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Exploring depression

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Contents

Introduction

If you are about to embark on this course, please be aware that this is the second part in a three-part series covering anxiety and depression. We would encourage you to start with *Exploring anxiety*.

Depression represents a major global health concern. The condition contributes significantly to the overall global burden of illness and is ranked highest (with over 63 million Disability Adjusted Life Years or 'DALYs'; see *Exploring Anxiety* Section 3.3) amongst the DALYs attributable to mental, neurological and substance use disorders, according to the WHO's Global Burden of Disease Study 2010 (Whiteford et al., 2015). The 12-month prevalence of depression in Europe is 6.9% (Bschor et al., 2014). Anxiety disorders can precede depression, and symptoms can overlap, with anxiety often co-existing with depression. The course of illness can be episodic with periods of ill health and recovery. More than half of depressed patients recover within six months, but in around 15% of affected individuals, depression can become chronic and severe and some may experience long-term depression lasting several years (Bschor et al., 2014).

Resistance to treatment is a common issue −a third or more of patients do not respond to trials of several weeks of treatment with antidepressant medications (Bschor et al., 2014). The 'Sequenced Treatment Alternatives to Relieve Depression' (STAR*D) trial, a large study involving 4,000 participants in the United States, found 37% to be in remission after a 12-week treatment period with antidepressants, and 67% after four treatment trials (Rush et al., 2006).

Risk factors for developing chronic depression include family history of mood disorders, early onset depression, and long duration of depressive episodes. Comorbidity with anxiety disorders, personality disorders, substance dependence or abuse, poor social integration and negative social interaction are factors commonly associated with depression (Bschor et al., 2014).

But what does it actually *feel like* to experience depression? Is depression on the increase? Is this an illness of modern civilisation? Can depression serve as an adaptive response – is there any benefit to depressed mood? Why do some people who experience traumatic life events become vulnerable to depression while others do not? Why is it that around a third or more of people with depression do not respond to current antidepressant treatments? What other emerging treatment options are out there? We will explore these and other questions next.

This OpenLearn course has been developed from the Open University course *S826 Introduction to mental health science* (Stage 1 in the Masters in Mental Health Science), and is suitable preparatory reading if you are considering moving on to postgraduate study in this area. A number of related free courses are also available on OpenLearn. They are recommended to complement your studies. They can serve as background reading, introduce you to underlying concepts, and provide a basis that will help to support and broaden your knowledge and understanding of topics further. You can find these in the Further Reading section.

Please note that a glossary of terms is not provided on this course. However, you may find it helpful to keep one as you study. The course may contain some specialist vocabulary, terms or ideas with which you are unfamiliar, such as a medical condition, a complex technical term or a specific procedure or assessment. At this more advanced level of

study, we expect you to use your initiative and find the missing information for yourself, perhaps using medical dictionaries or encyclopaedias, or by conducting an online search using a search engine. Searching for information is also an overt feature of study at Masters level, and will help you to better prepare for postgraduate study.

Learning Outcomes

After studying this course, you should be able to:

- understand depression from biopsychosocial perspectives
- discuss contemporary issues in mental health science related to depression
- recognise different lines of evidence and appreciate the uncertainty, ambiguity and limits of current knowledge in the study of mental health science.

1 The experience of depression

The main forms of depression and related conditions are summarised in Box 1. Diagnostic criteria also distinguish between less severe and chronic forms of depression. Treatment-resistant depression and dysthymia are defined in Box 2. We will look at diagnostic criteria later on, but the information provided here should serve as a useful reference as you read further and engage with the activities in that follow.

Box 1 Depression and related conditions

(based on NIMH, 2015)

Major depression involves severe symptoms that interfere with the ability to work, sleep, study, eat, and enjoy life. An episode can occur only once in a person's lifetime, but more often, a person will experience several episodes.

Persistent depressive disorder is where depressed mood lasts for at least two years. A person diagnosed with persistent depressive disorder may have episodes of major depression along with periods of less severe symptoms, but symptoms must last for two years.

Other forms of depression include:

Psychotic depression, which occurs when a person has severe depression plus some form of psychosis, such as having disturbing false beliefs or a break with reality (delusions), or hearing or seeing upsetting things that others do not (hallucinations).

Postpartum (postnatal) depression, which occurs in around 10-15% of women after childbirth, and is more serious than the 'baby blues' that new mothers may experience after giving birth.

Seasonal affective disorder (SAD) is characterised by the onset of depression during the winter months, when there is less natural sunlight. The depression generally lifts during the spring and summer. SAD can be treated with light therapy, but nearly half of those with SAD do not get better with light therapy alone, and will require antidepressant medication and psychotherapy to reduce symptoms.

Bipolar disorder is also sometimes called 'bipolar depression', but the condition is different from other forms of depression− someone with bipolar disorder will typically experience episodes of extremes of mood (highs and lows) as well as symptoms of psychosis (delusions, hallucinations, disordered or disorganised thoughts).

Box 2 Treatment-resistant depression and dysthymia − definitions

(adapted from Bschor et al., 2014)

Treatment-resistant depression* typically refers to depression which does not immediately respond to 'standard' treatment with antidepressant drugs (but note that drug treatment is only one of the therapeutic options available for depression). Depression is termed 'treatment-resistant' if two trials of drug treatment, each at an adequate dose administered over a sufficient course (period of time), have had no beneficial effect. This definition too is partly arbitrary, however; more specific

definitions grade treatment resistance according to the number of failed treatment attempts.

Dysthymia according to ICD-10, is a state consisting of a depressive syndrome, lasting for several years, with lesser severity than in depression as strictly defined (including chronic or 'major' depression). The once separated diagnoses of 'dysthymia' and 'chronic major depression' have been grouped together in DSM-5 on account that 'diagnostic and therapeutic differences between the two entities' were deemed 'too small to warrant separate classification' (Bschor et al., 2014).

NOTE: (*) The time at which depression becomes 'chronic' by definition has been set, more or less arbitrarily, at two years. Chronic and treatment-resistant depression present the same symptoms as an acute depressive episode, but the following tend to be most prominent: low affective variability, anhedonia (an inability to find pleasure in activities one would normally find pleasurable), lack of drive, social withdrawal, lack of self-esteem, hopelessness, loss of libido, sleep disturbances, cognitive impairment and chronic suicidality.

Activity 1 The experience of depression

Allow 60 minutes

Watch the video below and consider the questions that follow. You might wish to view the entire recording first and then watch the video again thinking specifically about the questions the second time around, or you can review the questions as you watch the video from the start. Choose whichever approach suits you best.

Video content is not available in this format.

TED Talk: Andrew Solomon – The Secret We Share

In this moving and eloquent talk given in 2013, Andrew Solomon, Professor of Clinical Psychology at Columbia University, and author of *The Noonday Demon: an atlas of depression* (Andrew also writes regularly for *The New Yorker* and the *New York Times*), describes his personal experiences of living with depression, recounts the stories of others he has come into contact with, and describes how he has come to terms with depression.

1. Andrew explains that he had always thought of himself as being tough, as 'one of the people who could survive if[he]had been sent to a concentration camp' as he puts it. What triggered his depression?
2. How does he describe his depression?
3. Andrew also experienced anxiety. How did his anxiety develop and how long did it last?
4. When his depression became severely disabling, Andrew managed to seek help and started with medication and therapy. What does he consider as his two advantagesgoing'into the fight'?

5. He describes the relentless relapsing and remitting course of his illness. How did Andrew come to terms with his depression at the time?
6. He points out that people tend to confuse depression, grief and sadness. What important distinctions does Andrew make?
7. Andrew set out to understand depression, to find out what causes some people to be more resilient than others, and to know what mechanisms allowed people to survive. He gives the example of his friend Maggie Robbins, a poet and psychotherapist. How did Maggie experience depression? What thoughts did she and others experiencing depression typically have?
8. What therapies for depression does Andrew describe?
9. Andrew says that he was struck by the fact that ‘depression is broadly perceived to be a modern, Western, middle-class thing’. He touches upon cultural views on depression and different approaches to treatment, talking about his visit to Senegal and Rwanda. How does he describe some of these differences?
10. Through researching Andrew says that he has discovered that ‘depression is the result of a genetic vulnerability, which is presumably evenly distributed in the population, and triggering circumstances, which are likely to be more severe for people who are impoverished’, but that this is not being picked up, treated or addressed. What explanation does he give for this?
11. How does Andrew respond to questions like ‘Isn’t depression part of what people are supposed to experience? Didn’t we evolve to have depression? Isn’t it part of your personality?’
12. What is his response to whether depression is 'continuous with normal sadness'?
13. What does Andrew think is the main mechanism of resilience?

Discussion

1. Andrew says that he had a series of losses in 1991. His mother died, a relationship he had been in ended. He moved back to the USA after being abroad and then three years later he found himself losing interest in almost everything. He didn’t want to do any of the things he had previously done, and he did not know why.
2. Everything he did seemed to involve so much work. He seemed to think about and view things differently. He gives an example of messages left by friends on his answering machine, and instead of feeling thrilled to hear from them, he would think to himself ‘what a lot of people that is to have to call back’. Carrying out everyday things such as having lunch would become a burdensome task – he would think about having to ‘get the food out and put it on a plate and cut it up and chew it and swallow it’. And he knew ‘it was ridiculous’ while he was experiencing these thoughts, and should not be a big deal, but when in its grip ‘unable to figure out any way around it’, so he began to feel himself doing, thinking and feeling less, which he describes as a kind of ‘nullity’.
3. Andrew talks about his acute anxiety ‘setting in’ after his initial bout of depression and lasting for six months, as a ‘sensation of being afraid all the time, but not even knowing what it is that you are afraid of’.
4. First, Andrew notes that ‘objectively speaking he had a nice life and that if he could only get well, there was something at the other end that was worth living for’. The second advantage was access to good treatment.

5. Andrew says that while thinking about biological (chemical), psychological or philosophical explanations for his depression, he also came to the understanding that it was ‘braided so deep into us that there was no separating it from our character and personality’.
6. Andrew notes that grief is explicitly reactive, and gives the example that if a person experiences loss, feels incredibly unhappy, and then six months later, they are still deeply sad but functioning a little better, it is probably grief which will ‘probably, ultimately, resolve itself in some measure’. If a person experiences a catastrophic loss, feels terrible and six months later can barely function at all, then it is likely to be depression triggered by the catastrophic circumstances. He defines depression as not simply ‘sadness’, rather ‘much too much sadness, much too much grief at far too slight a cause’, and that importantly the trajectory of the illness tells us a great deal.
7. Maggie had bipolar disorder (previously called manic depression), and coming off her medication plunged her into severe depression. Andrew describes her as sitting in her parents’ apartment ‘more or less catatonic, essentially without moving, day after day after day’. He recalls at the time of interview Maggie describing thoughts in her mind telling her ‘you are nothing. You are nobody. You don’t even deserve to live’, leading to thoughts about suicide. He describes other people living with depression having similar thoughts: ‘no one loves me’ or ‘no matter what we do,we’re all just going to die in the end’ or ‘there can be no true communion between two human beings. Each of us is trapped in his own body’. Andrew emphasises that depression can be exhausting and lack of communication about it could make things worse: ‘it takes up so much of your time and energy, and silence about it really does make the depression worse’.
8. Andrew mentions medication, psychotherapy, electroconvulsive treatment, meditation and even relatively simple things that could be done to lift someone’s mood or that they would do to make themselves feel better. He also describes the moving story of Frank Rukosoff, who underwent quite a radical surgical therapy (called a ‘cingulotomy’) to remove a small portion of brain tissue from his frontal lobe, after everything else he had tried had failed. Remarkably, his treatment was successful.
9. Andrew refers to a tribal exorcism in Senegal, how rituals differ between East and West Africa, and how the practice of Western mental health workers can be viewed differently as relayed to him in Rwanda, which he recounts in a lighthearted way: ‘Well, they would do this bizarre thing. They didn’t take people out in the sunshine where you begin to feel better. They didn’t include drumming or music to get people’s blood going. They didn’t involve the whole community. They didn’t externalise the depression as an invasive spirit. Instead, what they did was they took people, one at a time, into dingy little rooms and had them talk for an hour about bad things that had happened to them’.
10. Andrew says that if someone has a lovely life but feels miserable all the time, they would think ‘Why do I feel like this? I must have depression’ and they would set out to find treatment for it. If life is awful all of the time and the person also feels miserable all of the time, the way they feel would be commensurate with their life and it wouldn’t occur to them to think that maybe it could be treated. Note that this is one interpretation only, but it does draw out an important view, and touches not only on societal views but stigma as well.

11. Andrew says that mood – the ability to express sadness, fear, joy and pleasure − is adaptive, and major depression is what happens when that system gets broken. It becomes maladaptive.
12. Andrew agrees that there is a certain amount of continuity, but then gives an example using an iron fence analogy, where severe depression is an extreme form similar in his comparison to leaving the fence untended for 100 years until all that is left is ‘a pile of orange dust’.
13. Andrew believes that people who tolerate, can come to terms with and learn from their depression are the ones who achieve resilience. Those who deny their experience, shutting out their depression, strengthen it. He notes that:‘Valuing one’s depression does not prevent a relapse, but it may make the prospect of relapse, and even relapse itself, easier to tolerate. The question is not so much of finding great meaning and deciding your depression has been meaningful. It’s of seeking that meaning, and thinking, when it comes again "This will be hellish, but I will learn something from it".’

1.1 Understanding depression − key issues

The activities below will explore key issues to help you gain a broader understanding of depression.

Activity 2 Understanding depression: key issues

Allow approximately 1 hour

Listen to the podcast below and consider the questions that follow. You might wish to listen to the entire recording first and review this again thinking specifically about the questions the second time around. Alternatively, you can consider the questions as you listen to the recording the first time around. Choose whichever approach suits you best.

Audio content is not available in this format.

BBC World Service ‘Discovery’ − Depression part 1

In the first of two extracts on depression, broadcast in 2012 on the BBC’s ‘Discovery’ programme, Geoff Watts talks to researchers looking for clues to the origins of depression. In interviews with Professor Bill Deakin, Professor of Psychiatry at the University of Manchester, and Professor Randolph Nesse of the University of Michigan, he asks 'Why has natural selection not made us less vulnerable to psychological illness?', and questions whether depression can in some way be useful in our lives.

1. What proportion of the population will suffer from diagnosable symptoms of depression at some point in their lifetime?

2. Is depression on the increase in today's society?
3. Is depression an adaptive response? Can the capacity to become depressed be beneficial in some way?
4. Explain the 'smoke detector principle' (a concept proposed by Randolph Nesse). How can this explain the increasing prevalence of depression?

Discussion

1. About a fifth of the population, according to Professor Bill Deakin.
2. It appears to be increasing in frequency, according to Professor Deakin – 'the prevalence is increasing and the age of onset is getting less over succeeding decades'. Some of this may be due to more diagnoses being made, and a greater sensitivity to detecting symptoms when first presented to the family doctor (general practitioners). Professor Deakin explains that depression can be manifested in different ways and that some people who present with physical symptoms (e.g. aches and pains that won't go away, and feeling generally unwell) may have depression as the underlying problem.
3. Professor Randolph Nesse explains that many people assume that depression is abnormal, but the real question should be, why have we developed a 'capacity for mood'? 'There are times in life when investing a lot and taking a lot of risks pays off handsomely' and 'there are other times in life, unfortunately, however, when the more effort you put in the more you're wasting effort, and the more risks you're taking', and 'there are somethings that we're trying to do that just aren't working', so the more effort we put in the worse it gets. Dedicating energy towards a goal, and not making any progress can lead to depression. Geoff Watts comments that from an evolutionary perspective, natural selection has helped us to strive for goals to ensure that we survive, whereas 'the goals in our lives today [are] shaped by our desires and the society we live in' and these are equally powerful drivers of behaviour and emotion. The inability to fulfil these goals is, according to Randolph Nesse, what triggers low mood. Geoff Watts asks whether mild depression in itself could be a way of building some sort of resilience 'to help mitigate future bouts of low mood'.
4. Randolph Nesse views pain, nausea, fever, vomiting, anxiety and low mood as normal aversive, defensive responses, and that natural selection has shaped these responses to be greater than they really need to be. They are sensitive and similar to a smoke alarm in the sense that they can go off when they are not really needed (i.e. in the absence of imminent 'real' danger), but this is still a perfectly normal response, 'because the system has to ensure that they go off in situations when they are needed'. So according to Nesse, we 'put up with lots of false alarms on our smoke detectors because we want to be absolutely sure that it does go off when there is a fire'. Geoff Watts comments further that this is 'an ancient biological system, far too sensitive for modern life especially in the culture which proclaims that "only losers quit", even when quitting may be beneficial'. Randolph Nesse's hypothesis is that 'low mood helps us to recognise when our ambitions are too lofty'. The experience of depression should therefore help us to reflect, reappraise our situation, learn from experience and adjust or reset our goals.

Listen to the podcast below and consider the questions that follow. You might wish to listen to the entire recording first and review this again thinking specifically about the questions the second time around. Alternatively, you can consider the questions as you listen to the recording the first time around. Choose whichever approach suits you best.

Audio content is not available in this format.

BBC World Service 'Discovery' − Depression part 2

In the second of the two extracts on depression broadcast in 2012 on the BBC's 'Discovery' programme, Geoff Watts meets researchers studying the brain in people who have experienced traumatic life events, and explores the reasons why some people who experience such traumatic events are vulnerable to severe depression, while others are not. In this extract he talks with Professor Bill Deakin and Dr Rebecca Elliott from the University of Manchester, and Professor Randolph Nesse of the University of Michigan.

1. How does Dr Elliott define 'resilience' in the context of mental health? Are most people resilient to some degree?
2. What three general (hypothetical) aspects to resilience does Professor Deakin describe?
3. Professor Deakin and researchers at Manchester were 'working on the assumption that different states of mind, including vulnerability to depression, should correlate with different patterns of activity inside the brain'. What did they expect to find?
4. Aaron, who is featured in the podcast and has volunteered to take part in the Manchester study, demonstrates some of the characteristics described for resilience. What are these?
5. Dr Elliott talks about the involvement of the amygdala and the prefrontal cortex in resilience. Briefly explain how these brain regions are important in processing emotional information in people who are depressed.
6. Is it possible to 'promote' resilience?
7. How does Professor Deakin see 'neuroscientific' diagnoses operating in the future?
8. Does Professor Nesse support this approach? Does he offer any alternative perspectives?

Discussion

1. Dr Elliott defines resilience as 'how well somebody is able to adapt and function in the face of stressful or traumatic experiences'. She explains that resilience can also be viewed along a 'continuum' in her view, with, at one end, people who may be vulnerable and 'who in the face of quite low levels of stress or even potentially no stress at all will develop a mental health problem such as depression'. At the other end of the continuum there are those whom 'life can deal an appalling hand, they can have all sorts of terrible stressful experiences,

and yet remain positive and optimistic in the face of all that stress'. But most people are somewhere in the middle, 'and we all potentially have a tipping point where enough stress, enough difficulties could cause us to have at least some degree of a problem'.

2. Professor Deakin explains that (i) firstly some people may inherently be more sensitive to rewards in the environment (experiencing pleasurable events put us in a good mood), (ii) some people may be more flexible in how they solve problems (termed cognitive flexibility; i.e. more creative in how they get out of their difficulties), and (iii) some people may be more sensitive than others in how they respond to and process emotions, how emotional responses are triggered (the brain circuitry involved in controlling anxiety, our responses to threat and to loss). Geoff Watts notes that the characteristics of resilience could therefore be thought of in terms of neural circuitry, brain chemistry, previous experiences and genetic inheritance and their interaction.
3. Dr Elliott explains that 'at one end of the continuum, the 'vulnerable' people, we expect to show one pattern. The 'resilient' people at the other end another pattern' and that both should differ from the 'average pattern for people in the middle'.
4. Aaron says that his life can get fairly stressful at times, but that it doesn't affect him in the way that it perhaps affects others. He thinks that this may have something to do with his outlook on life. He tends to think that if there is a problem, a solution can always be found, so he can 'think' his way out of problems. Optimism as well as cognitive flexibility (in problem solving, for example) are aspects to resilience that have already been discussed.
5. Dr Elliott explains that 'in people who are currently experiencing depression, the amygdala is over-responding to negative information. So, if you see a picture of a sad face, and you've currently got depression your amygdala responds more strongly to that sad face'. She notes that the prefrontal cortex 'is important in our cognitive performance, and to some extent in controlling our emotions', and that 'regions of the prefrontal cortex will tend to inhibit functions of regions like the amygdala' and this is imbalanced in depression. She refers to early data suggesting that 'people who are more resilient are more likely to recognise happy faces and less likely to recognise sad or fearful faces', and that 'the more resilient somebody is, the better they remember positive words and positive pictures'.
6. Professor Deakin points to psychological therapies that are based around this concept. Once a person has had depression, the chances that they will have a further episode are substantially increased over that for the general population. Cognitive behavioural therapy, different psychological approaches and inter-personal psychotherapy are 'a good way of reducing the chance of having a further episode' if one has had depression.
7. Professor Deakin sees these working more effectively as tailored to the individual. He explains this using the three aspects he referred to earlier as underlying resilience. For example, in one person, their depression could have been a consequence of 'their reward mechanisms [being] fused', whereas from the 'emotional processing' and 'cognitive flexibility' points of view they would be fine. In such an instance the therapy would be around promoting the ability to respond to reward.
8. Professor Nesse is cautious of methods used to study resilience, but supportive of attempts to compare people who get depressed with those who do not. He notes the complexity of the underlying neurobiology, acknowledging that it turns out depression is not in one place or one neurotransmitter. It's

> distributed in systems, and those systems aren't there just to make us depressed because it's a problem. They're there because the capacity for mood is useful.' He also points to the importance of considering people with depression 'not just as people with diseases who are somehow less able, and less fit than others', but as 'people who have advantages as well as disadvantages'.

Before carrying on with the course, take 10 minutes to reflect on your learning so far.

- Reflect on your learning in this section.
  - What were the key issues or concepts that stood out for you?
  - Can you offer further or alternative perspectives, drawing on your own personal or professional experience?

We will look more closely at diagnosis and explore depression further in Section 2.

2 Exploring depression

Common signs and symptoms associated with depression are indicated in Box 3. Sadness is only one aspect of depression, and some people may not necessarily feel 'sad'. There are other, including physical, symptoms which if lasting for more than two weeks should trigger a person to seek professional support.

Box 3 Common signs and symptoms associated with depression

- Persistent sad, anxious or 'empty' mood.
- Feelings of hopelessness or pessimism.
- Feelings of guilt, worthlessness, helplessness.
- Loss of interest or pleasure in hobbies and activities.
- Reduced energy, fatigue or feeling 'slowed down'.
- Difficulty concentrating, remembering or making decisions.
- Difficulty sleeping, early-morning awakening or oversleeping.
- Changes in appetite and/or weight.
- Thoughts of death, suicide, self-harm or suicide attempts.
- Restlessness, irritability.
- Persistent physical symptoms (aches and pains).

Note: A 'sign' is objective evidence of, and a 'symptom' any subjective evidence of, an illness. Symptoms are experienced by the affected individual, whereas a sign is a phenomenon that can be detected by someone other than the affected individual.

2.1 Depression can affect people in different ways

Depression affects people in different ways (NIMH, 2015). Some people may experience a few symptoms, others many. The severity, frequency and duration of symptoms (how long they last) will vary depending on the individual. Symptoms may also vary depending on the stage of the illness. Depression is also more common amongst women than men (see Table 1).

Table 1. Age-standardised DALYs (per 100,000) attributable to major depressive disorder and dysthymia, 1990 and 2010 (adapted from Whiteford et al., 2015)

Disorder	1990 Male	2010 Male	1990 Female	2010 Female
Major depressive disorder[a]	694.8	689.9	1171.7	1161.2
Dysthymia[b]	135.3	135.8	189.7	190.0

(a) Major depressive disorder accounted for 63,200,000 DALYs (2.5% of all cause DALYs and 24.5% of DALYs attributed to mental, neurological and substance use disorders) in 2010; (b) Dysthymia accounted for 11,100,000 DALYs (0.4% of all cause DALYs and 4.3% of DALYs attributed to mental, neurological and substance use disorders) in 2010.

- From the data presented in Table 1, has there been any change in the relative proportion of DALYs attributed to major depressive disorder over the two decades?
- The relative proportion of DALYs for major depressive disorder and dysthymia have remained relatively constant for both males and females. However, the figure for females was also consistently higher than for males (1.7 fold greater than for males for major depressive disorder, and 1.4 fold greater for dysthymia).

The reasons for the gender disparity are not fully clear but may be linked with biological, psychosocial or other gender-related factors (NIMH, 2015). Women are also vulnerable to depression after giving birth (termed 'postpartum' or 'postnatal' depression), at a time when physical and psychological changes can be overwhelming. Women with depression describe experiencing feelings of sadness, 'worthlessness' or guilt, and men frequently report irritability (anger, or frustration) and insomnia and tend to turn more to alcohol or drugs when depressed. People who suffer from depression may avoid talking with family or friends about their mood or their feelings, and severe depression can turn to suicidal thoughts and suicide attempts.

Children and adolescents may also develop depression. Because typical behaviours associated with transition from childhood to adolescence to adulthood may vary, it can be difficult to identify whether a child or young person is going through a temporary 'phase' or suffering with depression (NIMH, 2015). Frequently, chronic mood disorders such as depression can begin as high levels of anxiety in childhood. The teenage years can be particularly difficult, as young people begin to form their identities, deal with social, gender, sexuality and relationship issues, and start making independent decisions. Severe changes in mood, anxiety, eating disorders or substance abuse could also indicate higher risk for depression and suicide among adolescents. Depression often persists, recurs, and continues into adulthood, if left untreated.

Depression in older adults may be difficult to recognise, as it may be less 'obvious'. Most older adults feel satisfied with their lives, despite living with illness or physical problems, so having depression is not a normal part of growing older (NIMH, 2015). Depressive symptoms may be linked to medical conditions (Alzheimer's disease, heart disease, stroke or cancer) in older people, and major depression can sometimes be difficult to distinguish from grief (the loss of a loved one). However, grief that is complicated and lasts for a very long time following a loss may require treatment. Older adults who have experienced depression when younger are also at higher risk of developing depression late in life. Factors commonly associated with depression are indicated in Box 4.

Box 4 Factors associated with depression

- **Genetic predisposition**
- **Life events** − trauma, loss of a loved one, a difficult relationship, an early childhood experience, or any stressful situation.
- **Early onset** − often in teenage years or young adulthood (early 20s or 30s); most chronic mood and anxiety disorders in adults begin as high levels of anxiety in children, and could mean high risk of developing depression as an adult.
- **Comorbidity with other medical illnesses** − depression can co-occur with diabetes, cancer, heart disease, neurodegenerative disorders such as Parkinson's, Alzheimer's or Huntington's diseases, and can exacerbate these conditions (or vice versa).

- **Medications** − often taken for comorbid illnesses, could contribute in some cases to depression.

2.2 The diagnosis of depression

Boxes 5 and 6 briefly outline the criteria for a depressive episode according to the ICD-10 and DSM-IV-TR classification systems (ICD, 2003; APA, 2000). The DSM-5 criteria for major depressive disorder and persistent depressive disorder are summarised in Box 7. The 11th revision of the International Classification of Diseases (ICD-11) became available on 18 June 2018 and has yet to be used more widely at the time of writing.

Depression is a broad and heterogeneous diagnosis. Low mood, loss of interest and pleasure in activities and loss of energy are among the key symptoms. Severity is determined by the number and severity of symptoms, as well as the degree of functional impairment (activities of daily living). Formal diagnosis of major depression requires at least four out of ten symptoms according to ICD-10 or at least five of the nine symptoms in the DSM (see Boxes 5-7). Symptoms should be present for at least two weeks, and at sufficient severity for most of every day. A depressive episode can be part of a lifetime pattern of disturbance associated with a diagnosis of recurrent depressive disorder, bipolar affective disorder or persistent affective disorder. Symptoms are considered persistent if they continue despite active monitoring or (low-intensity) intervention typically over a period of several months, and to meet the previous diagnosis of ‘dysthymia’, symptoms should be present for at least two years (NICE, 2018).

In DSM-5, what was previously termed ‘dysthymia’ now falls under the category of ‘persistent depressive disorder’ together with chronic major depressive disorder (see Box 7) (APA, 2013b). The reasoning behind this change was ‘an inability to find scientifically meaningful differences between these two conditions’ (APA, 2013b). The core criterion symptoms for a major depressive episode, and the specified duration of at least two weeks have not changed from the previous version (APA, 2013b).

In the UK, up until 28 June 2022, the National Institute for Health and Care Excellence (NICE) clinical guidelines for depression in adults (CG90 ‘Depression in adults: recognition and management’ published October 2009, replacing CG23 published in December 2004) still referred to both the ICD-10 and DSM-IV criteria. The NICE (CG90) guideline stated that:

…..classificatory systems are agreed conventions that seek to define different severities of depression in order to guide diagnosis and treatment, and their value is determined by how useful they are in practice. After careful review of the diagnostic criteria and the evidence, the Guideline Development Group decided to adopt DSM-IV criteria for this update rather than ICD-10, which was used in the previous guideline (NICE clinical guideline 23). This is because DSM-IV is used in nearly all the evidence reviewed and it provides definitions for atypical symptoms and seasonal depression. Its definition of severity also makes it less likely that a diagnosis of depression will be based solely on symptom counting. In practical terms, clinicians are not expected to switch to DSM-IV but should be aware that the threshold for mild depression is higher than ICD-10 (five symptoms instead of four) and that degree of functional impairment should be routinely assessed before making a diagnosis. Using DSM-IV enables the guideline to target better

the use of specific interventions, such as antidepressants, for more severe degrees of depression.

(NICE, 2018)

It is important to bear in mind that clinical guidelines are also periodically reviewed and revised in line with diagnostic classification systems and may therefore be subject to change over time. CG90 has since been updated and was replaced by NICE guideline NG222 'Depression in adults: treatment and management', published on 29 June 2022. NG222 now refers to both DSM-5 and ICD-11, and places greater emphasis on informed and shared (healthcare professional-service user) decision-making with regards to choice and preference of treatment options. This includes 'carrying out an assessment of need; developing a treatment plan; taking into account any physical health or co-existing mental health problems; discussing factors that would make the person most likely to engage with treatment; taking previous treatment history into account; addressing any barriers to the delivery of treatments due for example to disabilities, language or communication difficulties; ensuring regular liaison between healthcare professionals in specialist and non-specialist settings; and matching the choice of treatment to meet the needs and preferences of the person with depression, using the least intrusive and most resource efficient treatment that is appropriate for their clinical needs or that has worked for them in the past' (NG222, Section 1.4.1 and 1.4.2).

Box 5 ICD-10 criteria for diagnosis of a depressive episode

(based on ICD-10, 2003)

In typical mild, moderate, or severe depressive episodes, the patient suffers from lowering of mood, reduction of energy, and decrease in activity. Capacity for enjoyment, interest, and concentration is reduced, and marked tiredness after even minimum effort is common. Sleep is usually disturbed and appetite diminished. Self-esteem and self-confidence are almost always reduced and, even in the mild form, some ideas of guilt or worthlessness are often present. The lowered mood varies little from day to day, is unresponsive to circumstances and may be accompanied by so-called "somatic" symptoms, such as loss of interest and pleasurable feelings, waking in the morning several hours before the usual time, depression worst in the morning, marked psychomotor retardation, agitation, loss of appetite, weight loss, and loss of libido. Depending upon the number and severity of the symptoms, a depressive episode may be specified as mild, moderate or severe.

(F32 Depressive Episode: ICD-10 2003). [a]

Key symptoms

At least one of the following, most days, most of the time for at least two weeks:

- persistent sadness or low mood, and/or
- loss of interests or pleasure
- fatigue or low energy.

If any of the above are present, the individual is then asked about associated symptoms:

- disturbed sleep
- poor concentration or indecisiveness

- low self-confidence
- poor or increased appetite
- suicidal thoughts or acts
- agitation or slowing of movements
- guilt or self-blame.

The 10 symptoms then define the ‘degree’ of depression and management is based on the particular degree:

- **not depressed** (fewer than four symptoms)
- **mild depression** (four symptoms)
- **moderate depression** (five to six symptoms)
- **severe depression** (seven or more symptoms, with or without psychotic symptoms).

Symptoms should be present for a month or more and every symptom should be present for most of every day.

_____

Note [a] F32 major depressive disorder, single episode excludes: bipolar disorder (F31), manic episode (F30) and recurrent depressive disorder (F33).

Box 6 Diagnostic criteria for major depressive episode

(based on DSM-IV-TR, APA, 2000)

A diagnosis of depression is suggested if, during the same two-week period, a person experiences five (or more) of the following symptoms, which must include either or both of the key presenting symptoms.

At least one of the following **key symptoms**:

- persistent feelings of depressed mood (determined either by subjective report or observation made by others)
- loss of interest or pleasure in usual activities.

Plus three or more of the following symptoms:

- changes in appetite that result in weight losses or gains not related to dieting
- insomnia or oversleeping
- loss of energy or increased fatigue
- restlessness or irritability
- feelings of worthlessness or inappropriate guilt
- difficulty thinking, concentrating or making decisions
- thoughts of death or suicide or attempts at suicide.

Symptoms should not be counted if they are a direct physiological effect of a substance (drug abuse or medication); or a medical condition (e.g. hypothyroidism); or if they would be better accounted for by bereavement (i.e. after the loss of a loved one).

Box 7 DSM-5 criteria for major depressive disorder and persistent depressive disorder

(based on APA, 2013a)

Major depressive disorder[a]

Five or more of nine symptoms (including at least one of depressed mood and loss of interest or pleasure) in the same two- week period; each of these symptoms represents a change from previous functioning:

- depressed mood (subjective or observed)
- loss of interest or pleasure
- change in weight or appetite
- insomnia or hypersomnia
- psychomotor retardation or agitation (observed)
- loss of energy or fatigue
- feelings of worthlessness or guilt
- impaired concentration or decisiveness
- thoughts of death or suicidal ideation or suicide attempt.

Persistent depressive disorder[b]

Depressed mood for most of the day, for more days than not, for two years or longer.

Presence of two or more of the following during the same period:

- poor appetite or overeating
- insomnia or hypersomnia
- low energy or fatigue
- low self-esteem
- impaired concentration or indecisiveness
- hopelessness.

Never without symptoms for more than two months.

Note [a] in children and adolescents, mood can be irritable.

Note [b] in children and adolescents, mood can be irritable and duration must be one year or longer.

Medicalising grief?

The DSM was first published in 1952 in the United States as a guide to help mental health professionals communicate using a common diagnostic language. As understanding of mental health conditions has evolved, the manual has been periodically updated to remove elements that are no longer considered valid, adding newly defined conditions or revising existing criteria to reflect contemporary thinking. Prior to the publication of DSM-5 in 2013, concern had been raised in an open petition at the possibility that the proposed revisions to certain criteria would ‘medicalise’ patterns of behaviour and mood that would

otherwise be considered within the normal spectrum for human experience (Frances, 2012, 2013; Kamens, Elkins and Robbins, 2017).

These concerns were echoed by the British Psychological Society in their statement (BPS, 2011) supporting the open petition to the DSM Taskforce by the Society for Humanistic Psychology (see Kamens, Elkins and Robbins, 2017) in which they made clear the view that 'a major concern …. is that the proposed revisions include lowering diagnostic thresholds across a range of disorders. It is feared that this could lead to medical explanations being applied to normal experiences, and also to the unnecessary use of potentially harmful interventions', and further that 'we share concerns expressed in the open letter about the inconsistency of the proposed changes and their limited empirical basis' (BPS, 2011).

The petition specifically questioned 'proposed changes to the definition of mental disorder that deemphasize sociocultural variation while placing more emphasis on biological theory. In light of growing empirical evidence that neurobiology does not fully account for the emergence of mental distress' (Kamens, Elkins and Robbins, 2017). Expanding on this concern further, the statement clarified that 'advances in neuroscience, genetics and psychophysiology have greatly enhanced our understanding of psychological distress'; however, 'not one biological marker 'biomarker' can reliably substantiate a DSM diagnostic category. In addition, empirical studies of etiology are often inconclusive, at best pointing to a diathesis-stress model with multiple (and multifactorial) determinants and correlates'. And thus, 'in the absence of compelling evidence, we are concerned that these reconceptualizations of mental disorder as primarily medical phenomena may have scientific, socioeconomic, and forensic consequences', that 'psychopathology, unlike medical pathology, cannot be reduced to pathognomonic physiological signs or even multiple biomarkers', that 'hypothesizing a medical explanation for these symptoms will resolve the philosophical problem of Cartesian dualism inherent in the concept of 'mental illness', and that clinicians would be required 'to draw on subjective etiological theory to make a judgment about the cause of presenting problems' (Kamens, Elkins and Robbins, 2017).

Concerns around the 'bereavement exclusion' in DSM-5

One of the main concerns voiced by the petition, and by the former Chair of the DSM-IV Taskforce, Professor Allen J. Frances, was around the proposed removal of the 'bereavement exclusion' in the diagnosis of Major Depressive Disorder, which in the previous version was considered an important measure that prevented the 'pathologization of grief, a normal life process' (Frances 2012, 2013; Kamens, Elkins and Robbins, 2017). This has perhaps been one of the most contentious changes to the DSM. At the heart of the argument opposing the change, is the view that grief is a normal, albeit difficult and upsetting, part of human experience that should not require a formal diagnosis as a 'psychiatric illness' requiring treatment using medications such as antidepressants, and that its 'inclusion' is likely to lead to overdiagnosing or misdiagnosing of depressive illness.

Professor Frances wrote in 2013:

It is not at all pathological to have symptoms that closely resemble mild depression during bereavement. The Bereavement Exclusion is absolutely necessary to protect against the false positive over diagnosis of depression…two critical features of clinical depression are that it predicts a higher likelihood of later recurrence of new depressive episodes and a highly elevated rate of suicide attempts…there was no previous problem in DSM IV that needed fixing. Grievers who have severe and urgent symptoms−suicide risk, psychotic symptoms, severe agitation, inability to function−have always qualified for the diagnosis of Major Depressive Disorder; while those having typical symptoms of grief were appropriately regarded as having a normal, human reaction to a grave loss…Grief is a

normal and inescapable part of the human condition, not to be confused with psychiatric illness.

(Frances, 2013)

Professor Frances summarised his concerns around the wider changes proposed in DSM-5, in an online post in 'Psychology Today' in December 2012, noting that:

People with real psychiatric problems that can be reliably diagnosed and effectively treated are already badly shortchanged. DSM5 will make this worse by diverting attention and scarce resource away from the really ill and toward people with the everyday problems of life who will be harmed, not helped, when they are mislabeled as mentally ill.

(Frances, 2012)

But there have been arguments to counter the above view as well, noting for example, that for many people with a mental health problem, receiving a diagnosis can be helpful – it can give people access to other support and services, including benefits (NHS Choices, 2013).

Views amongst psychiatrists have also been mixed (Nemeroff et al., 2013). Carmine Pariante, Professor of Biological Psychiatry at King's College London noted that:

… removal of [the] bereavement exclusion will allow individuals who have been clinically depressed for less than 2 months after the loss of a loved one to be diagnosed with Major Depression…Does this mean that hordes of individuals who have just lost their partner or their parent will be started on antidepressants? Obviously not: first, because individuals will still need to fulfill the diagnostic criteria for depression, including the impairment in important areas of functioning such as their social or professional life; and second – and crucially – because no clinically competent doctor would do so…clinical competency and personalized decisions are, as always, key to clinical management, and diagnostic textbooks will not make good doctors take bad decisions.

(cited in Nemeroff et al., 2013)

On the other hand, Florian Seemüller, Consultant Psychiatrist at Ludwig-Maximillians-University in Munich noted that:

… in order to improve test-retest reliability and reduce the number of false positives, tightening of the diagnostic criteria for such problem diagnoses would have been desirable; however, with the elimination of the major depression bereavement exclusion in the DSM-5, the diagnostic boundaries have again been widened. Thus a major depressive episode can be diagnosed if a person grieves for a loved one for more than two weeks…clinical research further suggests that the risk for recurrent depression in people experiencing severe grief is not different from that of healthy controls. Although some individuals, especially elderly people with complicated grief, may benefit from this change, possibly by earlier receipt of intensive treatment after having lost a loved one, millions of other people might be unnecessarily labeled as having an illness, and consequently receive treatment that they do no need.

(cited in Nemeroff et al., 2013)

A further rationale for the 'bereavement exclusion' was provided by the American Psychiatric Association in a document highlighting changes from the previous version (APA, 2013b).

Although the DSM represents a useful guide for defining and communicating about mental health, and can be used to support clinical decision-making, limitations should also

be considered. Issues such as the one discussed here represent a legitimate area of debate and underscore the challenges of diagnosis, treatment and care for people affected by mental health conditions.

2.3 Consolidating your understanding of depression

The activity below will help to consolidate your understanding of depression and to expand this to consider some issues further. Please note that there is no discussion associated with this activity. The questions posed, however, will help you to structure your thoughts as you reflect on the issues raised in the podcast. You might find it useful to take notes and write down your answers to individual questions.

Activity 3 What is depression?

Allow 60 minutes

Listen to the podcast below and note down your responses to the questions that follow. You might wish to listen to the entire recording first and review this again thinking specifically about the questions the second time around. Alternatively, you can consider the questions as you listen to the recording the first time around. Choose whichever approach suits you best.

Audio content is not available in this format.

Oxford University Podcast – What is depression?

Professor Mark Williams and Dr Danny Penman from Oxford University discuss what is meant by ‘depression’. The podcast is part of a series on the ‘New Psychology of Depression’ from the Department of Experimental Psychology at the University of Oxford, produced in 2011.

1. What does it feel like to have ‘full-blown’ depression?
2. How does depression relate to anxiety and stress?
3. Why does it seem to be increasing globally?
4. Are a ‘relapse’ or subsequent ‘episodes’ of depression inevitable after recovery?
5. What is the link between depression and suicide?
6. Is it possible to prevent depression?

Provide your answer...

- Reflect on your learning in this section.
  - What were the key issues or concepts that stood out for you?

We will briefly explore psychological theories and take a look at the treatments for depression next.

3 Theoretical models and psychological explanations of depression

The prevalence of depression was already well-recognised more than half a century ago. Indeed, Martin Seligman (1973-1975) is often cited for having referred to depression as the 'common cold' of psychiatry because of the rate at which it was encountered at the time. Our understanding of depression began to transform in the 1950s and 60s with the introduction of tricyclic 'antidepressant' medications, and psychological theories of depression that were mainly based around behaviourist, classical conditioning and psychodynamic theories, which evolved during this period. Theoretical models and psychological explanations for depression (see Box 8) progressively developed over subsequent decades.

Box 8 Psychological theories and models of depression[a]

- **Behaviourist theory**: classical conditioning[b] (associating stimuli with negative emotional states) and social learning theory (behaviour learned through observation, imitation and reinforcement).
- **Operant conditioning (Lewinsohn, 1974)**.
- **Psychodynamic theories (1960s-70s)**.
- **Beck's (1967) cognitive model**: cognitive triad, cognitive distortions, core irrational beliefs.
- **Seligman's (1973-1975) 'learned helplessness' theory**.
- **Abramson, Seligman and Teasdale's (1978) 'attribution' model**.
- **Abramson, Metalsky and Alloy's (1989) 'hopelessness theory of depression'**.
- **Wolpe's (1986) model of 'neurotic depression'**: depression secondary to 'maladaptive anxiety' through classical conditioning[b]; all non-psychotic problems can be reduced to specific fears.
- **Various models of rumination**: the concept of depression as 'overthinking', e.g. Nolen-Hoeksema's (1991) 'Response Styles Theory' of rumination (consisting of repetitively thinking about the causes, consequences, and symptoms of one's negative mood), or Conway et al's (2000) 'Rumination on Sadness' theory (seeing rumination as repetitive thinking about sadness, and circumstances related to one's sadness).

———————

Note [a] A 'theory' can be considered to represent an 'explanation' or a 'hypothesis' that can be used to predict an event(s) or an observation(s), or to identify relationships or associations between concepts; theories can be inductive (a 'bottom-up' approach) or deductive (a 'top-down approach'); a 'psychological model' can be thought of conceptually as a simplified representation of a theory, often presented in diagrammatic form, that allows theoretical predictions to be tested (providing evidence for or against a particular theory). Note also that a 'biological model' is used in relation to experimental research (e.g. to denote cellular, molecular, animal or clinical 'models'). 'Models' used in this context can also be thought of as 'evidence-based representations' designed to test specific hypotheses.

Note [b] 'classical conditioning' is a learning process which occurs when two stimuli are repeatedly paired such that a response typically elicited by the second stimulus is eventually elicited solely by the first stimulus; 'operant conditioning' is a learning process in which the likelihood of a specific behaviour is modified (may increase or

decrease) as a consequence of (or in response to) a reinforcing stimulus such as a punishment or a reward.

The diathesis-stress model, which is central to an understanding of depression, considers depression to be triggered by a combination of negative or stressful life event(s) (e.g. loss of an important source of love, security, identity or self-worth; death of a loved one, breakdown of a relationship or a significant personal failure) and vulnerability factor (s) (termed 'diathesis') that make the individual susceptible to depression. The theory was predicated on observations that depressive episodes are often preceded by negative life events. A depressive episode therefore tends to occur when a person who is vulnerable to depression experiences a negative life event, and this can be characterised by feelings of hopelessness and/or worthlessness, which may resolve quickly or turn into long-term depressive illness.

Self-referent thoughts, feelings and excessive rumination (negative introspection, self-reflection) are prominent features of depression and depressive episodes. A century ago, Sigmund Freud (1917) published his seminal work titled 'Mourning and Melancholia' in which he argued that depression can take two forms: 'mourning' the loss of a loved one characterised by intense sadness and despair, but not (typically) guilt, shame or self-reproach; and what he termed 'melancholia' characterised not only by intense sadness, but feelings of self-recrimination and self-deprecation, a failure of living up to one's ideals or standards. Contemporary views see 'hopelessness', 'worthlessness' and 'helplessness' as self-referent perceptions that are prominent features of depression. People can feel hopeless if they believe there is nothing that can be done to bring about a desired outcome or to avoid a negative outcome (develop feelings of resignation), they can feel worthless when they feel weak, inadequate or flawed, and they can feel helpless when they feel powerless to change an undesirable situation. These perceptions are interlinked, and prominent features of depression.

3.1 Behaviourist theory and operant conditioning

Behaviourist theory focuses on observable behaviour and places emphasis on the importance of the environment in shaping behaviour. Operant conditioning (Lewinsohn, 1974) considers the cause of depression to be the removal of positive reinforcement from the environment, or situations that would serve to reinforce 'maladaptive' behaviour, leading to increased social isolation, and an inability to seek or respond to alternative sources of positive reinforcement. However, while these theories offer an explanation where the cause of depression is known (or observed), they are more problematic where the underlying cause is undefined ('endogenous' depression), and fail to take into account the influence of thought (cognition) on mood.

3.2 Psychodynamic theories

Psychodynamic theories view depression in terms of inwardly directed anger, loss of self-esteem or self-worth, egotistic or excessive narcissistic or personality demand, or deprivation in mother-child relationship (loss or rejection by a parent). Freud's (1917) psychoanalytic theory is an example of the psychodynamic approach. Repressed anger at a loss (symbolic or actual loss of a loved one during childhood, for example) is directed inwards, reduces self-esteem and increases vulnerability to further experiences in the future, causing the individual to 're-experience' (symbolic or actual) the loss when

encountering similar triggering stimuli during adulthood. The theory argues that people prone to depression have an excessively high interpersonal dependency (i.e. they seek approval and reassurance from others – to be loved, respected, admired, appreciated,etc. and depression arises when they fail to receive it). Those who may depend on others for their sense of self-esteem may therefore remain in a more vulnerable 'depression-prone' state. Alternatively, they may hold lofty ideals, standards and goals, in which case depression arises when they fail to achieve these. Congruency models view both a high dependency on social sources of approval, and a high dependency on achievement outcomes as important aspects of depression. The main problems with the psychodynamic approach relate to difficulties in testing the theories scientifically, using operational definitions that allow empirical (clinical and experimental) investigation. A lack of emphasis on distressing life events and conscious negative rumination and 'self-verbalisation' are further criticisms. Beck's model of depression, which we will examine shortly, was heavily influenced by psychodynamic theories.

3.3 Cognitive models

Cognitive theories consider the manner in which people think about and process personal information, by focusing on core beliefs (formed during early life experiences; unconscious beliefs about self, others and the world), underlying assumptions (spontaneous thoughts or prompts arising from core beliefs) and systematic negative bias in thinking. An assumption of this approach is that altered thinking processes precede the onset of depressed mood. Aaron Beck (Beck, 1967a and 1967b) proposed three mechanisms underlying the 'negative appraisal' of events in depression: the cognitive triad (negative automatic thinking), negative self schemas and errors in logic (altered processing of information).

Beck's (1967) cognitive triad model of depression identifies three common forms of negative (helpless and/or critical) self-referent thinking which occur spontaneously ('automatically') in individuals with depression: negative thoughts about the self, the world and the future. The three core beliefs (which encompass feelings of hopelessness and worthlessness) interact and interfere with cognitive processing, leading to impairments in perception, memory, problem solving and reinforce an 'obsession' with negative thinking. According to the model (see Figure 1), negative beliefs and expectations may be acquired in childhood as a consequence of a traumatic event(s) such as death of a parent or sibling, parental rejection, criticism or overprotective parenting, neglect or abuse, bullying or exclusion from a peer group. These can predispose the individual to depression. A subsequent stressful life event or a critical incident in later life can act to trigger the schema, and activate systematic negative (biased) thinking whereby the individual tends to focus selectively on certain aspects of a situation or event, while ignoring other relevant information. Negative thoughts will often persist even in the face of contrary evidence. These 'cognitive distortions' (i.e. systematic negative biases in thinking), can be self-defeating and a significant source of anxiety *or* depression for the individual (see Box 9).

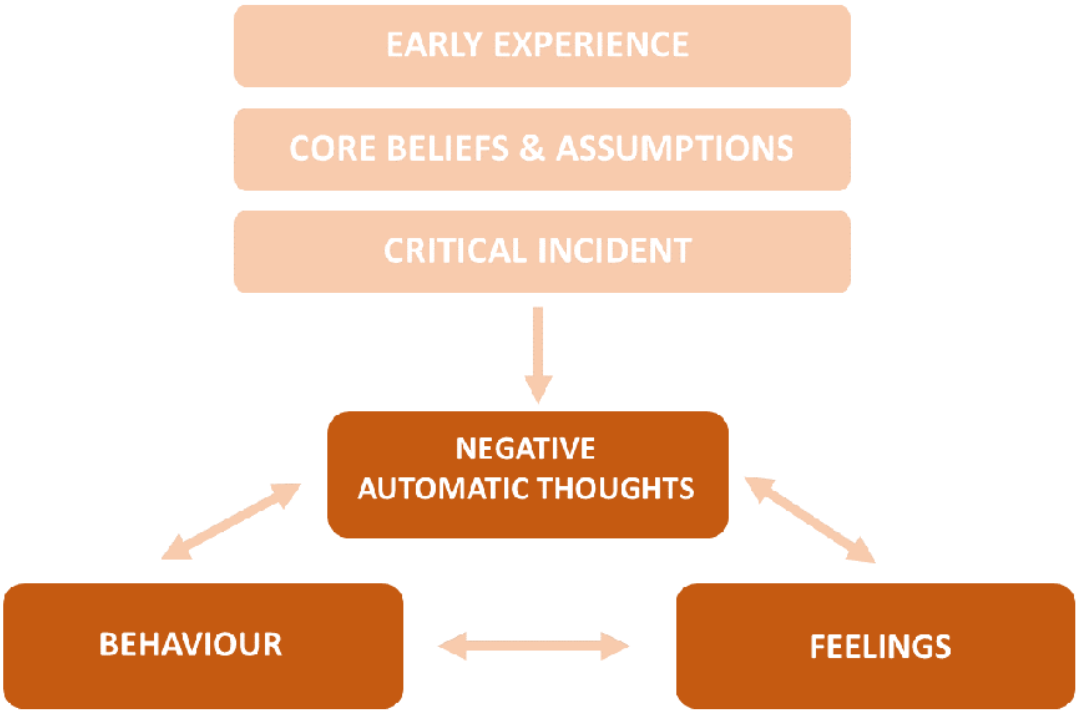

Figure 1 A cognitive model of depression (adapted from Beck, 1976, 1979)

Depression typically involves a negative view of oneself, the world and the future.

Box 9 Cognitive distortions (systematic negative biases in thinking) that can contribute to depression (adapted from Beck, 1967a; Burns, 1999 and 2000)

Dichotomous ('all or nothing') thinking	Looking at things in absolute ('black or white') categories with no middle ground, e.g. 'If I fall short of perfection, I'm a total failure'.
Overgeneralisation	Generalising from a single negative experience and viewing this as a never-ending pattern of defeat, e.g. 'I didn't get hired for the job, I'll never get any job'.
Mental filtering	Dwelling on the negatives, filtering out the positives, e.g. focusing on one or two things that went wrong, rather than all of the things that went right.
Disqualifying or discounting or diminishing the positives	Rejecting positive experiences, qualities or accomplishments, insisting that they 'don't count', e.g. 'I did well on the presentation, but that was just pure luck'.
Jumping to conclusions	Drawing negative conclusions even though there is insufficient evidence or not warranted by facts, such asassuming that people are reacting negatively to you when there's no definitive evidence ('mind reading'), e.g. 'I can tell she secretly hates me'; you

	arbitrarily predict that things will turn out badly ('fortune telling'), e.g. 'I just *know* something terrible is going to happen'.
Magnifying or minimising	Blowing things out of proportion or shrinking their importance.
Emotional reasoning	Reasoning from one's subjective feelings. Believing that the way you feel reflects reality. e.g. 'I feel like an idiot, so I really must be one', or 'I feel hopeless; this means I'll never get better'.
Catastrophising	Assuming extreme and horrible consequences of events. Expecting the worse-case scenario,e.g. 'The pilot said we're in for some turbulence; the plane's going to crash!'
'Should' statements	Holding oneself and others to strict rules of what should and shouldn't ('ought', 'must' or 'have to') be done; criticising or being hard on yourself for breaking any rules. Self-directed 'should' statements lead to feelings of guilt and inferiority'; directing 'should' statements at others can lead to feelings of bitterness, anger and frustration. Hidden 'shoulds' are rules that are implied by your negative thoughts.
Labelling	Labelling yourself based on mistakes and perceived shortcomings, e.g. instead of saying 'I made a mistake' you tell yourself 'I'm a failure, an idiot, a loser'.
Personalisation and blame	Assuming responsibility for things that are beyond one's control, e.g. 'It's my fault that my friend had the accident; I should have warned her not to drive in the rain'. Finding fault instead of solving the problem, e.g. blaming yourself for something that you weren't entirely responsible for (self-blame) or blaming others and overlooking ways in which you may have contributed, or denying your role in the problem (other-blame).

If negative interpretations of situations are not challenged, the patterns of thoughts, feelings and behaviours become increasingly repetitive and intrusive and can be repeated as part of a debilitating cycle (see Figure 2). However, while correlation between cognitive style and development of depression is suggested by this model, it is unclear whether maladaptive cognitive processes and negative thinking such as those described above are a *consequence* rather than a *cause* for depression (i.e. they may accompany and persist in depression, but do not predispose or predict the onset of depression).

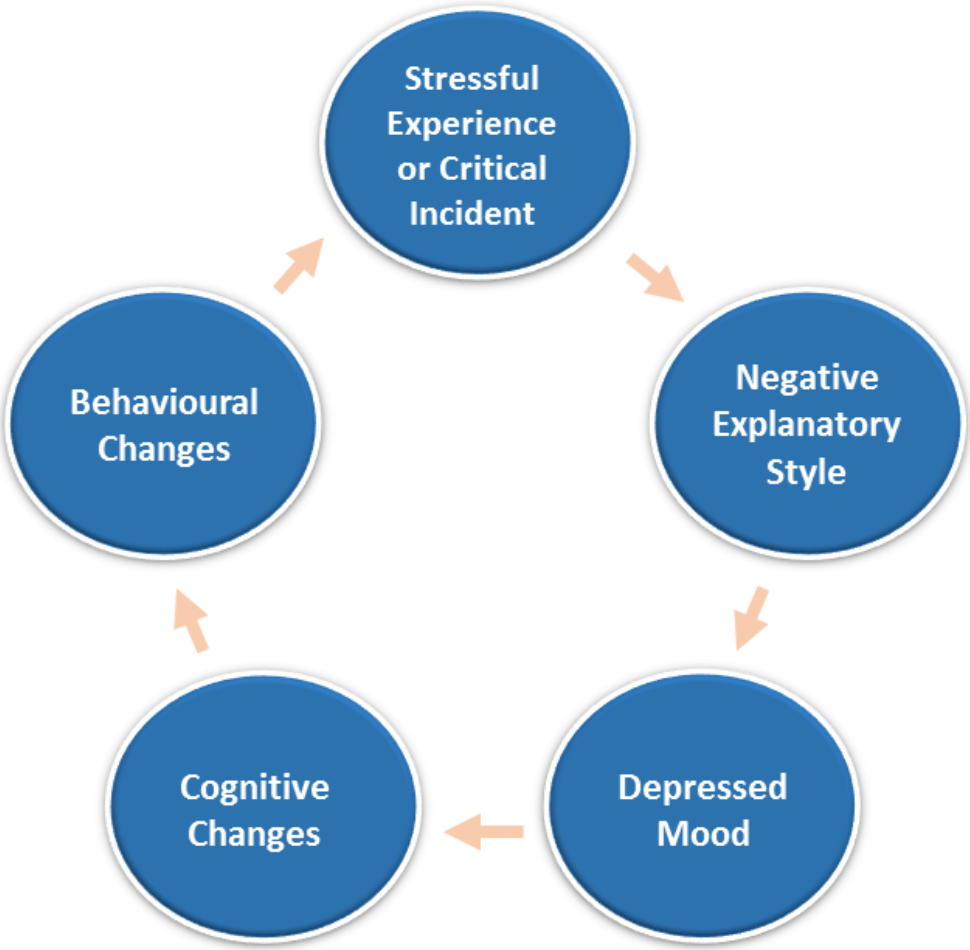

Figure 2 Depression 'cycle' based on the cognitive model of depression

Seligman's 'learned helplessness' theory, another psychological explanation for depression, considers depression to arise as a consequence of a person's futile attempts to escape 'negative' situations (Seligman, 1973-1975). Seligman based this theory on experiments conducted in dogs. When dogs were subjected to mild electric shock delivered through the floor of their housing, but had access to a partitioned area, escape was possible by crossing over to the 'shock-free' area. When restrained, however, and escape was no longer possible, they eventually stopped attempting to escape. When subjected to repeated 'inescapable' shocks in this way, they not only failed to escape even when it was later possible to do so, but also exhibited some symptoms associated with depression in humans (e.g. passive, lethargic behaviour in the face of stress and loss of appetite). While such experiments do raise ethical considerations, at the time they did offer an explanation for depression in humans as a condition whereby an individual would learn that they are helpless as a consequence of having lack of control over what happens to them.

Abramson, Seligman and Teasdale (1978) reformulated this hypothesis to include a cognitive process whereby an individual could 'attribute' or explain the 'cause' for an event. **The attribution model** is based on three 'causal' dimensions: (i) whether the cause is internal or external to the individual, (ii) whether the cause is stable and permanent, or transient in nature, and (iii) whether it is global (affecting all areas of life) or specific. Abramson et al. argued that people who attributed failure to internal, stable and global causes were more likely to become depressed, as they would come to the conclusion that they were unable to influence or control the situation for the better.

Attributions to internal factors are tied to feelings of worthlessness, whereas attributions to stable and global factors are linked to feelings of hopelessness and despair.

For example, if a person loses their job, and they attribute this to some failing on their part (internal dimension), and they also see things as not working out for them in other areas (global dimension), and view this as a long-term pattern of failure and disappointment in the future (stable dimension), then they are likely to become depressed. On the other hand, if they view the loss of a job as being due to circumstances beyond their control (external dimension), as an event that was unique to the situation (specific dimension), and as something that did not represent any pattern in the future (unstable dimension) they would be likely to handle this well emotionally, according to this model.

Abramson, Metalsky and Alloy (1989) further revised the model, integrating Beck's (1976) theory with a reformulated learned helplessness model to derive **the 'hopelessness theory of depression'**. In keeping with the diathesis-stress model of depression, the theory considers depression to arise when people with a negative attributional style interpret a stressful life event in negative terms. These interpretations give rise to hopelessness, seen as an immediate cause of a particular 'subtype' of depression. Once again, however, whether 'helplessness' or 'hopelessness' are *symptoms* (or manifestations) rather than a *cause* of depression, remain unclear.

3.4 Neurotic depression

Joseph Wolpe (1986) introduced the idea of 'neurotic depression' based on earlier concepts of 'neuroses' (a term that is no longer widely used) that made reference to forms of 'nervousness' − psychological or behavioural conditions in which anxiety was a primary characteristic (as opposed to 'psychoses' in which people experience distortion of reality or a disorganisation of personality). He viewed some forms of depression as 'neurotic' in the sense that they were 'secondary to learned maladaptive anxiety responses that are the core of neuroses'. Wolpe saw maladaptive anxiety (i.e. anxiety which was not 'useful') as the core problem. He hypothesised that maladaptive anxiety was acquired through classical conditioning, associated with an 'environmental' stimulus or trigger, and that generalised anxiety disorders and depression were both grounded by specific fears. He identified social anxiety as a common and specific 'cause'.

Wolpe distinguished two categories of depression, one which correlated highly with anxiety (termed 'neurotic depressions') and another which correlated less well with anxiety (which he referred to as 'endogenous depressions'). He considered neurotic depression to arise:

- secondary to a severe and prolonged conditioned anxiety
- as a consequence of a cognitively-based anxiety
- secondary to social anxiety or to a feeling of interpersonal intimidation
- as a result of unresolved bereavement.

He considered that treating the underlying anxiety or 'symptomatic neuroses' by relearning methods (using behaviour therapy based on the classical conditioning model) would also resolve the depression 'without [specific] measures being taken against the depression as such'. Once the focus of the maladaptive anxiety was identified it would then be treated as an anxiety problem which would also resolve the depression.

3.5 Models of rumination

People differ in the extent to which they ruminate when they are feeling sad. Excessive introspection and self-preoccupied thinking, however, are rarely effective 'mood-management' strategies, and are often associated with depression. Nolen-Hoeksema and others have studied aspects of rumination in depression and how this may differ between men and women (Smith and Alloy, 2009). The Responses to Depression Questionnaire is a scale that can be used to measure individual differences in 'ruminative coping styles'. Rumination in this respect does not involve a focus on the *causes* of depression (which is active and 'problem-focused'), but rather a preoccupation with the *state* of 'being depressed' (dwelling on negative mood states).

Individual differences in rumination have been found to relate to the duration and severity of depression. Nolen-Hoeksema (2012) has posed the notion that women are more likely to show a ruminative coping style compared to men. In addition to social, cultural and biological differences, differences in ruminative coping style may therefore represent another relevant factor which may contribute to susceptibility to depression, and to influence the duration and severity of a depressive episode.

People who are depressed find it difficult to consciously suppress (or unconsciously repress) negative thoughts, and to keep these from coming to mind. Negative thinking tends to resurge. Learning to replace negative biases and negative thoughts with more positive ones, however, is a key element in the recovery from depression, and a main goal for treatment (cognitive therapy in particular), the idea being that if negative thinking is eliminated, other symptoms of depression (intense sadness, loss of interest in activities, sleep disturbances, etc.) will also abate, allowing the individual to 'gain control' once more.

It is important to note, however, that there is no single unifying definition for rumination, and various theories and models have been proposed (Smith and Alloy, 2009). Among these are the 'Response Styles Theory' and 'Rumination on Sadness'. Nolen-Hoeksema's (1991) Response Styles Theory (RST) considers rumination to consist of repetitive thinking about the causes, consequences and symptoms of one's negative mood. The Response Styles Questionnaire (RSQ), although widely used and empirically supported, has been criticised however, for overlap with positive forms of repetitive thoughts (reflection) and worry, and the RST does not address how rumination fits with biological or cognitive processes such as attention or metacognitive beliefs (Smith and Alloy, 2009). Conway et al.'s (2000) 'Rumination on Sadness' defines rumination as thinking about sadness, and the circumstances related to one's sadness – proposed as a model for 'predicting' sadness. However, the Rumination on Sadness Scale is not widely used, so its specificity for use in depression remains unclear. Other models include the Stress-Reactive model (rumination on negative, event-related inferences), Goal Progress Theory (which views rumination as a response to failure to progress satisfactorily towards a goal), and Self-Regulatory Executive Function (S-REF) theory (which considers rumination to be a multi-faceted construct and attempts to integrate attention, cognition regulation, beliefs about emotion regulation strategies and interaction between various levels of cognitive processing as part of an overall conceptualisation of rumination).

An important issue to consider would be clarifying the extent to which models of rumination relate to and overlap with other psychological constructs that may be similar conceptually, including negative-automatic thoughts, private self-consciousness, self-focused attention, repetitive thought, intrusive thoughts and obsessions, worry, emotion regulation and coping, neuroticism, social and emotional competence and emotional intelligence (Smith and Alloy, 2009). It has also been proposed that 'rumination' should be differentiated from 'reflection'. The picture that emerges is therefore a complex one, in

which rumination is a multifaceted, multidimensional construct. Although rumination is important in the context of depression, it can be difficult to determine how it can be best measured and used to predict a clinical outcome.

Theoretical models continue to evolve, and it has been argued that new models are needed that take into account not only psychological constructs and life events but socio-cultural and environmental factors (such as poverty, conflict and violence), as well as genetic and biological predisposition which influence ‘vulnerability’ and ‘resilience’, and that importantly such models should be validated experimentally (empirically supported), including in suitable animal models where feasible, while recognising the inherent limitations.

4 Biological explanations for depression

Genetic, molecular and neuroimaging studies have continued to contribute to advances in understanding the neurobiological basis of depressive illness (Kupfer et al., 2012). Animal models for depression, and rodent models in particular (see Box 10), have been extensively used to test the efficacy of antidepressant medications leading up to clinical trials, and to inform and guide pharmacological therapies. Genetic variation in *SLC6A4* (the serotonin transporter gene), *FKBP5* (encoding a protein that helps regulate cortisol binding to the glucocorticoid receptor), *TREK1* (a potassium channel mediating SSRI mechanism of action) and *COMT* (catechol-O-methyltransferase) amongst others, have been associated with the response to several antidepressant drugs in clinical studies.

Molecular studies also support at least three categories of biological determinants associated with depression:(i) reduction in neurotrophic and related factors such as BDNF, VEGF and IGF-1; (ii) excessive production of pro-inflammatory cytokines (immune mediators),including IL-1beta, IL-6 and TNF-alpha; and (iii) impaired regulation of the hypothalamic-pituitary-adrenocortical (HPA) axis, which can be affected by antidepressant medication.

Box 10 Rodent models for depression

Despair-based: forced-swimming, tail suspension.

Reward-based: sucrose preference, intracranial self-stimulation.

Anxiety-based: novelty-induced hypophagia, open field, elevated plus maze, light/dark box.

Early life stress: maternal deprivation.

Stress-based models: learned helplessness, chronic mild stress, social defeat stress.

Others: genetically engineered mice; olfactory bulbectomy.

Neuroimaging studies have highlighted changes in neural systems involved in the processing and regulation of emotion, and in reward-seeking in people with major depressive illness. These include the amygdala and ventral striatum (brain areas that are involved in emotion and reward processing), medial prefrontal, orbitofrontal and anterior cingulate cortical regions (brain areas involved in ‘implicit’ regulation and processing of emotion), and the ventrolateral and dorsolateral prefrontal cortex (which are involved in cognitive control and the voluntary regulation of emotion) (see Figure 3).

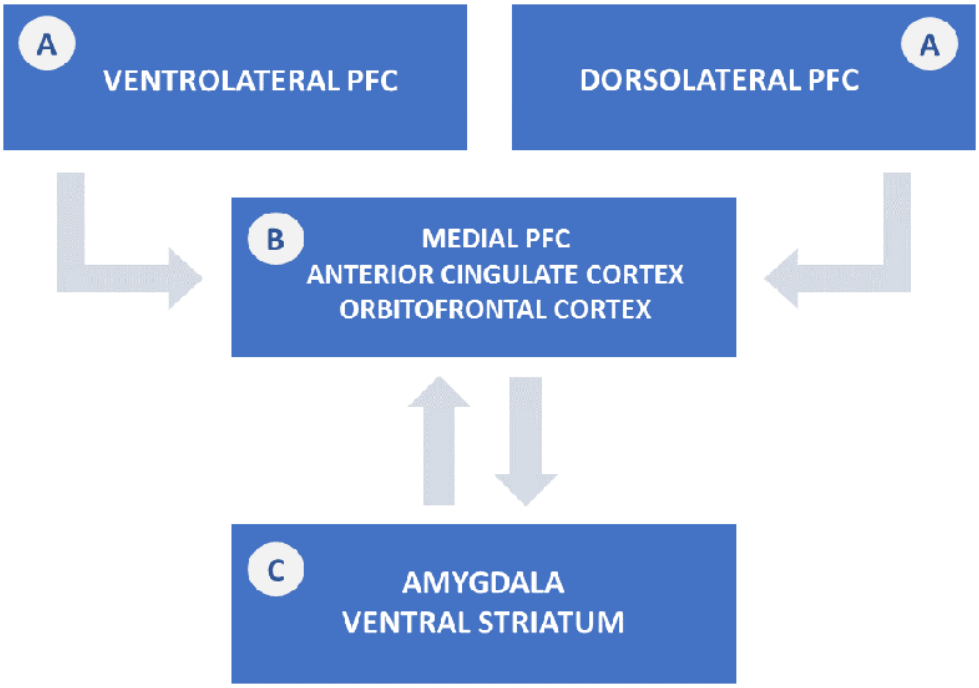

Figure 3 Neural systems involved in depression. [A] involved in cognitive control and the voluntary regulation of emotion; [B] involved in the implicit (automatic) regulation of emotion; [C] involved in emotion and reward processing; PFC: prefrontal cortex. Various brain imaging studies point to increased activity in neural systems supporting emotion processing (amygdala and medial prefrontal cortex), and reduced activity in neural systems supporting regulation of emotion (e.g. dorsolateral prefrontal cortex) in major depressive illness.

Functional connectivity between brain regions (the effect that activity in one region exerts over that in another) is altered in neural systems for emotion and reward processing and for regulation of emotion in major depressive disorder. The medial prefrontal-limbic neural network which regulates emotion (and includes the amygdala, anterior cingulate cortex and medial prefrontal cortex) is modulated by serotonin neurotransmission. The ventral striatum, and interconnected orbitofrontal and medial prefrontal cortices which are involved in reward processing, is modulated by dopamine.

People with major depression show a bias of attention towards negative emotional stimuli, and away from positive emotional and reward-related stimuli. Through measuring responses to fearful faces as a ‘negative stimulus’, activity within the amygdala, ventral striatal and medial prefrontal cortex was shown to be increased in people with major depression, whereas in response to positive emotional stimuli and receipt or anticipation of a reward, activity within the ventral striatum was atypically reduced in people with major depression when compared to those who were not depressed (see Kupfer et al., 2012).

Activity within the ‘default mode network’ (a neural system that involves several midline structures within the brain including the ventral medial prefrontal cortex, and can provide insight into brain function at rest and during self-reflection, associated with self-referential processing) is also altered in major depressive disorder.

Importantly, antidepressant treatment has been found to modulate activity within these neural networks: (i) reduced activity within the dorsolateral prefrontal cortex, anterior cingulate cortex and other regions implicated in cognitive control of emotion during the

resting state in depressed individuals returned to ‘normal’ levels (i.e. levels observed in individuals who are not depressed) following treatment with antidepressant drugs; and (ii) overactivation of the medial prefrontal cortex and subcortical regions in response to emotional stimuli in the depressed state was also returned to normal after antidepressant treatment (Kupfer et al., 2012).

5 Treatments for depression

The activity below provides an overview of antidepressant medications and psychological therapies that are available for depression, and is also useful for consolidating your learning in this section. Please note that there is no discussion associated with this activity. The questions posed, however, will help you to structure your thoughts as you reflect on the issues raised in the podcast. You might find it useful to take notes and write down your answers to individual questions.

Activity 4 How is depression treated?

Allow 60 minutes

Listen to the podcast below and note down your responses to the questions that follow. You might wish to listen to the entire recording first and review this again thinking specifically about the questions the second time around. Alternatively, you can consider the questions as you listen to the recording the first time around. Choose whichever approach suits you best.

Audio content is not available in this format.

Oxford University Podcast – How is depression treated?

Professor Mark Williams and Dr Danny Penman from Oxford University discuss how the treatment of depression has evolved to include antidepressant medications which directly affect brain neurochemistry, and psychological treatments including cognitive behaviour therapy. The podcast is part of a series on the 'New Psychology of Depression' from the Department of Experimental Psychology at the University of Oxford, produced in 2011.

1. Professor Williams says that 'so much of our functioning, our eating, our sleeping, our mood' is governed by the action of neurotransmitters in the brain. Depression can result when neurotransmitters are no longer in balance. Which two neurotransmitters that he refers to as having been studied extensively are important for antidepressant function? How do antidepressants affect the availability of these neurotransmitters within the brain?
2. Tricyclic antidepressants were introduced in the 1950s and 60s. Why are the 'new generation' of antidepressant drugs preferred over these earlier medications for the treatment of depression?
3. Professor Williams notes that about 60% of people will respond to antidepressant medication and recover from depression, and he refers to the STAR*D clinical trial which showed that changing medications can be useful to support treatment response. He also notes that antidepressant drugs can help in 'severe' forms of depression. What does he refer to as the 'main problem' with stopping taking antidepressant medications?
4. Depression tends to be episodic, 'it comes, it's crushing when it happens, but then you get over it'. What, according to Professor Williams, is the difference between a 'relapse' and a 'recurrence' of depression?

5. Psychological therapies can help to protect against relapse and recurrence of acute episodes of depressive illness. These include cognitive therapy, behavioural activation, interpersonal psychotherapy, psychodynamic psychotherapy and psychodynamic interpersonal psychotherapy, and problem-solving therapy for mild or acute depression. What does interpersonal psychotherapy involve, as described by Professor Williams?
6. Professor Williams explains that during the 1950s and 60s, with the recent availability of antidepressant medications ‘nobody thought that depression was the sort of thing that you could treat’ with a psychological approach. Irrational thoughts and negative thinking were considered a symptom of depression and that in order to treat depression the view at the time was that one would need to treat the underlying biological or psychodynamic problem first, and that ‘negative thinking,and so on,would naturally just dissolve’, and the thoughts 'clear up by themselves’. He refers to two major hypotheses that were proposed in the late 60s and early 70s that changed this view. What were they?
7. Is there a difference between ‘cognitive therapy’ and ‘cognitive behavioural therapy’?
8. Is it possible to know if someone would respond more favourably to cognitive therapy than to antidepressant medication?

Provide your answer...

5.1 Pharmacological and psychological therapies

Pharmacotherapy with antidepressant medications (see Table 2) and depression-specific psychological therapies (see Box 11) can be effective treatments for depression, either delivered as monotherapy or in combination (Kupfer et al., 2012). Cognitive behavioural therapy (CBT) is empirically supported for mild to moderate depression, and interpersonal psychotherapy can be as effective as CBT in some cases. For those who have had three or more previous depressive episodes, mindfulness-based cognitive therapy has an additive effect. Problem-solving therapy has also been shown to be effective in treating depression. Computer and internet-based therapies are also being developed.

Table 2. Antidepressant medications used in the treatment of depression (adapted from Kupfer et al., 2012)

Class of Drug	Examples	Proposed mechanism of action
Selective serotonin reuptake inhibitors (SSRIs)	Citalopram, Escitalopram, Fluoxetine, Fluvoxamine, Paroxetine, Sertraline	Selectively inhibit the reuptake of serotonin
Tricyclic antidepressants	Amitriptyline, Desipramine, Doxepin, Imipramine, Maprotiline, Nortriptyline, Protriptyline, Trimipramine	Nonselectively inhibit the reuptake of monoamines, including serotonin, dopamineand noradrenaline

Noradrenaline-dopamine reuptake inhibitor	Bupropion	Inhibits the uptake of noradrenaline and dopamine
Serotonin modulator	Nefazodone, Trazodone	Primarily antagonises 5-HT2 receptors
Serotonin-noradrenaline reuptake inhibitors	Desvenlafaxine, Duloxetine, Venlafaxine	Inhibits the reuptake of serotonin and noradrenaline
Noradrenergic and specific serotonergic modulator	Mirtazapine	Primarily antagonises alpha-2 and 5-HT2C receptors
Serotonin reuptake inhibitor and 5-HT1A receptor partial agonist	Vilazodone	Potently and selectively inhibits serotonin reuptake and acts as a partial agonist at 5-HT1A receptors
MAO inhibitors	Isocarboxazid, Phenylzine, Tranylcypromine, Selegiline	Nonselectively inhibits enzymes (MAO-A and MAO-B) involved in the breakdown of monoamines, including serotonin, dopamineand noradrenaline

MAO: monoamine oxidase; 5-HT: serotonin (5-hydroxytryptamine).

Ketamine, an NMDA receptor antagonist and analgesic/anaesthetic drug, has more recently been investigated as a new form of treatment for severe depression. At the time of writing, a derivative of ketamine (esketamine, marketed as the nasal spray ‘Spravato’) has subsequently been approved by the Food and Drug Administration (FDA) in 2019, for the treatment of severe depression in the United States. Heralded as the first ‘new’ pharmacological treatment for depression in over thirty years, it will be offered to patients who have tried at least two other treatments without success, and will be administered under medical supervision and monitoring, due to its potential for abuse (FDA 2019).

Box 11 Psychological therapies for depression

Cognitive-Behavioural Therapy (CBT)

CBTcan help to change negative thinking, to interpret the environment and interactions in a positive way, to recognise factors that may be contributing to the depression and to help change behaviours that may be making the depression worse. Longitudinal formulations such as the 5 ‘Ps’ (predisposing factors, precipitating factors, presenting difficulty, perpetuating factors, protective factors) can be useful.

Interpersonal Therapy (IPT)

IPT is designed to help an individual understand and work through troubled relationships that may cause the depression or make it worse, by exploring major issues that may add to depression, such as grief, or times of upheaval or transition.

Problem-Solving Therapy (PST)

PST can improve an individual’s ability to cope with stressful life experiences, and can be an effective treatment option particularly for older adults with depression. Using a step-by-step process, the individual identifies problems and comes up with

pragmatic (realistic) solutions. Typically a ‘short-term’ therapy, PST may be conducted in an individual or group format.

5.2 Treatment-resistant depression and somatic treatments

Switching between antidepressant medications is a common strategy for managing depression, but whether effectiveness can be improved remains controversial. ‘Treatment-resistance’ is said to occur where depression does not respond to (i.e. symptoms are not alleviated following) two or more trials with antidepressant medications. Combination therapies of antidepressant drugs can sometimes be effective (drugs that are recommended for treatment-resistant depression are aripiprazole, quetiapine fumarate and the combination of olanzapine with fluoxetine) (Kupfer et al., 2012). Two stage (augmentation and combination) approaches are based on the understanding that one treatment strategy alone is unlikely to treat the varied symptoms of depression and that switching to a different treatment is dependent on the individual’s response to the first approach and a reassessment.

Psychological therapies can be effective for mild to moderate depression, and although a combination of psychotherapy and medication may be more effective in reducing the chances of recurrence and relapse, this may still not be sufficient to address the symptoms in some cases of severe depression.

Nonpharmacological (somatic) treatments aimed at the underlying neurobiology for medication-resistant forms of severe depression include electroconvulsive therapy (ECT), brain stimulation techniques such as deep brain stimulation (DBS) and transcranial magnetic stimulation (rTMS). These procedures are believed to modulate the underlying neurotransmission affected in depression.

ECT is considered to be effective for treatment-resistant depression (with a 50-75% response in those who do not respond to antidepressant medication) (Bschor et al., 2014). Although usually transient (short-lived), side effects can include confusion, disorientation, and memory loss. DBS involves implanting electrodes, connected to a pulse generator, bilaterally within the brain. Electrical stimulation is believed to modulate neurotransmission in the cortico-striatal-thalamic-cortical circuit (including the cingulate gyrus, nucleus accumbens and ventral striatum,among other areas). Although deep brain stimulation is still in early stages of investigation and shows promise for treatment-resistant depression, it is not currently approved in the US or in Europe. rTMS has been approved in the US for the treatment of major depressive disorder (for those who have not responded to a course of an antidepressant drug). The procedure involves producing a magnetic field around target areas of the brain – the left and right dorsolateral prefrontal cortex – in depression (Kupfer et al., 2012).

Conclusion

Use the visual summary below to reflect on your learning for this course, and on the topic of depression. What were the key issues or concepts that stood out for you?

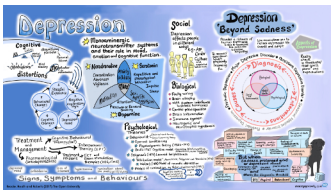

Figure 4 Visual Summary 'Depression'

We have provided you with a larger version of this image in PDF format.

This graphic illustration brings together many of the key themes discussed on the topic of depression in this course. Use the summary to reflect on your learning for this topic.

In the third and final course in this series, we will consider the overlap between anxiety and depression, take a look at a controversial treatment for depression, and leave you with some final thoughts and questions which you may wish to explore beyond this course, around the contemporary topics of diagnosis, causes and interventions for these conditions. To find out more about this series on OpenLearn, take a look at our Introduction to mental health science article page.

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Further reading

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Understanding depression and anxiety

Emotions and emotional disorders

Challenging ideas in mental health

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