Introduction

We have reached something of an impasse in the treatment and care of those of us who experience mental health problems. Despite the huge budget and resources allocated to research and treatment of mental health problems, the lifetime risk of being diagnosed with a major depressive episode is nearly 15% in wealthy developed countries (Bromet, et al., 2011). The World Health Organization (WHO) believe that the day-to-day burden of depression is increasing and will take second place in the global rating of disease burden by 2020. Approximately one in 100 people experience a psychotic episode; a statistic that has remained unchanged since Emil Kraepelin (1899) started to classify types of mental distress. Clearly, we are not making much acceptable progress in the prevention of such conditions.

For over a hundred years, psychiatrists and other mental health workers, as well as pharmaceutical companies and medical researchers, have been trying to find the evidence to support the paradigm of ‘mental illness’. So far, no unequivocal evidence to show that mental distress problems are caused by neurological and neurochemical changes has been found. We continue to search for this evidence, in part because the advocates of this paradigm are loud and strong, but also because we must find more effective ways to help people in mental distress. Mental health problems can be complex, life-changing and sometimes life-threatening. We have to stop and take stock, as we are not making discernible progress in identifying a biological cause for mental distress. We can identify the effects of distress in the brain, but have failed to show clear genetic and biological markers that signal the inevitable onset of a mental health problem.

The progress we have made in the treatment of such problems is not trivial. We have come a long way from dropping cold water on the heads of the mad, but not far enough to ensure that everyone experiencing mental distress can live productively. We talk about ending the stigma of mental health problems but, in fact, only seem to be making such stigma worse (Read and Harré, 2001). The surge to proclaim mental distress as illness seems defeated by the inability to cure these problems permanently. Indeed, many of the big pharma companies are withdrawing from research into mental health problems because they recognise that progress has
effectively stalled (Miller, 2010a; see Chapter 11). It is time to take a step back from the drive for medical intervention as a first line of treatment and try to make a rational decision about where to go next.

The aim of this book is to take a critical look at the existing paradigm and consider the options that could stand in its place. Before we start this process, it is important that we look at the biomedical model, and the philosophy behind paradigms. We will then move on to the major philosophical problems involved in the study and treatment of mental distress.

1.1 How Theories are Developed

Thomas Kuhn (1922–1996) was a physicist and philosopher who in 1962 published a book called *The Structure of Scientific Revolutions* that has had a considerable impact on the history and philosophy of science. It had been thought that science was a logical progression of theories. A particular theory holds sway until a theory that better explains the data comes along, and so on. Instead, Kuhn posited that scientific theories were underpinned by a set of beliefs or assumptions, called a paradigm. A paradigm is a collection of ideas scientists agree to believe in at any current time. A good example of a paradigm would be geocentricism – the Ptolemaic idea from the second century BC that the Earth is a stable body at the centre of the universe. People believed in geocentricism for around 1500 years, using it as a basis for astrological charts. The evidence for geocentricism included the idea that the Sun and Moon seem to revolve around us. Gradually the idea of Nicolaus Copernicus, published in 1543, that the Earth revolved around the Sun (heliocentrism) took over. Copernicus gave a set of new assumptions to support his idea and gradually these became established.

Kuhn used examples such as the move from geocentricism to heliocentrism to illustrate a pattern that has been repeated throughout the history of science. Someone has an idea and, if enough people subscribe to it, that idea becomes the dominant view. This theory then becomes the underpinning concept of a paradigm and all subsequent theories refer to and thus extend the paradigm. Occasionally other theories come along to challenge the current paradigm and, if enough people support this new theory, it will become dominant and supplant the first theory. For example, before Charles Darwin came up with his theory of evolution in 1859, the dominant theory was Lamarckism (that organisms pass on characteristics learned during their lifetime to their offspring, and that this was the main driving force for adaptation). Darwin constructed his theory based upon his observations of the natural world and on reading the ideas of others (including Alfred Wallace, who had earlier posited a similar theory of natural selection). Other scientists saw that Darwin’s theory of natural selection was better at explaining what was seen in
natural history and, with the adoption of this new theory, science moved on. Ideas become popular and will become the dominant explanation until all the phenomena that cannot be explained overwhelm the old theory and lead to someone proposing a new set of assumptions, thereby effecting what Kuhn terms a paradigm shift.

Kuhn summarised the way science works like this:

prescience → normal science → crisis → revolution → new normal science → new crisis → etc.

1.2 The Biomedical Model

The psychiatric or biomedical model of mental illness is currently the dominant paradigm of mental distress. Put very simply, the biomedical model considers the various manifestations of mental distress, which it labels ‘mental illnesses’ or mental disorders, to be caused by biological processes, specifically structural and/or chemical changes in the brain and nervous system. There are certainly clinical disorders characterised by changes in behaviour and mentation that do have an organic basis (e.g. vascular dementia, Alzheimer’s disease and Korsakoff syndrome), but in recent years proponents of the biomedical model have suggested that chemical imbalances in the brain cause various forms of mental distress such as depression and psychosis. However, to date, no biological cause or reliable biomarker has been identified for either of these conditions or indeed most of the other mental disorders listed in psychiatric classification systems such as the fifth edition of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; APA, 2013) and the tenth edition of the World Health Organization’s *International Classification of Diseases* (ICD-10; WHO, 1992) (Deacon, 2013).

Several consequences follow from subscribing to the biomedical model of mental distress. The first is that mental disorders, like physical disorders, can and should be classified. This implies that a clear division exists between normality and ‘abnormality’, an idea which many critics of the biomedical model contest. One of the most famous classifiers of mental distress was Emil Kraepelin (1856–1926), who in the late nineteenth century proposed two categories of insanity: dementia praecox (now known as schizophrenia) and manic-depression. Whilst Kraepelin considered dementia praecox to be a condition characterised by disorder of thought that was likely to deteriorate overtime, he thought that many of the affective disorders denoted by manic-depression were usually episodic and likely to remit. Kraepelin’s work and influence on the development of modern psychiatry are considered in more depth in the next chapter. Here, it is perhaps more important to note the medical naturalism implicit in the idea that separate forms of mental
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distress exist (as indicated by different diagnostic categories), that is, mental disorders exist as discrete entities in nature waiting to be discovered in patients. A number of propositions follow from this. The first is that separate ‘mental disorders’ like separate physical problems are each underpinned by distinctive pathological biological (in the case of the biomedical model) processes that give rise to particular clusters of symptoms and signs. The second is that making a diagnosis (that is, the allocation of a person to a specific category of mental distress) should tell us something about what that person is likely to experience in the future without therapeutic intervention. The third is that a diagnosis should indicate an effective approach to the treatment of that form of mental distress.

When it comes to treatment, the history of psychiatry is as chequered as any medical discipline. There have been notable lows, for example, the use of frontal leucotomy, a form of psychosurgery, as a treatment for psychosis and depression especially in the USA during the middle of the previous century (Braslow, 1999). Considered revolutionary at the time by its advocates, leucotomy was superseded by the discovery of the antipsychotic effects of a drug called chlorpromazine in the early 1950s, the introduction of the first antidepressant in the late 1950s and the use of other treatments such as electro-convulsive therapy (ECT). Braslow’s (1999) paper, which refers to case studies of the use of the procedure in depressed women, makes for particularly bleak reading. Forms of psychosurgery are, however, still in use, although clinicians have become selective about how, when and with whom it is used (Swartz, et al., 1998).

The early promise of newly discovered psychotropic medications was never fully realised, although the psychotropic drugs did play a part in the subsequent policy of deinstitutionalisation, as the primary location for the treatment of mental disorders shifted from large mental hospitals (formerly known as asylums) to the community. Early antipsychotic drugs made some people physically ill. Some medications gave such unpleasant side effects that many people stopped taking them. The early treatments for psychosis sometimes led to a syndrome called tardive dyskinesia (e.g. Jeste and Wyatt, 1981), which caused involuntary movements of the face and limbs. There is now plenty of evidence to say that psychotropic medication is not universally efficacious, but rather that certain people with specific characteristics can be treated more successfully than others. However, as long as we continue to consider things such as depression and psychoses as physical illnesses equivalent to diabetes or stomach ulcers, we are honour bound to continue to look for medicinal cures. You can equate this to the search for a cure for cancer because in the minds of most psychiatrists since the 1950s, mental illness is a chronic, life-threatening problem just as cancer is. Here in 2018, we sit with the knowledge that 60 years of drug treatment have made only limited inroads into the treatment of mental illness. Furthermore, this is totally out of proportion to the leaps in treatment made in other areas of medicine.
A further consequence of the dominance of the biomedical model in the field of mental health is the comparative neglect of psychological and social factors in explanations for mental distress and strategies for the prevention and treatment of such distress. In the United Kingdom and elsewhere, many psychologists (see, e.g., Bentall, 2004a, b; Boyle, 2002, 2006; Kinderman, 2014) and others are trying to change this. It is time for a paradigm shift that acknowledges that at least some of the problems and causes of mental distress are psychosocial and, furthermore, that they can be treated psychosocially.

1.2 The Biomedical Model

1.2.1 What is the Difference Between a Mental and a Physical Illness?

One of the problems with using the biomedical model for understanding psychopathology is that the evidence for the physical bodily nature of mental distress cannot be demonstrated easily. It has been argued that one reason why we have not found evidence of neural changes that would give rise to mental distress is that we are still relatively new at examining the human brain. As we continue to discover more, we might still discover the biological causes of mental health problems. Increasingly, however, there are those who suggest that organic changes in the brain will not be found to be a causal factor in the development of mental distress. Instead, they believe such problems are behavioural and can best be treated behaviourally. That it is difficult to collect evidence to support either of these viewpoints is an indication of just how complex the human brain, and human behaviour, is. The fact that we are not sure of how mental distress develops is a very old problem. The next section will explore the development of this problem.

1.2.2 Dualism

Dualism arose out of René Descartes’ (1596–1650) reformulation of Plato’s Theory of Forms. Descartes made a simple observation that although he could believe that his body was an illusion, he could not doubt that his mind was real. To him, this meant that the mind and body (including the brain) are separate things, hence the term dualism. Descartes discussed the nature of sensations like hunger and pain and concluded that he gave meaning to these sensations using thought and association; otherwise they are uninterpretable. Thought is the more important because without it, we could not interpret sensations and events (Cardinal, et al., 2005). Cartesian dualism was a prominent philosophical explanation in the early days of treating mental distress in the middle of the eighteenth century. It was thought, for example, that the behavioural changes seen in melancholia (a condition which corresponds roughly to what we now term depression) had to be the mental representation of a physical problem, and therefore melancholia should be treated in the same way as other physical complaints. However, both the lack of success in treating these
conditions with available medical treatment and a failure to find any physical manifestation of the distress condition at post-mortem contributed to the separation of mental and physical illnesses. Gradually, however, there was a swing back to the biological explanation of mental distress spurred on by Bayle’s 1922 discovery of lesions in the brains of those afflicted with general paresis of the insane. This was a specific disorder that caused skin lesions, followed by dementia, and finally turned into paralysis. We now know that this disorder is the process of untreated syphilis and it is treated with antibiotics. In the early nineteenth century, however, general paresis was seen as a form of insanity. The final recourse for people with general paresis was to enter an asylum. Then Bayle found physical evidence of the infection in the brain at post-mortem and suddenly there was visible evidence for the biological causes of insanity. At much the same time, Benjamin Rush in the United States was reporting that insanity was the result of pathological changes in the blood vessels of the brain. Little by little, the paradigm in favour of the belief that insanity had biological causes became established. More practitioners of the new medical discipline of psychiatry declared that it would be possible to find the neurological damage causing insanity; that insanity was just the same as bodily disorders such as asthma and migraine. Gradually, people let go of Cartesian dualism in favour of a belief that mind and brain (or mind and body) were the same thing. However, the causal nature of brain changes in mental distress disorders has not yet been uncovered, despite many years spent looking at the brains of those diagnosed with these conditions.

At this point, you might be wondering whether it matters if mental distress is a condition of mind or brain if we can treat it successfully. It probably does not matter to someone with clinical depression what causes his/her distress if s/he is assured that this distress will go away. In other words, the debate outlined in this section is merely a philosophical one. Do we need to be certain of the cause of a problem to treat it?

It is perhaps expedient to look for treatments for mental distress in the same way as for a more obviously organic illness. Whether a problem is psychological or physical need not interfere with any successful treatment. However expedient, the fact that in some ways we are no further forward in the treatment of mental distress than we were 65 years ago means that it is time to start looking for other treatments. The argument does matter if the use of pharmaceuticals to treat mental distress is obscuring the true cause or precipitating factor of that distress. It is time to examine the difference between brain and mind more objectively and to stop fearing the accusation of dualism.

1.2.3 Dualism and the Perils of Reductionism

John Searle (1984) said that ‘brains cause minds’. The mind and the brain are synonymous. If you believe this, then it is perhaps logical to believe that abnormal
thought and behaviour must have a biological basis; that abnormal thought is caused by abnormal biology. We assume that the brain underpins everything that we are. So, it is logical to think of mental distress as a biological abnormality that causes a problem with one’s behaviour and thoughts. However, what if this is a major oversimplification? What if mind and therefore self are separate or at least independent of brain? What if mind is parallel to brain?

These are not simple questions. Mind and thought are more than the sum of their parts (i.e. a side effect of normal processes, known as an epiphenomenon). Mind and thought are individual and unique to each of us, because we have individual and unique experiences. In fact, some people think of mind as consciousness (Jackson, 1982). It could be (it probably is, in fact) the case that when people talk of ‘mind’ they are talking about consciousness. Consciousness can be defined basically as the state of being aware of the implications of perceptions; of being aware of and active in the world around you. As we interpret our perceptions through the filter of our own past experiences, we can say that consciousness (in part) refers to the part of you that is unlike that of anyone else. (There is a mountain of books that argue whether consciousness is an epiphenomenon and generally all have something different to say. If you are interested in this, a good place to start would be Daniel Dennett’s 1993 book *Consciousness Explained*. Let us simply say that consciousness is an epiphenomenon in the sense that it has no distinct and unified locus in the brain that we have so far established.)

It can be assumed that normal neural functions would continue in the instance of abnormal thought. The brain would continue to operate relatively normally despite the person thinking about and responding to thoughts that are not ‘normal’. Obviously, should these thoughts continue for any length of time, the brain would change its functioning in some way to adapt to them. If you continue to think anxiety-provoking thoughts, eventually you will end up in a permanent or near constant state of anxiety. We would like you to consider that once this has occurred, those abnormal thoughts are no longer abnormal, but rather they become normal to that person. For example, the psychologist Peter Chadwick (2002) describes the process of his own psychotic episode beginning in such a way.

Think about it in a more prosaic way; you need to get up three days a week for a nine o’clock lecture. You decide that, to attend every session feeling at your best, it might be better to stop going to bed at 3am but go at 11pm instead and get your 7–8 hours of sleep. This is hell for the first few weeks; you lie in bed awake until you eventually fall asleep in the early hours. You are just as tired in lectures, and having much less fun outside of them. Eventually, however, you get used to it and find that it becomes normal to follow this routine even on the days where you have no 9am lecture. You have changed your habits.

This obviously is not just a case of stubbornness or self-control, even though they play an obvious part. Your body (including your brain) has adjusted to
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accommodate these changes. You have altered your circadian rhythms, specifically your sleep/wake cycle, to make sure you are alert at the most beneficial time for you. Behaviour has altered brain function.

This can be a double-edged sword. Imagine that someone is following you and you must get away from that person. It would not take long performing these behaviours before you started to suspect that someone is following you. Therefore, you become more vigilant or perhaps even paranoid. Perhaps you begin to check that you have locked your front door. The thought of the person following you entering your home makes you go back and try the door handle a couple of times. This also becomes routine, so much so that after a while you give the first check of the door the same attention you gave to locking the door in the first place. This means you need to go back and check again or live with the stomach-churning uncertainty of having an unlocked door all day. It is easier to go back and check. Thought has changed your behaviour and once you fall into this deceptively easy trap, it is very difficult to get out of it.

This example of how thought changes behaviour serves to illustrate one way that thoughts can alter behaviour gradually until the person starts to act in atypical ways. Descartes said: ‘I think therefore I am’ and, of course, living is all about thinking but it is also about perceiving and feeling. Thought is the result of the integration of many brain areas, working towards the common cause of creating and maintaining one’s ‘self’. If we can accept this, then the idea that thought can change the brain is surely possible; there is no dualism here. In fact, we know thinking changes the brain. Learning can be defined as gradual changes in part of the brain; this is the nature of neural plasticity (Baudry, 1998). It happens when a task you are learning, for example how to drive a car, becomes automatic and therefore smoother and less effortful over time. We would ask you to consider that mental distress is another example of the effects of neuroplasticity but this time not such a positive example. Once the disturbing or distressing thoughts and actions start to change the brain, it will become much more difficult to stop thinking/doing them. The thoughts have become automatic and are no longer controlled by the thinker.

This fits in with the comments of people who experience mental distress. Frederick Frese (2010), a clinical psychologist who has experienced several psychotic episodes, describes the normality of going from a mentally healthy to a florid psychotic state. Serving in the army during the Vietnam War, he puzzled over why the Vietcong should be so difficult to fight. One day, after working several long days and musing on this problem, he realised that it must be because the Vietcong were using ‘brain-washing’ techniques on not just the soldiers, but also the military command and the politicians in the United States. The more he thought of it, the more he became convinced that this was the logical answer. He went to explain this to the people in charge and the army psychiatrist and found himself committed to a padded cell as a result. He obviously believed his incarceration was
1.3 Do Discrete Mental Illnesses Exist?

Kraepelin’s work to identify and classify separate mental disorders towards the end of the nineteenth century was very much done in the spirit of the age: people were classifying everything in the eighteenth and nineteenth centuries from plants to physical illnesses. It was Kraepelin (1893) who first defined symptoms for the traditional concepts of melancholia, mania and hysteria and devised new concepts. He attempted to define conditions in terms of (a) symptoms at diagnosis, (b) aetiology (the potential causes) and (c) prognosis. This had the effect of essentially adding colour to the traditional term of insanity. People were no longer thought to be simply insane, but could be diagnosed more accurately. As already mentioned, Kraepelin famously proposed the existence of two separate forms of insanity: dementia praecox (rebranded by Bleuler in 1908 as schizophrenia) and affective psychosis (or manic-depression). Kraepelin, however, was never entirely satisfied with this division because it became clear to him that it was not always possible to distinguish easily between these two types of mental disorder (1920/1974).

The problem we have now is that having ignored Kraepelin’s doubts, we have gone on to build our entire diagnostic and treatment systems upon his initial distinction, and starting all over again is almost unthinkable. We will go into more depth about the development of the current diagnostic systems in the next chapter. Here we must question whether we need to formulate separate disease entities. Could we manage without them?

When we talk of discrete illness, we mean that it is possible to distinguish a medical condition from any other medical condition. Although symptoms may
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overlap (for example, in traditional medicine both influenza and measles will start with a raised temperature), the condition must also have unique symptoms or at least a unique pattern of symptoms (e.g. measles has a distinctive rash). The reason why a more specific diagnosis, such as measles, could be beneficial is that it would be easier to define treatments for a specific problem.

If diagnosis is used to establish medication type and regimen, then it is clearly important to get the diagnosis right. If a psychiatrist gives an incorrect diagnosis of, for example, bipolar disorder (manic-depression), then the resulting treatment with mood stabilisers may not be effective and the patient could become worse. If we are to stress the existence of discrete mental disorders, we must first show that each disorder has a set of distinct symptoms that can be diagnosed reliably over time and by different psychiatrists, regardless of where they are in the world. In the next sections, we will examine the value of a diagnosis of either functional psychosis or manic-depression.

1.4 Diagnosing Discrete Mental Distress Conditions

The most commonly diagnosed functional psychosis is the condition of schizophrenia. The concept of schizophrenia has been challenged on its reliability and its validity, and yet it is a condition with an incidence of 1.1% of people aged over 18 years according to the Diagnostic and Statistical Manual of Mental Disorders (DSM), although other sources state the incidence to be as low as 4/1000 (Bhugra, 2005). The validity of a diagnosis can refer to several components. Conceptual validity refers to whether the criteria used to make a particular diagnosis correctly identify people who have that particular mental disorder and distinguish them from those who do not, whereas construct validity refers to the idea of being able to differentiate between conditions with distinct causes. Predictive validity refers to the extent to which people with the same diagnosis follow the same clinical course over time. Reliability refers to the stability of a diagnosis over time and in different places, that is, the likelihood that an individual who provides the same information about the problems s/he has been experiencing would receive the same diagnosis from two different clinicians, or would receive the same diagnosis at different points in time.

Traditionally, psychiatric diagnoses struggle when it comes to reliability and validity. The first problem is whether patients can be diagnosed consistently as experiencing schizophrenia or any other disorder. For example, can clinicians reliably distinguish between schizophrenia and bipolar disorder (BPD)? Of course, this is not a straightforward question to answer because: (1) the patient may have good days when his/her symptoms are controllable and bad days when they are not, or (2) the patient many present with different behaviour on subsequent days. People