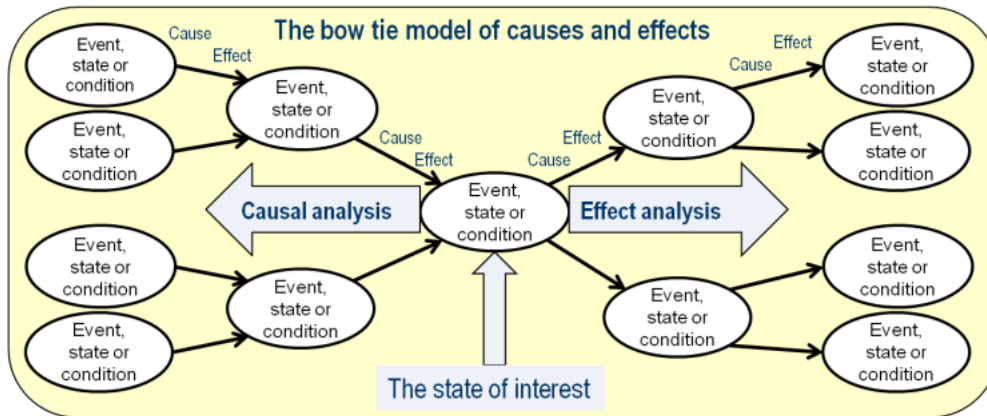


Figure Two: The Bow Tie Model of Causes and Effects
 (from <https://uk.linkedin.com/in/grahamberrisford>)

Figure Two shows the theoretical mode described by the systems analyst Graham Berrisford, which he refers to as the ‘bow tie model of causes and effects’ (my Figure 2), and is his version of the common cause model that I described in Chapter Three, Section Two, Subsection (h). Central to the bow tie model is the ‘event state or condition’ which is the ‘state of interest’. Effect analysis (on the right of the diagram) shows that, according to this model, this central (or what I choose to call the ‘parent’) *state of interest* causes effects on some (in this diagram, two) ‘daughter’ event *states or conditions*, which themselves can cause effects in ‘granddaughter’ event *states and conditions*. In the case of disease, I interpret this bow tie model as indicating the parent to be the underlying cause, the daughters to be the resulting pathological processes, and the granddaughters to be the manifest symptoms and course of the disease. The right hand part of the bow tie diagram can be likened to a branching tree, where the effects do not have causative interactions *with each other*, and where the root is the common cause of the disease.

This bow tie model can be applied to, say, myxoedema; the central state of interest is the failure of the thyroid gland to produce enough thyroxine. This is the *common cause*. All

people who have this common cause have myxoedema; all those without this common cause do not have myxoedema; all those who have the diagnosis of myxoedema have this common cause; all those who don't have this common cause do not have myxoedema. (Although, there may well be some marginal case regarding diagnosis in a small minority of people, as in many medical conditions.) Insufficiency of thyroxine produces various daughter physiological abnormalities, such as lowered serum thyroid level, raised thyroid stimulating hormone level, which are not themselves symptoms, but which act as biomarkers. These physiological changes produce the granddaughter symptoms of myxoedema, such as weight gain, reduced appetite, physical and mental slowness, tiredness, skin thickening, facial changes, and intolerance to cold.

The deficiency of thyroid hormone is the 'state of interest' in myxoedema, central to understanding, investigating, and treating this condition. That deficiency defines myxoedema, unifies myxoedema as one condition, demarcates myxoedema from other conditions that cause tiredness and the other symptoms, brings about the biochemical changes that are recognised as biomarkers, and leads to medication and management recommendations.

For an individual myxoedema patient, causal analysis (as illustrated on the left side of the bow tie model – my Figure 2), highlights the recognition that there are a number of known causes of myxoedema, such as deficiency of dietary iodine, surgical excision of the thyroid, or the effects of previous radiotherapy, which are all likely to be obvious in the patient's history, or auto-immunity (as demonstrated by investigations), any of which causes all have the effect of leading to the common cause of myxoedema.

A hidden variable is a hypothesized common cause, which is surmised but (as yet) unidentified. Multiple sclerosis and Parkinson's disease are examples of diseases that are assumed to have hidden variables, but where a unifying common cause has not yet been adequately identified. These two medical conditions are each fairly well demarcated and unified by their symptomatology and by their biomarkers (demyelination patches in magnetic resonance imaging of central nervous system in multiple sclerosis, degeneration in the substantia nigra in Parkinson's). These shared features suggest that all those with the diagnosis of multiple sclerosis are likely to share some underlying cause, although it is as yet hidden. A similar situation applies to Parkinson's disease. In the future, medical research may enable a that a common cause for each condition to be identified.

The hiddenness of a variable may be temporary; medical conditions that were hypothesised to have a hidden variable later became recognised as having a common cause. For instance, in the nineteenth century, John Snow hypothesised that Broad Street pump water was contaminated with 'cholera poison'; independently, Ignaz Semmelweis hypothesised that puerperal fever was due to contamination by what he called 'cadaveric particles or material' (Russo & Williamson 2007). However, neither of these doctors was in a position to identify the nature of these hypothesised causes, that is to say, the causative variables were hidden from them. The symptoms and prognosis shared by cholera patients suggested to Snow that these patients shared a cause for their disease; similarly, the symptoms and prognosis shared by puerperal fever patients suggested to Semmelweis that these patients shared a common cause for their disease. The success of Snow's and Semmelweis's actions to prevent these diseases implied they were correct, even though the common causes were hidden. (Some decades later, medical research enabled the common cause of cholera to be identified as

infection with *Vibrio cholerae*, and the common cause of puerperal fever was identified as Streptococcal infection.)

The hidden variable for CFS is assumed by many researchers and patient advocates to be discoverable. There are many hypothesised candidates; it may be a virus, an auto-immune process, some other malfunction of the immune system, a disruption to the gastro-intestinal tract biome, a malfunctioning of the mitochondria (the intracellular organelles that supply energy), some inflammatory process in the central nervous system, or some other abnormal biomedical dysfunction (the daughters in the bow tie causation model). These pathological processes are thought to cause the symptoms and course of CFS (the bow tie model's 'granddaughters') in all those who have the diagnosis of CFS. (There is some evidence for some pathological processes in some CFS patients (Muller *et al.* 2020).) The hidden variable model posits that one of these physiological abnormalities—or some other as yet unknown pathological process—it is the cause of all the other pathological processes.

The left half, the 'causal analysis' part, of the bow tie model (my Figure 2) reminds us that many medical conditions are thought to have more than one cause, sometimes acting in combination. For a healthy person to become ill with any particular medical condition, it may be that two or more different factors combine together to precipitate that disease. The continuing uncertainties about CFS sanction speculations about its causes. In the case of CFS, one can speculate that not all patients share the same precipitating factors. It may even be that there are no factors that are shared by all CFS patients, with the speculation being that CFS is merely the final common pathway of differing processes.

Given an analogy with the later progress of identifying common causes for cholera and puerperal fever, it seems reasonable to search for the identification of a common cause for chronic fatigue syndrome. However, I suggest that there is not enough evidence even to hypothesise the existence of a hidden variable for CFS, because the lack of demarcation and homogeneity, the lack of a unique symptom, and the lack of a biomarker all give reduced confidence that all those with the symptoms of CFS share the same causative pathological processes.

7.5: The Network of Symptoms Model

I next introduce the hypothesised network causal structure of disease, wherein some medical conditions are postulated to consist of a network of interacting symptoms. The network model is applied by some researchers to mental disorders (Borsboom 2017; Hevey *et al.* 2013; Nuijten *et al.* 2016; Borsboom, 2017, p. 6; Blanken *et al.* 2019; Robinaugh *et al.* 2020). Many of these references include Denny Borsboom as an author, or cite the work of Borsboom and his colleagues, so for the sake of simplicity I sometimes refer here to ‘Borsboom’s network model’. Later in this section I speculate that this model may help towards understanding and managing chronic fatigue syndrome, even though CFS is not considered to be a mental disorder.

The concept of interactions between symptoms falls outside the scope of the bow tie model, and are not consistent with it, since this the bow-tie model assumes that the various causes are independent of each other, and do not cause each other.

The psychiatrist and psychology theoretician Danny Borsboom and his colleagues postulate a hypothecated network causal structure of diseases (Borsboom 2017). They and other researchers apply this model as a theoretical explanatory model for mental disorders (Borsboom 2017; Hevey *et al.* 2013; Nuijten *et al.* 2016; Borsboom, 2017, p. 6; Blanken *et al.* 2019; Robinaugh *et al.* 2020), as an alternative to the assumed common cause model that is analogous to the conceptualisations of many non-psychiatric medical conditions. Borsboom and colleagues' concept of mental disorders is that the symptoms *cause each other*:

[I]nstead of being *effects of a common cause*, psychiatric symptoms have been argued to *cause each other*' Borsboom, 2017, p. 5) [Italics in original].

According to Borsboom, the causal interactions between symptoms in mental disorders, "... can be understood as a network, in which symptoms are *nodes* and causal interactions between symptoms are *connections between nodes*" (Borsboom, 2017, p. 6) [Italics in original]. Borsboom suggests the nature of these causal interactions:

At the heart of the theory lies the notion that symptoms of psychopathology are causally connected through myriads of biological, psychological and societal mechanisms. If these causal relations are sufficiently strong, symptoms can generate a level of feedback that renders them self-sustaining (Borsboom, 2017, p. 5).

Borsboom calls this conceptualization *the network approach to psychopathology*' (Borsboom 2017, p. 6) He gives a diagrammatic representation of this concept:

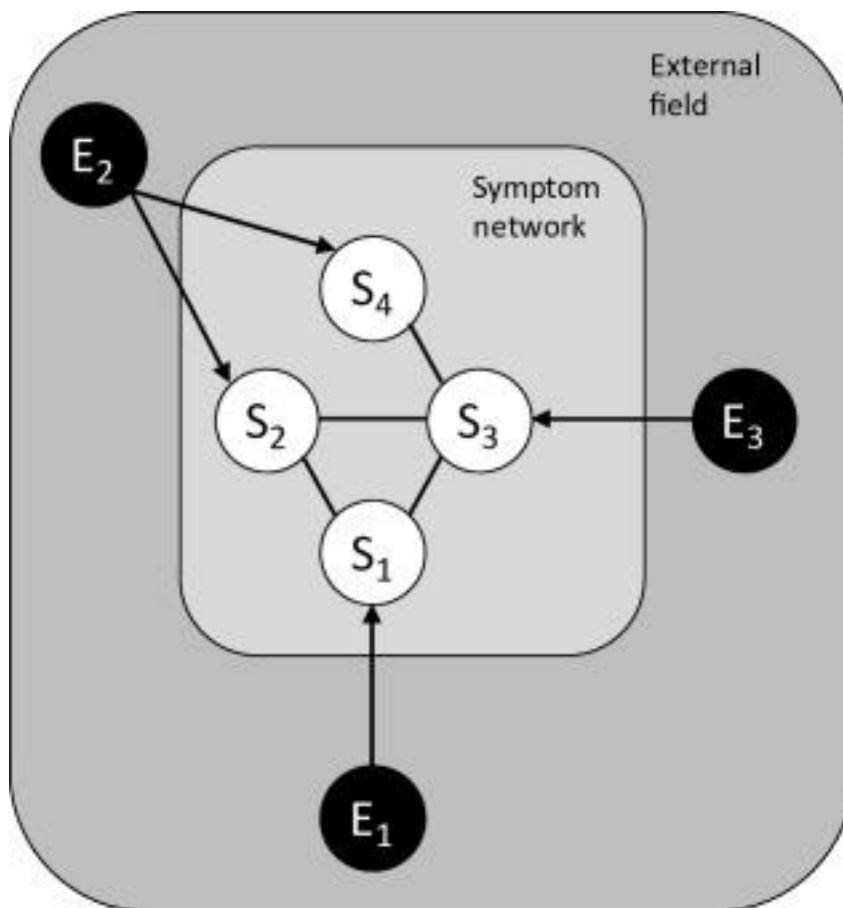


Figure Three A symptom network of four symptoms (from Borsboom 2017, Figure 1, p. 6).

(S_1 - S_4). If two symptoms have the tendency to activate each other, they are connected by a line (e.g., S_1 - S_2). Symptoms that are not directly connected to each other (e.g., S_1 - S_4) can still synchronize if they share a common neighbor in the network (e.g., S_3). External factors that affect the network (e.g., adverse life events) are represented in the external field. These may be symptom-specific (E_1 , E_3) or shared across symptoms (E_2) (Borsboom, 2017, Figure 1, p. 6).

My Figure Three is Borsboom and colleagues' hypothesized and simplified model of a psychological disorder. According to this diagram, a mental condition may be precipitated by some unknown or unspecified external event(s), E_1 , E_2 , and/or E_3 , which

cause[s] the symptoms (S₁, S₂, S₃, or S₄) to develop, following which these and other symptoms interact causally with each other to perpetuate the disorder (Borsboom 2017, p. 6). The precipitating event are merely triggers; they subsequently may not take part in the disease mechanism or in the causation of the symptoms.

Borsboom and colleagues tell us that this model (my Figure Three) shows a dynamic bidirectionally interactive network. Borsboom and colleagues postulate that (unlike the known mechanisms of many non-psychiatric conditions, for instance, my example of myxoedema) mental symptoms can reciprocally cause each other, with those causal connections being physical, psychological, and/or social. As Borsboom and his colleagues write,

[N]etwork models being applied to psychological data sets [demonstrate the] emergent behavior of complex, dynamical systems in which psychological, biological, and sociological components directly affect each other (Eskamp *et al.* 2018, p. 453).

Feedback loops between any two symptoms that are connected by an edge can cause them to exacerbate and sustain each other; similarly, feedback loops between a number of symptoms can exacerbate and sustain groups of symptoms, in a cycle. (Additionally, in the recovery phase of the illness, these symptoms can interact to improve each other.) The postulated network is complex, with many nodes and many edges, and it is dynamic, all of which features accommodate the variability of symptoms in some mental conditions over time, and the differing patterns of symptoms between patients.

Borsboom and colleagues' understanding of their network of symptoms is that it can be stable, giving symptoms that remain relatively unchanged over time, which would give a pattern that enables the allocation of a label in the form of a diagnosis. Borsboom tells us that specific patterns of symptomatology shared between different people can be labelled as a particular psychological disorder, such as,

... depression, anxiety disorders, post-traumatic stress, complex bereavement, autism, psychotic disorders, substance abuse, the general structure of psychiatric symptomatology, diagnostic manuals themselves, health-related quality of life, and personality traits (Borsboom, 2017, p. 6).

Here, Borsboom suggests wide applicability for the network of symptoms model. The identification of a specific individual's pattern of symptom-symptom interactions (Borsboom, 2017, p. 10) could lead to treatment strategies for that individual: “[T]he network theory has direct implications for how to understand diagnosis and treatment” (Borsboom, 2017, p. 5), where, if the network model is correct, treatment can be individualised to any particular patient's ostensive network of symptoms.

However, these mental diagnoses are considered by Borsboom and colleagues to be, “... a range of constructs ...” (Borsboom, 2017, p. 6), rather than specific distinct diseases. The network model postulates that some mental disorders may well not have a central disease mechanism (Borsboom, 2017, p. 5); they may not be associated with an identifiable common cause or latent variable:

... we do not need underlying latent disorders to explain psychological symptoms: the disorder is nothing more – but also nothing less – than a causal network of observable symptoms (Nuijten *et al.* 2021, p. 74).

So, Nuijten *et al.* tell us here that, unlike some better-understood medical conditions (such as myxoedema), it may not be necessary, or worthwhile, in some medical conditions to search for any structure apart from the network, because the disorder is a network disorder.

Nuijten and colleagues' (2016) and Borsboom's (2017) theoretical network of symptoms model is similar in structure and function to a theoretical state space model (Bradley & Drechsler 2014), which I described in Chapter Two, Section Two; but, the state space model is expected to be much more fully populated, the connections may be quantified, and therefore the state space model can give more robust predictions.

Borsboom's Figure 1 (my Figure Three) also has some similarity in appearance with a Bayesian causal network, in that there are nodes—the circles representing symptoms— and edges—the lines connecting the symptoms. However, there are major differences between Borsboom's network approach and Bayesian networks. The first is that the connections between symptoms in Borsboom's scheme are posited to be *bidirectional*, rather than unidirectional as in a Bayesian network. This means that, rather than being acyclic as in a Bayesian network, the network of symptoms approach can accommodate *cyclic causation*; that is to say, two symptoms, or groups of symptoms, can exacerbate (or improve) each other, in a feedback loop. Secondly, Borsboom's network is *dynamic*, changing over time (which may not necessarily be a feature of a Bayesian network); Borsboom's network of symptoms model accommodates our knowledge that the severity of psychological symptoms and the

strength of their connections change over time, leading to the recognised changes in the course of symptomatology in individuals.

Borsboom and colleagues restrict the application of his network theory to psychiatric conditions. In contrast to that restriction, I now explore its value as a model in conditions that are usually classified as being non-psychiatric. As a demonstration of the value of the network of symptoms model, I show its application to a condition that is not classified as a mental disorder, temporary insomnia. I define temporary insomnia to be inadequate length of sleep for less than a week, which is an experience common to all, and usually does not prompt the seeking of medical help. We have all experience of this, often precipitated by an understandable external cause such as work or social stress, or jet lag. After few days, we find that we have returned to our normal sleep pattern.

Cha *et al.* (2022) give a dynamic interactive network diagram for insomnia:

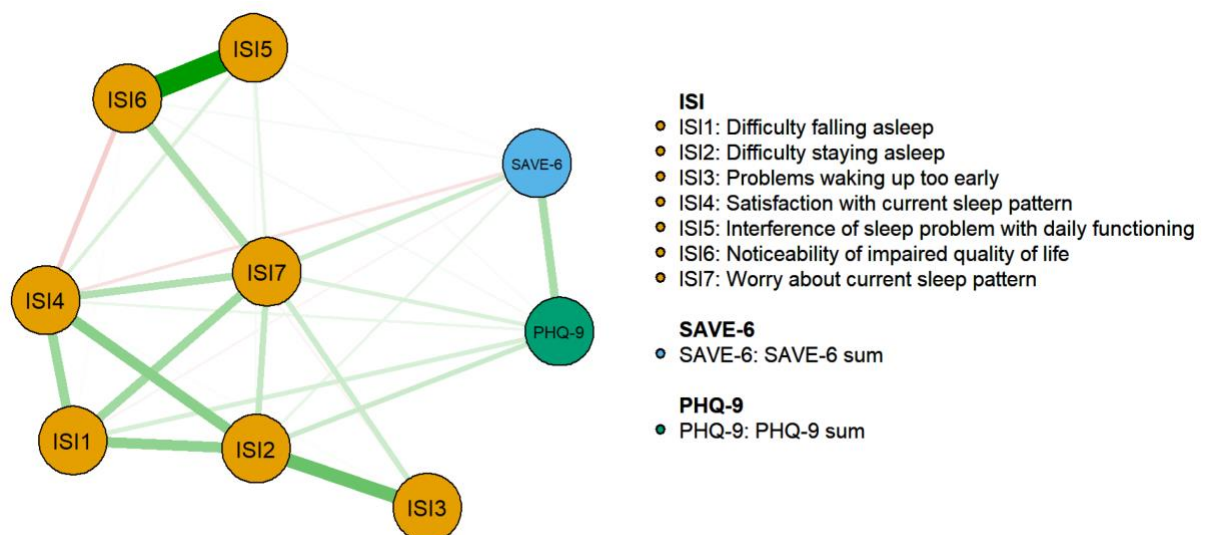


Figure Four: A dynamic interactive network diagram for insomnia (from Cha *et al.*, 2022, p. 5)

In my Figure Four, Cha *et al.* label each node with an ‘ISI’ code, referring to ‘insomnia severity index’, scored according to a self-reported questionnaire (Cha *et al.*, p. 3). ISIs 1 to 3 are the different classifications of sleep disturbance, that is to say, difficulty falling asleep, difficulty staying asleep, and waking too early. ISIs 5 and 6 refer to the daytime effects of poor sleep, that is to say, problems with daily functioning and impaired quality of life. ISIs 4 and 7 refer to cognition relating to the present sleep pattern, [dis]satisfaction with current sleep pattern and worry about current sleep pattern. All these nodes are connected by edges to two or more of the others.

Cha and colleagues, “... used network analysis to investigate interactions between insomnia symptoms... It conceptualizes a disorder as a network of interacting symptoms that reinforce each other in varying degrees” (*ibid.*, 2022, p. 1). Strongly connected nodes are placed closer together in the diagram. The thickness of the lines reflects two things, the empirical findings of co-occurrence of the symptoms represented by the two nodes, and the claim that connected nodes affect each other. I take this description of the network to be incorporating the postulation of two-way dynamic causal links between each node.

According to this diagram, the three types of sleep disturbance, difficulty falling asleep, difficulty staying asleep, and early waking are strongly linked. Together they are linked to reduced satisfaction and increased worry about the current sleep pattern, and indirectly linked to poor daytime functioning and quality of life. The edges in Cha *et al.*’s diagram are intended to represent two-way interaction and causation, so that poor sleep pattern and dissatisfaction with sleep have causative effects on each other; similarly, daytime problems because of poor sleep and dissatisfaction with sleep have similar causative effects on each

other. The other important feature of these links between insomnia is that they are dynamic, changing over time.

Our understanding of ordinary temporary insomnia, using Cha *et al.*'s analysis, is that the insomnia episode is initiated by some disturbance, such as jet lag causing an unsatisfactory sleep pattern, or some daytime problem causing worry that interferes with sleep at night; the interaction between the nodes leads to a short episode where poor sleep pattern, poor daytime functioning, and worry about sleep are present for a few days. However, our experience is that usually these problems reduce, and the sleep pattern returns to our normal. This experience is accommodated in the characteristic of the diagram that allows the causation in the links to be positive or negative, that is to say, each symptom can make its neighbours worse or better, and this effect of better or worse can change over time. Temporary sleep disturbances can then improve within a few days.

Cha *et al.* are also interested in the connection of poor sleep with anxiety and depression, represented in their diagram with SAVE-6 and PHQ-9, which refer to patient-completed questionnaires regarding anxiety and depression. Their finding is that sleep disturbance that does not improve is associated with anxiety and/or depression. I don't use that part of the diagram here, because quotidian sleep disturbance that I am using as an example is not usually associated with medically diagnosable anxiety or depression. Borsboom's network theory suggests a mechanism for the fortunate usual failure of sleep disturbance to progress to clinical depression or anxiety. Borsboom uses the concept of 'symptom thresholds': symptoms have to be of a certain severity before they can have a causal effect along certain connections (Borsboom 2017, p. 11). In this case the threshold refers to those nodes and edges that could lead to longer-lasting psychological disturbances, anxiety and/or depression.

This dynamic two-way interactive network of symptoms model seems plausible in relation to temporary insomnia, and seems to fit with our own experience. If our sleep problem is temporary, we are likely to suffer some of the symptoms as highlighted by Cha *et al.*, and then, within a few days, unless we are unfortunate, we recover. The practical use of the network depends on its demonstration of the centrality of some symptoms. In the case of Cha *et al.*'s model, the main centrality is in the symptom ISI 7 (Cha *et al.*, 2017, p. 7), which they describe as our worry about our current sleep pattern. The centrality of this one symptom gives us a guide to where our self-treatment should be directed. If we manage to convince ourselves that our present episode of insomnia is likely to be temporary, then this worry and all the other symptoms soon reduce, and our sleep pattern soon returns to normal. As Cha *et al.* say, "... a symptom with high closeness may become an intervention target, as reducing the symptom would likely lead to reductions in most other symptoms within the network" (*ibid.*, p. 2). By contrast, if worry about the current unsatisfactory sleep pattern does not diminish, then the other symptoms also are likely not to diminish, and the insomnia is more likely to continue.

The network of symptoms theory thus seems to have value in a condition such as temporary insomnia, a condition that is not considered to be psychiatric, so the network theory may also have similar value when applied to another condition that is classified as non-psychiatric for instance, CFS/ME.

7.6: Applying Network Models to Chronic Fatigue Syndrome

I have shown the use of the concepts of network theory in mental disorders and in temporary insomnia. CFS/ME is a more complex condition than temporary insomnia, with many more symptoms, and these symptoms could well be interacting; so, a CFS network model would be more complex than the temporary insomnia network model. (It may also be that there are some, as yet inadequately understood, connections between physiological or biochemical pathological processes in CFS and the symptoms and course of CFS, but here I concentrate on ostensive symptoms.) In this section, I first describe a non-dynamic associational model for CFS that eschews causal claims. Despite this eschewing, my view is the empirical associations shown in a non-dynamic non-causal model could be used to populate a dynamic and causal network model.

Kujawski *et al.* apply a network concept to the symptoms of chronic fatigue syndrome (Kujawski *et al.*, 2021), using empirical data; however, in contrast to Borsboom (2017) and Nuijten *et al.* (2021), Kujawski *et al.*'s network illustrates only associations between symptoms and does not include causal claims. In Kujawski *et al.*'s network diagram the connections between symptoms are non-directional and not assumed to be causal:

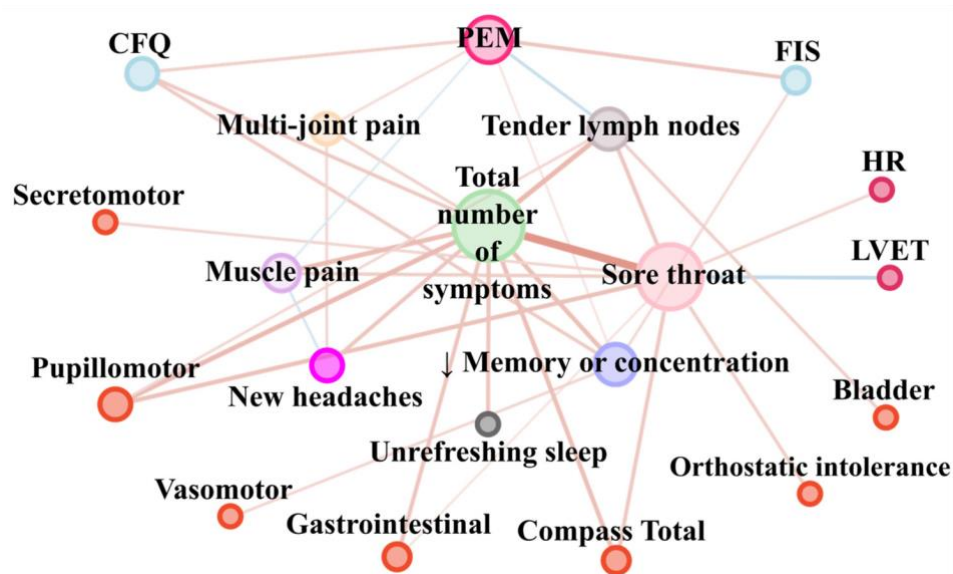


Figure Five: Diagram of the CFS association model (Kujawski *et al.*, 2021, p. 6)

In Figure Five, CFQ is chronic fatigue scale, PEM is post-exertional malaise, FIS is fatigue impact scale, HR is heart rate, LVET is left ventricular ejection time, Compass total refers to autonomic symptoms such as postural hypotension. Edge width and intensity of colour denote strength of relationship. Blue edges indicate a negative relationship.

Kujawski *et al.*'s analysis shows that, "[t]hree of the most densely connected nodes were the total number of symptoms, sore throat and PEM [post exertional malaise]" (*ibid.*, 2021, p. 9). Despite the absence of causal claims in their article, Kujawski *et al.* see the value of their research to future research: "Future investigations may employ this analytic approach in identifying correlations between symptom occurrence with pathophysiological findings" (*ibid.*, 2021, p. 9).

I speculate that a 'dynamic network of symptoms model', based on the work of Nuijten *et al.* (2016) and Borsboom (2017), may be valuable for the understanding the complexity of a

non-psychiatric medical condition such as chronic fatigue syndrome. If Borsboom's model were to be applied to CFS/ME, the nodes would refer to patients' symptoms. One could speculate that many of the symptoms of CFS/ME, such as poor sleep, daytime tiredness, reduced energy, physical deconditioning, muscle aches after exertion, poor memory and concentration, low mood, etc., are interacting with other. These interactions are likely to be exacerbating and perpetuating each other; so, conversely, improvement in one could lead to improvements in those symptoms to which it is connected, according to a model such as that of my Figure Five (Kujawski *et al.*, 2021, p. 6). Consequently, in an individual patient, if a therapeutic intervention can be successful in improving of one or more of these symptoms, then that intervention could well give improvement in many of the other symptoms, including those symptoms that are indirectly connected, through other symptoms.

However, the possibility of a 'hysteresis' (a delayed effect) (as hypothesised by Nuijten *et al.* (2016) for psychopathological symptoms) in CFS patients would mean that an effect may not be detectable during the same time period as its original cause, so an investigation at a particular time may not detect both the cause and the effect, further hindering understanding that they are connected. This significant area of uncertainty gives us an extra explanation for how decades of research has failed to find a cause, a biomarker, or effective treatments for CFS/ME.

Medical researchers do find pathological findings in some CFS patients, to which I alluded in Chapter Three, Section Three, Subsection (j). Areas of research based on these abnormal findings have not led to adequate progress in understating of or treatments for CFS. It may be that one or more of these specific abnormal pathological process may explain the symptoms and course of CFS. However, these processes are disparate, and are not integrated

It may be that these abnormal pathological findings indicate pathological processes that are linked in some way to each other, and to patients' symptoms in a dynamic causally interactive network, such as that of Barabási *et al.* (2011), which they apply to the pathological processes of non-psychiatric disease.

Borsboom and colleagues' network theory of symptoms discounts the necessity for hypothesising the existence of a latent variable, that is to say, an (as yet unknown) common cause. However, this discounting may not be appropriate in chronic fatigue syndrome, as there is an absence of evidence that CFS does *not* have a common cause. Amelioration of the disputes concerning the nature CFS and its research projects may be helped by not rejecting the possibility of a common cause, as many CFS patients and their advocates seem to assume that there *is* a common cause for CFS. A hybrid latent variable network model for CFS therefore seems to be a less oppositional conceptualisation than a model without a latent variable for explaining the symptoms and course of CFS.

Bringmann and Eronen's (2018) *hybrid* network model is a development of Borsboom's network of symptoms model that incorporates the possibility of the presence of a '*latent variable*', that is the primary cause of the other pathological processes and the symptoms, along with a dynamic causative network of symptoms. They apply this model to psychopathology. However, finding a latent variable may not be straightforward because relevant nodes can also be latent: "In such networks, the nodes are not only (only) observed variables, but latent constructs" (Bringmann & Eronen 2018, p. 610). My understanding of Bringmann and Eronen here is that networks can contain a number of relevant but undetected *latent nodes* as well as observable nodes. Linderman and Adams (2014) similarly argue that, "... in many systems it is difficult or impossible to measure the network directly" (Linderman

& Adams 2014, Abstract). My understanding of Linderman and Adams here is that networks can contain a number of relevant *latent edges* and nodes as well as observable nodes and edges. These latent nodes and edges could have causal connections to other nodes in a similar way to observed nodes, but are not necessarily the common underlying causes of all the other symptoms and pathological results. The existence, and the quantification, of latent nodes and latent edges in CFS are at present un-surmisable: they are the ‘unknown unknowns’ of Djulbegovic *et al.* (2011, p. 301) that I described in Chapter Two, Section Two. If there are significantly functional latent nodes and latent connections in CFS pathological processes, then lack of knowledge of these latent features could well hinder medical research on CFS; researchers would not know where to look for these features, let alone know how to measure them or their effects. I now supply a diagram that illustrates this complex model with latent features.



Figure Six: PowerPoint stock diagram

My Figure Six is a stock diagram from PowerPoint, meant merely to be background of a slide. As it happens, I feel that it can also be taken as a representation of the hybrid model of a complex medical condition that is subject to latent relevant features, such as CFS/ME. This diagram consisting of lines and points could represent a three dimensional structure that could be taken to represent the nodes and edges of a network model. The lines and points that are in focus could be the manifest symptoms of CFS/ME, connected causally to each other; these manifest symptoms are also connected causally to the lines and points that we can see are out of focus, which represent as the as-yet-not-well-enough-understood pathological processes that are causing the symptoms. What we can't see in this the diagram, are the lines and nodes that are out of sight, where we postulate the still unknown underlying latent causes and connections exist. These underlying causes may be basically infective, inflammatory, immunological, auto-immune, mitochondrial, gastrointestinal, circulatory, some other abnormal process, or some combination of these. At this stage of knowledge about CFS, surmising about these causes and their connections may not be reliable. What is not made clear in this this simple diagram is the nature of the edges, which in the network theory of disease causation are two-way causal, with the manifest symptoms having the propensity to influence other nodes, either positively or negatively, and upwards and downwards, thereby having the property of causally affecting the underlying pathological processes. In addition, I emphasise that the network model is dynamic, changing over time, and maybe subject to 'hysteresis' (meaning a delayed effect – perhaps delayed for decades), as in Borsboom's model (2017, p. 8).

The network of symptoms model (and its hybrid version) accommodates the existence of wide variations in symptoms between patients, and variation of symptoms in individual patients over time, so these models do not *expect* precision about disease definition or

diagnostic criteria; furthermore, network models seem to predict that research into disease definition and diagnostic criteria may well be unsuccessful because patients' patterns of symptoms are likely to be disparate, and patterns for individual patients are likely to change over time. Therefore, these models give explanations for the lack of adequate progress of research into definition and diagnostic criteria, and consequently give explanations for other failures of research into CFS.

However, in the present absence of effective treatments for CFS, the network of symptoms models could guide treatment recommendations of an *individual* with CFS/ME, by identifying those symptoms at person that seem most prominent and seem have the most connections to other symptoms, that is to say, have centrality.

7.7: De-emphasising Disease Mechanisms.

In this section, I show the value of an alternative approach to uncertainties about CFS, which is to de-emphasise disease mechanisms:

Persistent physical symptoms (synonymous with persistent somatic symptoms) is an umbrella term for distressing somatic complaints that last several months or more, regardless of their cause. These symptoms are associated with substantial disability and represent a major burden for patients, health-care professionals, and society.

Persistent physical symptoms can follow infections, injuries, medical diseases, stressful life events, or arise *de novo*. As symptoms persist, their link to clearly identifiable pathophysiology often weakens, making diagnosis and treatment

challenging. Multiple biological and psychosocial risk factors and mechanisms contribute to the persistence of somatic symptoms, including persistent inflammation; epigenetic profiles; immune, metabolic and microbiome dysregulation; early adverse life experiences; depression; illness-related anxiety; dysfunctional symptom expectations; symptom focusing; symptom learning; and avoidance behaviours, with many factors being common across symptoms and diagnoses. (Löwe *et al* 2025, p. 2649)

In this passage, the psychiatrists and psychologists Bernd Löwe and his colleagues see the value of an alternative term for the continuation of symptoms in some medical conditions, that they call ‘persistent physical symptoms’ (PPS). This term de-emphasises disease mechanisms (because it may not be possible to identify those mechanisms); PPS is merely a description of the patient’s experience. These researchers give the examples of, “... fatigue in multiple sclerosis or chronic kidney disease ... diarrhoea in irritable bowel syndrome ... [and] headache in major depressive disorder” (Löwe *et al* 2025, p. 2650). In all these conditions the pathological mechanisms for these particular persisting symptoms have not yet been identified, even though these symptoms are common, unpleasant, and burdensome. Other patients with less identifiable disorders may have other symptoms, such as, “... fatigue, insomnia, sleep problems, loss of energy, concentration difficulties, impairment of memory, headache, and pruritus” (Löwe *et al.* 2025, p. 2650). In these circumstances there may or may not be an explanation for, for instance, the fatigue; there may or may not have been an identifiable precipitating cause for the present symptoms. However, the symptoms persist regardless of cause(s), and regardless whether a cause still present, or whether the cause is completely or only partially explained, or is even unexplained. Even if the precipitating cause is known, that knowledge becomes less relevant over time:

[T]he association between persistent symptoms and their original biomedical cause typically becomes weaker as the condition persists or progresses ... (Löwe *et al.* 2025, p. 2649).

Here Löwe *et al.* are de-emphasising the value of finding a continuing underlying cause, as the association between original cause and present symptoms may well have become weak, perhaps making the original or precipitating cause now undetectable. Using the interpretation of Löwe *et al.*, uncertainty about the precipitating cause in any particular patient becomes less problematic.

The division into somatic diseases, functional somatic disorders, and mental disorders is artificial, given the many overlaps between diseases, and promotes a misleading mind–body dualism (Löwe *et al.* 2025, p. 2651).

In this context disease categories and classification of disease that rely on cause are less helpful. Löwe *et al.* (2025) are using what they call a ‘transdiagnostic’ view, where they focus on symptoms and how to understand and manage them rather than on medical diagnoses and mechanisms of disease (which often rely on biomedical evidence, which may well be inadequate to explain the present symptoms). The persistent physical symptoms model shares with the network of symptoms model the acceptance of a wide variation in symptoms between patients, and in individual patients over time, so does not *expect* precision about disease definition, diagnostic criteria, or homogeneity. So, according to Löwe *et al.*’s interpretation, uncertainty concerning the nosological classification of an individual’s symptoms becomes less necessary and less problematic.

Continuing mechanisms for the persisting symptoms may not be discoverable:

Causal mechanisms of persistent physical symptoms appear to be complex and multifactorial, and might *vary between individuals with the same symptoms*. Neither purely biomedical nor purely psychological models are sufficient, and both represent overly simplistic and outdated dualistic views ... (Löwe *et al.* 2025, p. 2650). [My italicisation].

Here, Löwe *et al.* are de-emphasising the value of discovering the mechanisms that are perpetuating the symptoms, because the mechanisms for that perpetuation may be too complex, and anyway may well be individual to that patient. Löwe *et al.*'s interpretation implies that uncertainty about mechanisms becomes less problematic; the concept of persistent physical symptoms is agnostic about disease mechanisms. The application of PPS does not require the identification of the mechanisms for the continuing symptoms; so, in the case of chronic fatigue syndrome, PPS does not assume that, for instance, that there is a biomedical explanation, or, alternatively, that physical deconditioning (Walitt *et al.* 2024) due to the avoidance of exercise and resting too much (Vercoulen *et al.* 1998) is leading to reduced tolerance of activity (S. Butler *et al.* 1991, p. 153). PPS is therefore not in opposition to other models of disease; it is compatible with the biomedical model, the biopsychosocial model (which I described in Chapter Four), and the bow tie model, the network of symptoms model, and the hybrid latent-variable-network-of-symptoms model, all of I described earlier in this chapter. Consequently, PPS does not block or discourage using any of these models if they are used to guide the direction of research into the mechanisms of and treatments for CFS.

According to Löwe *et al.*, treatment options can be individualised, rather than being fitted to the original cause or the possible mechanisms for the perpetuation of symptoms:

Basic care consists ... using person-centred communication techniques with validation, appropriate reassurance, and biopsychosocial explanation. If basic care is insufficient, targeted psychological and pharmacological interventions can be beneficial. (Löwe *et al* 2025, p. 2649).

Löwe *et al.*'s (2025) approach to PPS could also have benefits in CFS healthcare encounters, that is to say, those healthcare encounters where the patient has symptoms that could fit with the diagnosis of chronic fatigue syndrome. Instead of specifying a diagnosis, the patient and health care professional could accept that the persistence of the patient's symptoms is under-explained. The persistent physical symptoms model does not expect or even *require* precision about disease definition, diagnostic criteria, or disease mechanisms, as routes to help with patient management recommendations. Uncertainty remains (because the mechanisms of persistent physical symptoms are acknowledged to be uncertain), but there is no impetus for there to be oppositional responses to that uncertainty. The advantages of applying the label of persistent physical symptoms in these cases is, firstly, this label facilitates a shared (and non-oppositional) understanding of the *tasks* of the encounter, which are the addressing of the symptoms and their effects. These tasks then become central to the encounter, rather than arguing for one disease model over another disease model. Secondly, the reality or not of CFS as a well-defined, demarcated, and unitary distinct entity becomes less relevant; instead, the *reality of the symptoms* and of their deleterious effects are relevant and are not doubted. Thirdly, the allocation of a disease label and its classification become

less important, thereby obviating *disputes about the name*, nature, and status of the patient's condition. Fourthly, a referral to a CFS specialist clinic does not have to depend on the primary care health professional deciding that the most appropriate diagnosis is CFS; if the label instead becomes persistent physical symptoms the healthcare professional is less likely to *delay making a 'diagnosis'* of PPS and arranging a referral, while waiting for more evidence from further tests. Fifthly, *stigma* for CFS patients is reduced, and validation can be increased, because the label 'persistent physical symptoms' can be applied to a large number of medical conditions, not just those which are classified by some as being psychosomatic (in the current meaning of that term, referring to physical symptoms that have a psychological origin). Sixthly, there may be common factors in patients with the many different examples of persistent physical symptoms, which may *guide research* into mechanisms of PPS.

7.8: Conclusion to Chapter Seven

In this chapter, I have responded in various ways to some of the problems of chronic fatigue syndrome and in CFS healthcare encounters. As is to be expected, I endorse those generic skills that others advocate for reducing epistemic injustice in healthcare encounters. I then suggested that oppositional responses to the uncertainties about CFS could benefit by using the precepts of epistemic virtue. I suggested that re-conceptualisations of chronic fatigue syndrome as being merely a network of causatively interact symptoms, or simply persistent physical symptoms, can reduce healthcare injustice. I also suggested that each of these conceptualisations can explain the continuing uncertainty regarding CFS. I promoted the value of the network of symptoms models and the persistent physical symptoms model as routes to reducing stigma concerning CFS and conflicts concerning disease mechanisms and

treatment recommendations. However, I accept that research into biomarkers, mechanisms, and treatment of CFS is hindered in the absence of knowledge of biomedical disease mechanisms, thereby permitting uncertainty about all these issues to remain.

Chapter Eight: Summary, Conclusion, and Applications

8.1: Introduction to Chapter Eight

This conclusion consists of a summary of this thesis, an underlining of concepts I have used and the values of these concepts, and my recommendations for wider applications of some of the arguments and concepts in this thesis.

I commence with my summary, where I recapitulate the arguments in this thesis concerning explanations for epistemic injustice in CFS healthcare encounters. I next reiterate my suggestions for amelioration. I summarise my responses to some possible counter-claims. I then itemise the concepts that I am promoting, and for each concept I show its value in helping to understand some of the problems of chronic fatigue syndrome and of CFS healthcare interactions, and how these concepts can promote routes to amelioration. Some of these concepts are under-emphasised elsewhere. I then summarise my critiques of others' contributions to debates concerning CFS. Finally, I suggest how the lesson of this thesis could be built on and extended.

8.2: Summary of This Thesis

In Chapter One, "Introduction, Preliminaries, and Outline", I gave my methodology and theoretical frameworks. I clarified my use of terminology in this thesis, and I gave some explanations for my choices. I then stated my caveats and limitations, and I gave some reasons for those.

In Chapter Two, “Radical Uncertainty, and Uncertainties in Healthcare”, I described and then accepted the conception of radical uncertainty as identified by some economists. I then gave examples of uncertainty in healthcare that do not fulfil the criteria for being *radically* uncertain.

I argued in Chapter Three, “Chronic Fatigue Syndrome and its Uncertainties”, that chronic fatigue syndrome is indeed subject to radical epistemic uncertainty, for which I gave the explanation that there is existence of a complex network of uncertainties regarding CFS that interact with each other and hinder the reduction of those uncertainties.

In Chapter Four, “The Biomedical and Biopsychosocial Models, and their Dichotomous Applications to CFS and Its Treatments”, I described and then critiqued the biomedical and the biopsychosocial models of disease; I then described their dichotomous applications to CFS.

In Chapter Five, “Stigma and Chronic Fatigue Syndrome and ‘Medical Disease Dualism’”, I showed evidence of, and some explanations for, stigma towards CFS and towards CFS patients, where they are blamed and stigmatised. A prominent explanation is the attribution by some healthcare professionals of the significance of psychological factors in the causation of the perpetuation of CFS symptoms, and a concomitant recommendation by these HCPs for treatments aimed at the CFS patient’s psychological processes and behavioural responses to those processes. I noted with disapproval that some people conflate the concept of a psychological attribution with a psychiatric attribution. Some people mistakenly assume that psychiatric medical conditions are less real than physical medical conditions, and mistakenly believe that psychiatric patients have some control over their symptoms, to some extent, so

psychiatric patients are blameworthy and are therefore stigmatised. I criticised the concept of ‘medical disease dualism’.

I argued in Chapter Six, “Epistemic Injustice in Healthcare, and in Chronic Fatigue Syndrome”, that epistemic injustice in some CFS healthcare encounters is partially explained by oppositional responses to those uncertainties, in those cases where the CFS patient favours the biomedical model and rejects the biopsychosocial model for CFS, while, in the same encounter, the healthcare professional favours the biopsychosocial model and rejects the biomedical model. There is similar rejection of oppositional treatment recommendations. I argued that these conflicting rejections explain epistemic injustice in CFS healthcare encounters. I have critiqued both of these oppositional responses, arguing that radical uncertainty makes support for each model inadequate, and, concomitantly, the rejection of each model is inappropriate. I then presented my central argument, that uncertainty-driven healthcare epistemic injustice can be explained by the combination of radical uncertainty, dichotomies of views, and power differentials, with prejudice and stigma also contributing.

In Chapter Seven, “Responses to the Problems of Chronic Fatigue Syndrome”, I showed how responding to some of the problems concerning chronic fatigue syndrome could reduce healthcare epistemic injustice in CFS healthcare encounters. For mitigation:

- *Firstly*, as others do, I suggested a reduction in the power differential in healthcare encounters, moving towards a partnership approach to understanding both the patient’s experience of illness and the health professional’s medical understanding;
- *Secondly*, I recommended a modification of rhetoric concerning the disputes about CFS in the public sphere as well as in healthcare encounters. (These two recommendations are, of course, not original);

- *Thirdly*, for amelioration, there needs to be a shared appreciation that the biomedical conceptualisation for CFS, and the symptom management recommendations that follow from it, are derived not unreasonably from patients' experiential perspective; and, the oppositional biopsychosocial conceptualisation and treatments are derived, also not unreasonably, from healthcare professionals' epistemological perspective (*c.f.* Pomey *et al.* 2015). Consequently, there needs to be an acceptance that each of these conflicting conceptualisations is not unreasonable. However, in the present state of uncertainty about CFS, the rejection of the opposing conceptualisation is *not reasonable*, according to the tenets of epistemic virtue: and
- *Fourthly*, as an alternative to these divergent conceptualisations, I suggested two alternative models for CFS.

The first hypothetical model that could have value when applied to chronic fatigue syndrome is the network of symptoms model of Borsboom and his colleagues (2013), which can give explanations for the persistence and variety of symptoms between patients, and the change in symptomatology over time in individual patients. This network of symptoms model is compatible with many of the attributes of the biopsychosocial model; additionally, along with allowing the possibility of a hypothetical latent variable, the network of symptoms model is compatible with a form of the biomedical model. The second hypothetical model that could be applied to CFS is the 'persistent physical symptoms' model (as advocated by Löwe *et al.* (2025)), which is already applied to some inadequately understood medical situations. The persistent physical symptoms model reflects the longer time-span of symptoms and their severity in some patients, compared to other patients with the same initiating causes. This model defuses to some extent the stigmatising distinction between some people's persistence

symptoms and disability after injury and the persistence of symptoms and disability in chronic fatigue syndrome.

I responded to a number of possible challenges to the arguments in this thesis:

- *Firstly*, in of Chapter Three, Section Six, I responded to a possible counter-claim that chronic fatigue syndrome is not necessarily subject to radical uncertainty;
- *Secondly*, in Chapter Three, Section Six, I responded to a possible counter-claim that this uncertainty is not due to a network of uncertainties;
- *Thirdly*, in Chapter Three, Section Six, I responded to a possible counter-claim that this uncertainty is contingent on the present state of knowledge, by arguing that, following hypothetical future advances in knowledge about CFS, the symptoms of some patients with CFS symptoms would be likely to remain in uncertainty;
- *Fourthly* in Chapter Five, Section Five, I responded to a counter-claim that dualistic conceptualisations and categorisation of diseases and their treatments are appropriate and helpful;
- *Fifthly*, in Chapter Six, Section Six, in Chapter Six, Section Seven, Subsection (b), and in Chapter Six, Section Eight, I responded to a possible counter-claim that CFS patients are not subject to epistemic injustice;
- *Sixthly* in Chapter Six, Section Eight, I responded to a possible counter-claim that that epistemic injustice is not explainable by oppositional responses to that radical uncertainty; and,
- *Seventhly*, in Chapter Seven, Section Three, I identified and then responded to some counters to my recommendation of coming to mutual understandings of chronic fatigue syndrome. I responded to a possible counter-claim that the rejection of an oppositional conceptualisation is *not* epistemically vicious..

8.3: Summary of the Concepts in this Thesis and The Values and Applicabilities of Those Concepts.

In order to support my arguments, I have relied on the application of a number of relevant concepts, some of which are insufficiently highlighted elsewhere in the literature. I now itemise those concepts.

In Chapters Two and Three, I showed that there is value in the inter-disciplinary application of the economists' concept of radical uncertainty and similar terms to healthcare knowledge, which application is not usual in the literature. In Chapter Two, I showed the recognition of, the features of, and some of the effects of, radical epistemic uncertainty in some areas of healthcare. In Chapter Three, I argued that knowledge about chronic fatigue syndrome exemplifies radical and deleterious uncertainty because there are many basic features of it that are underdefined or underdetermined – its definition, demarcation, homogeneity, prevalence, diagnostic criteria, objective estimates of severity, pathological processes, treatments, measurements of responses to treatments, and biomarkers for these basic features. I therefore showed that there is *radical epistemic uncertainty regarding chronic fatigue syndrome*. This attribution of radical uncertainty to chronic fatigue syndrome is underpromoted elsewhere in the literature. Uncertainty regarding a medical condition hinders the obtaining of probability estimates on mechanisms of, or treatments for, CFS. Consequently, the uncertainties about CFS are ineliminable (in the Knightian (1933, 1921) sense, where it is not appropriate to compare cases), or radically uncertain (in Kay and King's (2020) sense, where further relevant information is not available (which uncertainty may be

contingent on possible future knowledge)). I have also showed a distinction between uncertainty about a medical condition and uncertainty about a particular patient in a specific healthcare encounter. Uncertainties concerning the illness of an individual patient with symptoms that could be labelled as CFS are at the present time *unresolvable* in the way that I defined the term in Chapter Two, Section Two, because those uncertainties cannot be resolved by comparing cases, and further relevant information cannot be obtained within the timescale of healthcare encounter. So, this uncertainty is categorical rather than contingent.

The value of recognising the presence of radical uncertainties regarding CFS is that these uncertainties explain the existence of oppositional conceptualisations for CFS and oppositional appraisals of treatment recommendations, thereby negatively impacting healthcare encounters. I have therefore shown the applicability of the economists' concept of radical uncertainty to problems regarding medical epistemology and medical practice.

The second concept that I promote is that of *a complex network of uncertainties* as an explanation for this situation of radical uncertainty about CFS. In Chapter Three, Section Four, "The Interactions Between the Differing Areas of Uncertainty in Chronic Fatigue Syndrome", I argued that research concerning CFS is hampered by the existence of an under-recognised concept, a mutually reinforcing network of uncertainties in CFS, in which the many basic features of CFS that are uncertain, indeterminate, or underdefined, themselves hinder research and knowledge-creation concerning the other basic features. Again, this attribution of a network of uncertainties about chronic fatigue syndrome is not generally emphasised elsewhere in the literature.

The value of the concept of a network of uncertainties is that this concept increases the recognisability of radical uncertainty in some medical conditions, and gives an explanation for the elusiveness of research solutions to the radical uncertainties regarding CFS and other medical conditions, such as fibromyalgia, long covid, Candida, chronic Lyme disease, and multiple chemical sensitivity. This is a further contribution to medical epistemology.

The third concept that I promote that is not well-promoted in the literature is *uncertainty-driven healthcare epistemic injustice* (UHEI). In Chapter Six, I argued that epistemic injustice in some CFS healthcare encounters is partially explained by differing responses to radical uncertainty. UHEI is additional to the other already-recognised explanations for healthcare epistemic injustice that cite the power differential between patients and healthcare professionals and the differences in their phenomenological and epistemological perspectives. Patients' and healthcare professionals' dichotomous and oppositional positions regarding the nature of CFS and its treatment recommendations (that I described in Chapter Four) exist because of patients' and healthcare professionals' differing responses to radical uncertainty about CFS. I criticised both of these dichotomous stances because, in my view, they can (in combination) lead to healthcare encounters that are epistemically unjust.

The value of the concept of UHEI is that it encourages a re-analysis of unsatisfactory healthcare encounters, in order to *recognise and then evaluate* the contributions of under-recognised and under-acknowledged radical uncertainty in some medical conditions, combined with the unsatisfactorily differing and oppositional responses to that uncertainty. The recognition this UHEI concept can lead to recommendations for the amelioration of certain instances of healthcare epistemic injustice (unconnected with chronic fatigue syndrome) that are additional to the recommendations of other HEI commentators. My own

recommendations include the mitigation of the effects of that uncertainty, the acceptance of the reasonableness of others' differing responses to that uncertainty (using the precepts of epistemic virtue), and the forging of new shared conceptualisations and treatment recommendations. Thus, this thesis adds to the understanding₇ and amelioration₇ of healthcare epistemic injustice, and similarly benefits medical ethical understanding, particularly regarding the principles of patient autonomy and non-maleficence.

In Chapter Two, Section Three, I promoted the application of *epistemic pluralism* (which is promoted by Mormina (2022)) to medical conditions where there is uncertainty.

The value of the concept of epistemic pluralism is that it is an acknowledgement of the reality and relevance of evidence from differing sources in healthcare, valuing both objective and subjective evidence; furthermore, epistemic pluralism discourages the rejecting of differing sources.

In Chapter Seven, Section Six, as an alternative to both the biomedical model and the biopsychosocial model, and to avoid 'medical disease dualism', I promoted a hypothesised network model of the symptomatology of chronic fatigue syndrome. This model is not usually applied to non-psychiatric illnesses in the literature. According to this network of symptoms model, symptoms in a particular medical condition manifest *a dynamic reciprocally interactive network of mutually reinforcing (or weakening) symptoms*; the interactions between symptoms can be positive or negative. This network of symptoms model is itself not original: Borsboom and his colleagues (2013) apply a version of this model to medical conditions where psychological symptoms predominate such as depression. However, unlike me, Borsboom *et al.* do not apply the model to medical

conditions where physical symptoms predominate, such as CFS. This network of symptoms model still allows for the possibility of the existence of a (at present hypothetical) ‘latent variable’, that is to say, an as-yet-undiscovered common cause for those symptoms within a hybrid network model.

The value of the application of this network of symptoms model (and its hybrid variant) to the understanding of the medical condition CFS is that the complexity of the model can explain the (otherwise puzzling) diversity of symptomatology between people whose symptoms are labelled as CFS; and, the dynamic nature of the model can explain the (similarly puzzling) variability over time in symptomatology in any particular CFS patient.

The value of a network of symptoms model in a CFS healthcare encounter is that (in the present absence of knowledge of a common cause) it encourages the concentration of treatment recommendations for CFS onto symptoms that seem to have centrality, that is to say, with more and/or stronger associational, causative, or mechanistic connections to the other symptoms. The reduction in the severity or negative effects of a symptom with centrality may well reduce the severity and negative effects of adjacent symptoms, because the reciprocal interactions between those symptoms.

The value of a hybrid network model is that is compatible with the two conflicting conceptualisations of the mechanisms that are attributed to CFS, the biomedical model that postulates a single biomedical cause in any particular patient, and the biopsychosocial model that postulates that many factors, including cognition and behaviour, contribute to the perpetuation of symptoms. The concepts of both the network model and the hybrid network model of disease are contributions to debates concerning CFS ontology.

In Chapter Seven, Section Seven I promoted the concept of *persistent physical symptoms* (PPS) as a possible conceptualisation for the perpetuation of CFS symptoms. Describing the symptoms of CFS in terms of PPS allows the medical condition CFS to be viewed as not explicitly different from other medical conditions where some patients have physical symptoms that persist much longer than others' symptoms. This perspective may well reduce stigma, and be acceptable to those who focus on a physical nature for CFS. The concept of PPS suggests that therapy for an individual patient with PPS should address the symptoms, rather than to wait for an understanding of the underlying pathological processes.

8.4: Summary of My Critiques of Others' Contributions to the Debates Concerning Chronic Fatigue Syndrome

In order to support my arguments, I have critiqued others' conceptualisations of chronic fatigue syndrome and their responses to its uncertainties. I summarise my critiques in the following paragraphs.

I have critiqued both of the oppositional models for CFS, the biomedical model and the biopsychosocial model, as each having inadequate empirical support from research on mechanisms or on treatments.

In my scrutiny of others' support for the biomedical model in Chapter Five, Section Three, I critiqued (as other commentators have) a concept that I call *medical disease dualism*, that is to say, the assumption of the existence of a (global or local) strong distinction between

physical and psychological medical conditions. This concept is present in the literature concerning the biomedical model, but is under-acknowledged, and often only implied by those who support the biomedical model. I have argued that the biomedical model of CFS (as supported by some CFS patients, their support and advocacy groups, supporting websites, and ME research organisations) is dependent on the assumption of this local medical disease dualism, because of their claim that CFS is a physical, not a psychological, condition. This medical dualistic claim refers to the assumed mechanisms of CFS, treatment recommendations for CFS, and as a guiding principle for directing CFS research. Those people who advocate this local medical disease dualism in regard to CFS may be accepting a global distinction between physical and psychological medical conditions, although this assumption is not usually made explicit. There is usually no explicit connection made between medical disease dualism and metaphysical mind-body dualism.

In contrast to some people's assumption of 'medical disease dualism', surveys show most healthcare professionals see that CFS, like many other medical conditions, has both physical and psychological features, with biomedical, psychological, social, and behavioural aspects in its symptomatology, in its mechanisms, in treatment recommendations, and in the necessary directions of research. Even though the biopsychosocial model is explicitly multifactorial, it is mistakenly interpreted by those who oppose it and have a local medical dualistic conceptualisation as implying that patients' symptoms are being almost exclusively attributed to psychological, psychiatric, cognitive, and/or behavioural processes, with biomedical processes being down-played or even rejected. Oppositional support for either the biomedical model or the biopsychosocial model facilitates disputes between some CFS patients and some healthcare professionals in CFS healthcare encounters.

The value of restraining the concept of medical disease dualism is that this restraint can lessen the adversarial dichotomy between the biomedical and the biopsychosocial models of chronic fatigue syndrome (and similar conditions) and their treatment recommendations; additionally the restraining of a mental disease/physical disease dualism gives a positive counter to some people's unfortunate perception that a mental condition is stigmatising. This concept is therefore a contribution to debates concerning disease ontology.

In Chapter Four, Section Four, I critiqued some applications of the biopsychosocial model to CFS, and the inadequate success of applying treatments based on BPS to CFS patients . I criticised Sharpe and Greco's (2019) concept of 'illness without disease' in Chapter Four, Section Four, because this concept does not incorporate the concept of a continuum of knowledge about medical conditions, nor does it incorporate the meta-inductive understanding that knowledge about many medical conditions improves over time.

In Chapter One, Section Six, I critiqued the categorisation of some under-explained conditions such as chronic fatigue syndrome and others as being '*medically unexplained*', which epithet can be stigmatising, and may mistakenly be interpreted as meaning '*medically unexplainable*', thereby reducing incentives for research. Instead of being described as medically unexplained, I have suggested an alternative description of the symptoms of CFS as being 'persistent physical symptoms' (Löwe *et al.* 2025). Persistent physical symptoms can be a feature of the illness experience of people suffering with many kinds of medical conditions, including those that have pathological mechanisms that are well understood.

In Chapter Seven, Section Three, I noted that many of the dichotomies concerning chronic fatigue syndrome are unfortunately *debated eristically* (and non-productively) in healthcare

encounters and in the public field, where there is arguing to win rather than a searching for the understanding of others' not-unreasonable perspective.

The value of disputing non-eristically in CFS healthcare encounters and in public debates is that these disputations and debates are more likely to produce the appreciation of opponents' views and lead to mutual understanding.

In Chapter Seven, Section Three, I critiqued the rejection by some people of oppositional conceptualisations of, and treatment recommendations for, CFS as failing to show appropriate *epistemic virtue*.

The value of epistemic virtue is that it discourages the rejection of other's understanding of disease processes.

8.5: Summary of the Contributions of This Thesis, and Recommendations for Future Study

In this thesis I have promoted the interdisciplinarity of economics and medical knowledge in regard to uncertainty; I have given contributions to medical epistemology, medical ethics, epistemic injustice, disease ontology, and the application of the concept of epistemic virtue and vice to disputes in medicine. Thereby, I have demonstrating how the philosophical study of one medical condition (and similar conditions) can contribute to the philosophy of medicine, and some of the ways in which philosophy of medicine can benefit the practice of medicine.

For the future, I would suggest that the issues in this thesis could be built on and extended by: (1) using the insights on scientific practice and scientific justification of Kuhn, Laudén, Lakatos, Latour, and others (Hepburn 2021), to survey and critique the differing research programmes on chronic fatigue syndrome; or (2) studies of the phenomenology of responses to uncertainties in medicine, from the perspective of patients, healthcare professionals, and research scientists; or (3) using the lessons of intersectionality (Buchman *et al.* 2017; Collins 2017; Bamed *et al.* 2019; Wilson *et al.* 2019; Mitova 2020; Radoilska 2020; L. Smith & Archer 2020; Bogaert 2021; Pot 2022; Radoilska 2022) to extend the understanding of epistemic injustice in the face of uncertainty; or (4) using the lessons of Standpoint Theory (Wylie 2015) to incorporate the epistemic privileging of the oppressed as a route towards adjudicating between differing responses to uncertainty. The findings from each of these studies may provide suggestions for reducing uncertainty-driven healthcare epistemic injustice.

Appendix – The Testimony of ‘Suzanne’

‘Suzanne’ (2024), ‘Experiences of epistemic injustice regarding chronic fatigue syndrome and Long Covid’, *Anonymous personal communication via E-mail*.

The following is a personal communication to me from ‘Suzanne’ (not her real name) who kindly agreed that I can use her testimony anonymously in my thesis. My question to Suzanne was, “Do you have views on why there is so much epistemic injustice in regard to CFS or Long Covid?”. This is her reply. For convenience, I have numbered her five paragraphs.

‘Suzanne’s’ Testimony:

§1. I think there is so much epistemic injustice regarding CFS and Long Covid because there is no one proven link that the illness is physical, psychological or both, most sufferers don’t look particularly ill and in my experience, most sufferers are women. And so some people (including medical practitioners) don’t necessarily believe it exists at all. And this disbelief is gut-wrenching for sufferers. Because you know, as a sufferer, that it’s physical and anti-depressants don’t tend to work. Unless you have suffered yourself, you can’t imagine how it really feels, as sufferers can’t fully explain themselves and no one can point to a positive CFS test result. So I do understand why some medical practitioners dismiss sufferers as being depressed and so only offer psychological interventions. Because they can’t prove it with a physical test. And the stigma associated with having a mental health condition is very real, which is even worse when you, as a sufferer, really don’t believe that it is only a mental health condition, but that you have something very physically wrong with

you. However, it is vital that medical practitioners understand that their reactions to sufferers needs to improve, that they need to consider how unjust it is for sufferers to be treated in this way, as it does have a very negative effect on them. You feel labelled as a worrier, a hypochondriac, a time waster, when you just want to understand what is wrong and do whatever you can do to improve.

§2. Sufferers really need to be heard and understood that it's more than symptoms of feeling depressed/anxious. Yes, you can get fatigue from depression, but the depths of chronic fatigue are quite extraordinary. It's when there is no respite from it, despite rest, which really negatively affects you psychologically. I went through chronic fatigue with Long Covid recently whilst a friend was having chemotherapy for breast cancer treatment. It was extraordinary to hear from her how she felt sorry for me, as her terrible fatigue at least came and went and having had a glimpse of it, she couldn't understand how I could cope with it, mentally, continually. And she had cancer! In both instances when I've got ill, it was following a harsh virus and a period of stress running up to those incidents. I then had an extreme physical reaction which stayed a long time (years in the first instance.) I then (believe) I had psychological problems of anxiety (first time around) and both anxiety and depression (2nd time around after covid) in the months following the initial symptoms. The physical symptoms happened first, and there was no obvious reason why I should suffer this reaction compared to others who got better after a virus. Following that, my inability to work and the collapse of social and family structures around me absolutely affected my mental health negatively. Add in a puzzled, though sympathetic look from my GP (and I moved surgeries to see him as my first surgery didn't believe me at all), the offer of anti-depressants only following negative blood tests of anything abnormal,

the advice to never join a support group because it will make you worse and an off you go attitude, and then whenever I went back looking for answers and was treated as an ‘oh well, she’s here again’ case, made me fall apart. And even if I had news to bring of what appeared to help, I was pretty much dismissed.

§3. My reaction to getting Long Covid, in my late-40s, after 16+ years of being well following 6 odd years of Chronic fatigue, I think, is quite telling, having gone through the stigma and symptoms of having chronic fatigue in my mid-20s. The traction that Long Covid was getting, felt like I could say that I had Long Covid and feel legitimate, and not Chronic Fatigue, because Long Covid had a very obvious beginning, a Covid infection, and as a new virus there was next to nothing known about it, and huge numbers of people worldwide were getting Long Covid symptoms like me. I thought I would be taken more seriously than I was in my 20s – by the medical profession, colleagues, family, friends. And I was. I refused to say I had chronic fatigue (because of the old associated stigma and I couldn’t mentally face the potential years of feeling horrendous and practically house bound and the loss of my social and family life), and so in my head it was absolutely Long Covid (as a legitimate, seemingly new illness). But when the Long Covid clinic told me that I had Chronic fatigue again because as a former sufferer I would always have Chronic Fatigue Syndrome, I couldn’t, psychologically, handle this very well. I was in complete denial. Once I fully recovered from chronic fatigue in my early-30s, following pregnancy, I buried it in the far recesses of my mind. Because it had been pretty traumatic and the stigma from the first time around had a hugely negative effect. I had an extremely difficult stressful period for around 6 years, whilst I was well from chronic fatigue and had a very young family, that could have tipped anyone

into anxiety/depression, yet I was ok. So I was convinced that I had fully recovered from the first bout of Chronic fatigue (due to hormone changes in pregnancy.) But Covid did it to me again. And I was terrified as any former sufferer will tell you, your biggest fear is that it will come back. So of course the anxiety of this made me feel much worse. The bonus this time around, that 20 years on from my first chronic fatigue diagnosis,

§4. I felt that I had more avenues to try to get better and that things were improved. I was referred, a year and a half in, to a chronic fatigue clinic, which didn't exist the first time around, and I was told there, that although it was based in neuro-psychology, they believe that chronic fatigue sufferers have weak immune systems and it can re-occur through your lifetime. So the Covid infection was the catalyst for those symptoms to re-appear. They couldn't tell me anything I didn't already know about how to improve my symptoms (and in fact, I was giving them ideas!), but I am now 85% better, 2 years after getting covid, clearly back at work and coping physically and mentally with a great social and family life—and I have just been given a diagnosis of CFS again. Which given my personal circumstances, where my life is back on track and I don't need to apply for benefits, I'm not sure whether this is helpful or not. But my understanding is that I am prone to bouts of fatigue, a syndrome is not an illness, I can be fine for ages until a trigger like a virus comes along, and, most importantly, I know I can get better from it.

§5. Anyway, as you can tell from my long ramble, anyone who has ever had this has a story to tell! I think this is because it can affect people differently in slightly different ways and you can't point to a test result – yes you definitely have/have not got

chronic fatigue. But you want to be believed, to tell your truth. To be believed and to be treated empathetically with pointers for good information on how to help yourself, and to be told that it is very much a physical illness which can and does affect your mental health, can only be positive for sufferers. Because being believed is half the battle. I'm in a Facebook group for Long Covid sufferers (who may suffer from chronic fatigue or other symptoms which I never had, such as heart issues and breathlessness), and the messages there, from people all over the world are heartbreaking. Because they are not being believed by their medical professionals. As time goes on, Covid is becoming less of an issue, and I fear that Long Covid sufferers are becoming part of the worried well brigade, and badged by some medical professionals as such as there's no known cause and it looks too much like post-viral syndrome right now. If you are heard and listened to, you don't feel like you are losing your mind, and you can eventually get back to your life and be a productive member of society again. That belief in the first place in you is crucial.

Best wishes, 'Suzanne'

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