Essential Abnormal & Clinical Psychology

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5 MOOD DISORDERS

General introduction

In this chapter we describe the diagnostic criteria for major depressive and bipolar disorders in *ICD*-10 and *DSM*-5, and we discuss some controversial changes to the diagnostic criteria that occurred between *DSM-IV* and *DSM*-5. We then also discuss the causes of these disorders, including heritable and environmental factors, and gene × environment interactions. The largest section of the chapter is devoted to the cognitive distortions that underlie mood disorders, their relationship to abnormalities of brain function, and a critical evaluation of cognitive theories which posit that negative thoughts and cognitive biases are the most important causes of mood disorders. Finally, in the last section we take a look at how mood disorders are treated, with a consideration of the psychological mechanisms of the action of 'talking therapies', and a critical look at the effectiveness and mechanism of the action of medications.

Assessment targets

At the end of the chapter, you should ask yourself the following questions:

- Can I explain how major depression and bipolar disorder are diagnosed?
- Do I understand the key psychological processes that characterise major depression and bipolar disorder?
- Are mood disorders heritable and can I explain their biological basis?
- Can I explain how distorted cognitive processes might play a role in the initial development of mood disorders, and in the maintenance of those disorders?
- Do I understand how mood disorders are treated, and can I relate those treatments back to theoretical models?

Section 1: What are depression and mania?

This section will look at how we diagnose the two major types of mood disorder. These are *depression*, which is known as 'recurrent depressive disorder' in *ICD*-10 and 'major depressive disorder' in *DSM*-5, and *manic depression*, the official name for which is 'bipolar disorder' (*DSM*-5) or 'bipolar affective disorder' (*ICD*-10). We will refer to them as major depression and bipolar disorder for the remainder of this chapter. The disorders are characterised by extreme mood states (deep sadness and manic over-excitability, respectively), and these extreme moods correspond to distinct patterns of behaviour and cognition. You can probably remember a time when you felt sadness, ruminated on bad things that had happened, and felt pessimistic about the future. You can probably also remember times when you felt full of energy, very confident, and optimistic about your future. These are normal human characteristics, but they are taken to the extreme in people who suffer from mood disorders.

The *ICD*-10 diagnostic criteria for recurrent depressive disorder are shown in Box 5.1. Bipolar disorder is diagnosed if a person has experienced at least one manic episode (Box 5.2) and at least one depressive episode (Box 5.1). The experience of living with a close family member who has bipolar disorder is depicted in Box 5.3.

Box 5.1 Essential diagnosis

For major depression

According to *ICD*-10 (World Health Organisation, 1992), Recurrent Depressive Disorder is characterised by recurrent *depressive episodes*, but there must be no history of manic episodes (see Box 5.2). Typical symptoms of depressive episodes, which should be present for at least two weeks to warrant diagnosis, are as follows:

- Depressed mood to a degree that is definitely abnormal for the individual, present for most of the day and almost every day, and largely uninfluenced by circumstances.
- Loss of interest or pleasure in activities that are normally enjoyable.
- Decreased energy or increased fatiguability.
- Loss of confidence and self-esteem.
- Unreasonable feelings of self-reproach or excessive and inappropriate guilt.
- Recurrent thoughts of death or suicide, or any suicidal behaviour.
- Diminished ability to think or concentrate.
- Change in psychomotor activity, with agitation or retardation.
- Sleep disturbance of any type.
- Change in appetite (decrease or increase) with corresponding weight change.

The *ICD* also distinguishes between mild, moderate and severe depressive episodes, depending on the number and severity of the above symptoms that are present during the episode.

The *DSM*-5 (American Psychiatric Association, 2013) applies a different label (Major Depressive Disorder) but the diagnostic criteria are similar to those described by *ICD*-10. As with other disorders, both *ICD*-10 and *DSM*-5 require the symptoms to be associated with clinically significant distress and/or impairment in social and occupational functioning.

Box 5.2 Essential diagnosis

ICD-10 diagnostic criteria for a manic episode (World Health Organisation, 1992)

ICD-10 distinguishes between three degrees of severity of manic episode, which are hypomania (least severe), mania without psychotic symptoms and mania with psychotic symptoms (most severe). The criteria for *mania without psychotic symptoms* are:

A. A mood which is predominantly elevated, expansive or irritable and definitively abnormal for the individual. The mood change must be prominent and sustained for at least a week.
B. At least three of the following must be present (four if the mood is merely irritable), leading to severe interference with personal functioning in daily living:

- Increased activity or physical restlessness.
- Increased talkativeness ('pressure of speech').
- Flight of ideas or the subjective experience of thoughts racing.
- Loss of normal social inhibitions resulting in behaviour which is inappropriate to the circumstances.
- Decreased need for sleep.
- Inflated self-esteem or grandiosity.
- Distractibility or constant changes in activities or plans.
- Behaviour which is foolhardy or reckless and whose risks the subject does not recognize e.g. spending sprees, foolish enterprises, reckless driving.
- Marked sexual energy or sexual indiscretions.

The *DSM*-5 criteria for a manic episode (American Psychiatric Association, 2013) are similar, and both *ICD*-10 and *DSM*-5 require symptoms to be present for at least one week and to be associated with social or occupational impairment in order to warrant diagnosis.

Both *ICD*-10 and *DSM*-5 recognise subcategories and specifiers of major depression and bipolar disorder. For example, one specifier of major depressive disorder in *DSM*-5 is 'with seasonal pattern', which is more commonly known as 'seasonal affective disorder' or the 'winter blues'. Subtypes of bipolar disorder distinguish the severity of manic episodes. For example, manic symptoms can be 'hypomanic' (less severe) or 'with psychotic symptoms' (more severe). In the latter case, inflated self-esteem and grandiose ideas develop into grandiose delusions, or irritability and suspiciousness can develop into paranoid delusions. At this point the distinction between a severe manic episode and other psychotic disorders such as schizophrenia (Chapter 4, see Box 4.3) becomes very blurred indeed!

The diagnostic criteria for major depressive disorder in the previous version of the *DSM* (*DSM-IV*) contained a 'bereavement exclusion' criterion, which meant that a person could not be diagnosed with major depression if they were recently bereaved (a similar exclusion criterion applies in *ICD*-10). However, this exclusion criterion was removed from *DSM*-5. This has divided opinion among the scientific community. On the one hand, it seems reasonable because although it is normal to feel depressed when suffering a bereavement, why shouldn't it be possible to develop major depression after such a traumatic life event? On the other hand, critics argue that this change to the criteria will lead to a massive over-diagnosis of major depression among people who are having a normal and predictable reaction to an unpleasant life event, but who are likely to recover by themselves in time (Wakefield & First, 2012). We saw in Chapter 1 that this general widening of diagnostic criteria is likely to result in much 'normal' human experience being sufficient to warrant a diagnosis of a psychological disorder, and this is one of the main criticisms of categorical approaches to psychological disorders in general, and *DSM*-5 in particular.

Box 5.3 Essential experience

Living with bipolar disorder (Eyers & Parker, 2008)

Depression has the Black Dog. In our home, bipolar has the Polar Bear. A code word created between my sister and me when she was first diagnosed as bipolar aptly describes the illness and our experience of living with this 'animal'.

Polar bears look cute and cuddly, and most of the time my sister is open, funny and playful. Polar bears enjoy company, and my sister has a wide circle of friends, enjoys sport, movies and going out. Polar bears are also versatile, living on land and in deep waters, and my sister is managing her illness extremely well, aged 21, having had the illness since she was 14, attending university and singing in a local church group.

However polar bears also have a predatory side, and this is when we see the illness emerge and my sister goes from gorgeous to grizzly.

The early warning signs for us are over-reaction, over-emotion and extreme irritability. My sister is aggressive with my parents and myself and favours her friends over us. This is brought about by a lack of sleep and racing thoughts. The racing thoughts are ones of anxiety and worries such as being late, organising her room, meeting people, failing in her studies – all mixed up in her mind. As a result she stays up late and cannot relax.

My sister's illness is quite brittle. Within a couple of days she is in a full bipolar episode, and a few days after the inevitable hospital admission she is back home and in recovery. She has experienced far more manic episodes than depressive episodes. Seeing other friends with depression the manic episode is actually easier to handle, believe it or not. Although it requires a higher level of prowess and fitness (like when her thoughts become too overwhelming and she decides to leave home regardless of the time of night, with the result that my family and I have had some stealthy midnight strides across the local shire where we live!), the mania is highly transparent. Depression is hidden and darker: we can't tell what she is thinking or feeling, and it is far harder to help and takes far longer to resolve.

As the episode progresses we also see a change in clothing: haphazard dressing where she tries to wear everything that she likes … all at once, regardless of the weather and venue, like a haute couture model! The final indicator for my family and me is the episode's 'theme tune'. My sister listens to her iPod constantly throughout her illness, and chooses one song that she will play repeatedly: J-Lo, Eminem, Dido, have made up our 'bipolar soundtrack' over the years.

Prevalence, course, comorbidity and the financial burden of mood disorders

The lifetime prevalence of major depression has been estimated at between 7% and 17%, although there is much debate about the true figure which could be even higher (Richards, 2011). The incidence of major depression is about twice as common in women as it is in men. The lifetime prevalence of bipolar disorder is much lower, at approximately 1% (Merikangas & Pato, 2009), and the prevalence of the most severe subtype of bipolar disorder is equal in men and women. However, female bipolar sufferers may have more depressive episodes than males.

Symptoms of both major depression and bipolar disorder first appear in late adolescence and early adulthood (Merikangas & Pato, 2009; Richards, 2011). Depressive episodes last for around six months on average, and most people will make a full recovery after experiencing a depressive episode. Unfortunately, and in common with the chronic nature of other psychological disorders, the majority of people will have at least one additional major depressive or manic episode at some point after recovering from the first one, although they could be symptom-free for many years before experiencing a recurrence of symptoms (Richards, 2011).

Both major depression and bipolar disorder are often comorbid with anxiety disorders and substance use disorders. For example, 70% of people with a diagnosis of bipolar disorder also have panic disorder, and 50% will also have social anxiety disorder (see Chapter 8) (Merikangas & Pato, 2009). With regard to substance use disorder, chronic substance use lowers brain 'reward thresholds', and this leads to a syndrome that looks very similar to major depression (see Chapter 9).

The disabling effects of chronic mood disorders cannot be overstated: the global burden of disability attributable to major depression is second only to that which can be attributed to heart disease (Murray & Lopez, 1996). The economic burden of major depression (attributed to healthcare costs, work absenteeism and reduced productivity) was estimated at 118 billion euros in 2004 (Richards, 2011).

Section summary

We have seen that both *ICD*-10 and *DSM*-5 recognise two broad types of mood disorders, characterised by extreme sad mood (major depression), or alternations between extremely sad and manic states (bipolar disorder). Bipolar disorder is relatively rare and in its extreme form it can be difficult to distinguish this from other psychotic disorders such as schizophrenia. On the other hand, major depression has been called 'the common cold of psychiatry' because it is one of the psychological disorders that psychologists will see most often.

Section 2: How do mood disorders develop?

Do mood disorders run in families?

Both major depression and bipolar disorder run in families, although the heritability of bipolar disorder (estimated at around 70%) is much greater than that for major depression (about 30% to 40%). This issue is complicated because there is shared heritability for the two disorders. This means that a child born to a parent with bipolar disorder is at increased risk of developing both major depression and bipolar disorder, and the same is true for a child who has one or more parents with major depression (Lau & Eley, 2010; Merikangas & Pato, 2009).

Specific genetic variants associated with bipolar disorder have not yet been identified (Merikangas & Pato, 2009). However, polymorphisms (variations) in genes that are involved in serotonin function have been linked to the heritable risk for major depression. For example, genes that code for serotonin transporters seem to differ in people with major depression and unaffected controls, and these genetic variants are associated with personality traits such as neuroticism that are in turn related to major depression (Lau & Eley, 2010). Unfortunately, as with much of the research on psychiatric genetics, many findings in this area cannot be replicated across different studies. Even when we consider the findings that do seem to be reliable, closer examination of individual studies reveals that there is little consistency between different studies (Lau & Eley, 2010; see Box 5.4). Other researchers have identified a relationship between major depression and genes associated with Brain Derived Neurotrophic Factor

(BDNF), which is involved in the regeneration of neurons that are damaged during exposure to stressors (Lau & Eley, 2010). There is undoubtedly a heritable risk for both major depression and bipolar disorder, but our search for the specific gene variants that are involved seems unlikely to succeed until we have a better understanding of how genetic polymorphisms are related to the structure and function of the brain.

Environmental influences

It is easy to think of life events that can cause us to feel sad (e.g. the break-up of a relationship) or ecstatically happy (e.g. passing your exams). However, one-off life events such as these are probably insufficient causes for the development of major depression or bipolar disorder by themselves, because the majority of people will return to 'normal' in time. The risk of developing major depression is increased if people experience a series of negative life events in quick succession (e.g. losing their job, then getting divorced, then being diagnosed with cancer), probably because it is difficult to cope when faced with multiple stressors at the same time (Bender & Alloy, 2011). Similarly, chronic stress associated with poverty, unemployment and low social

Figure 5.1 One-off stressful life events, such as divorce, can trigger major depressive episodes but they are unlikely to lead to major depressive disorder unless experienced as part of a sequence of negative life events

status is associated with increased risk for major depression, and this has also been attributed to psychological coping resources being overwhelmed (see Bender & Alloy, 2011).

Another explanation for why multiple stressful events can lead to chronic major depression or bipolar disorder is the kindling hypothesis, proposed by Post (1992). 'Kindling' is a biological term, which refers to a progressive decline in the strength of the electrical current that is needed to trigger a seizure in mice. At first a high voltage would be needed to cause a seizure, but a second seizure could be triggered by a slightly lower voltage than that needed to cause the first one. The threshold continues to drop until eventually the animal can experience seizures in the absence of any electrical stimulation at all.

The basic idea of the kindling hypothesis of mood disorders is that a major life stressor is needed to trigger the initial depressive or manic episode. However, once the person has recovered from this episode, their threshold for experiencing a subsequent episode is lowered. This kindling process continues until at some point a very minor stressor (e.g. having a minor argument) is sufficient to cause a major depressive or manic episode. After that episodes might occur spontaneously without any environmental trigger at all. Although this idea was originally inspired by a biological process (kindling), we should note that cognitive theories can also account for kindling effects in terms of a progressive strengthening of depressogenic cognitions over time (see Section 3).

In support of the model, there is a declining relationship between life stress and major depressive episodes over time: the first episode is almost always linked to a major stressor, but recurrences can occur independent of life stress (Monroe & Harkness, 2005). However, the model has been less well-supported when applied to bipolar disorder, because there are many inconsistent findings in the literature (Bender & Alloy, 2011).

Gene–environment interactions

In Chapter 1 (Box 1.9) we introduced the many ways in which genes and environment can interact (commonly termed 'G×E' interactions). As reviewed by Lau and Eley (2010) there are many examples of G×E interactions in relation to mood disorders. For example, individuals with a heritable risk for mood disorders are more likely to report symptoms of depression, but only after a stressful life event. In the absence of a stressful life event, there is no relationship between heritable risk for depression and experiencing the symptoms of depression. Attempts to extend this work by identifying the specific genes that moderate the influence of stressful life events on depression have so far yielded inconclusive findings (see Box 5.4). It has also been demonstrated that the gene and environment cannot be disentangled as easily as we would like, because people at increased heritable risk for depression also tend to have more stressful environments (Lau & Eley, 2010), as we discussed in Chapter 1. The study of G×E interactions is a burgeoning area of research, but these interactions are likely to take many forms. In the future major advances are likely to come from the study of epigenetics (how gene expression is influenced by environmental factors; see Chapter 1) and attempts to identify the depressed endophenotype, that is, the characteristics of people who are at increased familial risk for mood disorder, but have not yet developed a disorder.

Box 5.4 Essential research

When an apparently reliable finding isn't so reliable after all

A landmark study by Caspi et al. (2003) has pride of place in many psychology textbooks, because it demonstrates a very clear GxE interaction that seems to play a major causal role in depression. The authors studied a genetic polymorphism of a serotonin transporter gene, and explored how individuals who differed on this genetic polymorphism reacted to stressful life events. They reported that the number of stressful life events that people experienced was directly related to the number of depressive symptoms that they reported (and the severity of those symptoms), but this was only seen amongst people who possessed a certain polymorphism of this serotonin transporter gene. People who had a different genetic polymorphism seemed to be 'immune' to depression, in other words no matter what life threw at them, they just didn't get depressed.

In the years that followed, other reports were published which seemed to directly replicate these findings. Had we found the gene which determined who would be vulnerable (or resilient) to depression? Unfortunately not. When Munafò and colleagues (2009) conducted a meta-analysis of all of the studies that had investigated this issue, they found no reliable evidence for the GxE interaction reported by Caspi et al. (2003). To make matters worse, some of the studies that claimed to directly replicate the findings from the original study had actually shown a completely different pattern of results, but they had interpreted those results incorrectly. This example illustrates the dangers of over-interpreting results from a single study, and the problems that arise when we are too quick to interpret an apparent replication of a novel finding. Perhaps most crucially, it demonstrates the power of meta-analysis to provide the bigger picture on a given topic (see Chapter 1).

Section summary

We have seen that both major depression and bipolar disorder are influenced by stressful life events and that both have a heritable basis, although the heritable basis of bipolar disorder is likely to be more substantial than that for major depression. The roles of nature and nurture are less important than the interaction between the two, and we are beginning to recognise how gene × environment interactions have the potential to explain why people develop mood disorders.

Section 3: Biological and psychological mechanisms in mood disorders

Neurotransmitters

According to the monoamine hypothesis (for an historical overview, see Heninger et al., 1996) depression is caused by reduced levels of serotonin, noradrenalin and dopamine

(the monoamines) in the limbic system. Several pieces of evidence support the theory. First, the most commonly prescribed antidepressants are selective serotonin reuptake inhibitors (SSRIs) and noradrenalin reuptake inhibitors (NRIs). These drugs work by increasing activity in serotonin and noradrenalin systems, respectively, and both are effective at alleviating the symptoms of major depression (see Section 4). Neuroimaging and post-mortem studies of depressed patients' brains show that they have fewer serotonin receptors, and their receptors tend to be less sensitive compared to healthy controls (Drevets et al., 2008). Second, other studies have investigated the effects of depleting tryptophan, a biological precursor of serotonin. If we give an amino acid drink that depletes levels of tryptophan (and therefore levels of serotonin in the brain) to formerly depressed patients who have recovered, this can trigger a recurrence of their depressive symptoms (Drevets et al., 2008). Finally, the recreational drug ecstasy increases monoamine activity, and when people take it they report strong feelings of euphoria. On the basis of all of these observations, it can be argued that reduced levels of these neurotransmitters play a causal role in major depression, and that increased levels might cause mania.

However, some evidence is not consistent with a simplistic monoamine hypothesis. First, the SSRIs alter serotonin function immediately, but people need to take them for several weeks before they see an improvement in symptoms. Second, the observed deficits in serotonin function might reflect changes in the brain that occur as a consequence of chronic depression, rather than being a cause of it. Finally, and more fundamentally, we know that neurotransmitters do not function in isolation, and we have to consider the interactions between different neurotransmitters (including glutamate, GABA and acetylcholine) in order to understand mood disorders properly (Drevets et al., 2008). This is why current biological theories of mood disorders focus on abnormal function in different regions of the brain, rather than the activity levels of different neurotransmitters.

Abnormal brain function in depression

Drevets et al. (2008) proposed that a network of brain structures was implicated in major depression and bipolar disorder. These are regions included within the medial prefrontal cortex (MPFC), the limbic system, and the connections between the two. In mood disordered patients we can see reduced grey matter volume (a structural deficit) and glucose metabolism (a marker of brain activity, i.e. a functional deficit) in specific regions of the MPFC, particularly the left anterior cingulate cortex (ACC). This is important because the MPFC normally inhibits activity in the limbic system, so one indirect consequence of reduced activity in the MPFC would be increased activity in the limbic system. This is exactly what we see in mood disorders: activity in the limbic system (particularly the amygdala) is increased in major depression and bipolar disorder patients when they are in the middle of a major depressive episode, and the level of increased activity in these regions is associated with the magnitude of emotional processing biases in depressed patients (Drevets et al., 2008; emotional processing biases are discussed later in this chapter). Furthermore, antidepressants normalise

activity in these regions (Goldapple et al., 2004). Cognitive behaviour therapy (CBT) may work in a different way, by increasing activity in the MPFC and therefore indirectly increasing inhibition of the limbic system. In essence, both antidepressants and CBT can normalise an overactive limbic system, but their mechanism of action is very different: a 'top-down' action for CBT (the MPFC changes first, and then influences activity in the limbic system) versus a 'bottom-up' action for antidepressants (the limbic system changes first, and then activity in the MPFC changes afterwards) (Goldapple et al., 2004).

Drevets et al.'s (2008) model represents a notable advance on simplistic monoaminergic theories of mood disorders: it acknowledges that serotonin plays a key role, but it also shows how it is important to investigate the function of different networks in the brain, and the interplay between many different neurotransmitter systems within these networks.

Cognitive factors: Learned helplessness

Martin Seligman and colleagues undertook several experiments in the 1960s to demonstrate how the lack of control of aversive outcomes was linked to helplessness in dogs. An overview of these experiments is shown in Box 5.5.

Box 5.5 Essential research

Classic studies of 'learned helplessness' (e.g. Seligman et al., 1968)

Seligman's basic paradigm used chambers that had two compartments separated by a barrier. Both sides of the chamber had a metal floor through which a strong electrical current could be passed – thus shocking anything that happened to be in that compartment. They studied two groups of dogs. The first group could escape the electric shocks by jumping across the barrier to the opposite compartment, in which the shock was not activated. This group could escape the shocks by jumping, so they had some control over what happened to them. For the second group, however, both compartments were electrified, so this group of dogs received a shock no matter what they did – they had no control over negative events.

In the second stage of the experiment, both groups of dogs could escape the shock by jumping the barrier and entering the opposite compartment. However, the dogs that had no control in the first part of the experiment didn't do this; instead they cowered passively in a corner and whimpered. Even when the experimenters dragged them across the barrier into the safe chamber, they did not learn that they could escape the shock. These dogs had learned to be helpless.

Subsequent studies replicated the basic learned helplessness effect in animals and demonstrated that this also occurs in humans (for a review and some discussion, see Forgeard et al., 2011). For example, Hiroto and Seligman (1975) demonstrated that if participants were exposed to an aversive noise that they could not turn off, they were slow to learn to press a button in order to terminate the noise in a second phase of the experiment. The learned helplessness theory of depression (Seligman, 1975) evolved from these experiments: it suggests that depression arises from a perception that environmental events cannot be controlled. For example, loss of a loved one or repeated abuse may lead to passivity and a belief that the person is unable to prevent negative things that might happen to them in the future.

When people learn to be helpless, this can be seen in cognitive changes (they believe that whatever they do, bad things will happen), motivational deficits (they have no motivation to try to change things) and emotional changes (depressed mood). Seligman's subsequent studies with dogs showed that helpless animals showed other biological changes that were associated with depression: reduced aggression, a loss of appetite and reduced serotonin function (Maier & Seligman, 1976). Therefore, learned helplessness was a plausible explanation for why uncontrollable negative life events could lead to the development of major depression.

Unfortunately, the original formulation of learned helplessness theory struggles to explain the importance of 'dependent' versus 'independent' life events on the development of major depression. Think of someone who loses their job. This might happen because their employer went bankrupt, in which case this would be 'independent' of how the person behaved. Alternatively, the person might have been a bully and they were sacked when their employer had finally had enough of them. This would be a 'dependent' life event, because the person's behaviour was directly responsible for the outcome (i.e. losing their job). Based on learned helplessness theory, we would expect people to be more likely to become depressed after experiencing independent rather than dependent negative life events. But this is not generally the case: people are more likely to become depressed if they experience a negative life event that they had some influence on (a dependent event) rather than if they experienced an independent negative life event (Hammen, 2005).

Learned helplessness theory had a cognitive makeover when it was revised by Abramson, Seligman, and Teasdale in 1978. According to reformulated helplessness theory, the experience of helplessness is not enough to cause depression. Instead, 'when a person finds that he is helpless, he asks *why* he is helpless. The causal attribution he makes then determines the generality and chronicity of his helplessness deficits as well as his later self-esteem' (Abramson et al., 1978: 50).

Reformulated helplessness theory posits that people with major depression make causal attributions about negative life events that have the following characteristics:

- Events are attributed to *internal* (rather than external) factors, for example: 'It's my fault that I fell out with my friend, because I behaved badly.'
- Events are attributed to things that are *stable over time* (rather than something that was specific to that particular time), for example: 'I fell out with my friend because I am a bad person, and I will always be a bad person.'

- Attributions are *global* (rather than something that was specific to that occurrence), for example: 'I am a bad person and not only does this make my friends dislike me, it also makes me perform badly at work.'

Abramson et al. (1978) suggested that people who make these types of causal attributions are more likely to blame themselves for negative events and to expect to experience negative events in the future. The resulting expectations lead to increased helplessness, a loss of self-esteem and feelings of hopelessness. There is good evidence that depressed people think in this way, which has been called a 'depressogenic' attributional style. For example, Quiggle et al. (1992) reported that depressed children were more likely than nondepressed controls to attribute negative life events to internal, stable and global causes. Furthermore, prospective studies show that a depressogenic attributional style predicts the onset of depressive symptoms in response to a negative life event (Metalsky et al., 1993). A large longitudinal study from Alloy et al. (2006) found that a depressogenic attributional style predicted depressed mood at later time points, and similar findings have been reported in children (Abela, 2001) and adolescents (Auerbach et al., 2014)

Reformulated helplessness theory may describe the way that depressed patients think, but it doesn't explain why they think in this way (and non-depressed patients do not). What we need to know is why some people acquire this depressogenic cognitive style in the first place. Alloy et al. (1999) extended the theory by proposing that attributions have a developmental origin. Given that depression tends to run in families, they proposed that depressed parents have a depressogenic cognitive style and their own children acquire this attributional style as they are growing up, which ultimately causes them to develop depression themselves. After reviewing the evidence, Alloy et al. identified four pathways by which children could acquire attributional styles from their parents:

- *Modelling*: children might learn to explain environmental events simply by copying their parents' attributions. The evidence for this was mixed, with some studies finding an effect and others failing to do so.
- *Parental feedback*: depressed parents provide depressogenic feedback to their children about the causes of negative life events ('You fell off your bike because bad things always happen to our family'). There was some evidence for this happening in interactions between depressed parents and their children.
- *Parenting style*: parents who suffer from depression are likely to adopt a critical, commanding and threatening style of parenting, and this can lead their children to develop depressogenic cognitions.
- *Childhood maltreatment*: neglect and emotional, physical and sexual abuse during childhood are associated with a depressogenic cognitive style during adulthood. This relationship may be particularly strong for emotional abuse, for example being told 'Of course you didn't get invited to the prom. You're ugly'. Alloy et al. (1999) speculate that comments such as this could be internalised, leading to the formation of depressogenic cognitions.

Figure 5.2 Children may learn to explain life events by copying their parents' attributions for them

Beck's cognitive model of depression

Beck's (1967) theoretical model of depression and his formulation of cognitive therapy (Beck, 1976) are based on the idea that during childhood we acquire a set of schemata, a 'world view', based on our early experiences and/or by modelling the world view held by our parents (as discussed in the previous section). If children have negative experiences such as major trauma (e.g. the death of a parent, parental divorce), rejection or criticism from friends, parents or teachers, or if their parents have a negative view of the world, then they are likely to acquire dysfunctional beliefs about the world. These negative schemata will usually lie dormant but can be 'reactivated' by negative life events in the future. So, for example, failing your psychology exam might trigger a set of beliefs that were formed during childhood, such as 'I am stupid' and 'I always disappoint the people who love me'. Once these negative schemata are (re)activated, this leads to a stream of what Beck called negative automatic thoughts (NATs), which are negatively valenced intrusive thoughts that the person cannot control. Other symptoms of depression such as negative mood and reduced motivation, together with automatic cognitive biases for negative information (see below), follow on from this barrage of negative thoughts. This depressogenic cognitive style is then maintained by a number of cognitive distortions or logical errors that influence how the person will interpret life events (see Box 5.6).

Box 5.6 Essential experience

Examples of cognitive distortions ('logical errors') proposed by Beck (from Field, 2003)

- *Arbitrary inference*: if you visit your friend but they do not answer the door, you assume that they are ignoring you.
- *Selective abstraction*: in the early days of a new romantic relationship, the person tells you they would really like to see you again and that they really like you but that they're busy for a few days. You interpret their unavailability as a signal of their 'true' feelings, that is, that they don't really like you.
- *Overgeneralisation*: you have an argument with an acquaintance and this causes you to think that all of your friends dislike you.
- *Magnification and minimisation*: magnification would be taking a relatively minor incident and blowing it out of proportion; for example, if you are late to meet someone this makes you think 'All of my friends will think I'm always late'. Minimisation would be playing down positive feedback; for example, if someone tells you that 'You look good', you take it to mean 'They are telling me that I look slightly less disgusting than normal'.
- *Personalisation*: this is the 'world revolving around me' syndrome. For example, if nobody seems to be having fun at a party you assume that it must be your fault.
- *Absolutistic dichotomous thinking*: for example, 'If I fail my exams, my life is ruined', or 'Without my girlfriend, I am nothing'.
- *'Should' and 'must' statements*: for example, 'I must be best at everything' and 'I must be liked by everyone'. Even for the most high achieving and popular person, these statements are unlikely to be true *all* of the time.

There is a great deal of overlap between Beck's model and reformulated helplessness theory (Abramson et al., 1978; remember that Beck's theory was published first!). Beck's theory can incorporate Abramson et al.'s ideas about attributional style, but arguably the additional cognitive distortions specified by Beck (e.g. those in Box 5.6) make it a more complete cognitive theory of depression. Many clinical reports suggest that depressed patients do think in the way described by Beck (Haaga et al., 1991). There is also good evidence that depressed patients have automatic cognitive biases for negative information, as predicted by the theory. For example, people with major depression have a memory bias: they preferentially remember negative information. They also have an interpretive bias: they are more likely to infer negative information from ambiguous scenarios. Finally, they have an attentional bias for negative information, in the sense that they struggle to disengage their attention from such

information once they have focused on it (Gotlib & Joormann, 2010). However, while this evidence suggests that Beck's model is a useful way of *describing* the depressive thinking style, it doesn't tell us whether depressogenic cognitions are the *cause* of depressive episodes.

If depressogenic cognitions have a causal influence on depression rather than being an irrelevant by-product of negative mood, then the onset of depressive episodes should be explained by an interaction between depressogenic cognitions and the occurrence of stressful life events. Results from several longitudinal studies support this prediction, and many of these were discussed in the previous section as support for the reformulated helplessness model. For example, Brown et al. (1995) looked at depressive symptoms in students who didn't do as well as they expected to in their exams. As they predicted, they found that depressogenic cognitions (which were measured before the students got their results) interacted with the extent to which the students underperformed in their exams to predict the severity of depressive symptoms shortly after the students got their results. In another study, Kwon and Oei (1992) found that the interaction between depressogenic cognitions and negative life events predicted symptoms of depression three months later. Alloy et al.'s (2006) cognitive vulnerability to depression (CVD) project studied a large sample of undergraduates over a five-year period. They found that people with high levels of depressogenic cognitions at the start of the study were much more likely to be diagnosed with major depression at the end of the study than participants with low levels of depressogenic symptoms. Furthermore, any participants who were cognitive 'high risk' and had a history of major depressive episodes in the past were much more likely to experience a recurrence of depressive symptoms (i.e. another major depressive episode) than participants who also had a history of major depressive episodes but were cognitive 'low risk'. However, one review of the evidence concluded that the effects from these longitudinal studies were small and many of the methods used were inadequate (Lakdawalla et al., 2007).

Mood priming experiments are another way to test Beck's theory. In these experiments, researchers compare the effects of a laboratory mood induction (e.g. listening to sad music or watching a sad film) on depressogenic cognitions in remitted-depressed and control participants. According to Beck's theory, all participants should report depressed mood after negative mood induction, but only people who have a cognitive vulnerability to depression should show an increase in depressogenic cognitions. Several studies have demonstrated such findings, and therefore this is strong support for Beck's theory (reviewed by Scher et al., 2005). For example, Miranda and colleagues (1998) reported a study in which healthy controls and remitted depressed patients initially completed the Dysfunctional Attitudes Scale (DAS), a questionnaire that measures many of the negative thinking styles that are central to Beck's theory. Participants then watched either a depressing or a neutral film before completing the DAS again. DAS scores increased in the remitted depressed patients who had seen the depressing film (but not those who had seen the neutral film), while DAS scores did not change in the group of healthy controls regardless of which film they had watched. Therefore, depressogenic cognitions can be 'reactivated' by a depressing life event in patients who have experienced major depressive episodes in the past. Importantly, other studies have shown that the extent to which depressogenic cognitions reappear after negative

mood induction in remitted depressed patients predicts the recurrence of major depressive episodes in the future (Segal et al., 1999). These studies support Beck's theory because they show that depressogenic cognitions are 'latent' in patients with a history of major depression, just waiting to be activated by a negative life event.

More recently, Beck (2008) showed how his model could be integrated with genetic influences on depression (see Section 2) and abnormalities of brain function (earlier in this section). For example, variations in serotonin transporter genes have been linked to hyperactivity of the amygdala, which in turn is related to negative cognitive biases. Finally, the clear evidence that CBT is an effective treatment for major depression strongly suggests that depressogenic cognitions play a role in maintaining depression, because changes in these depressogenic cognitions that occur during a course of CBT are what cause a patient's mood to improve during treatment (see Section 4).

Cognitive processes in mania and bipolar disorder

While the cognitive processes involved in major depressive disorder have been well researched and are fairly well understood, we cannot say the same for manic episodes and bipolar disorder. During depressive episodes, patients with bipolar disorder display the same types of depressogenic cognitions as those with major depression (Scott et al., 2000), and much of the evidence discussed above applies here. One influential theoretical model of cognitions in bipolar disorder (Winters & Neale, 1985) proposes that when patients experience a negative event this would normally trigger a major depressive episode. Alternatively, in some (not clearly specified) circumstances, bipolar disorder patients may have a defensive reaction to these negative cognitions that takes the form of a manic episode. Evidence in support of this theory comes from a study (Lyon et al., 1999) which showed that bipolar disorder patients had implicit negative beliefs about themselves regardless of whether they were experiencing a depressive or manic episode at the time. However, patients who were in the middle of a manic episode reported positive beliefs about themselves, whereas patients who were in the middle of a depressive episode reported negative beliefs about themselves. Therefore, both manic and depressive episodes are associated with negative automatic thoughts, but bipolar disorder patients during manic episodes may try to compensate for this by reporting that they feel positively about themselves ('manic defence'). Other theories have focused on the behavioural approach system (BAS), which is implicated in motivational responses to rewarding stimuli. BAS activity is elevated during manic episodes, and therefore it may be useful to consider this individual difference in order to understand the root causes of manic episodes. However, while the model can explain mania, it cannot account for the cycling between manic and depressive episodes that is seen in bipolar disorder (Johnson et al., 2012).

Section summary

In this section we have shown that we can investigate brain dysfunction in mood disorders at two different levels: individual neurotransmitters, and different regions of the brain. Whilst serotonin and noradrenalin function seem to be disrupted in major depression and bipolar

disorder, this does not provide a complete picture. It is more useful to think about different regions of the brain, and interconnections between different brain networks, in order to understand what goes wrong in the brain in mood disorders. This doesn't mean that we can ignore neurotransmitters – this is how different brain regions communicate with each other, after all – but it does mean that neurotransmitters are only one part of the overall picture.

Cognitive theories of major depression are able to explain the core symptoms of the disorder, and they provide a convincing explanation of how the disorder develops and how the symptoms are maintained. There is overwhelming evidence that depressogenic cognitions can be triggered by negative life events, and that once activated they play a causal role in vulnerability to depression. These theories are beginning to be integrated with biological models of mood disorders. Cognitive distortions can also explain why bipolar disorder patients experience major depressive episodes, although our understanding of why bipolar disorder patients cycle between manic and depressive episodes is limited.

Section 4: How are mood disorders treated?

Pharmacotherapy (drug treatments)

Antidepressant medications for major depression increase the activity of the monoamines, particularly serotonin and noradrenalin. The various types are monoamine oxidase inhibitors (MAOIs, e.g. phenelzine), tricyclics (e.g. amitryptiline) and selective serotonin reuptake inhibitors (SSRIs, e.g. fluoxetine; the best known brand of this is Prozac) and noradrenalin reuptake inhibitors (NRIs, e.g. venlafaxine). Although different types of drugs have different mechanisms of action (and MAOIs are now rarely used because of their side effects), they all ultimately work because they prevent the reuptake or breakdown of serotonin or noradrenalin from the synapse. This means that these drugs increase the amount of serotonin or noradrenalin activity in the brain. When taken for long periods of time, they enhance transmission within these neurotransmitter systems.

There is little doubt that antidepressants are effective in the sense that people who take them are more likely to get better: around 50% of those who take them report significant improvements in mood (Anderson et al., 2008). Furthermore, antidepressants reduce the risk that patients will experience a recurrence of symptoms (i.e. another major depressive episode) by about 70%, compared to receiving no treatment at all (Anderson et al., 2008). There is also an emerging body of evidence showing that patients treated with antidepressants show improvements in cognitive biases, for example improvements in memory biases such that tendencies to recall more negative rather than positive material are reduced in people who have received antidepressants (Harmer et al., 2004). Harmer and Cowen (2013) showed that antidepressant-induced improvements in cognitive biases were seen slightly before the drugs led to an improvement in depressed mood. They suggested that improvements in cognitive biases might ultimately explain why antidepressants work: the drugs alleviate automatic cognitive biases, and this takes a while to filter through and influence subjective mood.

However, all types of antidepressants have side effects, although these are less severe with the SSRIs. In Box 5.7 we discuss the controversial topic of the role of the placebo effect in the response to antidepressant drugs. Another important point to note about antidepressants is that people are very likely to relapse (i.e. experience another major depressive episode) when they stop taking them, whereas patients who receive CBT seem to be more resilient to depression after they have finished the treatment (see Box 5.8 later).

Box 5.7 Essential debate

The drugs don't work … or do they?

A meta-analysis of clinical trials of antidepressants hit the headlines in 2008 (Kirsch et al., 2008). Unlike previous meta-analyses, this one was based not just on published trials but also on unpublished trials from pharmaceutical companies. Their results were startling: compared to a placebo, antidepressants were only minimally effective at alleviating the symptoms of depression, and the effects were moderated by the severity of depression. In severely depressed patients, antidepressant drugs had a modest effect on symptoms. But in mildly and moderately depressed patients, the drugs were not effective at all! This isn't to say that patients who receive an antidepressant in the real world will not get better (many of them will). Instead most people will improve if they receive a placebo, and only a minority will show an additional improvement if they receive an antidepressant drug.

The implications were clear. To begin with, the majority of depressed patients (who are not classed as 'severely' depressed) may as well take a sugar pill, and added to this, the apparent effectiveness of antidepressants could be explained by pharmaceutical companies hiding the results from trials that did not show a benefit for their drugs over a placebo.

However, other researchers – most of them not connected to the pharmaceutical industry – have been very critical of this study. A subsequent meta-analysis from Horder and colleagues (2011) argued that Kirsch et al. (2008) had used inappropriate data analyses, and when more appropriate analyses were used (on the same data) the benefits of antidepressants over a placebo were much larger. Horder et al. (2011) were also very critical of many of the assumptions made by Kirsch et al. (2008), and the way in which they interpreted their results. Both papers are an entertaining read (and not too technical) and come highly recommended.

One thing that researchers can agree on is that there is a big placebo response to antidepressants, in other words many depressed patients who take part in drug trials will get better even if they receive a placebo. It is also clear that patients who receive an antidepressant drug will show a bigger improvement than patients who receive a placebo. The ongoing debate is how large, and how clinically significant, that difference between antidepressants and a placebo actually is.

Lithium and other medications for bipolar disorder

As with many psychiatric medications, the psychological effects of Lithium (a common salt which used to be an ingredient in 7-Up!) were discovered by accident. The drug is effective at stabilising mood in about 60% of patients, and it prevents bipolar disorder patients from oscillating between depressive and manic episodes (Geddes et al., 2004). Its mechanism of action is unclear, but it may ultimately work because of a general effect on neurotransmission throughout the brain. Given its effectiveness, it is currently recommended as a first-line treatment for bipolar disorder. However, it is more effective at blunting manic episodes than alleviating depressive episodes, and for this reason many bipolar disorder patients may be prescribed other medications at the same time. Importantly, the side effects of lithium (which can be toxic) mean that patients require careful monitoring when they are maintained on the drug, and many patients stop taking the drug because of the side effects. Other medications, such as Valproate (an anti-seizure medication originally developed for the treatment of epilepsy) and some antipsychotic drugs, are also effective for the treatment of manic episodes (Goodwin, 2009).

Cognitive behaviour therapy (CBT) for major depression

The primary aims of CBT are to educate clients about the role of negative cognitions in mood disorders, and to teach strategies that will help them to think in a more positive (or optimistic) way (Beck, 1976). There is also a behavioural element to this: given that depression is associated with anhedonia and a reluctance to engage in activities that might otherwise be viewed as pleasurable, behavioural activation and event scheduling are used to increase activity and engagement in activities, such as going to the shops or socialising (Kanter et al., 2010). Clients usually receive between 6 and 20 one-to-one sessions with a qualified CBT therapist, although the homework that clients do between sessions and afterwards is recognised as a vital component of therapy.

In order to challenge depressogenic cognitions, a client is taught exercises that can help them evaluate their pessimistic attributions for negative life events rather than accepting them uncritically. Some of the techniques that a therapist might use are as follows:

- *Thought catching*: the client recalls a recent incident that led to depression and then lists their thoughts and feelings at the time. The therapist and client then determine which thoughts were reasonable reflections of reality, and which were negative automatic thoughts (NATs) brought on by the incident.
- *Task assignment*: the client thinks of activities that they are avoiding (e.g. they don't go to social events) and then makes predictions about the bad things that would happen if they were to engage in these (e.g. being ignored by others). After the therapy session, the client completes the activity, and at the next session the client and therapist discuss the extent to which the client's predictions were accurate. The client will usually have overestimated the negative consequences of engaging in the activity.

- *Reality testing*: this is similar to the above but is focused on disproving specific beliefs. The client generates tasks to test the reality of a given belief (e.g. phoning a friend to disprove the belief that the friend does not want to speak to them). The client completes the activity and at the next session the client and therapist discuss the outcome; again, the idea is that the client will realise that their expectations were unrealistically negative.
- *Cognitive rehearsal*: once the client has performed some of these tasks, they move on to using cognitive rehearsal. For this they think of potentially negative situations that are likely to arise in the future, and plan for how they will apply task assignment and reality testing to a situation. The general idea is that the client starts to use these cognitive techniques in their everyday life, in a range of situations. Eventually, they will get better at identifying and challenging their negative automatic thoughts, and thinking in a more positive and optimistic way during challenging situations.

CBT for depression is undoubtedly effective. In a meta-analysis of previous meta-analyses (serious amounts of data!) Butler et al. (2006) reported overall large effect sizes for CBT in comparison to other treatments, including other psychological therapies (also see Tolin, 2010). There is some evidence that CBT may be more effective for patients with less severe depression, that patients with severe depression may respond better to antidepressants first, and that they may be more receptive to CBT once their mood has stabilised.

Direct comparisons of CBT with antidepressants generally reveal that the treatments are equally effective in the short term, but CBT has a more enduring effect. In a now-classic study, Hollon et al. (2005) followed up remitted depressed patients for two years after they had finished a course of either antidepressants or CBT. Of those who were symptom-free one year after treatment had finished, 80% of patients who had received CBT remained symptom-free one year later, in contrast to 50% of patients who had received antidepressants. Studies such as this have led to suggestions that CBT might work because it offers a kind of cognitive 'vaccine' against future episodes of depression (see Box 5.8).

Box 5.8 Essential treatment

Is CBT a cognitive vaccine?

Clients who receive CBT are at reduced risk of experiencing a relapse (i.e. another major depressive episode) in the future (Hollon et al., 2005). This might mean that CBT works as a cognitive 'vaccine', because it stops NATs from re-emerging whenever clients experience a negative life event. A study by Segal et al. (1999) suggests that this might be the case. Remitted depressed patients who had previously been treated with either CBT

(Continued)

(Continued)

or antidepressants completed measures of depressogenic cognitions before and after exposure to a negative mood induction procedure. They found that the negative mood induction caused depressogenic cognitions to increase in both groups, but to a much smaller extent in the group that had previously received CBT. Perhaps most notably, individual differences in cognitive reactivity to the mood induction procedure predicted a recurrence of depressive symptoms later on. Taking these findings together, it seems that CBT can reduce the activation of depressogenic cognitions in response to negative events, and this in turn is associated with a reduced risk of recurrence of symptoms.

Other studies of CBT have shown that improvements in depressive symptoms only happen after depressogenic cognitions have changed, and that for patients who receive CBT, cognitive change mediates the subsequent change in negative mood (see Garratt et al., 2007). By contrast, patients who receive other types of treatment (including antidepressants and other psychological treatments) also show improvements in depressogenic cognitions, but these changes occur after improvements in negative mood in these patients (Garratt et al., 2007). In other words, different types of treatment might ultimately result in improved mood and a more optimistic way of thinking. The important difference is that the cognitive change is what causes the improvement in mood among patients who receive CBT, but patients who receive other types of treatment show improvement in mood first, and changes in cognition follow on afterwards.

Figure 5.3 Adopting a more positive, optimistic cognitive style may explain why CBT leads to improved mood in depressed patients

Other treatments for major depression

You have probably heard about electro-convulsive therapy (ECT), which involves the application of a high voltage electrical current to the dominant brain hemisphere, usually over repeated sessions. It is not a first-line treatment for major depression (because of its side effects), but may be offered to patients with severe depression who have not responded to antidepressants or are at high risk of suicide. It is considered effective for such patients, and its beneficial effects are seen more quickly than with antidepressants (McCall, 2001). Other types of psychological treatment, including mindfulness (Piet & Hougaard, 2011), are also effective, although CBT usually emerges as the most effective treatment when compared with others.

Psychological treatments for bipolar disorder

CBT for bipolar disorder utilises many of the same approaches as those used in regular CBT for depression (see above), but with additional components aimed at challenging cognition and behaviour during manic episodes. A meta-analysis from Szentagotai and David (2010) concluded that this approach had small but reliable effects on reducing symptoms of bipolar disorder, although the effects at follow-up (and prevention of recurrence of manic or major depressive episodes) were not impressive. As a consequence, CBT (and other psychological therapies) may be recommended as well as medication, but there is no convincing evidence that they should replace medication as the primary treatment for bipolar disorder.

Section summary

In this section we have shown that both antidepressant medication and cognitive behaviour therapy are effective treatments for major depression. Although both are equally effective in the short term, CBT has the edge in the longer term, perhaps because it has more enduring effects on depressogenic cognitions. Drug therapy remains the first-choice treatment for bipolar disorder because it improves symptoms in the majority of clients who take it.

Essential questions

Some possible exam questions that stem from this chapter are:

- Why do negative life events cause depression to develop?
- Critically evaluate the role of gene × environment interactions in the development of depression.
- Do depressogenic (pessimistic) cognitions play a role in the development of major depressive disorder, and in the recurrence of major depressive episodes after recovery?
- Do antidepressants work?
- Does cognitive behaviour therapy improve the symptoms of major depressive disorder, and if so how does it work?

Further reading

Beck, A. T. (2008). The evolution of the cognitive model of depression and its neurobiological correlates. *American Journal of Psychiatry*, 165(8): 969–977. (An excellent overview of the cognitive model of depression from the guru of this topic, with some accessible explanations of how the theory relates to brain dysfunction in mood disorders.)

Garratt, G., Ingram, R. E., Rand, K. L., & Sawalani, G. (2007). Cognitive processes in cognitive therapy: Evaluation of the mechanisms of change in the treatment of depression. *Clinical Psychology: Science and Practice*, 14(3): 224–239. (Engaging discussion on the role of cognitive change in the mechanism of the action of different treatments for major depression, with a focus on CBT.)

Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and construct diatheses in unipolar depression. *Clinical Psychology Review*, 25(4): 487–510. (Very readable dissection of the evidence on the causal influence of depressogenic cognitions on depressed mood.)